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PLANT DISEASES

ERNST GRAM AND ANNA WEBER

PLANT DISEASES

IN ORCHARD, NURSERY AND GARDEN CROPS

Edited and adapted for English conditions by
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Translated from the Danish by
Evelyn Ramsden

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F. KOLPIN RAVN

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EDITOR'S PREFACE

The Danish authors undertook the preparation of this work in 1933 but the first edition was not published until 1940, in Copenhagen, under all the difficulties imposed by the late war. In a community enduring the state of siege imposed by war and blockade, however, elimination of wastage by plant diseases becomes more than ever important and it is not surprising that a second Danish edition was required by 1944. The English translation has been made from the latter. The long experience and international reputation of the authors is a guarantee of the reliability of their work and in editing the English edition only a few paragraphs of purely local interest have been omitted or replaced by others more applicable to English conditions. Not all the plant diseases known in Britain have yet been met with in Denmark, however, and the editor has thought it desirable to add brief accounts of most of those omitted from the Danish text. He has also taken the opportunity, with the consent of the authors, to include references to much English work, published since 1940, not available in Denmark until after the second Danish edition was published.

It is very difficult to describe a plant disease so clearly that a person unfamiliar with it will recognize it on sight. Hence the value of a book of this kind depends largely on its illustrations. The colour plates are those of the original edition, but in preparing the English edition 32 of the original illustrations have been omitted and replaced by 27 new ones. Of the latter, Figures 16, 124, 138, 139, 180, 185, 189, 190, 200, 206, 215, 216, 217, 227, 236, 272, 274, 286, 287, 292, 293 are the property of the Department of Agriculture for Scotland to whom thanks are due for allowing publication in this work. The editor's thanks are due also to Mr. W. C. Moore for revising the account of legislative control measures in England and to the Director of the Royal Botanic Gardens for allowing him to undertake this work.

Royal Botanic Gardens, Kew.
October, 1951.

R. W. G. DENNIS.

PREFACE

We originally undertook the preparation of this book in 1933 when we became impressed by the expansion in fruit growing and market gardening. These industries have gradually come to regard disease control as an essential part of successful cultivation, and in private gardens, too, growers are aiming at higher standards of appearance and quality.

By 1939, when we were ready for publication, a World War had begun, bringing general uncertainty and curtailment of purchasing power. During the actual printing of the work the difficulties seemed insuperable to us all. In good times one can settle down in peace and quiet to think over recent improvements in control measures, but in periods of austerity and stress it becomes essential both for the individual and the community to avoid any wastage in food value of fruit and vegetables. At such times, too, we appreciate more than ever the cheering influence of beautiful flowers.

Plant diseases are important not only by the destruction they cause but also by the time, labour and expense required to combat them.

Rational control methods must be based on a general understanding of the nature of plant diseases, such as we have tried to supply in Section I, supplemented by specialized information about the individual diseases. We have dealt with these under the names of the plants they affect, arranged alphabetically under three sections. Section II: Tree and Bush Fruit; Section III: Vegetables and Herbaceous Fruit; and Section IV: Ornamental Plants. Therefore, a disease may be described more than once and cross-references have been provided to help the reader to find the most detailed account. For the most important crops a summary of the diseases has been worked out in the form of an analytical key to the symptoms. We urge readers to persevere in the use of these keys for it is a common experience that the better a grower knows his plants and the more he uses this kind of key, the more he profits by it.

Prevention is often the surest means of control and today this is more than ever the case, in order to keep down expense. In the instructions for use of fungicides (which are dealt with in alphabetical order in Section V: Control Measures), we have been guided mainly by the " 250 Statements and Lists of Proved and Recognized Special Preparations " issued by the Danish State Experiment Authorities, but we have tried to include also promising results obtained from abroad and some derived from practical experience even when the results of final experiments were not yet to hand.

The book was written in the hope that it would be of help not only to agricultural advisors but also to practical growers and we hope the illustrations will go far to make this possible. The book will also be useful for evening schools and courses where demonstration of living material is not practicable. Towards the cost of most of the original figures we received help from the Laurits Andersens Fund to which both readers and authors owe a debt of gratitude.

The experience gained in our work at the State Plant Pathological Experiment Station has been of great value to us in preparing this book. Many enquiries come to the station from fruit growers, market gardeners and gardeners. In this way and by means of our many journeys about the country for over 20 years we have had great opportunities of observing the effect

of diseases in practical horticulture. We have to thank many fruit growers, gardeners, land owners and their consultants and other kind helpers; in this connection we are glad to thank particularly Professor Anton Pedersen, Docent H. K. Paludan, Konsulent Verner Nielsen and Konsulent Lars Hansen for the help they have given us in reading the manuscript and supplying much valuable data.

The coloured plates and the numerous drawings in the text were made by the painter Ellen Olsen of Lyngby, with a care and interest that calls for our special thanks; some of the drawings have already done service in smaller publications. Many of the photographs were taken by Louis Hertz, Lyngby, and have been given us by gardener Willy F. Hansen. Some photographs belong to the State Plant Pathological Experiment Station and have been taken by our colleagues or by the authors themselves; they are marked S.P.F. or with the name of the appropriate author if they have been published previously. For other valuable original photographs thanks are due to Professor C. A. Jorgensen and to a number of foreign colleagues and institutions, notably O. Banga, Holland, W. Buddin, England, Dr. E. Jamalainen, Finland, Professor M. L. Massey, U.S.A., Dr. Heinrich Pape, Germany, Director N. Van Poeteren, Holland, Professor E. Van Slogteren, Holland, Professor Johanna Westerdijk, Holland, Professor H. H. Whetzel, U.S.A., and the Biologische Reichsanstalt, Germany.

Finally, while we anxiously watched the mounting size of the manuscript and the cost of illustrations, the Court Printer, Egmont H. Petersens, by his experience in deciding a suitable lay-out of the text and by untiring personal supervision of every detail, greatly enhanced the appearance and value of the book.

ERNET GRAM. ANNA WEBER.

Lyngby, May 1940.

The second edition contains few alterations. A few new control methods have been added and references have been included to various experiments, practical observations and publications in the current literature.

E. G. A. W.

Lyngby, August 1944.

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I
THE NATURE OF PLANT DISEASES

1 DEFINITION AND CLASSIFICATION OF DISEASE

Before discussing the different kinds of plant diseases it is necessary to decide on a classification of them, though it must be recognized that the rigid outline of any classification will be liable to modification for practical considerations. Also, at the outset, it is difficult to draw a clear-cut distinction between health and disease in plants, as will be seen from the following examples:

The Advisory Department at Lyngby once received an enquiry about an outbreak of " rust " which had rendered unsaleable a consignment of *Araucaria* received from abroad. Examination soon showed this to be truly an instance of rust but not of attack by a rust fungus. The plants had been sprayed with water rich in iron and were coated with a layer of rust, easily removed by dipping them in a very weak acid solution. The *Araucaria* was dirty but not diseased.

Another enquiry related to an apple tree which had only weak growth and leaves with scorched brown edges. Winter application of a potassic fertilizer induced normal growth with healthy green leaves the following summer. The tree had been starved but it was not diseased in the sense of being attacked by a parasite.

In a particular garden where the potato plants had always yielded clean smooth-skinned tubers the potato plot received a dressing of lime. Next season the tubers were scabby and the scabs contained one particular kind of ray fungus (*Actinomycete*). Generous application of sulphate of ammonia neutralized the lime, and the amount of scab on the tubers was reduced. Most people would agree to call the scabby tubers diseased; the ray fungus was the cause of the disease, but the intensity of the attack depended on the calcium content of the soil.

As a general principle one may regard as healthy any developments normal to the life history of a plant species, and any deviation from the normal as constituting disease. But some deviations, such as variegated foliage in elder or maple, are the result of a change in the hereditary constitution of the plant, which is not usually classed as a disease. The abnormal conditions usually regarded as diseases are either induced by infection with a parasite, and hence are transmissible to hitherto normal plants, or are due to unfavourable environmental factors, as in nutritional disorders. In horticultural practice a condition which would be regarded as " disease " if induced accidentally is " normal " if produced at will. For example, the etiolation which is purposely produced in bleaching asparagus, endive, celery and similar vegetables for market is a normal reaction of the plants to darkness. In other plants where bleaching had not been deliberately carried out its appearance would be regarded as a " disease " due to lack of light.

In commercial practice one's attention is focused mainly on the economic losses which decide whether a disease has to be reckoned with or not. But a few diseased plants are deliberately propagated and form profitable commercial propositions; such are parrot tulips, with parti-coloured flowers, and *Abutilon* with variegated leaves. Both these plants are actually diseased, owing to their infection with specific viruses.

Plant diseases can be classified in various ways.

A classification according to causes, fungi, bacteria, viruses, environmental factors, etc., is logical but not altogether practical. Often several factors are involved, and it is necessary and often difficult to decide on the primary cause, and of course there is likely always to remain a heterogeneous residue of " diseases of unknown origin."

Classification by symptoms is attractive to the practical man but cannot be rigidly applied. Even if we were to keep to the three main groups—necrosis, death of the tissues; hypotrophy, under-development; hypertrophy, over-development—many diseases would still be difficult to place or their position in the scheme would depend on their stage of development. For instance, in such a disease as club root of crucifers there is first hypertrophy of the roots, followed by underdevelopment of the foliage, while finally there is premature death and rotting of the swollen roots.

Classification according to host plants is the least logical and necessarily involves considerable repetition in the case of diseases common to many plants. It is nevertheless the most helpful to the actual grower and is that adopted here.

2 CAUSES OF PLANT DISEASES

Vague traditional ideas that diseases resulted directly from the wrath of God, from conjunction of the planets, from injurious vapours emitted by the soil or from deleterious juices within the plants were replaced in the middle of the last century by the theories of the German mycologist De Bary, based on his epoch-making researches on the life histories of fungi. For a time these great discoveries tended to obscure the fact that discovery of a parasitic agent in disease did not altogether exclude an influence of environmental factors on its development. Plants and potential parasites often exist side by side without any appreciable amount of disease resulting. A slight change in the environment, rendering it either less favourable to development of the host or more favourable to that of the parasite, may then lead to an extensive outbreak of disease. In such an outbreak the active agent of disease is the parasite but the immediate cause of its activity is the change in the environment. Hence it is often necessary to decide whether control measures are likely to be most effective in a direct attack on the parasite, as by the use of fungicides, or indirectly by modifying some environmental factor, such as temperature, humidity or nutrition of the host.

Accurate diagnosis is in all instances an essential prerequisite in a programme of control. For example, it may have to be decided whether a valuable consignment of plants has been damaged by parasitic fungi or by heating in transit. Moreover different fungi have very different life histories, and control measures are only likely to be effective if they are directed against some particularly vulnerable period in the life of the parasite.

Among the causes of plant disease the first to be considered is the hereditary factor or gene, which is a part of the plant itself and may therefore be regarded as an internal source of disease. Next come the environmental factors responsible for non-parasitic diseases, especially in cultivated plants grown under unnatural conditions, for example with excessive, deficient or unbalanced supplies of nutrients. Finally there are the many living organisms that cause parasitic infections or contageous diseases.

INHERITED DISORDERS

Researches in heredity have brought to light numerous instances where a change in the inherited constitution of the plant has led to the appearance of variegated leaves, deformities—sometimes of commercial value, as in the fasciated form of cockscomb (*Celosia*

cristata)—or even complete absence of chlorophyll. In the latter case the seedling inevitably dies as soon as the food stored in the seed has been exhausted. Change in the hereditary constitution may also take place in individual buds (bud sports). Something of this kind is probably responsible for the appearance of the " wilding " type of growth in potatoes.

" Degeneration " or " running out " is a popular expression used to cover any incomprehensible decline in vigour of a crop. Originally the terms were applied to the unfavourable and weakening effect that continual vegetative propagation was imagined to have on certain kinds of plants. Subsequently it was thought that continued cultivation under unsuitable conditions could have a cumulative injurious effect on the constitution of a plant. Critical investigations have yielded little or no evidence in favour of either of these hypotheses. Degeneration in vegetatively propagated crops is due in most instances to the spread of virus diseases, especially in potatoes, strawberries and raspberries. These diseases are seldom transmitted by the true seed but their casual agents permeate the entire vegetative body of infected plants and are inevitably transmitted by all their tubers, runners, cuttings or offsets. Other so-called types of degeneration are expressions of self-sterility or the result of inbreeding.

ENVIRONMENTAL FACTORS

Shortage of Water

Transpiration or evaporation of water from the leaves and other above-ground parts is an essential function in land plants. Not only is it one of the factors which brings about the ascent of sap but, owing to the large consumption of latent heat in changing liquid water into water vapour, transpiration helps to cool the plant. In hot sunny weather there is intense radiation from the surface of bare soil which scorches the moisture still more out of herbaceous plants. The effect varies greatly between different plants. Potato leaves wilt helplessly, but beetroots, owing to their great ability for obtaining during the night the moisture they require from the soil and dew, have their leaves as erect as ever next morning. Sunflowers transpire especially through their lower leaves which wilt first; begonias, on the other hand, mainly through their flowers. There is little restriction to evaporation from the surface of most petals and it is a common experience that large showy flowers like chrysanthemums and roses wither very readily when cut. In extreme drought, plants, and even deciduous trees, shed their leaves. A second crop of leaves is then produced when the drought is over. Similarly in very dry summers apple trees shed a large proportion of their young green apples (see p. 131). The dry air of living rooms tends to production of " long-legged " pot plants with only a few leaves near the top of their stems. Out of doors a dry wind may shrivel the edges of leaves, especially of the newly opened leaves of pear (see p. 187), apple and red currant in the spring. Such marginal scorching becomes more pronounced the further one gets from shelter. Marginal scorch of fruit-tree leaves is often, however, a sign of potassium deficiency, especially later in the season. Less often, drought injury becomes apparent in the centre of leaves, as in tomatoes, where it appears as angular or irregular leaf spots. Normal autumnal leaf fall of deciduous trees is less a protection against cold than against drought. Leaves of evergreens have anatomical devices to restrict and control transpiration but even in conifers dry spring winds, coming before the roots have begun to function adequately, too often give rise to the well-known " red fir trees." The terminal buds generally survive on the branches of such trees and later produce the normal shoots with young green needles of the current year, behind which is a bare zone where the needles of one or two previous years have been scorched, dried and fallen off.

Premature shedding of flower buds or flowers, especially common in begonias, epiphyllums and camellias, is often a sign of drought. In the lily and amaryllis families the flowers react to drought in a peculiar way, by adhesion of the tips of the petals.

Fruits also have their own characteristic ways of reacting to a shortage of water. Tomatoes develop blossom-end rot (see p. 389), pears stone cells in the flesh, and apples bitter pit (see p. 134).

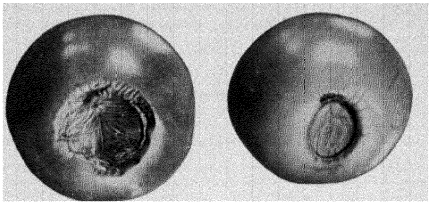


Fig. 1. Tomatoes that have developed blossom-end rot as a result of drought (*S. p. F.*).

During severe drought potatoes develop tubers as soft as indiarubber, either at the heel end only or throughout, with a tendency to a sprout in early autumn soon after lifting. Indirect effects of drought are the encouragement it gives to some insect pests, especially aphides, and to a few parasitic fungi, notably the powdery mildews. Absorption of certain nutrients from the soil is also hindered, especially of minor elements like boron. Such deficiency diseases as heart rot of sugarbeet and canker or black spot of garden beet are thus often associated with dry weather. The boron-deficiency diseases of apples are similarly encouraged and were hence known as " drought spot " before their primary cause was understood.

Because of the severe losses so often caused by drought various methods of artificial water supply have been devised, but there are other less direct ways of mitigating the effects of dry weather. These include destruction of weeds, keeping the surface soil loose by hoeing, planting shade crops or supplying artificial shade, mulching the soil, etc. When plants have been raised under glass it is important to harden them off gradually if they are later to be exposed to a dry atmosphere.

Excess Water

Roots must be able to breathe, and in waterlogged soil the fine absorbing roots of most plants are choked and die so that absorption of nutrients and even of water is impeded. Water plants have a system of internal air channels which supply the roots with oxygen from above the water level. Potato tubers are very sensitive to waterlogging and may be killed after a day's complete immersion in water. Occasionally tubers planted in wet soil do not rot but become glassy, very hard, and fail to sprout. Detrimental chemical changes may take place in saturated soil and the population of soil micro-organisms may also be adversely affected. Changes of this kind can only be slowly corrected after thorough drainage.

Besides producing the suffocated, brown, sour-smelling roots found in waterlogged soil, excessive water supply, whether in a plantation or a flower pot, is often manifested by yellowing of the leaves. This indicates, amongst other things, that the nitrogen in the soil is not being absorbed by the suffocated roots and partly that it has been washed or leached out of the soil and lost. In house plants this " drowning " sometimes induces a bright yellow colour in the foliage (aurigo), especially in dracaenas and the like. An instance of high death-rate among fruit trees and bushes in Kent is described on p. 130.

Often, especially in greenhouses, the soil contains only a moderate amount of water but, owing to inadequate ventilation, the air becomes overcharged with moisture. If this condition persists the check to normal transpiration leads to a number of peculiar outgrowths on the above-ground parts of the plants. Oedematous eruptions composed of greatly enlarged cells full of sap appear on vines and tomatoes (see pp. 220, 392). In potatoes grown on benches under glass this swelling and eruption of the leaf cells may occur to such an extent that the whole leaf tissue turns mealy and falls away in patches leaving large holes, while the whole leaf becomes curled and deformed. Such a condition appears very alarming but with sunshine and proper ventilation fresh normal leaves are

quickly produced and the plants recover. On leaves of *Pelargonium zonale* wart-like intumescences may be produced by enlargement of the cells which ultimately collapse and die leaving a scab of corky tissue. The inner surface of pea and bean pods is often covered by a layer of such inflated cells, looking like a coating of white mould. Cacti may react to excessive humidity by becoming in places clear and glassy (see p. 425).

In damp situations the breathing pores or lenticels in the bark of many trees become enlarged into corky warts. The same kind of lenticel proliferation may happen in potato tubers produced in wet, though not completely saturated, soil and is often mistaken for a parasitic infection.

In some trees the loosening of the texture of the bark may break out in blisters or involve large patches of the trunk or branches which become covered with a loose corky powder the colour of tan, called by the Germans " tan disease " (*Lohkrankheit*). The condition is probably harmless and may disappear when the trees are transplanted to drier situations but it is disfiguring and hence reduces the market value of nursery stock. It is advisable to be very careful in winter-spraying trees which show these symptoms.

When several big branches are removed together from an old tree the pressure of the rising sap within the trunk may become so high that cracks appear in the bark. It is therefore best to remove undesirable branches a few at a time

An excess of water in the soil favours attack by many parasites, especially where a motile swarm-spore stage occurs in their life history. Hence powdery scab in potatoes (p. 324) occurs mainly in ill-drained soils and is only prevalent in the wetter parts of the country and on heavy soils; club root of crucifers is also encouraged by a waterlogged soil or a heavy spring rainfall. Some bacterial diseases such as potato black-leg are also favoured by a high soil-moisture content; black-leg is usually only prevalent in ill-drained parts of a field.

High atmospheric humidity, followed by condensation of moisture on the roof and dripping on to the plants, frequently leads to an outbreak of grey mould (*Botrytis*) diseases in glasshouses and conservatories. Even out of doors the still damp air of warm muggy days favours an outbreak of many diseases, especially if it is accompanied by dew at night. Well-known diseases encouraged in this way are chocolate spot of beans and potato blight.

Sudden changes in water supply from drought to great humidity damage many garden products. Splitting in plums and other tree fruit is all too common after heavy rain (see p. 205); so also are cracked gooseberries, fallen hazel nuts, split tomatoes, telescoped apples (described on p. 138), cracked heads of cabbage and carrots and parsnips split right to the core.

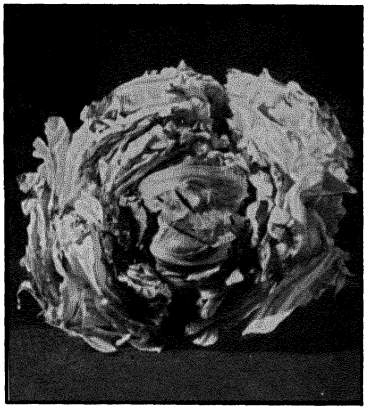


Fig. 2. Head of white cabbage which has cracked because the weather has suddenly become very damp.

The potato tuber seems particularly unfitted to cope with a sudden increase in the water supply. In mild cases the skin splits all over in a fine network of cracks, but often the splitting goes much deeper and opens the way to various tuber rots. Splitting generally takes place before lifting but it may also occur during or after lifting. Sometimes a clear transparent patch of tissue develops at the centre of the tuber which soon dries and

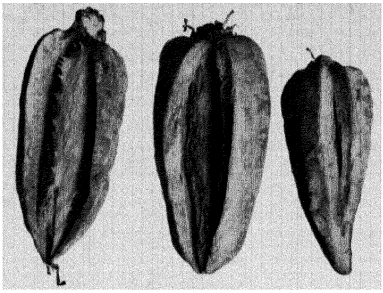


Fig. 3. Carrots which have split after heavy rain (*Photo F. Ballard*).

cracks to form a lens-shaped hollow, lined with dead brown tissue (hollow heart). This condition is most prevalent in big tubers and is often associated with heavy nitrogenous manuring and wide spacing of the plants. Close planting, balanced manuring and thorough and early cultivation of the potato bed should prevent damage of this kind (Moore, 1926; Wollenweber, 1922). Boron deficiency or infection with some viruses may also increase the tendency to splitting.

When heavy rain follows drought towards the end of the growing season potatoes are very liable to second growth. The already-formed tubers develop swollen ends or fantastically shaped outgrowths and the stolons produce an abundance of small new tubers, often several in a row; this last condition is also, however, often associated with the "wilding" condition irrespective of changes in water supply. The condition known as Jelly End Rot of potato tubers is apparently due to the roots continuing to function and supplying water to the tubers after the haulm has died or been killed. Such tubers grow out at the rose end and withdraw starch from the tissue at the heel end which becomes glossy and ultimately decays.

Under glass a sudden change from drought to high humidity leads to oedema, bitterness in cucumbers, breaking of chrysanthemum stalks just below the head and splitting of the calyx in carnations.

CLIMATIC AND OTHER PHYSICAL FACTORS

Hail Damage

Hail damage, varying in extent with the size of the hailstones and the intensity of the storm, is to be regarded as a purely mechanical injury. The local cooling of the tissues due to temporary contact with the frozen drops is of little or no consequence.

When the hailstorm occurs during the blossoming of fruit trees, the blossom is usually destroyed and the loss irreparable. Once apple fruit has set and begun to grow the damage depends on the force of the blow. Light hail that does not break the skin will cause only small sunken spots (Fig. 4) below

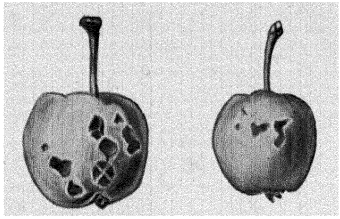


Fig. 4. Apples with wounds caused by hail.

which dry, dead tissue will be found. If the skin is broken *Monilia* often gains a footing or large scars are formed from which pieces of dead tissue gradually fall away or, in the worst cases, the whole apple may be knocked to pieces by the hail. Hail causes dead dry spots on gooseberries and strawberries if it falls when the fruit is half ripe, but here also ripe fruit is liable to be smashed to pieces in a hailstorm (Esbjerg, 1926a). Large leaves may be badly torn by hail (Fig. 5) and sometimes both leaves and fruit are shed. Plants especially liable to hail damage are: rhubarb, beet, leek, onion (Fig. 7), cucumber, spinach and lettuce. Rows of peas may be beaten flat and appear to be ruined by a hailstorm, but with good growing weather they recover with amazing rapidity though they may not altogether lose the marks of hail on the pods. Badly battered potatoes may similarly recover and yield quite well in the end.



Fig. 5. Apple leaf torn by hail.

The bark of trees may be marked by small, well-defined hail scars (Fig. 6); as a rule the fresh wounds are deep enough to expose the bast or wood, but in the course of a few years the wounds become beautifully sealed by ingrowth of callus tissue from their edges. This often happens in apple trees, but in trees with scaly bark, like the cherry, the wounds are larger, and soft bark, like that of the elder, may be peeled off in large flakes.

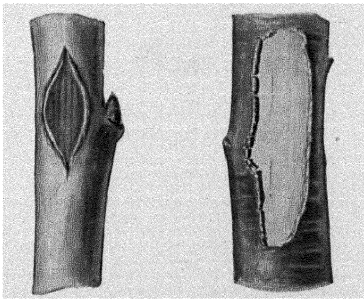


Fig. 6. Injuries caused by hail to bark of apple (left, × 2) and cherry (right, × 1½).

Hailstorms form a special hazard in seed production, especially towards ripening, partly because they beat down the plants and partly because of the great shedding of seed that may result. Besides the direct damage to fruit, seed and other marketable products, a reduced crop must be expected from plants whose leaves have suffered extensive injury from hail. Heavy hailstorms may also beat the surface layers of the soil together and necessitate additional cultivation or hoeing (Esbjerg, 1926b).

The one-sided character of hail lesions, due to the hailstones having been driven from a particular direction, is a help in diagnosing hail damage and in differentiating it from somewhat similar injuries due to other causes; especially spots and shot-holes caused by fungi and bark scars due to chafing. The amount of loss from any particular storm cannot be estimated immediately, but only after comparisons with the yield of undamaged crops have been made and when it has been seen how far the shattered plants have been able to recover.



Fig. 7. Hail damage to onion leaves.

Injuries by Low Temperatures, Frost and Severe Winters

Some plants are readily killed by low temperatures. Certain tropical plants even die at 41° F., others not until the air temperature falls far below the freezing point of pure water, 32° F.; but frost, even when it involves formation of ice within the plants, is often less fatal than is generally supposed.

Much so-called "frost damage" is the result of drying out of the tissues; the dry winter winds deprive pine needles and evergreen leaves of so much water that their tissue dies at the tip, round the edges, in patches or even over the whole leaf. If the ground around the roots is frozen they cannot be supplying water to the above-ground parts of the plant or tree. The benefit derived from planting shelter belts often demonstrates the great effect drying winds can have during a frost. The natural autumnal leaf fall of deciduous trees is to a large extent a protection against water loss in winter. Bud scales protect the tender next year's shoot against drying more than against cold. The final function of the deciduous leaf stalk is to lay down a layer of cork to seal the scar left when the leaf falls; if they are killed by a very early frost the leaves may hang on the trees until far into the winter.

When cold weather comes many plants convert starch, an insoluble food store, into sugars or fats, especially in bark and evergreen leaves. Because the presence of dissolved substances depresses the freezing point of a liquid this may be regarded to some extent as a protection against ice formation in the living cells. This is not the main reason for storage of reserve foods in the form of sugar, however, as is shown by the facts that sugar beet is not particularly hardy while sugar cane is a tropical plant. In apple trees, raspberries and gooseberries, hardiness seems to be correlated with pentosan content (Hooker, H. D., 1921).

Ripening or maturing is a comprehensive term covering the changes that take place in autumn in the current year's growth of a tree or bush. Normal ripening is of great importance in avoiding winter injury. Ripening includes the production of protective substances, a considerable fall in the water content of the tissues and changes in the protoplasm of the living cells. Plant cells do not contain pure water but strong solutions whose freezing point is a good deal lower than 32° F.

Ripening is affected by many conditions. It is retarded by an excessive supply of nitrogen, and in raspberry plantations it has been shown that excessive liming weakens the cane's resistance to frost. Common spruce has been found to suffer frost damage when growing in a very acid soil of pH 3.9, but not in a less acid soil of pH about 5. In New York State some correlation has been found between a shortage of lime and frost injury to apple shoots (Collison, 1934). Conditions of terrain, such as deep or shallow soil, light or heavy soil, may also influence the amount of frost damage. In fruit farming the earlier ripening of fruit in grass orchards helps it to escape frost. Parasitic diseases may also have an influence on the frost resistance of plants. Hauch and Ravn have shown that oak mildew retards ripening of the shoots and hence increases their liability to injury by frost. Apple scab reduces the production of starch by the leaves, and so retards ripening of the shoots, and makes frost damage, often followed by canker, more likely. In the cold damp summer of 1923 the trees at Spangsbjerg Experiment Station near Esbjerg had their leaves beaten off by the storm of 30th August; in the following winter the trees were destroyed by frost, their bark peeling off in sheets. Only those trees that had been protected by the shelter belt survived. The fact that cold weather early in the winter is more destructive than severe frost in February is certainly due to the former's occurrence before ripening has been completed.

It is not known whether the prevalent red coloration of alpine and arctic plants affords them any protection against cold. In alpines, at least, it is more likely to be a protection against the intense sunlight experienced at high altitudes.

In spring, as light and warmth increase, the sugar in perennial plants tends to be replaced

by starch (Müller-Thurgau, 1880, 1882, 1886) and the buds show signs of life. They then become more exposed to injury from the cold, as for example the female flowers of the hazel and the buds of the cherry tree (see p. 159). Cold-induced chlorosis is a common spring phenomenon, due to the green pigment being absent from the parts of the plant that were making most active growth when the cold spell set in. An example of this yellowing of the growing zone in hyacinth leaves is shown in Fig. 8, and similar yellowing often occurs in the first leaf of cereals and in the shoots of early potatoes.

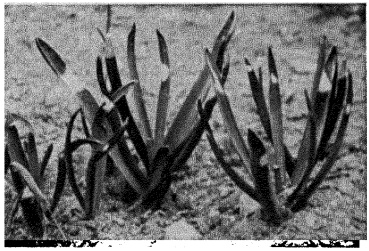


Fig. 8. Hyacinth leaves damaged by low temperature (S. P. E.).

Bolting of celery, beet and other biennial vegetables is in part governed by hereditary characters but the tendency is much increased by spring frosts. Cold air is heavier than warm and it tends to flow towards low ground. In winter if the sky is clear and the air still the cold air gradually collects in hollows and valleys. In such circumstances it may freeze in the hollows while on higher ground there is no frost. " Frost pockets " of this kind are a well-known problem in fruit growing. The tissues to suffer most from spring frosts are the tender young growing organs full of sap, such, for example, as young potato shoots, young beans and the flower buds of fruit trees. In the latter case, however, blossom blight due to *Sclerotinia* is very commonly mistaken for frost damage. Flowers of black currants and other fruit buds are liable to be destroyed by frost, while in strawberry blossoms the pistils are blackened so that the fruits do not set or are misshapen. Streaks, rings or spots of corky skin on apple and pear fruit (p. 140) and green blotches round the nose of ripe apples may also be due to frost; so are blistered leaves of apple and other trees and small blisters on young bark. Half-open chestnut and elm leaves may have their exposed surfaces scorched by frost and whole patches of tissue may be killed so that when the leaves finish unfolding they split and fray like feathers (Ferdinandsen and Buchwald, 1936). In other leaves only the margins or tips may be scorched, depending on how the leaves are folded in the bud and which parts were exposed when the frost occurred.

Frost cracks may be produced not only in green, juicy tissue but also in wood. A sudden great fall in temperature may lead to such high tension in a tree trunk that it splits from crown to root, with a loud report. When the temperature rises again the crack closes. Often the adjacent cambium and bark are almost undamaged and the wound is healed by formation of a frost callus. The cracked place may, however, remain weak so that the annual rings covering it are split again in the same place when the next hard winter comes.

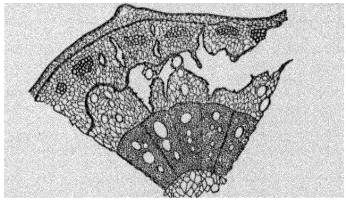


Fig. 9. Frost crack in raspberry cane. Cross-section, greatly enlarged (after Rainio).

In wet ground when ice is formed in the surface layers of the soil the crust is heaved upwards and the roots of plants in it may be torn apart. Rolling the ground before and after a frosty spell may save the lives of many plants. When a night frost is followed by a drying wind, plants transpire freely but are unable to absorb water from the frozen soil to replace that lost from the leaves. The roots of many plants also cease to absorb water

freely at temperatures below 40° F. This inability to maintain their water supply explains why many half-hardy species cannot survive our winters out of doors (Laflin, 1938).

Great differences in susceptibility to frost damage may exist even between varieties of the same plant, and this is an important factor for the consideration of the plant breeder, as for example in winter broccoli. Among apples Worcester Pearmain, Allington Pippin, Grenadier and Lane's Prince Albert suffer less from frost than Bramley's Seedling and Cox's Orange Pippin. Conference is notoriously frost resistant among pears and Czar among plums. Ice formation in the tissues takes place most easily when water has seeped from the cells into the air spaces between them. Freezing of this intercellular water then often leads to the formation of columns of ice, often so small that they can only be seen under a microscope, but they are a useful indication of frost damage. Some plants do not suffer from ice formation in their tissues, though only a few are like *Larix sibirica* which can stand 30–50° C. of frost for six months of the year. A number of tissues are not damaged by freezing if they are allowed to thaw slowly. Thus, edible roots frozen into the ground should be left undisturbed until the ground has thawed. This may be partly connected with the phenomenon of super-cooling, i.e. there may be no ice formation even when the temperature has fallen several degrees below the freezing point of the sap, but if the plant is then shaken the whole tissue freezes suddenly. Of course, if the tissues have not merely been frozen stiff but also frozen to death no caution in thawing is of any use.

Death of the living cells by freezing may be due to their being crushed and torn by the expansion of ice when the water in the intercellular spaces freezes. More often it is due to excessive withdrawal of water from the cells into these spaces as the ice forms. This withdrawal of water may proceed so far that the concentration of soluble substances in the cell sap reaches an injurious level. Moreover the protein substances of the living cells occur in a colloidal condition, and excessive withdrawal of water may lead to a coagulation of these colloids which is irreversible and cannot be overcome when the ice melts and the cells reabsorb water. Oxidation of some of the constituents of these damaged cells turns them brown or black. When the cells have not been killed they are able to reabsorb the water from the spaces between them provided thawing proceeds slowly. If it occurs suddenly these spaces become flooded with water, however, and the vital processes of the living cells are impeded by lack of air.

Freezing to death may occur either in a whole plant or only in parts of it. Green and juicy parts then become glassy and when they thaw they are limp and the water may be squeezed out of them. All resistance to bacterial and fungus infection is lost. Several weak parasites like *Botrytis*, *Valsa* and *Nectria cinnabarina* gain a footing in frost-damaged shoots and are able to spread thence into the adjacent living tissues. Gummosis and die-back of branches may sometimes be the direct result of frost injury, sometimes of frost damage aggravated by feeble parasites of this kind. Sometimes frost-damaged trees burst into leaf but weeks or even years later the affected branches wither or the whole tree may die.

Avoiding injury from cold and frost. Mulches of leaves or manure, with branches of evergreens or mats to cover espalier trees, are commonly used to counteract too great variations in temperature on warm days in early spring. Planting shelter belts may be effective in the long run. In places where night frosts are a problem it may be possible to deflect the cold currents by suitably planned hedges (air drainage). Earthing up in various ways may be undertaken in autumn to give shelter. Fruit blossom may be saved from frost at the critical period by a judicious use of orchard heaters at the rate of 50 to 60 per acre. If this is attempted, however, it is necessary to heat a considerable area. It is useless to attempt to control frost in one acre of a large frost pocket as there will be too much cold air accumulating all round it. The smoke drifting from a heated orchard may also afford

some protection from frost to field crops in the vicinity.

In countries with a continental climate the goods wagons are generally heated and in Denmark heated lorries are used. In England serious losses often occur during the transport of seed potatoes from grower to merchant or if severe frosts set in after the pits have been opened. An ample supply of straw as insulating material may reduce these losses. Roots or tubers frozen in transit or in storage should be left undisturbed to thaw gradually, when it can be seen which have survived. Those that have been killed can then be boiled, strained and used for fodder.

In the case of shrubs and trees balanced manuring to ensure proper ripening and a thorough disease-control programme will go far to minimise frost damage. Damaged branches should be cut out to leave a clean, smooth surface to prevent their being used by fungi as avenues of attack.



Fig. 10. Oak damaged by lightning (after Rostrup).

Lightning Damage

When lightning hits a tree it often only ploughs a furrow in the bark (Fig. 10). Less frequently large pieces of bark are sheared off or the wood is splintered. Old trees with much decayed wood may even be set on fire. In a general way it may be said that tall trees are somewhat more likely to be struck by lightning than low ones, and in addition, those with rough bark like ash, oak, elm, willow, poplar and conifers tend to attract lightning more than trees with smooth bark, like sycamore, beech, birch and horse chestnut whose bark is more quickly and evenly wetted by rain, thus making them better conductors of electricity.

Lightning injury to growing potatoes has often been described (Burr, 1933; Samuel,

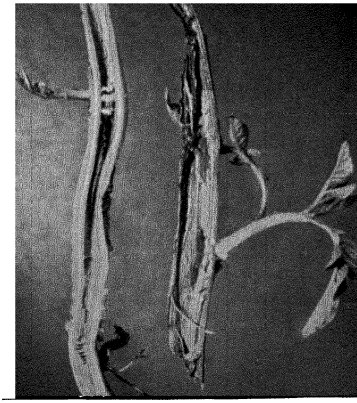


Fig. 11. Potato plants damaged by lightning (*S. p. F.*).

1940). In a patch some three to ten yards across, where the lightning struck, the plants look as if they had been rolled or boiled. Round this is a zone of damaged haulm with flattened or split stems, and the pith may be killed, though the leaves still remain green. When the injury occurs late in the season the tubers are also damaged. One end is often shattered and there are depressed blackened patches of flesh over which the skin may be still intact or broken later by shrinkage. These are apparently " burns " due to the passage of the electric current. Both on the haulm and in the tubers secondary attack by *Fusarium* and other fungi may occur.

Light

Light is essential to the life of green plants. Only in very exceptional cases, such as sycamore cotyledons, do tissues turn green in the absence of light. Moreover light is the source of energy whereby the green colouring matter (chlorophyll) is enabled to transform carbon dioxide and water into sugars and starch. The quality (colour) and intensity of light affect the growth of leaves and flowers and the process of transpiration. Moreover, the daily duration of light is of great importance in determining whether a plant will flower or not (photo-periodism). Some plants flower only during long days; others, which normally flower only in the short days of autumn, can be induced to do so in summer if they are artificially darkened for the requisite number of hours a day.

Lack of light. Lack of light generally induces etiolation (blanching). In extreme cases there is a yellow colour, long fragile stalk and small leaves; weak light induces straggling soft growth with few leaves, light green in colour.

Too dense a seeding in seedbeds and seed-boxes has a similar effect in producing weak, semi-etiolated plants particularly liable to damping off.

Excess of light. Injuries due to excessive sunlight are seldom seen in Denmark or England and in practice it is difficult to differentiate between injury due to too intense a light and that due to the accompanying heat. Sun scald of greenhouse tomatoes in bright sunny weather is almost certainly due to the latter. Too intense a light is, however, known to have a destructive effect on chlorophyll and it may be for this reason that plants suddenly moved into full sunlight make red pigment (anthocyanin) in their leaves. Such red colouring is regarded as abnormal in cacti and begonias. Aspidistras, camellias, *Cissus antarctica*, etc., do not flourish in strong light as their chlorophyll is gradually destroyed by it. Tropical species of *Selaginella* with their delicate leaves cannot endure full sunlight, and many ferns require moderate shade. Camellias in strong light often develop yellow spots on their leaves.

Mechanical Damage

Some wounds are caused intentionally, as during pruning of fruit trees and in lopping undesirable branches. All such wounds offer possible courts of entry to parasitic fungi and the larger ones should always be given a protective covering (see p. 192). Where branches have been torn off by wind the wound should be cut back to a smooth surface and covered in the same way. Accumulation of snow may break down many branches especially of evergreens and conifers. Occasionally when light rain falls through a cold atmosphere it becomes



Fig. 12. Tomato stem constricted near soil by too tight a tie.

supercooled and freezes immediately it touches a solid object. In such circumstances even small twigs may be surrounded by a layer of ice four inches thick. Newbold (1940) records an instance of a branch which bore 64 times its own weight of ice in the great storm of 27th January, 1940. Such storms naturally cause great destruction even to deciduous trees. Branches broken by snow or by a heavy fruit crop should either be cut off or forced back into place and held in position by binding and propping according to circumstances.

Bruising of fruit during picking and in transit is not only disfiguring but often opens the way to weak fungus parasites that cause rotting.

Damage by Heat

It is often difficult to distinguish heat damage from that due to too intense a light or to drought. During winter temperatures may be injurious to glasshouse plants which would be quite harmless with the brighter light of summer. A day's bright sunshine after a period of dull weather may scorch the leaves of plants under glass. Shedding of flower buds and even of leaves may be a reaction to excessive heat, especially in azaleas and camellias. On outdoor plants, especially trees and bushes, hot dry weather is associated with high red-spider infestation. Pear leaves (p. 187) and apple and gooseberry fruits (pp. 139, 175) are all liable to sun-scorch. Sometimes raspberries may be sun-scalded and then have a grey hue which leads people to confuse them with mouldy berries.

Most green leaves and stems are killed at temperatures of 100–120° F. though some succulents can endure rather more. On hot summer days such high temperatures may occur near the surface of soil and rock. Some plants, such as flax and coniferous seedlings, are then liable to develop heat cankers at or near the soil level. In countries with a Continental climate, such as the North American prairie states, even trees may be thus injured near the base of their trunks. In such regions it is advisable to grow apple trees with very short trunks so that the branches always afford shade to the base of the trunk.



Fig. 13. Beech with sun scald on the bark after surrounding trees had been cut down.

When tree bark is marked by sun scald in Denmark it is generally due to too rapid thinning of timber or the felling of trees that gave shade. Sun scald of bark is not usually to be ascribed to the heat of the sun alone but to a combination of spring frosts and bright sunshine.

Local scalding of leaves in glasshouse plants is sometimes due to an air bubble in the glass which acts as a lens concentrating the sun's rays on a single point. As the sun moves round this focus moves across the leaf leaving a seared line across it.

Heating during storage is often blamed for decay in vegetables and potatoes but often unjustly. Cool storage conditions are, however, desirable since vital processes are slowed down at low temperatures and the reserve food in tubers and similar storage organs is less rapidly used up. Evaporation is also slower at low temperatures and parasitic fungi and bacteria are usually less active. Storage at high temperatures, on the other hand, leads to premature shrivelling and sprouting.

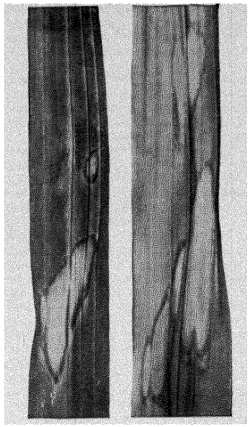


Fig. 14. Sun scald on palm leaves due to concentration of the sun's rays by bubbles in the glass.

NUTRITION AND OTHER CHEMICAL FACTORS

It has long been known that plants require ten nutrient elements, access to which is absolutely essential if they are to live and grow. These major nutrients are oxygen, carbon, hydrogen, potassium, phosphorus, nitrogen, sulphur, calcium, magnesium and iron. The first two are obtained from the air, all the others are absorbed in solution from the soil. In addition to these we now know that most, probably all, green plants require minute quantities of other elements, especially boron, manganese, copper and zinc, which must also be obtained from the soil. While all green plants need all these elements and probably also a trace of molybdenum they differ in their individual requirement of each. This is one of the factors governing the distribution of plants in nature and the restriction of some species to particular soils.

Lack of one or more of these essential elements leads to some form of plant disease, but it is not always easy to diagnose the exact deficiency involved from the appearance of the diseased plant. Precise information on the symptoms caused by particular deficiencies has been obtained by cultivating plants under controlled conditions, usually in sand or water cultures to which pure nutrient salts have been supplied. Omission of one element from a culture medium then usually induces a distinctive disease symptom in the plants which is cured by restoring the missing element to the culture. Symptoms of deficiencies in the field, however, do not always correspond precisely to those produced in artificial culture, especially as the former are usually attributable to an inadequate supply of the element concerned rather than to its complete absence.

Deficiency of several nutrients at once usually results in a general slowing down of growth and stunting of the plant. Absence or shortage of a single element more often induces a recognisable disease. An excessive supply of any element is also injurious, and in the case of some minor elements such as boron there may be quite a narrow zone of favourable concentrations between an inadequate supply on the one hand and an injuriously high one on the other. A correct balance of all the essential nutrients is the end to be aimed at, and in artificial manuring it is important not to supply large quantities of some elements without the others. Obviously if an element like boron is present in the soil in the quantity required for slow growth, sudden stimulation of growth, as by nitrogenous manuring, will lead to a suddenly increased demand for boron by the plant. If this cannot be met a deficiency disease will result, though from another point of view the disease may be attributed, indirectly, to an excessive supply of nitrogen.

Also to be taken into account is the strange phenomenon of " antagonism " between calcium and potassium. Each of these can be poisonous when supplied alone, but when present together in the soil solution they " neutralise " each other. In glasshouses excessive use of artificial manures may lead to an accumulation of salts in the soil which may be one cause of root diseases. This is estimated by the conductivity figure ($X10^4$). For most outdoor soils this index is usually below 1, never over 2. In hothouse soils

figures of 4–10 have been obtained, associated with browned roots and discoloured leaves of vine, tomato, cucumber, cyclamen, begonia, hydrangea, etc. (V. Nielsen and L. Hansen, 1935; *Plantesygdomme i Danmark*, 1935 and 1936).

There is also a peculiar inter-relationship between calcium (the active element in " lime ") and minor elements like manganese, boron and iron. Thus in swedes, sugar-beet and celery heavy liming may be followed by a diseased crop; brown heart in the case of the swedes, heart rot in the sugar-beet, cracked stem and sometimes heart rot in the celery. The crops may then be said to be suffering from excess " lime," but their diseased condition can be corrected by supplying borax at the rate of about one ounce to twenty square yards. It may then be found that the lime was harmless and even beneficial if boron is supplied with it. Many factors appear to be involved in this inter-relationship of lime and other elements. To some extent lime brings about chemical and physical changes in the soil which " fix " the minor elements and render them unavailable to the plant roots.

In addition to the essential nutrient elements there are many others, some of which may be beneficial, like sodium which has been shown to be able partly to replace the potassium required in some plants, such as sugar-beet. Others play no known useful part in plant nutrition but are injurious when present in excess; these are plant poisons in the strict sense.

In the following pages we shall deal with problems arising from deficiency or excess of the various major nutrients, minor elements and plant poisons.

Lime

Plants need a small quantity of the essential element calcium, but comparatively few soils, except on very acid moorlands, are actually deficient in calcium. The agricultural uses of " lime " depend on its influence on the reaction and texture of a soil and on the supply of other nutrients. Unfortunately " lime " is a very vague and unsatisfactory term, covering such chemically diverse substances as quicklime and its product slaked lime or hydrated lime, calcium carbonate or carbonate of lime, ground limestone, chalk, calcareous marl and various industrial by-products and refuse. All these substances have the common property of neutralising acids but their activity in this respect varies greatly.

Liming also changes the physical properties of soils, especially stiff clay soils in which it develops a crumbly structure which makes them much easier to work. Light soils, on the other hand, require little lime and are very frequently over-limed. They then become too loose, dusty and dried up, and such nutrients as boron, manganese and iron become unavailable to the plants. Liming counteracts the harmful effects of excess aluminium, iron and free acids in moor and marsh soils. A suitable lime content also prevents ammonia and potassium from being washed out of soil. A shortage of lime is also often associated with a shortage of phosphorus, in which case the two deficiencies must be remedied together.

Another indirect influence of lime on plant growth lies in its encouragement to growth of soil bacteria and hence to their conversion of ammonia to nitrates (nitrification). Over-liming, on the other hand, " burns " away humus and thus reduces the soil's capacity for retaining water and nutrients in solution.

Some plant diseases, such as club root, are directly encouraged by a lack of lime and are, at least partly, preventable by liming. Over-liming encourages other diseases such as scab in potato and beet.

The degree of acidity or reaction of the soil moisture is a useful indication of the lime status of a soil. This depends on the concentration of hydrogen ions present, which is measurable with great accuracy by laboratory methods and roughly by a colorimetric test. The concentration is expressed as a negative index of 10, the pH value. A pH value of 7 indicates a neutral reaction; figures lower than this indicate high concentration of hydrogen ions, and hence acidity; figures greater

than 7 indicate low hydrogen ion concentration and a state of alkalinity. Soil very poor in lime may have a pH as low as 3 or 4, more often 5 or 6. By heavy liming it may be possible to raise the pH of a soil to 8.3 and correspondingly high figures may occur naturally in chalk soils. Experience has shown that in nature many plants are found only on soils of a particular pH range. Sometimes, as in the case of some moor and heath plants, this is because they cannot live in soils rich in lime. More often it merely means that at other pH levels, though they may survive when grown in isolation, they are unable to meet competition of other plants and are smothered or crowded out.

Soil reaction alone does not indicate the amount of lime required to bring a soil to neutrality. The reaction of some soils changes rapidly after liming; that of others changes more slowly and to a lesser extent because their soil solution is "buffered" against change. Laboratory analysis can, however, give an approximate indication of the amount of lime likely to be required to bring an acid soil to the pH desired.

As regards the kind of lime to use, quicklime or slaked lime gives a quick result. Calcium carbonate or ground limestone is likely to be slower acting and is required in greater quantity. Calcareous marls have the advantage of often containing traces of minor elements and, on light soils, their clay content may be definitely beneficial. In any case liming should not be undertaken indiscriminately and preferably not until the soil has been analysed and advice obtained from an Agricultural or Horticultural Research Station or the advisory service of the local Agricultural College. Not only do wild plants differ greatly in their reaction to soil acidity but so do many common weeds of cultivation. The presence of corn marigold, or great quantities of sheep's sorrel or spurrey in a crop is a fair indication of an acid soil.

Most crop plants are rather tolerant in this respect and can be grown successfully over a wide pH range provided the soil is kept in good heart and well manured. Most, however, flourish best in soils of a particular reaction and a few, like rhododendrons and ericas, will not grow in the presence of lime. The following tables give the optimum pH ranges of some common garden plants.

ACID-LOVING PLANTS THAT SHUN LIME (CALCIFUGES)

Azalea (below 5.5)
Begonia (below 6.5)
Ferns (about 6)
Camellia (4–4.5)
Cyclamen
Erica (not over 6)
Gardenia (about 5)
Raspberry (6–6.5)
Hydrangea (4.5–6)
Hydrangea cuttings (4–5)
Potato (4.8–6)
Rhododendron (below 5.5)
Various species of Spruce

PLANTS LIKING NEARLY NEUTRAL SOILS

Antirrhinum (6–7)
Asparagus (a little under 7)
Mountain Pine
Mushroom (7)
Chrysanthemum (6–7)
Lily of the Valley (6.5)
Dianthus (6)
Carrot (6.5–7.5)
Strawberries (6–7)
Cabbage (6–7)
Swede
Stock (7)
Narcissus (6.5–7)
Parsnip (7)
Horseradish (7)
Pelargonium (6.5–7)
Pelargonium cuttings (4.6–7)
Primula
Radish
Rose (at the most 6)
Syringa (7)
Tomato (6–7)
Apple (6–7)
Fir Tree

LIME-LOVING (CALCICOLOUS) PLANTS OF ALKALINE SOILS

Cucumber (hothouse 7)
Cucumber (outdoor 7.8)
Plum
Beans
Cactus (7.5)
Endive (under 8)
Peach
Ash
Iris
Cherry
Chervil (7.5)
Sweet Pea (7.5)
Onion (7.5)
Melon
Parsley (7.5)
Leek (7.5)
Pear
Brussels Sprouts (under 8)
Beetroot (7.5)
Lettuce (under 8)
Scorzonera (7–8)
Spinach (7–8)
Gooseberries
Tulip
Elm
Peas (7.5)

Over-liming can be counteracted by dressings of sulphate of ammonia, or, if it has been very heavy, by mixing finely ground sulphur with the soil. When preparing compost for glasshouse use it must be remembered that leaf mould contains calcium while peat is acid. Where glasshouse soils have been rendered so alkaline that the plants suffer from chlorosis we have sometimes found good results to follow watering with a minor element solution. We recommend a solution of 10 oz. iron sulphate, 3 oz. manganese sulphate and 1 oz. potash in 6¼ gallons of water. Out-of-doors watering with a solution of 10 oz. of iron sulphate and 10 oz. of manganese sulphate in 6¼ gallons of water has proved most beneficial to raspberries, peas and leeks on calcareous soil (see *Plantesygdomme i Danmark*, 1933). These substances can also be applied in sprays (see pp. 595, 597, 598).

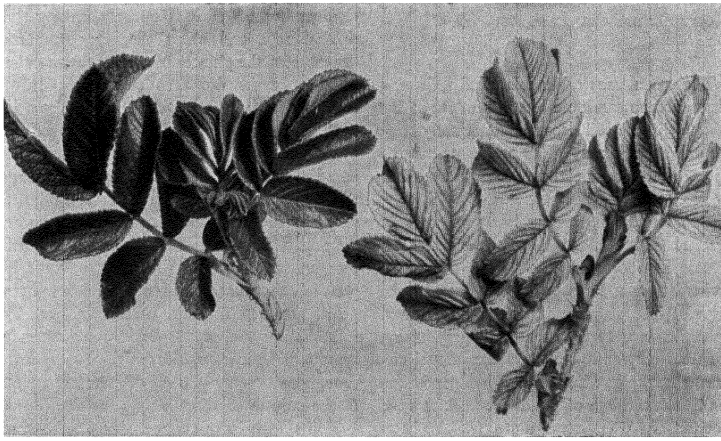


Fig. 15. Lime-induced chlorosis in rose shoots; shoot on left healthy, that on right from limed soil suffering from a deficiency of iron.

NUTRITIONAL DEFICIENCIES AND EXCESSES

Aluminium

A trace of aluminium may be essential to plant growth but it is never likely to become deficient in any ordinary soil. The amount of free aluminium is, however, closely bound up with the soil reaction. At low pH readings aluminium occurs as free ions in the soil solution, and lime-loving plants like lettuce and garden beet become prone to damping off, while their roots may become thick and lacking in root hairs (Bortels, 1941). Aluminium ions have even been regarded as a root poison but this does not apply to all chalk plants (C. Olsen, 1936). If poisoning of this kind does occur it can be checked by applying lime; manuring with superphosphate may also have a beneficial effect. Conversely, when rhododendrons or other lime-shunning plants have to be grown in a soil containing lime, addition of aluminium sulphate (and also of magnesium sulphate) may assist their growth.

It is well known that hydrangea flowers turn blue if alum is supplied to the soil or dissolved in the water used for watering them; the same result may be obtained by injecting the alum solution into the stalks or spraying it over the umbels.

Arsenic

There is a trace of arsenic in all soils, and plants absorb a minute quantity of it. If the quantity in the soil becomes abnormally large the soil bacteria may suffer and roots may be injured, but the quantity of arsenic absorbed by green plants does not increase appreciably. Such a condition is only likely to arise in practice when a considerable amount of arsenic has been supplied to the soil of an orchard over a long period of years in the form of lead arsenate in insecticidal sprays. Even then, apart from immediate spray injury, the trees only suffer in quite exceptional circumstances, e.g. when arsenic has penetrated the trunks through cracks in the bark or open wounds. In Washington State, U.S.A., however, barley and lucerne grown on the site of an old cleared orchard have been found to suffer from arsenic poisoning when the quantity of easily soluble As_2O_3 in the soil rose to 5–12 parts per million. Injuries from this source can be counteracted by the use of superphosphate or lime.

Sporadic reports of beneficial results in increased growth of plants like radish and maize as a result of manuring with white arsenic at the rate of 5 lb. per acre have never been substantiated and it is not certain that arsenic is essential or beneficial to green plants.

Another possible source of arsenic poisoning lies in the use of arsenic-containing baits for caterpillars. Pines in a nursery have developed scorching of the needle tips after lead arsenate has been distributed at the rate of 600 lb. per acre, and Paris green is said to be much more dangerous. The amount of free arsenic in soil depends partly on its constitution; clay and lime bind a certain amount of the arsenic present, sand rather less. Cucumbers, beans, tomatoes and peas are among the plants most liable to be injured by arsenic.

Boron

Water and sand culture experiments with highly purified salts have shown that probably all green plants require a trace of the element boron. If it is entirely lacking the tips of root and shoot die, the cambium degenerates, the phloem portion of the vascular bundles breaks down so that transport of sugars is impeded and starch accumulates in the leaves. Plants completely deprived of boron sooner or later cease growth altogether and die. On the other hand plants differ enormously in their need of boron; cereals require very little indeed and seldom suffer from boron deficiency in the field. Tomatoes require comparatively little, and in water cultures in glass jars two successive generations of tomato plants may obtain all the boron they need by solution from the glass, so that boron deficiency symptoms appear only in the third crop even though no boron has been deliberately added to the culture solution.

Plants with large storage organs, like beet and turnip, however, readily show signs of a lack of boron.

Under field conditions, though a normal amount of boron may be present in a soil, environmental factors, especially heavy liming or prolonged drought, may render it unavailable to the plants. Boron deficiency diseases will then set in. It has also been shown that the lignin liberated by rotting wood decaying in soil may bind boron so that it becomes inaccessible to plants (Midgley and Dunkley, 1940). In Danish gardens boron deficiency is most familiar as a cause of heart-rot in celeriac; in English gardens it sometimes causes brown heart of turnips (Jacobs, 1941). There is a trace of boron in farmyard manure, however, and an appreciable quantity in compost so that boron deficiency diseases are not usually a problem in well-cultivated garden soils. In farming practice, especially if only artificial manures are used, they may become important in sugar-beet and mangolds (heart rot and dry rot), swedes (brown heart) and celery (cracked stem). In parts of Canada, U.S.A., Australia and New Zealand apples suffer from boron deficiency (internal and external cork or drought spot of the fruit, dieback of the twigs, etc.). Similar troubles occur in fruit trees on calcareous soil in Switzerland and have been noted in Finland but are not known with certainty in English orchards, though an instance of fruit pitting probably due to boron deficiency has been described by Levy (1947). Potatoes in sand cultures without boron develop a stiff rolling of the upper leaves, due to accumulation of starch (van Schreven, 1939), and there is some evidence that the non-parasitic form of leaf-rolling characteristic of the variety Gladstone may be connected with boron deficiency. This, however, is not a condition of any economic importance, and, though there have been numerous reports of yield increases on particular soils as a result of manuring potatoes with borax or boric acid at the rate of 10 or 20 lb. per acre, general adoption of such a practice is not to be recommended. All plants are liable to suffer from boron poisoning if the required amount of free boron is appreciably exceeded, and potatoes appear to be particularly prone to injury of this kind.

In horticultural practice boron is usually supplied in the form of borax ($Na_2B_4O_7 .10H_2O$) at the rate of 15–20 lb. per acre or boric acid (H_3BO_3) at the rate of 10–15 lb. per acre. It is usually unwise to exceed these rates of application but in Denmark up to 30 lb. of borax per acre may be used in spring to control heart rot and hollow crown in celeriac.

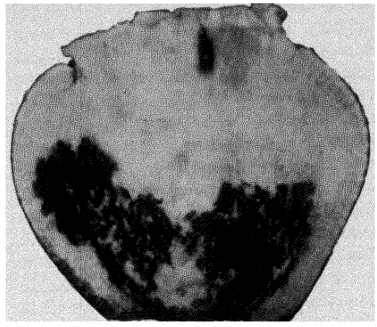


Fig. 16. Brown heart of swede, due to boron deficiency (*Photo Dept. Agric. Scotland*).

Calcium

In addition to the usefulness of calcium compounds (quicklime, chalk, marl, etc.) for improving the reaction and texture of soils, calcium is an essential element for plant-growth. It plays an essential part in all living cells and is of importance also to plants in its antagonistic effect to other elements. Plants grown in water or sand cultures without calcium develop red root tips and dead shoot tips somewhat reminiscent of the symptoms of boron deficiency. Potatoes in cultures without calcium also developed brown spots in the flesh of their tubers reminiscent of the internal brown-spot condition sometimes encountered on sandy soils in the field (van Schreven, 1934). Indirect effects of a lack of calcium include damage by free aluminium, manganese and iron ions, as is indicated under the heading of each of these elements. Diseases due directly to a deficiency of calcium are very rare in agricultural practice except on very acid soils. On reclaimed moorland of pH about 4, however, failure of potatoes to

come above ground, owing to death of the young shoots so that the seed tubers remain hard and " blind " in the drill, has been shown to be a direct result of calcium deficiency (Wallace and Hewitt, 1948).

Chlorine

It is well known that a number of garden plants (including cucumbers, strawberries, red currants, tobacco) grow less luxuriantly when a small amount of chlorine is supplied, as when muriate of potash is used, than when the corresponding chlorine-free manure, such as sulphate of potash, is used. On the other hand, many plants, especially those native to seashores and deserts, can tolerate large amounts of chlorine without injury. Asparagus and beet are derived from wild forms native to seashores and a little chlorine seems to be beneficial to them. Spinach is said to taste better and keep longer when a chloride has been used, but it nevertheless does well in warm and rainy weather if sulphate of potash is used instead. Beneficial effects of chlorine have been reported also with lucerne, onion, leek, radish, and other crucifers and with celery. On the other hand sulphate of potash has been found to be better than the chloride for potatoes, cabbages and rhubarb (Schupan, 1941).

Rainwater introduces a certain amount of chloride to the soil, particularly near the sea and when the wind is off the sea. At Spangsbjerg in Denmark a deposit of 80 lb. of chloride per acre a year from rain has been measured and at Askov an annual deposit of 27–36 lb. per acre (Frode Hansen, 1931). Scorching of leaves by winds off the sea is due at least in part to the salt they bring with them, for during gales spray is carried considerable distances inland. According to Laflin and Phillips, (1939) this scorching is due to rapid concentration of salt on the leaves as the drops evaporate, leading to withdrawal of water from the underlying cells. During a gale deposition of fine spray would be continuous over a considerable period and the salt deposit on the leaves would accumulate. Plants that suffer little damage from gales off the sea are those whose leaves have their cuticles impregnated with oily or waxy substances, e.g. cabbage, holly, ivy, pines, olearia, laurel and escallonia. Of apple varieties Philippa is said to be resistant to spray damage in Denmark.

Chlorine poisoning may also be observed when manure from latrines or stables disinfected with chloride of lime is used. A much commoner cause of poisoning in gardens and nurseries is the use of chlorates as weed-killers. Sodium chlorate used in this way is gradually converted to the chloride in the soil and is eventually washed out by degrees. If, however, treated soil is used too soon for crops characteristic symptoms of poisoning appear. They also occur when potted plants such as chrysanthemums are stood on paths or gravelled areas that have recently been treated with chlorates. The first effect of chlorate poisoning in chrysanthemums is a paling of the veins, followed by crinkling and distortion of the leaf, which turns grey-green all over. Growth becomes stunted and may cease altogether (Owen, 1936). Tomatoes react similarly, and in addition the leaves dry up and fall, while brown streaks appear in the stems and petioles (Bewley and Corbett, 1928). Winter cherry and dahlia are also very susceptible to chlorates, aster, cineraria and wallflower less so, while antirrhinums and ericas are comparatively resistant.

Chloride injuries to vegetable crops take the following forms:

Peas: yellow leaves, shrivelling first along the veins and ultimately withering altogether.

Beans: faint leaf spots (Fig. 7).

Carrot: yellow, faded tops.

Outdoor cucumber and strawberries: with large doses, the leaves wither as though frosted.

Indoor cucumber: withered shoot tips, shrivelling of the fruit tips, pale spots between the leaf veins. We have observed these symptoms where there was only 1.5 pM of common salt in the soil (1.6 tons per acre). When the sodium chloride content of the soil was reduced to 0.3 pM by leaching the growth of cucumbers became quite good again.

Certain kinds of tree have survived watering with water containing up to ½% common salt, apple among them, but whether it could stand

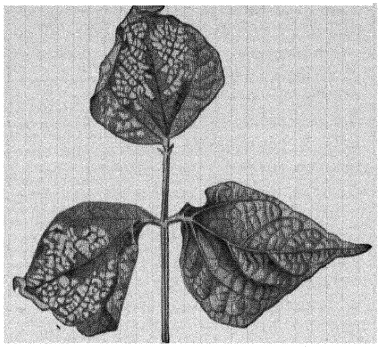


Fig. 17. Bean leaves with chlorine poisoning. The plant has grown on soil treated with sodium chlorate.

this year after year, it is hard to say. Cabbage, celery and leek also tolerate brackish water while carrots do not. In many parts of the arid south-western states of U.S.A. the presence of dissolved salts, especially chloride, in irrigation waters is a serious problem.

In this country reservoir water used for human consumption is often chlorinated for hygienic reasons. Many plants can endure 50 parts of chlorine per million, 100–150 are on the border line, while 200–300 parts per million give rise to visible disease symptoms on the leaves and with 500–1,000 the roots are affected also. The common freshwater weed elodea cannot endure 3 parts per million and goldfish not one.

Extensive and prolonged flooding by the sea occasionally occurs in parts of Lincolnshire, Norfolk and Essex. After the sea floods in the Horsey district of Norfolk, when much land was inundated with salt water for three months, commencing 12th February, 1938, all conifers and deciduous trees in the affected area died during the following summer. Even birches, which appear to suffer least, were nearly all dead by the end of June. Experience in the Netherlands shows that a few young oaks may survive such a prolonged immersion, while poplar and beech are more resistant to salt water than ash, alder, willow, horse chestnut or conifers. Sea floods also kill worms which only reappear in the third year after a flood. In the Norfolk floods land which had been under sea water for only 7–10 days had up to 0.3% of salt in its top soil in the week after the floods receded, but the concentration of salts due to evaporation gradually increased to a maximum of 1.6% in the top three inches during June. By the following December autumn rains had reduced it to 0.2% in the surface layers, with a corresponding increase lower down. In Holland no cropping is attempted until the salt concentration in the soil is reduced to 0.3% in the spring, and for normal farming a much lower concentration is required (Sainty *et al.*, 1939).

Efficient drainage is an important factor in washing out the residual salts, and this may be assisted by digging drainage channels at frequent intervals across each field. More serious than the actual presence of salt is its effect on the texture of the soil, particularly of the clay fraction. The lime normally held by the clay particles is replaced by sodium and the granular structure of the soil is destroyed, so that it becomes impervious to water, slimy in wet weather and hard and cindery in dry weather. Regranulation may be hastened by dressing with burnt lime or gypsum at the rate of 1–3 tons per acre, but only light harrowing to break the surface crust should be practised until the soil structure is restored. Ploughing will only make matters worse and delay recovery, especially on heavy land. After the salt content has been reduced to a low level and the structure has been restored a crop of rape, kale or mustard may be sown, followed by one of mangolds, sugar-beet, oats or barley. As garden crops spinach and possibly celery may be tried. After the Humber floods of 1921 in North Lincolnshire grassland recovered to some extent in 2–3 years, but much arable land was semi-derelict for 7 years and complete recovery took about 10 years (Cranfield, 1938). Because the flooding washes out nitrogen, potash, lime, magnesium and phosphates the reclaimed land will require considerable manuring.

When using sodium chlorate as a weed-killer care should be taken to spray only on

a calm day, otherwise the drifted spray may scorch adjacent hedges or crops. When watered over paths care should be taken not to exceed the recommended applications and not to go too near trees and shrubs or border plants. Otherwise, especially in rainy weather, it may penetrate to their roots and kill them; we have seen whole avenues destroyed by sodium chlorate in this way. Flower pots seem to retain sodium chlorate for long periods and are not freed from it by scrubbing or prolonged immersion in running water. The most effective means of removing it from pots is to soak them for 2 hours in a 5% solution of photographic hypo and then rinse in cold water (Owen, 1939).

Copper

Within recent years it has been shown that a trace of copper is necessary for normal plant growth. Under exceptional circumstances, especially on reclaimed moors and bogs, cereals suffer from the so-called "reclamation disease," characterized by bleaching and withering of the leaf tips and failure to head. This is due to copper deficiency and is corrected by a dressing of about 50 lb. of copper sulphate (blue vitriol) per acre. Symptoms of copper deficiency in tomato include stunting of growth and rolling of the leaves, which develop dead spots in their palisade tissue (Reed, 1939). Other horticultural crops reported to have shown poor growth on very humic soils deficient in copper are lettuce, onion and sunflower. In South Africa chlorosis of peach, plum and apricot due to the same cause has been reported, and in extreme cases copper deficiency may cause dieback and a disease of the bark in pear and citrus trees. All these conditions are controllable by broadcasting small quantities of copper sulphate or by using a copper spray such as Bordeaux mixture. Copper deficiency in vines has been described from Western Australia (Teakle *et al.*, 1943).

Drought, liming and excessive cultivation all accentuate a deficiency of copper.

In Danish experiments some response to copper manuring has been reported with strawberries, parsley and tomatoes (Lars Hansen, 1939), but there is no evidence of lack of available copper in any British arable soils.

While copper deficiency is injurious to many plants an excess of easily soluble copper is equally so. Spray damage to fruit trees by Bordeaux mixture is well known (see p. 45). In English experiments copper sulphate mixed with compost in the proportion of 1 : 160 was injurious to root growth and prevented germination of tomato seeds. The copper in Cheshunt compound and in Bordeaux mixture is in a far less toxic form. Experiments have shown that up to 0.07% of copper as Cheshunt compound for soil is quite harmless to tomato seedlings. This is far more than is applied in normal practice with this fungicide. Mature plants are far less susceptible to copper poisoning, and even 0.19% copper as copper sulphate in the soil had little effect on them. Where copper is present in excess tomato seedlings develop blue or violet stems and dark green cotyledons. The true leaves are first dark green but soon turn yellow with prominent veins. Young potted tomato seedlings have brown streaks on the stems and yellow top leaves, often purplish on the under side. Heavy watering with good drainage will remove much of the excess copper, but the best treatment for copper poisoning is a light top dressing with hydrated lime (Owen, 1929).

Iron

As long ago as 1843 the French botanist Gris demonstrated that if the leaves of plants suffering from chlorosis were painted with ferrous sulphate they soon turned green. Chlorosis due to iron deficiency is often serious in vineyards on limestone hills and also in pineapple cultivation where the soil has an excessive manganese content. In Danish gardens and nurseries it occurs from time to time, especially in roses, fruit trees, willows, hydrangeas, etc., and rapid recovery has been obtained by spraying the affected foliage with a solution of ferrous sulphate alone at a rate of 1: 500 or 1: 1,000 (approx. 1 oz. in 4–6 gallons). Some plants can stand a spray 10 times as strong. The iron can also be supplied in Bordeaux mixture at the rate of 1 oz. of

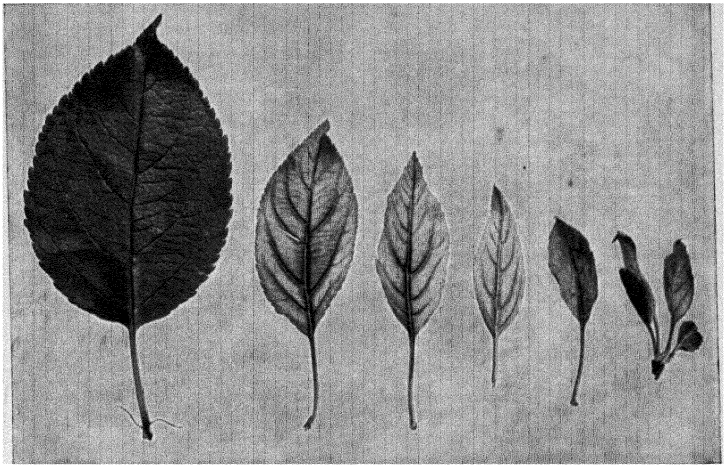


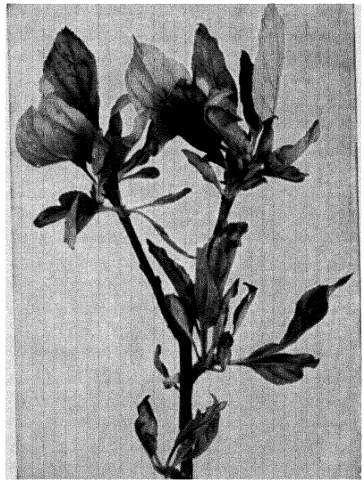
Fig. 18. Iron deficiency. Left healthy apple leaf. The others, leaves from trees on soil which has a high reaction and large content of phosphoric acid.

ferrous sulphate in about 3–4 gallons of spray. In France a winter spray is used on peaches and vines at a rate of 1–2 lb. of ferrous sulphate per gallon of water.

As a summer spray for vines they use 1 lb. in 10 gallons, a solution that etches a number of scarcely visible holes in the leaf and thus assures rapid entry of the iron into the tissues (Ravaz, 1925).

In a number of instances we have found chlorosis, associated with the production of rosettes of narrow leaves, on apple and pear trees which have received excessive quantities of superphosphate (or basic slag) and lime (see Figs. 18 and 19). In these cases the apple leaves are often rolled and the pear leaves are curled, while red or brown discoloured patches occur between the veins. Occurrence of a phosphoric acid index up to 100 or more with a pH nearer 8 than 7 indicates that the iron is probably rendered unavailable to the trees. It is not surprising that such soils show a very low manganese content.

In other cases the lack of available iron is due to the ground being too rich in lime. On calcareous soils the whole vegetation sometimes assumes a light-green hue. Presence of potassium and chlorine ions may also induce



iron deficiency. Chlorosis of this kind tends to be worst on dry loose soils lacking humus. In English orchards it has been controlled by the growth of a cover crop such as grass or lucerne.

Our own experience shows that chlorosis can be corrected by adding ferrous sulphate to the summer Bordeaux spray at the rate of 1 oz. in 4–6 gallons. In many cases amazingly good results have followed spraying with 1 oz. of both ferrous sulphate and manganese sulphate per 4 gallons of both the pre- and post-blossom sprays. In particularly obstinate cases a little zinc sulphate should be added as well.

Broadcasting ferrous sulphate (200–250 lb. per acre), zinc sulphate (200–250 lb. per acre) and manganese sulphate (100 lb. per acre) may also be effective but will act more slowly. We have also seen satisfactory results in an orchard watered at regular intervals with ferrous sulphate solution.

It must, however, be remembered that lack of iron is only one cause of chlorosis. It may also be a reaction to a lack of copper, zinc, manganese, magnesium, potassium or sulphur or to saturated soil, cold or lack of light. Slight differences in the disease pattern may help in deciding the cause of the trouble. With a lack of potash or magnesium the chlorosis is first visible, as a rule, on the lower leaves. When it is due to lack of iron it shows first in the upper, younger leaves. When sulphur is lacking the portions of the leaf blade along the main veins are yellower than the rest of the tissue whereas with a lack of iron this pattern is reversed. In manganese deficiency in addition to the pale colour a large number of spots generally appear on the surface of the leaves. With potassium and magnesium deficiencies the spots appear later and occur rather round the leaf-edges.

At East Malling Research Station methods have been worked out for rapidly diagnosing the cause of nutritional deficiencies by injecting shoots or even single leaves with solutions of the various nutrient salts.

Even when lack of iron in the leaves is the cause of chlorosis it cannot be assumed that iron is necessarily wanting in the soil concerned. The iron may be there but rendered unavailable by too much lime, oxygen, as in loose soils, manganese or phosphoric acid. Indeed, excess of lime may render several elements unavailable at once, e.g. iron, manganese and boron.

Less often, in waterlogged soils, iron may be present in a toxic form. Such a condition can be corrected by drainage and ventilation of the soil.

Lead

The lead present in lead arsenate has not so far been found to be injurious to plants. In experiments in Washington State, U.S.A. (*47th Annual Report*, 1937), large quantities of lead nitrate and lead carbonate were added to soil, but only small quantities of easily soluble lead could be detected in the soil afterwards and the effect on plants was, on the whole, favourable. On the other hand isolated instances have occurred where lead has been dissolved by water passing through lead pipes and 0.1 pM of lead in solution has been found to stop growth in cress and hyacinth (Dilling, 1926; Bell and Patterson, 1926).

Lithium

There is no evidence that lithium is required for plant growth but some interest has been aroused by reports that plants supplied with small quantities of lithium salts were rendered resistant to attack by powdery mildews. This is only the case at concentrations which are slightly injurious to the plants themselves and the discovery has probably no practical significance.

Magnesium

Magnesium is a constituent of chlorophyll, the green colouring matter of plants. It is, therefore, not surprising that a deficiency of magnesium results in chlorosis. In Denmark, however, it has only been noted in corn (Zebra disease) on sandy soil very deficient also in lime. In experiments at Hornum, brussels sprouts heavily manured with sulphate of ammonia developed very pale leaves, with stripes of green along the veins, possibly due to lack of magnesium. Magnesium deficiency also seems to be accentuated by high potash

manuring, for in the presence of excessive amounts of potash there may even be a reduction in the amount of magnesium absorbed by plants (Walsh and O'Donohoe, 1945). In recent years it has been shown that magnesium deficiency is widespread in English orchards and also in field and garden crops. It may also occur in greenhouse tomatoes (Cromwell and Hunter, 1942) and appears to be widespread in Irish potato crops.

The usual foliar symptom is the appearance of chlorotic spots between the veins, often associated with a downward curling of the tips and margins.

Some experiments have indicated that addition of about ¼ oz. of magnesium sulphate per pound of soil may correct the symptoms of lime injury in azaleas and rhododendrons grown on calcareous soil. A similar result has, however, been obtained by addition of aluminium sulphate. On a field scale magnesium deficiency can by corrected by a dressing of 250 lb. magnesium sulphate per acre. It can also be added to Bordeaux sprays.

Manganese

In the absence of an adequate supply of available manganese many plants develop yellowish-green, mottled, spotted or curled leaves. The condition is probably best known in cereals, especially oats, where it induces the disease known as " grey speck," in which grey patches appear on the leaf-blades, often causing them to bend over. Manganese deficiency has been much studied in Denmark and throughout northern Europe and it has been shown that drought and liming both contribute to render the manganese in the soil unavailable to plants (Steenbjerg, F., 1933, 1934, 1935). It is particularly likely to occur on bog, moorland and marsh soils, less so on pure sand and least on clays. When it is necessary to lime susceptible soils the manganese index number should be determined to indicate how much lime can safely be applied. During the ploughing-up campaign in the Second World War crop failures due to manganese deficiency were reported from the West of England on newly ploughed old grassland situated on alluvial silt of pH 6.6–7.0 (Wallace, 1941). Other soils liable to suffer from this condition are thin peats on calcareous subsoil, e.g. the " skirt " soils of the fens, alluvial soils derived from lower lias clay, wet patches of soil on carboniferous limestone, reclaimed heath soils too generously limed and old black garden soils which have received stable manure and lime regularly for many years. All are characterized by a high organic matter content and a high pH.

Crops highly susceptible to manganese deficiency are oats (grey speck disease), sugar-beet and mangolds (speckled yellows), garden beet, spinach and spinach beet (chlorotic mottling of leaves) and peas (marsh spot). Less susceptible are potatoes (speckling of leaves), parsnip (chlorotic mottling), parsley (browning of leaf edges), cos lettuce (chlorosis and leaf scorching round the edges), onions (white streaking of leaves), dwarf beans, runner beans and marrows (chlorotic mottling of leaves). Apples and raspberries also show chlorosis between the leaf veins (Wallace, 1941).

The symptoms of manganese deficiency are discussed in detail under the individual crops.

Onset of manganese deficiency may be prevented by the use of acid fertilizers, such as sulphate of ammonia in place of nitrate of soda, by manuring with fresh (not wind-dried) eel-grass or seaweed (15–20 tons of seaweed correspond to about 130–175 lb. of manganese sulphate) and by keeping the ground as firm as possible. Where manganese deficiency is very well developed any excess of lime may be neutralized by sprinkling powdered sulphur over the ground before digging. The amount to use, usually several hundredweights per acre, must be determined by titration of a soil sample. For apple trees affected with chlorosis due to manganese deficiency, Wallace and Jones (1942) recommend adding 3 lb. manganese sulphate per 100 gallons of the lime sulphur spray applied at petal fall. Research at Long Ashton has demonstrated certain disease symptoms associated with acid soils which are in fact due to manganese poisoning (Wallace *et al.*, 1945). Among these are intervenal chlorosis and brown spotting of runner bean leaves and

incurled margins of cauliflower leaves. Such conditions are controllable, at least in part, by liming and by the use of phosphatic fertilizers.

Mercury

When corrosive sublimate (mercuric chloride) or organic compounds of mercury are used for disinfecting soil some scorching may develop under exceptional circumstances, e.g. on hydrangea leaves and rose-buds at high temperatures in closed glasshouses. The damage can be overcome by watering the soil and providing adequate ventilation.

Molybdenum

It has lately been shown that molybdenum is probably an essential element for normal growth of green plants. Among the species for which this has been demonstrated is lettuce, but the quantity required is so small that it is very unlikely that deficiency of molybdenum will be found to be a cause of lettuce disease in normal horticultural practice. On certain soils in Australia, however, molybdenum deficiency is an important factor limiting the growth of many crops (Piper, 1940; Anderson, 1942, 1946; Anderson *et al.*, 1946; Teakle, 1944). Plants readily absorb molybdenum when it is present in the soil, and in the " Teart " pastures which cover some 20,000 acres of Somerset the herbage contains so much molybdenum as to be poisonous to dairy cows and to cause scouring in other cattle and sheep (Ferguson *et al.*, 1938) (see also whiptail of cauliflower, p. 259).

Nitrogen

Most crops suffering from nitrogen deficiency are pale green in colour, fading to yellow in extreme cases, especially in the leaves. Red tones may also occur, particularly on the stalks and leaf veins. Growth as a whole is stunted and " hard." In tomatoes it has been shown that nitrogen starvation reduces the number of blossom buds formed, the percentage of buds that open and the number of fruit per truss. The rate of development of successive flower trusses is retarded and the time that elapses between flowering and ripening the fruit, which may be shortened by slight nitrogen deficiency, is lengthened when the deficiency becomes acute (White, 1938).

The poor condition of apple trees on lawns is often attributable to lack of nitrogen, especially when they show weak shoot growth, proportionately small and open foliage and small brightly coloured fruit. Leaching, removal of more nitrogen in produce than is replaced by fertilizers, and progressive impoverishment of the soil by perennial crops all contribute to bring about a deficiency of nitrogen.

Manuring with sawdust or digging in straw as a source of humus in place of farmyard manure may also lead to sudden impoverishment in nitrogen. This is due partly to the fact that the bacteria which are active in decomposing the straw consume most of the nitrogen in the soil. This is easily put right by a dressing of a nitrogenous fertilizer. In breaking down the straw, however, they may also liberate toxins (vanillin, salicylic and stearic acid compounds) which may bring about a form of soil exhaustion, especially on sandy land (Collison and Conn, 1925). For nitrogen deficiency in relation to fungus attacks see potato blight, p. 312. Where there is an excess of nitrogen, especially if potassium and phosphoric acids are in short supply, most plants develop a dark-green luxuriant growth which gardeners term " soft." Such plants form numerous new vegetative shoots, but



Fig. 20. Tomatoes misshapen because the plant has grown on soil containing excess of nitrogen: perhaps, also, it has been too dry.

ripening of wood, flavouring and fruiting are all retarded. Because of the delayed or defective ripening an excessive nitrogen supply is associated with increased sensitivity to frost. Such plants are also more liable to suffer from invasion by many fungi. Tomatoes grown with excess nitrogen tend to produce hollow stems and monstrous ribbed fruits (Fig. 20) and cucumbers under the same condition are apt to have blind shoots. On the other hand hydrangea and other plants of acid soils may grow best with abundant nitrogen.

Oxygen

Poorly ventilated soils whose roots have an inadequate oxygen supply are not uncommon in nature and are occupied by characteristic plant associations. Plants of bogs and wet moorlands are able to thrive on soils deficient in oxygen. Seeds also differ greatly in the amount of oxygen they require before they can germinate.

When oxygen deficiency supervenes in a normally well-ventilated soil many plants are killed, e.g. after a cloudburst or a prolonged covering of snow (see also rot of potato tubers, p. 335). Many potted plants succumb to overwatering because their roots become suffocated.

It will be noted that in all these cases it is a deficiency of oxygen for respiration of the root system that is involved. The above-ground parts of the plants are able to respire normally. Not only does lack of oxygen in the soil check root growth and impede respiration of the living cells, but in water-logged soils there often accumulates excessive quantities of carbon dioxide, sulphuretted hydrogen and ammonia that are poisonous to roots and may even kill them without assistance from any parasites. Flooding for a short period may cause death of the fine lateral roots; only the tap-rooted plants may survive and put out fresh laterals after the soil has become ventilated again.

Plants or parts of plants often suffer from a lack of oxygen during storage or in transit. Within limits, respiration rate and hence oxygen consumption increases with temperature; moreover rapid respiration liberates heat and still further raises the temperature, a vicious circle that has caused much damage in the " heating " of potatoes, cabbages, etc., stored in heaps or piles of sacks. The final stages of destruction are usually then brought about by fungi.

Phosphoric Acid

Manurial experiments have shown that certain traits are common to all plants lacking phosphoric acid. Their chlorophyll becomes dark, and relatively insoluble, and there is a tendency to production of violet, red or golden tints. The plants often have an upright, stiff habit of growth and their consumption of water is reduced. Root growth is weakened and the roots become more liable to attack by species of *Fusarium* and other root-rotting fungi. Rust and mildew may also be encouraged, especially if the deficiency of phosphoric acid is correlated with an abundant supply of nitrogen. In some plants fruiting may be accelerated. Phosphoric acid-deficient potatoes appear to be abnormally susceptible to blackleg and tuber rots but less prone to blight. Onions are also more liable to rot. Plum trees grown in tubs with an inadequate phosphoric acid supply become chlorotic. Vine leaves under these conditions acquire a dirty yellow margin and there may even be a narrow dead marginal zone (Konrads, 1936), while black-currant leaves become spotted (Wallace, 1930).

Red colouring is characteristic of phosphoric acid-deficient lettuce and tomatoes; tomato seedlings show stiff shoots with violet-coloured leaves pointing upwards, and in older plants there is a tendency to production of cracked fruit.

Chrysanthemums lacking phosphoric acid are said to produce loose flower buds, and observations in 1934 during manurial trials at the plant pathological experimental ground suggest that their flowering may be delayed.

The interaction of nutrient elements in plant growth is very complicated and the results of a relative deficiency of one essential element may be far-reaching. With a lack of phosphoric acid compounds of iron and aluminium may become toxic while sometimes even if

phosphoric acid is available in the soil plants are unable to utilize it because they are short of calcium, magnesium or potassium. In all schemes of manuring the ideal to be arrived at is a balanced supply of all essential nutrients with none present to excess or in short supply.

In Danish experiments it has been found that apple trees become chlorotic when heavily manured with phosphoric acid (see Iron, p. 37, Fig. 18). Excessive doses of phosphoric acid may also reduce frost resistance by increasing the phosphatic constituents of the plant cells, as in winter spinach (Wilhelm, 1935).

Potassium

Potassium is an absolutely essential nutrient for plants. It is required for growth of all young cells, for formation of starch and proteins, for balancing the reaction of the cell sap and especially in regulating the water relations of the cells, promoting water absorption and controlling evaporation. Experiments have shown that with a deficiency of potash water absorption is decreased and the stomata tend to open, thus facilitating water loss by transpiration. Apple trees generously manured with potash, however, tend to develop a shallow root system. While the water supply remains favourable they produce heavy crops, but during drought they suffer more than neighbouring potash-deficient trees with a deep root system (Grubb, 1928).

Mildew is said to be encouraged by potassium deficiency in apple, gooseberry and redcurrant, and most fungus attacks are likely to be encouraged if deficiency of potassium is associated with a high level of nitrogen. Seeds produced on potassium-deficient plants may have a low germinative capacity. Potash manuring sometimes enhances frost resistance (Wilhelm, 1935). In tomatoes potash starvation has no effect on the number of flowers produced but does reduce the quantity of fruit set. It also lengthens the period between blossoming and ripening of the fruit (White, 1938).

Liming and a free, open soil seem to intensify a shortage of potassium. Potash deficiency also tends to be accentuated by low light intensity, e.g. in hothouses during winter. Among plants with a high potassium requirement may be mentioned cabbage, potato, garden beet, celery, carrot, rhubarb and pineapple.

Discoloured leaves and especially a scorching round the edges are a very general indication of a lack of potash. The leaf colour often first becomes unnaturally dark blue-green, due perhaps to a relative excess of nitrogen, and then often assumes a brownish-purple hue. In herbaceous plants lacking potash the root system is often poorly developed, carrots and garden beet tend to be woody and carrots, cabbage and onions become less suitable for winter storage. Symptoms of potassium deficiency in cabbage, potato, redcurrant, tomato and apple are described in detail under these crops (see also plates 1, 2 and 3).

Like any other fertilizer potash can be applied in excess. Manurial experiments at Hornum (Denmark) showed that growth of mountain pine was restricted by potash manuring when that of other trees was favoured by it (Hakon Sorensen, 1936). Common spruce may develop reddish-brown needles when supplied with excess potash.

Because potash has a beneficial effect on tomatoes suffering from the virus disease streak it has sometimes been applied to glasshouse crops in unreasonable quantities. The resultant accumulation of large quantities of salts in the soil has certainly contributed to the outbreak of various root diseases so far not properly understood.

Sulphur

Plants require considerable quantities of sulphur. This applies especially to leguminous species which store large amounts of protein while crucifers are said to need as much sulphur as phosphorus. Where superphosphate, sulphate of ammonia or sulphate of potash are used as fertilizers plants will certainly never be exposed to lack of sulphur. Even the sulphur supplied in Bordeaux mixture and lime-sulphur sprays is accessible to plants sooner or later. Symptoms of sulphur deficiency have been described from water

culture experiments. Under these conditions tomatoes develop long slender stems without side shoots, thick, firm leaves, pale green above, purplish on the under-side, and plentiful development of woody tissue. Tobacco shows a copious development of white roots, and slender stems with yellowish-apical leaves in which the veins are markedly yellow. Other plants suffering from sulphur deficiency display similar symptoms.

In ill-ventilated soils, especially muddy, boggy or swampy soils, sulphuretted hydrogen may accumulate, easily detectable by its smell of rotten eggs. This may be toxic but is easily removed by drainage and ventilation. Presence of pyrites in the soil may be more serious as in the presence of oxygen this yields sulphuric acid, by which the pH of a soil may be reduced to 3 or even 2. Before such soil can be used for any crop, even those preferring acid soils, heavy liming is required, probably at the rate of 10–20 tons per acre. As humic soils are easily ruined by over-liming, the lime requirement should first be determined by analysis and only the recommended quantity of lime applied.

Zinc

In California chlorosis and " little leaf " or " rosette " of citrus and other fruit trees may be due to a deficiency of available zinc, especially on soils rich in phosphoric acid, potash and soda. Such conditions are curable by zinc sulphate sprays, by placing a few cupfuls of zinc sulphate in holes bored in the trunks or even by knocking zinc-galvanized nails into the bark. Zinc has also been used with success in a vine disease (Dufrenoy, 1935). Simple zinc deficiency in fruit trees has not been identified in England, but in Denmark apple trees with narrow, chlorotic leaves recovered when supplied with zinc and iron together. A somewhat similar instance of acute chlorosis in cherry in the Sittingbourne district of Kent has been shown to be due to a multiple deficiency of iron, manganese, and zinc (Thompson and Roberts, 1945).

Water cultures have shown that traces of zinc are essential for normal growth of many vegetables and probably for all green plants. The quantity required is so small, however, that it is always likely to be supplied in normal horticultural practice.

Where plants are grown under wire netting cages to protect them from birds they may occasionally suffer from poisoning by zinc dissolved in rainwater dripping from the galvanized netting. This can be much more extensive in glasshouses where sulphur has been burned as a fumigant. The sulphur dioxide produced may not only dissolve in drops of water on overhead galvanized wires and thus produce drops of zinc sulphate solution, but similar solutions may also be formed on the surface of paint containing zinc oxide. These drops cause dark-brown patches, often with a white deposit round the edges, on any leaves on to which they fall (Read and Orchard, 1934).

Ammonia Poisoning

Sulphate of ammonia is an excellent fertilizer for many crops, but fumes of free ammonia are poisonous even at very low concentrations. In hothouses ammonia may sometimes be released from manure that has been used while too fresh, and in such cases the fumes may cause a bleaching of the edges in the lower leaves of cucumber plants. Damage may also be caused to seedlings, dark green or blackish leaf spots may appear on tomato plants and the leaf stalks of begonias may become soft and easily snapped. Contamination of the gas with a small amount of ammonia may lead to damaged foliage when cylinders of carbon dioxide are used in hothouses. White variegation of the leaves may appear in tomato seedlings that have received an overdose of Cheshunt compound.

The extent of the damage depends on various external factors; it is worst in bright light, less severe in shaded plants and at night. Well-nourished plants growing well suffer less than weak ones. In all cases the green leaf tissue is most affected and the flowers the least. Susceptibility to damage by ammonia fumes also depends partly on internal factors. Plants with weakly acid cell sap (pH over 5) have their growth retarded by 1 part of ammonia

in 7–8,000 parts of air, whereas plants with very acid sap (pH under 5) are uninjured by such small quantities (Garber, 1935).

Where ammonia is used in the freezing plant leakage of fumes may damage fruit and onions in cold storage. They then develop brown or dark-green stains. In such cases the cause of the trouble can be verified by microchemical tests.

Poisoning by Coal-gas

Gas-poisoned trees are not uncommon in the streets of towns. In Copenhagen there is said to be an annual mortality from this cause of 27–28 trees or about 0.17% of the 16–1700 in the city (Østergaard, 1932). No gas pipe is absolutely free from leaks, and when the ground is disturbed by heavy frost or by the shaking of heavy traffic the joints are strained and breakages may occur. With old gas mains corrosion may cause leaks. The effect of a gas leak on neighbouring trees depends on the time of year and the amount of gas escaping. The leaves unfolded in spring may be unusually small, wither from the edges and finally hang shrivelled and dead or sudden yellowing and falling of the leaves may occur. On the branches the bark dies and that of the main roots flakes away. The root tips are killed and in older parts of the roots the cortex swells up to three times the normal thickness, and the bark becomes split, bluish or violaceous grey in colour and often stinking and slimy. Lenticels show abnormal growth (Woffenden and Priestley, 1924).

The leak in the pipe should be sought in the vicinity of the dead trees with the aid of a gas detector. Often when the ground is broken the smell of gas is at once noticeable. In other cases when the cause of death is uncertain samples of the soil may be taken to the laboratory and examined for oxygen deficiency or for the presence of hydrogen and other constituents of coal-gas.

In hothouses also poisoning may result from leaks in gas mains nearby. The usual symptoms are: first, yellowing of the leaves which shrivel along the margins and veins; secondly, shedding of leaves and buds. In cases of weak chronic poisoning cessation of longitudinal growth may be observed, followed by the growth of short side shoots from dormant eyes. Bark may develop swollen lenticels or become powdery (tan bark) and flower buds may open with papery withered corollos. If the fumes are in the soil the roots may swell near their tips, with splitting, shedding and death of their bark or the whole root may become curled and contorted. Similar symptoms are often seen when pot plants in bloom are brought into living-rooms illuminated by gas or into rooms adjoining a kitchen where a gas stove is in use. Experience has shown that all plants are not equally susceptible to damage by coal-gas. Aucuba, aspidistra, billbergia, dracaena, *Ficus elastica* and several palms can endure gas, hence the popularity of many of them as pot plants in Victorian houses (Ferdinandsen and Buchwald, 1936); the following are known to be sensitive: Azalea, camellia, crassula, cyclamen, hyacinth, lily, narcissus, pelargonium, primula, roses, tulips and ivy.

If the leak is very large so that the soil becomes filled with gas, roots may be killed by suffocation (oxygen deficiency). More often the damage is due to traces of by-products which are highly poisonous to plants. Traces of cyanide are believed to be the main cause of injury operating through the root system. In houses where it is gas in the air that causes the trouble traces of carbon monoxide and ethylene are usually implicated. Investigations at the Boyce Thompson Institute, New York, have shown that the latter is particularly injurious. Gas can only be smelt in a house at or over a concentration of 1 in 200 parts of air. Chemical tests for hydrogen or carbon monoxide can detect gas at a concentration of 1: 100,000, and at concentrations over 1: 20,000 it can be measured exactly. Many plants are damaged at concentrations as low as or lower than these. Tulips and other bulbous plants develop rolled leaf tips and have their growth checked at 1: 40,000; tomatoes and pelargoniums are damaged in 24 hours at a concentration of 1: 30,000. Tomatoes are very useful indicators of the presence of gas, for strongly growing seedlings react to a

concentration of about 1 : 200,000 in a closed room. Their leaf stalks curve so that the leaves bend downwards. Cinerarias, another useful test plant, have their leaves curved upwards (Hitchcock *et al.*, 1934; Krone, 1937). Carnations are especially sensitive to traces of ethylene; 1 part in 2,000,000 of air may cause open buds to close and 1 : 1,000,000 will prevent buds from opening.

Prevention of tree poisoning by gas leaks ought to be a consideration when gas mains are laid. At least two yards of undisturbed soil should separate the line of trees from the gas main. A further refinement that has been tried consists in laying the main inside an earthenware pipe which has air vents leading to street level at regular intervals. In Frederiksberg Allé (Denmark) the joints of the gas pipes are laid in shingle within shafts closed by a cover which allows easy access for control of gas leakage. Better than the usual method of joining pipes by filling the muffs with molten lead is that of employing lead shavings. This is a little more expensive but withstands shaking better. It is important to keep a watch on street trees so that gas poisoning can be detected at the outset. The soil can then be dug and ventilated in time to save the tree. If a tree killed by gas poisoning at the root is to be replaced a large hole must be dug and filled with water for several days to wash out the gas, or the hole must be left open for ventilation for at least a year. The bottom and sides of the hole may then be covered with a layer of clay stamped hard and at least 10 inches thick to form a gas-tight cavity which can then be filled with fresh soil (Østergaard, 1932). If clay soil has become permeated by gas during a prolonged frost, when the gas spreads undiscovered under the frozen crust of soil, through which it is unable to escape, planting of fresh trees may be hopeless for several years to come. Even after holes have been dug in such soil and left to air for a long time, fresh soil added to them will still be poisoned by gas from the surrounding soil. As mentioned above, young tomato plants may be used with boxes of earth dug from one or two spits deep as indicators of the lingering presence of gas in toxic concentrations (Hitchcock *et al.*, 1934).

Where gas pipes run parallel to a hothouse, infiltration of gas may be prevented by digging a channel between the main and the house and filling it with coarse slag to form a kind of gas drain (Krone, 1937).

Slag from gasworks, sometimes used as a weed-killer on footpaths, may poison neighbouring trees or bushes, which then show yellowed leaves with shrivelled edges. They will usually recover if the quantity used was not excessive.

Injury by Therapeutant Chemicals

Chemicals used as sprays, dusts or fumigants may be regarded as " medicines " having a beneficial effect against the pest or fungus causing a particular disease but liable also to have a subsidiary injurious effect on the crop. This generally consists in a scorching, especially of leaves. Usually, but not always, young tissues are more sensitive than old ones.

Treatments usually harmless may at times cause scorching and, with two sprays each of which is harmless in itself, scorching may sometimes occur when the second is applied while traces of the first still remain on the plant. Method of spraying, weather conditions and general health of the tree also influence the liability of leaves to scorching. Leaves of undernourished trees or of those in cramped surroundings or in dry soil are more liable to damage than those grown under optimum conditions. Trees weakened by winter injury such as occurred especially during the winters 1940–1 and 1941–2 are particularly sensitive to chemicals.

Moore (1936) found that Bramley's Seedling apples were more damaged by the post-blossom Bordeaux spray when the trees had been manured with nitrogen alone than when they had received a complete fertilizer or had not been manured at all. The root stock may also have an effect; trees on Malling Nos. I, IV and V were more injured than those on Malling Nos. II, III, VI, VII, and X. Malling No. V (Red Doucin) has been shown to be conducive to Bordeaux injury in Denmark also.

Menzel (1935) found that manured apple trees were less sensitive to copper sprays than unmanured trees and that those that did not fruit were more sensitive than trees that bore well. According to Danish experience, however, fruiting trees endure spraying worse than do non-bearing ones.

The results of experiments at one locality cannot be assumed to hold good everywhere until they have been checked. For example, spraying tomatoes with Bordeaux mixture has invariably had only beneficial results in our experiments, yet in New York State it may cause so much damage as appreciably to reduce the yield in spite of the control of fungi achieved. Bordeaux mixture 8:8:100 is said there to kill the growing points, cause misshapen leaves, shedding of flowers and the growth of more or less damaged scabby fruit (Horsfall *et al.*, 1938). Whether the great divergence between the results obtained in Denmark and in New York is to be ascribed to the use of different tomato varieties, to differences in climate or to other factors is not clear.

Besides the injury that may be caused by chemical " medicines " used strictly in accordance with the most recent recommendations one has also to reckon with injuries due to mistakes. One should always be accurate in measuring or weighing both water and chemicals; guesswork involves risks. When a grower takes a chance in estimating quantities he may apply too weak a solution and so waste his time and labour, or it may prove too strong and scorch his plants. Mixing a spray with the dregs of a previous one left in containers or sprayer may also have disastrous results.

Confusion of chemicals may be fatal. We know of one instance where all the plants sprayed were severely scorched because sodium chlorate had been used instead of lead arsenate. Care should be taken not to lose labels on jars, bags or tins of chemicals. Random modification of sprays is another fruitful source of damage. In one instance a large number of Czar plum trees lost nearly all their leaves because this variety, sensitive to nicotine, was sprayed with a nicotine soap solution to which 1 pM bird lime had been added " because we happened to have the bird lime."

Symptoms of spray injury are best known on apple, probably because this is the most extensively grown fruit and the one most thoroughly sprayed. In apples and in other plants, spray damage to the leaves appears as yellow or brown spots or scorched brown edges. In severe cases the leaves tend to fall. As a general rule perhaps yellowing results mainly from lime-sulphur sprays and brown spotting and brown-edged leaves mainly from Bordeaux mixture, but it is impossible in practice to tell from single leaves which spray has damaged them unless traces of it remain. Indeed it is usually impossible to tell from looking at the leaves whether the damage was due to spraying or to unfavourable weather or conditions of growth. Especially is this the

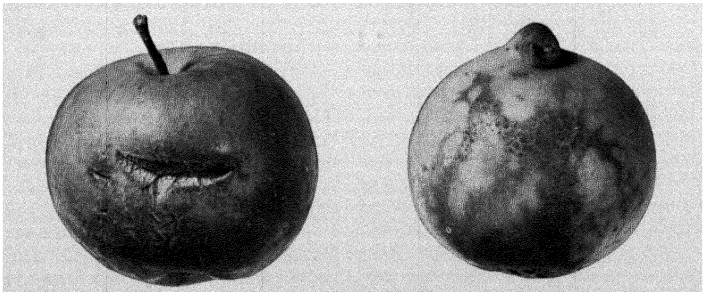


Fig. 21. Spray injury to apples.

case when one does not see the trees in question but has only a few leaves submitted without detailed information about their history and environment.

The way the spraying is done often greatly influences the amount of damage. One should work with a spray at high pressure so that the atomization gives a fine mist on the leaves, but the nozzle should not be held close to the parts being sprayed. If this is done the force with which the liquid strikes the plant may be sufficient for it to penetrate the skin of both leaves and fruit, and thus cause mechanical injury, aggravated later by the chemicals present. Where a spray gun is available it should only be used as a jet and then only where necessary to reach the tops of trees.

Lime sulphur injury leading to shedding of leaves is more likely to follow spraying in bright sunshine on a warm day than spraying done under cool cloudy conditions. On the fruit lime sulphur may cause russetting, or, more often, small red spots. Bordeaux mixture mainly causes damage to apple fruits while they are still downy. The skin is roughened and in the worst cases it cracks (Fig. 21). Bordeaux sprays late in the summer may cause small red spots on the fruit. The damage occurs especially when the weather is and has been cold and damp.

Susceptibility of Apple Varieties in Denmark to Injury by Fungicidal Sprays and Dusts

\+ slight ++ moderate +++ high susceptibility

	Bordeaux Mixture		*Lime Sulphur*		*Sulphur Dust*	
	Leaves	*Fruit*	*Leaves*	*Fruit*	*Leaves*	*Fruit*
Adams Pearmain		+++		++		
Beauty of Kent (Springgrove)		+				
Belle de Boskoop		++	+	+		
Blenheim Orange		++				
Bramley's Seedling		+	+			
Cox's Orange Pippin	++	++	+	+		
Cox's Pomona		+		+		
Frogmore Prolific	+		+++		+++	
Gladstone	++		++			
Golden Delicious	+++	+++	+++	+++		
Hawthornden	+		+++		+++	
Keswick Codling			++		++	
Lanes Prince Albert			+++		+++	
Lord Grosvenor			+++		+++	
Lord Suffield			++		++	
Manks Codlin			++		+++	
Newton Wonder			++			
Pigeon			+		+	
Queen		++	++		++	
Rev. W. Wilks			++		++	
Rival			++			
Rivers Early Peach	++					
St. Cecilia			++		++	
Stirling Castle			+		++	
White Transparent		+	+			
Wealthy		+	+			

Note: The reactions of nineteen Danish varieties unknown to Taylor (*Apples of England.* 1946) have been omitted.

Damage by winter tar-oil sprays. If winter sprays are applied before March 1st at the prescribed strengths they cause no injury. Yet winter oil sprays applied several years in succession may in time damage the bark and cause the development of large scalded patches. The apple varieties Lord Suffield and Sønderskov are particularly sensitive to late winter sprays. Newly planted trees stand these sprays very badly.

Among chemicals in general use one must refer also to damage by nicotine, lead arsenate and calcium arsenate sprays. Further information will be found under the various kinds of fruit in the section dealing with fungicides.

Almost all plants can stand a nicotine spray, but see pp. 46, 585.

Lead arsenate may cause spray injury, especially shedding of leaves when used dissolved in water and still more when added to a lime sulphur spray.

Calcium arsenate, much used in Denmark to replace lead arsenate during the war, has a rather greater tendency to cause injury, especially to stone fruit trees and to sensitive apple varieties like Cox's Orange Pippin.

Damage to apple varieties by sprays and dusts. The table on p. 47 has been drawn up partly from the answers to the 55 questionnaires sent out in 1933 and 1934 to fruit growers and experts and partly from additional experience gained in Denmark during the past few years. Little reliance can be placed on statements regarding relative susceptibility to sprays published by foreign experts because of the great influence of local conditions already mentioned.

In the table "Bordeaux mixture" covers both strengths 4:8:100 and 8:8:100 as it has not always been possible to tell from the replies which of these was used. Bordeaux damage is usually caused by the post-blossom spray or by one on the young downy fruit. Lime sulphur is usually employed in Denmark at a strength of 2 gallons per 100 gallons of water. Some susceptible varieties may well be able to endure a lime sulphur spray at half that strength. This 1:100 spray is that usually recommended for the post-blossom spray in England. As a rule apple leaves are more sensitive to lime sulphur during the summer than before flowering, when a 1:40 spray is often quite safe to use. Under English conditions scab can be controlled by a pre-blossom spray on most varieties sensitive to lime sulphur. In addition to those indicated on p. 47, Beauty of Bath and Duchess's Favourite are regarded as sensitive to sulphur sprays in England. Stirling Castle is regarded as extremely sensitive and lime sulphur should never be used on that variety in England. Lord Derby and Worcester Pearmain are considered highly sensitive to Bordeaux mixture.

Summer petroleum sprays ¾:100 have been successfully employed to control red spider on many varieties in a big orchard. Elsewhere at strengths of 1:100 and 1¼:100 they have caused injury to some varieties, notably Cox's Orange Pippin, Queen Louisa, Keswick Codlin and Pederstrup, while others were uninjured. Much depends on thorough emulsification of the oil and on its remaining emulsified; otherwise plants are easily scorched by it. It is better to pour the water into the spraying oil than the other way round, stirring very vigorously all the time. A mixed petroleum and Bordeaux spray has severely damaged the fruit of Bellefleur de France, even though applied after they had passed the downy stage. A petroleum spray should not be mixed with lime sulphur as this combination may cause scorching. Similarly a petroleum spray should not follow closely on a lime-sulphur spray or a sulphur dust or vice versa. Three to four weeks should elapse between these treatments.

A table like that given here can only be regarded as a rough indication of the kind of spray likely to be safe on a particular variety. Where trouble has been experienced or where other varieties are being grown it is well to seek the advice of the nearest Horticultural Research Station which will be able to recommend sprays likely to be safe under local conditions. As a general rule, however, varieties marked +++ in the table for a particular spray should not be subjected to that treatment. If circumstances render it necessary to do so, one should proceed very

cautiously, preferably trying out the spray on a few trees first or, when time will not allow this, spraying very carefully so as to avoid soaking with a strong jet. Also it must be remembered that even when a variety has endured a treatment unscathed once it does not follow that it will always do so under all circumstances.

We know of no instance where a sulphur dust has damaged apple fruit, but this column is included in the table for growers' own use in case they have such an experience and wish to record it for future reference. The other columns can also be supplemented or modified according to their own local experience.

Spray injury to pears. Pears stand spraying better than apples, but leaf scorch or slight shedding of leaves has been observed to follow a 2:100 lime sulphur spray on Bonne Louisa, Clapps Favourite, Colorée de Juillet, Comice, Giffard, Grey Pear, Count Moltke, Josephine de Malines, Juli Dekan, Nouveau Poiteau, Seigneur and William's. Josephine de Malines will not endure Bordeaux mixture, which may also injure Conference and Netis.

At one centre in 1934 shrivelled fruit were reported on Giffard, Nouveau Poiteau and Tongres after a post-blossom spray with 4:8:100 Bordeaux. The pear variety Colorée de Juillet has also once been reported to be injured by a 10:90 tar-oil spray applied before 15th January. In England it is considered particularly susceptible both to sulphur and copper sprays (Marsh, 1933).

The post-blossom 4:1000 lead arsenate spray formerly used against the larvae of the pear sawfly causes the appearance of numerous small holes in the leaves, as though damaged by hail, and many of these leaves drop. The damage is not apparent until six weeks after spraying. It is especially severe on River's Early Prolific, but other varieties, such as Reine Claude, also suffer from it. The damage occurs both where lead arsenate has been used alone and in combination with Bordeaux mixture or lime sulphur, whether with or without added lime.

Nicotine sprays may damage various kinds of plum, especially Czar. They suffer least when growing vigorously, as applies also to the lead arsenate injury on River's Early Prolific.

Spray injury to bush fruit. The washing soda-soft soap spray, often recommended for control of American gooseberry mildew, may scald both leaves and fruit, especially if it is not carefully prepared. The likelihood of damage is greatly increased if the spray is applied in strong sunlight.

Most kinds of blackcurrant and raspberry cannot stand lime sulphur, nor can yellow or hairy-fruited varieties of gooseberry.

Injury by fungicidal dusts is less general. Some apples are sensitive to sulphur dusts (see table, p. 47), but several varieties, like Cox's Orange Pippin, which can scarcely tolerate a lime sulphur spray and still less a Bordeaux mixture, can easily endure a lime sulphur dust, which gives the fruit a fine shiny skin. Sometimes, however, varieties that generally tolerate sulphur dusting may develop spots or a rough or broken skin if a thick coating of the dust is allowed to remain on wet leaves and fruit. Most blackcurrants and raspberries are unable to tolerate sulphur dusts any more than a lime sulphur spray. The same applies to most gooseberries, especially the yellow-fruited and hairy types.

Damage by fumigants. When various chemicals are evaporated inside hothouses, especially as insecticides, scorching of the plants may occur. The degree of airtightness of the house and the strength of the wind both have an effect on the effectiveness of the insecticidal fumigant and in its liability to scorch plants.

Hydrocyanic acid, which is especially used against aphids, white-fly and thrips may scorch many plants. In Denmark a course of instruction has to be attended before permission is given to use this gas for fumigation. Details of the commoner susceptible plants are usually given with the fumigant when it is sold and mention need only be made here of the transverse stripe that appears across the uppermost part of recurved carnation leaves where this fumigant has been used.

Naphthalene used against red spider and thrips may scorch various plants if used at high concentrations. Especially susceptible

are: phyllocactus, cyclamen, echeveria, gardenia and pelargonium. Roses may remain unaltered for 2–3 days after fumigation and then become quite colourless during the ensuing 8–14 days. Leaf tips of carnations are also liable to be scorched especially during the winter, when their growth is soft.

Nicotine, used as a fumigant especially against aphids, scorches or otherwise slightly damages some plants, especially adiantum, young cucumber plants, asparagus, astilbe, chrysanthemums (whose leaves acquire a yellow edge, Fig. 22), red cyclamen (whose

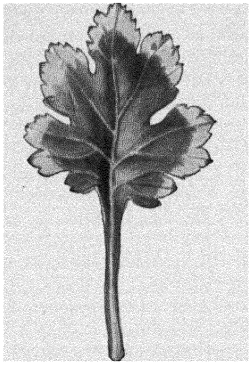


Fig. 22. Chrysanthemum leaf with yellow edge caused by nicotine fumigation.

flowers turn violet), heliotrope, hydrangea, orchid flowers, young rose shoots, *Salvia splendens*, senecio (cineraria), sinningia (gloxinia), streptocarpus, red tulip flowers and violets.

Paradichlorbenzene, used against aphids, wood-lice, grass-hoppers, slugs, etc., is tolerated by most plants. It has, however, been seen to injure *Bryophyllum tubiflorum*, chrysanthemums and a number of other plants. On several occasions, where the paradichlorbenzene has accidentally caught fire, plants have been severely scorched by the fumes.

Sulphur burnt to fumigate hothouses emptied for disinfection may damage plants in adjacent houses. The sulphur dioxide produced in this way is extremely poisonous to plants and the panes of glasshouses are not usually airtight enough to keep in the fumes.

In houses where the paint contains zinc oxide, sulphur-dioxide fumes from burning sulphur may react with this to form zinc sulphate which later may dissolve in moisture condensing on the roof and drip on to leaves, scorching them. To prevent this, walls and roof should be washed down with water after the sulphur has been burnt. Galvanised pipes may react with sulphur dioxide in the same way. Titanium white does not cause scorching.

Tetrachlorethane used against white-fly may scorch the buds of hydrangea and cause them to become quite black.

Injury by Other Chemicals

Artificial manure used carelessly may cause scorching if, for example, it is scattered just before a heavy dew, or falls on damp leaves of potatoes or roses. Apparently no harm is done by scattering it in wet weather. Sulphate of ammonia is particularly liable to cause scorching. The safest way is to apply fertilisers before the plants develop leaves or at least before they are fully expanded. If fruit trees need a top dressing of nitrate in summer it should be applied carefully so that it is not blown over the leaves. For summer applications it is best to use granulated salts and to spread them in dry weather.

Seed potato tubers may also be damaged by artificial manures applied in the drill. In various experiments with harrowing in fertilisers during the summer injury to the growing tubers has been noted. Potatoes may also be damaged after lifting if they are placed in unwashed bags that previously held fertiliser or are heaped against a wall where fertiliser formerly stood. The damaged tissue is first soft and watery and later becomes hard, dry and brown or black. Lesions of this kind afford opportunities for the entrance of tuber-rotting organisms.

Damage by Industrial Fumes and Smoke

In districts where much smelting is done the smoke from the furnaces has caused much damage to crops and involved the firms concerned in heavy expenditure for compensation.

Hence there have been many careful investigations of the problem which afford a basis for a reasonable estimation of the type and extent of this kind of injury. Much time and money has been spent in improving smelting methods to reduce damage of this kind and in purifying the smoke by recovery of useful by-products. Nevertheless in industrial districts it is advisable to cultivate only hardy plants. In places particularly liable to be covered by drifting smoke low-growing plants can be protected by hedges or by planting shelter belts of hardy trees and shrubs.

An expert can often diagnose smoke damage by the appearance of the plant. He may also detect characteristic symptoms under the microscope. The presence of phaeophytin can be detected spectroscopically, though this alone is not an infallible sign of smoke injury, and tar and asphalt residues may be observed under the quartz lamp (Dvorak, 1930). Analysis of air and soil samples may be made but the results are liable to dispute. Analysis of leaves for their sulphite or sulphate content in comparison with that of similar leaves from other districts may also be useful. On the spot the situation of the damaged area should be noted especially in regard to its relative altitude and the amount of shelter, together with any one-sidedness in the distribution of the damage over trees and hedges. This must be correlated with information on prevalent winds. Where more than a trifling amount of damage is involved impartial assessors, familiar both with the symptoms of smoke damage and with the ruling value of the crops, should be called in as soon as possible, so that the damaged area can be defined and specimens taken before acids or other substances present have disappeared. The possibility of any other cause of the trouble must also be thoroughly investigated, for example lack of lime, lack of manganese, errors in cultivation, damage by parasites, frost, wind or hail. Particularly instructive in this connection is the claim made by farmers in Washington State for damage to their lucerne crops alleged to be due to smelter fumes from across the Canadian border. The damage was shown by experiment to be due to boron deficiency in their own soils. Estimate of the extent of loss is easiest when it is due to a single acute attack, due perhaps to a temporary change in the factory. In weak chronic smoke damage the symptoms are less obvious and comparison must be made with similar crops grown under comparable conditions free from smoke. Bacteriological analyses have shown the acidity of the smoke may in time affect the number and activity of the soil bacteria by its effects on the soil reaction (Bredemann and Radeloff, 1932).

The usual cause of smoke damage is the small amount of sulphur dioxide (SO_2) contained in smoke from the burning of coal and the smelting of ores containing sulphides. Most plants are damaged by exposure for one hour to one part of SO_2 in a million parts of air (Zimmerman and Crocker, 1934).

On conifers the predominant symptom is a red discoloration starting at the tips of the needles. Affected needles turn brown, shrivel and fall if the injury is acute. If it is chronic their life may be shortened from the normal 4–5 years to 2 or 3 in the case of spruce, and from 10–12 years to 4–5 in firs.

In the leaves of deciduous trees yellowish-brown or dark brown patches appear between the veins, often forming striking colour patterns, quite distinct from the marginal scorching due to wind or chlorides.

When sulphur has been burned in a greenhouse at the rate of 1 gm. per 100 cubic metres (=6 parts SO_2 in 1 million of air) we have seen tomato plants develop silvery patches in the middle of the leaf in the course of an hour while the leaf edges became flabby and later dried up (Fig. 23). Cauliflower treated the same way developed large flabby dull patches between the main veins.

Plants differ greatly in their susceptibility to SO_2 injury, and the amount of damage may be influenced by many factors such as nutrition, stage of development (young or mature leaves or flowers) and soil and weather conditions. Dry plants suffer rather less than those full of sap. Mist and dew tend to concentrate the fumes but strong sunlight is not always desirable either. Certain very sensitive

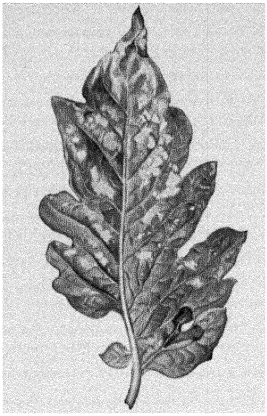


Fig. 23. Sulphur-dioxide injury to tomato leaf.

plants have been recommended for use as indicators of SO_2 injury. Among these are rhubarb, French beans, peas, ox-eye daisy, willow herb and *Lupinus angustifolius*. The smoky atmosphere is responsible for the absence of lichens on trees in or near large towns.

Among economic plants the following are particularly sensitive: stone fruit trees, beech, birch, hornbeam, rose, lilac, vine, redcurrant, tomato and most conifers, though the common yew is resistant and *Abies concolor* and several bluish varieties of fir can endure a lot. Douglas fir, *Pinus contorta* and common juniper are particularly sensitive to smoke. Rather less sensitive are: potatoes, apples, pears, plane trees, elm, gooseberry, raspberry, holly, rhododendron and various ornamental shrubs with tough leathery leaves, notably species of *Olearia*. Relatively insensitive to SO_2 are poplar, oak, maple, alder, privet, various species of willow, the common weeds sheep's sorrel, couch grass, soft grass (*Holcus*), bent grass (*Agrostis*) and above all the common heather (*Calluna vulgaris*). Jerusalem artichoke is also hardy and can be grown quite close to factories.

Smoke from the fires may also injure greenhouse plants if it leaks into the houses.

Chlorine and hydrochloric acid fumes cause discoloration of leaf edges which must be carefully distinguished from that due to potash deficiency or to drying winds. Myrtle and various kinds of cactus are particularly sensitive to these fumes as also, out of doors, are hornbeam, hazel, oak, beech, birch and maple.

Damage by ammonia fumes has been mentioned on p. 43.

Acid fumes from grease smeared over pipes in hothouses to protect them from rust have caused damage in new houses. The pipes can be cleaned by wiping them with a rag dipped in benzol (O. P. Juul Jørgensen, 1925). Other substances painted on hot pipes have caused damage at times.

Fumes from tar may damage tree leaves in spring if the tar engines used in road spraying are brought too near the crowns of trees bordering the road. Also if roofing felt, asphalt paper or any other substance impregnated with tar is burnt, obvious damage may be done by the smoke. Considerable injury, e.g. to begonias, may occur in hothouses when tarred woodwork or walls become heated, even though the tarring may have been done a considerable time before. The damage may be due partly to ammonia, partly to phenols or other constituents of the tar, and the symptoms are correspondingly variable. Besides various scorched yellow or brown patches on the leaves they may acquire a strange lacquer-like polish and often become spoon-shaped (Fig. 24).

The effect of tarring road surfaces on roadside trees has been investigated by a Dutch commission which has particularly stressed the danger that roots under this airtight covering may be suffocated, dried out or poisoned by small gas leaks. An untarred belt 2–3 yards broad along the line of trees should be aimed at where the soil is kept loose and eventually protected by a grating (Gerlings, 1930).

Asphalt fumes are similar to tar fumes and produce similar scorching and rolling of leaves. Ash, roses, paeonies and strawberries are particularly sensitive to them.

Industrial dust. Besides coal dust and soot, which blacken and disfigure the leaves of plants

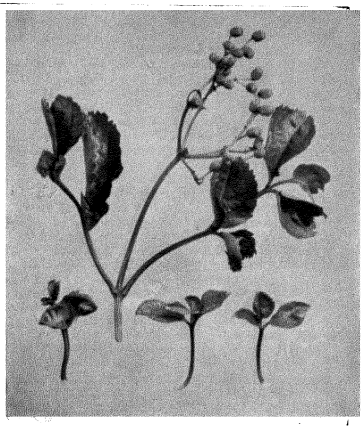


Fig. 24. Elder leaves damaged by tar fumes.

in large towns, cement dust and other dusty particles from factories may be troublesome, partly by coating plants directly, partly by their deposition on the glass of hothouses. The glass may be corroded and the amount of light admitted may be reduced so much as to hinder carbon assimilation by the plants within.

Tobacco smoke is not generally injurious to glasshouse plants. In laboratory experiments, however, it has been shown to check the growth of various seedlings and cause drooping leaves in *Impatiens* and shedding of leaves in *Mimosa* and other legumes. The damage can hardly be attributed to nicotine.

SOIL SICKNESS

It is well known that under continuous cropping with a particular plant an area may become " tired " or " sick " so that the yields progressively decline. This does not happen to the same extent with all crops nor is it always due to the same cause. Pea sickness may be due to root eelworm or to various soil-borne fungi, and is often associated with a shortage of lime. Clover sickness may be due to another eelworm or to *Sclerotinia* infection. Sometimes the cause is malnutrition, the plants having exhausted the available nutrients in the soil. The term may also be used very loosely to cover the results of bad farming practice. Thus, the old German turnip sickness was due to eelworms, but reported cases of turnip sickness in Denmark have been found to be the result of careless thinning; the poor yields were due to too few turnips being left per acre.

The gradual onset of soil sickness in tomato houses is a serious problem not yet fully understood. In some experiments it has been found that digging fresh green grass into the soil, together with good stable manure without much straw in it, increased yields to some extent (Bacher, T., 1942).

Soil sickness is a well-known problem in forestry nurseries. Sometimes a certain kind of tree may make the soil unfit for its own growth for ten years or so; we have even seen injurious after-effects persisting for twenty years. In such instances it is not yet clear whether the harmful substances present come from the roots or from micro-organisms in the soil. As a general principle pome fruits* should never follow pome fruits and preferably should not follow stone fruits. It is also undesirable to follow pome fruit by stone fruit (H. v. Bronsart, 1931). Experiments by Anton Pedersen show, however, that it is not absolutely necessary to plant tired nursery soil with agricultural crops to " rest " it. In his investigations common spruce, elm, myrobolan, hawthorn, apple and white beam were cultivated continuously and in succession. Spruce throve well after all five other species and also after itself. Elm and myrobalan both did poorly by repeated cropping. Hawthorn did best after spruce, worst after itself, and white beam did worst after apple. White beam and apple were not unfavourable precursors of apple in this particular experiment but have been found to be so in nurseries. As is usually the case, white beam proved worthless when grown after itself or after hawthorn but did splendidly after spruce.

Replacement with fresh soil is a common practice with tomato-sick glasshouses, and addition of fresh soil is sometimes helpful in

* Apple, pear, hawthorn, *Sorbus* spp.

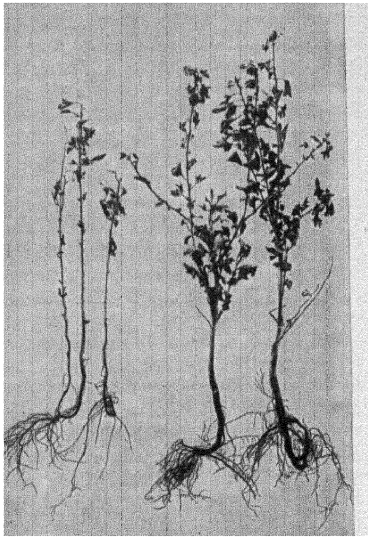


Fig. 25. Soil Sickness. The small hawthorns on the left are from rows on a nursery site which for many years carried young stone-fruit trees. The large plants on the right belong to the same planting but were grown on land that had never been used for stone fruit, only for other kinds of nursery stock (*S. p. F.*).

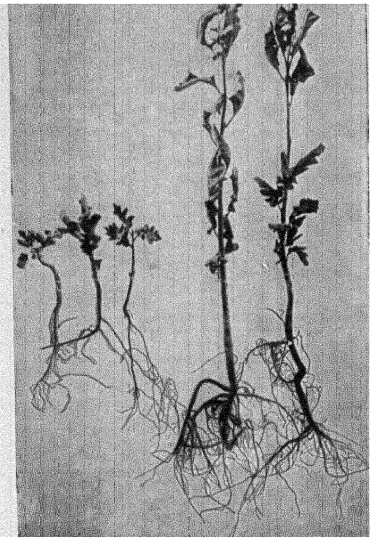


Fig. 26. Soil Sickness. Young oaks grown under the same conditions as the hawthorns in Fig 25 (*S. p. F.*).

soil-sick nurseries. Steam sterilising the soil or treating with formalin or other disinfectants may also be effective. In Swedish experiments the sickness in abandoned nurseries was cured by putting formalin or tar oil into the trenches in spring. When planting took place six to seven weeks later the earth still smelt a little but the roots did not suffer (Johansson, E., 1943). The high cost of the treatment limits its usefulness. The cause of the beneficial effect was not investigated. It might be due to disinfection (destruction of injurious bacteria, fungi, eelworms, or soil protozoa) or to liberation of minor nutrients. It can hardly have been a question of improved nitrogen, potash or phosphoric acid supply, for the analysis in the above experiment showed no significant changes in these three nutrients or in soil reaction. The possibility that manganese supply may have been involved is under investigation; it is known that soil sterilisation sometimes corrects a deficiency of manganese.

In the case of certain plants (e.g. walnut, *Juglans nigra, J. cinerea*) the roots have been shown to excrete substances poisonous to other plants (Schneiderhan, 1927). The roots of many forest trees are normally closely associated with certain fungi in the specialized structures called mycorrhizas and it has been suggested that they leave behind them an inheritance of fungi unfavourable to their successors.

The fact that soils " closed " by water or by hard pan formation become injurious is due to " soil wax " (stearin-like compounds) enveloping the tiny soil particles and cutting the plants off from the food contained in them. In such cases treatment with carbon disulphide should dissolve and remove the " soil wax " (Greigh-Smith, 1911). Poor growth on soils of this type may also, however, be due to simple suffocation of the roots (oxygen deficiency).

LIVING CAUSES OF DISEASE

The living organisms responsible for disease in plants (pathogens) are mostly either fungi or bacteria. As a rule they are parasites, living either on or inside the plants and drawing nourishment from them. Occasionally some damage may be done by non-parasitic organisms like the sooty moulds which grow on leaves. These do not withdraw anything from the plant but are injurious because they form a dark blanket shutting out light from the leaf.

Infectious diseases must needs be due to parasites, with the proviso that it is a moot point whether the viruses causing mosaic diseases are living or not, though they are often highly contagious.

Both in nature and under cultivation most diseases are not active during the resting period of the host, i.e. during winter in temperate climates. Many fungi survive this period in the form of long-lived resting spores or sterile resting bodies (sclerotia). Others, especially parasites of annual crops, pass the winter as mycelium in the seed of the host or as spores on its surface; others again persist as mycelium in the buds of the host (many powdery mildews), or in perennial organs such as root stocks or tubers, while some pass the winter as mycelium living freely in the soil. All parasites adapted to a temperate climate can easily survive a single winter or a single drought. The important question in practice often is " How many years must one leave an infected plot free from susceptible crops in order to starve out the parasite?" This depends partly on what kind of resting body the latter possesses, but also on its biological specialisation.

BIOLOGICAL CLASSIFICATION OF PARASITES

Obligate saprophytes or non-parasites are fungi or bacteria that live by decomposing dead organic matter and cannot in any circumstances attack the tissues of living plants. They may occasionally be injurious or unsightly as with sooty moulds, but their activities are generally entirely beneficial to green plants.



Fig. 27. Mycelium of *Fusarium* in conducting vessel of potato stem (× 200) (after Foex and Lansade).

Facultative parasites normally live as saprophytes but when conditions are favourable to them may attack living plants. Thus the common coral spot fungus (*Nectria cinnabarina*) growing in a dead branch may pass from it into an adjacent living one. The common grey mould (*Botrytis cinerea*) thrives on dead leaves and may spread from them to living ones. Probably in some such way the first parasites evolved.

Facultative saprophytes normally live as parasites but are able to survive saprophytically for a time in soil, on dead plant debris, etc. As a rule, however, they are not able to compete with the true saprophytic flora of soil and soon become starved out in the absence of a host. Sometimes there is a saprophytic phase in their life history. Thus the apple scab fungus (*Venturia inaequalis*) regularly spends the winter months in dead fallen apple leaves, maturing perithecia so that the ascospores are ripe and ready to infect the fresh leaves next spring. Not only are facultative saprophytes soon starved out but they may be directly attacked by other fungi and destroyed in most soils. Some, however, can produce resting bodies that survive for years.

Obligate parasites develop only in their host plants and survive the winter either as resting spores or as mycelium if their host is a perennial one. Usually, unlike the fungi of the other three groups, they can be grown in artificial media only with difficulty or not at all.

Many of these obligate parasites, especially the rusts and powdery mildews, live in or on their hosts for months or years without killing the cells. They may even stimulate the host tissues to excessive growth, as in witches'

brooms. Most of the parasites kill the surrounding tissues and must then invade new ones or spread to new host plants. Many fungi and bacteria excrete soluble substances that kill the host cells around them and themselves really live a saprophytic existence in the dead tissue. Parasites of this kind have been called internal or endo-parasites; the powdery mildews grow over the surface, only thrusting minute suckers at intervals into the outer cells of the host, and are called ecto-parasites.

Sometimes one parasite prepares the way for another with such regularity that we can speak of successional diseases, e.g. apple scab—canker, or apple scab—frost damage—canker.

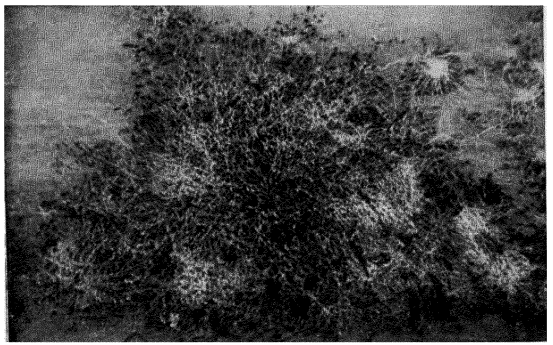


Fig. 28. Patch of powdery mildew on stem of kale (× 7).

FUNGI

To start a mushroom bed one usually buys some "spawn." This consists of the vegetative body or mycelium of the mushroom fungus and, if all goes well, the fine threads or hyphae that make up the mycelium grow into and permeate the manure of the bed. Ultimately from these threads the fruit bodies, or mushrooms, are produced. The fruit bodies or fructifications of fungi vary enormously in size, shape and structure, but the vegetative body consists of hyphae in all except the very minutest, simplest species. A hypha is a cylindrical tube usually divided into segments by partitions at regular intervals; each segment contains protoplasm and one or more nuclei but never contains chlorophyll. Because they lack this green colouring matter fungi cannot utilise the carbon dioxide of the air as green plants do but must derive their organic matter at second hand from green plants or from animals.

Occasionally individual hyphae can be distinguished under a hand lens, as in mildews. Sometimes they are woven together to form rope-like strands visible to the naked eye (rhizomorphs) or are matted together into hard seed-like resting-bodies (sclerotia), and ultimately they form more or less complex fructifications containing the reproductive bodies or spores. Actively growing mycelium requires a considerable amount of water, but sclerotia and many kinds of fructifications are very resistant to drought and can sometimes survive in a dry state for years. In some species exposure to frost or drought is even necessary to stimulate them to renewed growth or to bring about germination of the spores.

A mycelium usually feeds by secreting chemical substances (enzymes) that attack the cell walls and contents of living or dead plant tissues. These enzymes are easily demonstrated in some saprophytes or facultative parasites like the common grey mould *Botrytis cinerea*. Other more delicately adjusted obligate parasites, like the powdery mildews and

rusts, have special sucking hyphae (haustoria) which are inserted into the living cells of the host and withdraw nourishment without killing them.

Mycelium grows at the tips and by branching. When a spore germinates on a favourable medium the mycelium grows out in all directions to form a circular mat with the youngest hyphae at the margin. This is how fairy rings arise; the mycelium in the soil is perennial, and grows a little farther out each year and dies away inside the ring. Monilia pustules on apples form in similar rings and so do the minute fructifications on many leaf-spots. Theoretically a fungus mycelium could go on growing eternally but in nature it is probably always checked sooner or later by competition with other organisms or because it has exhausted the available food. Some fairy rings, however, are certainly very old. Usually wherever food runs short some kind of reproductive body or resting body is formed.

Spores are of two kinds, the asexual or vegetative, produced on what are called by mycologists the " imperfect " fructifications, and sexual spores produced on the " perfect " fructifications. For many fungi, however, only asexual or " imperfect " fructifications are yet known.

Asexual spores, usually known as conidia, correspond roughly to offsets, runners, bulbils and the like in flowering plants, but in most fungi they are the most abundant reproductive bodies and the most efficient agents of dispersal.

A patch of powdery mildew on a leaf starts as a fine cobweb-like growth radiating from a centre. When it is only a few days old the middle of the patch becomes mealy. This is due to the hyphae there sending short vertical branches into the air. The cells of these vertical hyphae are short, and separate from one another like a string of pearls to form the conidia which are then easily blown away by the wind or detached and carried off by man or animals. Any conidium which falls on a suitable leaf can, under favourable conditions for germination, give rise to a new patch of mildew. Somewhat similar chains of conidia, borne on more complicated hyphae, give *Penicillium* its green colour. The conidia of the common moulds are produced in millions and are always floating about in the air and settling on every exposed surface. Almost all of them must be wasted by falling on substances on which they cannot germinate or grow. Usually when a conidium does land on the right substance and receives the right

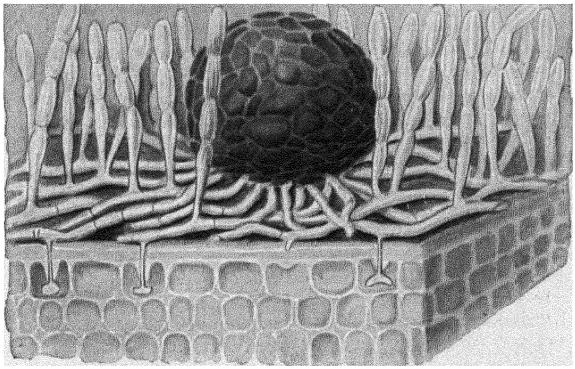


Fig. 29. Diagrammatic sketch of a powdery mildew. In the centre is a perithecium seated on the superficial mycelium and surrounded by chains of conidia. In front note the haustoria penetrating the host cells beneath (× 333).

amount of moisture it germinates to form a vegetative hypha straight away. In potato blight (*Phytophthora infestans*) and its allies, however, a conidium that falls into a drop of dew does not produce a hypha but functions as a sporangium instead. Its contents divide up to form several zoospores able to swim about in the drop of dew. If they are on a potato leaf each of these zoospores can settle down and grow out into a hypha to start a fresh spot of blight. Some fungi produce asexual spores with thick walls and oily contents (chlamydospores) able to survive for a long time. These are not usually as well fitted for dispersal as the thin-walled conidia.

The conidia of mildew, *Penicillium* etc., described above, are dry and dusty and blown about by wind. Others, as of *Verticillium* among the moulds and the conidia produced in pustules or tiny flask-shaped bodies (pycnidia), as in *Phoma*, are sticky, held together in globules and are dispersed by rain splashes or by sticking to the bodies of insects.

Perfect fructifications. In fungi like *Pythium*, the common cause of damping off, and its allies, one can detect, under the microscope, a fertilisation of a female cell by a male, with formation of an egg or oospore. This has a thick wall and oily contents and is resistant to drought. In other lower fungi, like the "pin moulds" *Mucor* and *Rhizopus*, there is no visible difference between the male and female cells but there are often two kinds of mycelium, called + and —. Sexual spores are then only formed when hyphae from a + and a — mycelium meet. In the majority of fungi, the ascomycetes and basidiomycetes, sexual organs are very rudimentary, but the formation of eight spores inside an ascus in the former and of four spores on the outside of a cell called a basidium in the latter is regarded as resulting from the equivalent of a sexual process. In ascomycetes there are two main types of fruit body, the flask-shaped form (perithecium), always minute, and the cup-shaped form (apothecium), which in a few species may be two or three inches across. Most basidiomycetes have their basidia either on the surface of gills as in mushrooms and toadstools, or lining the inside of pores, as in the bracket fungi. Some of the latter produce almost incredible numbers of spores. For example, the tinder fungus *Fomes fomentarius* may shed 21,000,000,000 spores in twenty-four hours. A common mushroom may shed 1,800,000,000 at the rate of 40,000,000 per hour and a giant puffball (*Calvatia gigantea*) has been calculated to contain 7,000,000,000,000 spores (Buller, 1909). The spores of toadstools and bracket fungi are shot off the basidia into the air and then drift passively until they come to rest. Those of ascomycetes are also shot violently into the air.

Fungi imperfecti. Important and interesting as the perfect stages of fungi are, the fungus classification is based almost entirely on them, it is the imperfect conidial stages that are most important in most plant parasitic fungi. Often only an imperfect stage is known and then the fungus has to receive a name based on the structure of this conidial stage. When, however, the perfect stage of the fungus is found it is possible to remove it from its temporary position among the fungi imperfecti, and allot it a place in the permanent classification of fungi. This necessarily involves a change of name. Thus, the common "imperfect" stage of apple scab on the living leaves was first named *Fusicladium dendriticum*. Later it was discovered that the "perfect" state of the same fungus occurred on the fallen leaves during winter and early spring. This had been described under the name *Venturia inaequalis*, and this name applicable to the perfect stage has to supersede *Fusicladium dendriticum* because they are the same fungus and only one name is needed for it. Sometimes names have to be changed because a fungus has been described independently by more than one mycologist. The rule then is that the earliest name applied to a perfect stage must be used but often a considerable time elapses before it is realized which is the earliest name. In critical mycological work it is usual to add the name of the author who first described the species. Usually a contracted form is used to save space. When increased knowledge has made it desirable to alter the

systematic position of the species the name of the author who first described it is quoted in the brackets and that of the person who changed its systematic position follows.

Close study of some parasitic fungi has shown that a species may include many different races. Sometimes, as in *Botrytis cinerea*, these can be distinguished by slight differences in growth rate, appearance of the culture and kind of sclerotia when grown on artificial media. Sometimes they differ in their response to various environmental factors such as temperature or reaction of the medium or in their capacity to produce particular enzymes and utilize certain substances as food. In the obligate parasites, rusts, smuts and mildews, physiological races can be distinguished. These look exactly alike as a rule but they can be distinguished because they are capable of parasitising different cultivated varieties of their host plants. In the cereal rusts, where a rudimentary sexual process has been discovered, it has been possible to produce new physiological races by hybridizing the old ones. The existence of these specialized races and the ability of the fungus to produce new ones, by crossing and no doubt by mutation, makes it very difficult to breed cereal varieties immune to rust. A variety immune to the physiological races common in a district may be susceptible to another race present perhaps in only minute quantities. In a few years, however, if the new variety is extensively grown the race capable of attacking it will automatically be " selected " and become the dominant race in the district. Even when no race capable of attacking the variety already exists there is always a chance of one arising by hybridisation or mutation from the old ones.

In the case of parasites it is often possible to differentiate between aggressiveness, the readiness with which the fungus attacks its host, and virulence, its capacity for inflicting damage when infection has taken place.

Most important parasites attack only one host plant or only a few related species. There are, however, a few which have a very wide host range. Among these are:

Grey Mould (*Botrytis cinerea* Pers. ex Fr.)

Botrytis cinerea can probably attack any herbaceous plant and may occasionally infect woody tissues as well. Among the economic plants known to be affected by it are:

Ornamentals	*Crop plants*
Begonia	Apple (fruit)
Cacti	Artichoke
Calceolaria	Broad bean
Calendula	Cabbage
Chrysanthemum	Capsicum
Cyclamen	Carrot
Dahlia	Cucumber
Gladiolus	French bean
Gloxinia	Gooseberry
Helianthus	Jerusalem artichoke
Heliotrope	Lettuce
Hyacinth	Marrow
Iris	Onion
Lily	Potato
Lily of the Valley	Raspberry
Lupin	Redcurrant
Narcissus	Rhubarb
Paeony	Strawberry (fruit)
Pelargonium	Sunflower
Primula	Tomato
Rose	Vine
Salpiglossis	
Snowdrop	
Solanum	
Stock	
Tulip	
Wallflower	
Zinnia	

In addition several of these plants, notably gladiolus, narcissus, paeony, snowdrop, tulip and onion, are attacked by related but distinct species of *Botrytis* peculiar to themselves and much more actively parasitic than the common omnivorous grey mould.

This very common fungus is easily recognized as a loose grey coating of mould from which a fine white cloud of spores rises when the mouldy leaf or flower is picked. Under a hand lens the long stiff dark conidiophores can be clearly seen, bearing clusters of white

conidia near their tips. Many different strains of the fungus have been distinguished in artificial culture but there seems to be little host specialization. Wormald (1941) showed that strains of grey mould from dandelion flowers, bramble fruit, black bryony leaves, sweet chestnut cupules, dwarf beans, African marigolds and garden scabious were all able to cause a rot of apple fruit.

The sclerotia or resting bodies, once regarded as a separate species, *Sclerotium durum*, form rather flat, hard cushions, first white, then grey and ultimately jet black on the outside, on the surface of dead plant tissue. When formed inside hollow stalks, as of the potato, they may be round or nodular. From them, under moist conditions, grow tufts of the familiar grey mould. Circumstantial evidence has long suggested that the perfect stage of this common fungus is the cup fungus, *Sclerotinia fuckeliana*, and this has recently been experimentally confirmed (Groves and Drayton, 1939). The perfect stage, however, is very rare and plays no part in the normal life history of the species.

Botrytis cinerea is a typical facultative parasite, living in dying leaves and dead flowers or bud scales from which it penetrates living parts of the plant. This is, for example, the way it causes a destructive disease of greenhouse lettuce, becoming established first on fading outer leaves and then entering the stem and killing the whole plant.

Moisture is essential to it and it is favoured by excess nitrogen and lack of light, both of which reduce plants' resistance to infection. Its minimum temperature for growth is about 36–41° F. and its maximum between 86° and 104° F., while it flourishes best at about 71–77° F. (Link, 1924). If the air or the plants become too dry it forms sclerotia to tide it over till better times.

Grey mould is a great enemy of the fruiterers and greengrocers. Berries and other fruit, artichokes, beans, lettuce and tomatoes become dusted with spores from dead leaves or shoots when they are picked but the mould often does not become visible until after they have been graded and packed. It may then spread rapidly throughout a package especially if there has been any bruising or wounding and the surface of fruit is covered with leakages of juice.

Control measures include hygienic precautions, keeping dead leaves, flowers and stalks cleared away from both plants and soil. Vegetables like beans, tomatoes and cucumbers should if possible be packed when dry, kept cool and well ventilated and should not be tightly packed. All suspect material should be removed before produce is placed in cold storage and chilled vegetables should be kept at about 40–42° F.

In glasshouses ventilation is all-important, especially the avoidance of condensation and dripping from the glass by careful watering and ventilation. Firing to avoid condensation at night and careful manuring, keeping the nitrogen supply carefully controlled, will also help. Thin sowing in seed boxes will prevent the damping-off phase of the disease and produce sturdy plants less likely to succumb to it later on. Protection against frost damage, against biting and sucking insects and any form of injury which results in dead leaves or spots or shrivelled leaf edges will reduce the chances of infection.

Sterilization of the soil will destroy sources of infection left over from a previous crop, but one must remember that it will soon be dusted all over with a fresh crop of the spores of this ubiquitous mould. Heavy " cold " soil encourages *Botrytis* and should be lightened by mixing in sand, compost, charcoal or lime, taking care not to over-lime.

Bordeaux mixture, flowers of sulphur and other copper and sulphur fungicides are not very much use against *Botrytis*. Shirlan, which contains salicylanilide, is often effective and a number of other proprietary dusts have been prepared for its control.

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

Symptoms. On herbaceous plants the first symptom of *Verticillium* wilt is usually a flagging of the lower leaves. Next the leaves turn yellow from the tip and margin inwards.



Fig. 30. Grey mould (*Botrytis cinerea*) on raspberry cane. Note the clusters of conidiophores, each tipped by a head of conidia (× 5).

Usually the affected plant soon dies and all the leaves hang limp and shrivelled. If conditions are not favourable to the parasite the wilting lower leaves may apparently recover during the night, when transpiration is less active, and sometimes the plant may recover especially if it can be induced to form fresh roots from the base of the stem. In greenhouse tomatoes similar flagging of the lower leaves may be due to various root diseases or to severe infestation by the root eelworm, and it is advisable to confirm the diagnosis of *Verticillium* wilt by looking for the brown discoloration of the wood mentioned below.

The fungus can also attack and kill woody plants, usually entering through the roots. Sometimes, as in myrobolan, it may enter the wood through a branch scar and its spread may be checked if the branches first attacked are cut away at once.

When the plants have been killed the fungus grows out to the surface of stems or roots and forms fine white mould composed of innumerable slender short conidiophores bearing whorls of branches each tipped by a sticky globule of spores.

The wood of affected stems is always stained brown, and in the later stages of the disease this brown discoloration can be traced through the wood almost to the tip of the plant. A very similar wilt disease can be caused by some species of *Fusarium*, but this appears to be much less common. It is difficult to distinguish microscopically from *Verticillium* wilt but can be controlled in much the same way,

except that raising the temperature does not help plants to resist *Fusarium* attack.

Verticillium albo-atrum and its near relative *V. dahliae*, believed by some authorities to be only a sclerotium-forming strain of the same species, may attack almost any plant. Hence one cannot starve out the fungus once a soil has become infected by it. Plants belonging to over 70 different families have been found to be susceptible, including species of: *Acer* (maple and sycamore), *Aesculus* (horse chestnut), *Ampelopsis* (Virginia creeper), *Amygdalus* (almond), *Antirrhinum*, *Apium* (celery), *Aster*, *Begonia*, *Beta* (beet), *Callistephus* (China aster), *Capsicum*, *Chrysanthemum*, *Cucumis* (cucumber, melon), *Dahlia*, *Dianthus*, *Fagus* (beech), *Fragaria* (strawberry), *Hydrangea*, *Juglans* (walnut), *Lathyrus* (sweet pea), *Lupinus* (lupin), *Lycopersicum* (tomato), *Matthiola* (stock), *Nicotiana* (tobacco), *Paeonia*, *Parthenocissus*, *Phlox*, *Prunus*, *Rheum* (rhubarb), *Ribes* (currant, gooseberry), *Rhododendron*, *Rosa*, *Rubus* (raspberry), *Sambucus* (elder), *Senecio* (cineraria), *Ulmus* (elm), and *Vitis* (vine).

Verticillium usually enters a plant through its roots, penetrates to the xylem tissue, or wood, and thence spreads upwards through the wood vessels. It is able to attack quite healthy undamaged roots. The wilting is due to the production of poisons by the fungus which are carried through the plant in the sap stream. A temperature between 60 and 75° F. favours development of *Verticillium* and its optimum temperature is from 70–73° F. Plants that have begun to wilt may recover if the temperature is raised to 77° F. because the fungus ceases to produce its poisons at high temperatures.

It is for this reason that the disease usually appears in tomato houses in spring and autumn but seldom during the heat of summer. Similarly, out of doors, it is a disease of temperate countries, not of the tropics. According to a Cheshunt report of 1935 it appears most often on tomatoes planted out when the soil at a depth of six inches has a temperature of less than 57° F. Kadow (1934) has shown that seed-borne infection may occur in tomatoes and egg-plants.

This disease was formerly very destructive in English tomato houses. As soil sterilization has become a routine practice and other methods of checking the loss have been worked out it is now far less prevalent. It appears to be more common in greenhouses in Denmark than in England, perhaps because the summers are cooler there.

Control. Where a serious outbreak of *Verticillium* wilt has occurred under glass, as for example in a tomato or cucumber house, the soil should be sterilized, preferably by steam. Change of soil alone affords no guarantee that the next crop will be healthy, for outdoor field or garden soil may well contain the fungus. Soil that has grown potatoes should especially be avoided for resoiling tomato or cucumber houses. Similarly old cucumber soil should not be used for tomato houses. Where infected soil cannot be sterilized it may be used for growing beans, cabbage or cress.

If only scattered diseased plants occur they should be removed, care being taken to dig the roots well out. If more are attacked the temperature should if possible be raised to at least 77° F. for fourteen days. The house should be shaded and the air kept moist by light spraying of both soil and plants, but heavy watering with cold water should be avoided and the soil kept rather on the dry side. When outdoor crops are attacked little or nothing can be done to save them. If seed is being saved care should be taken that only fruit from perfectly healthy plants is used.

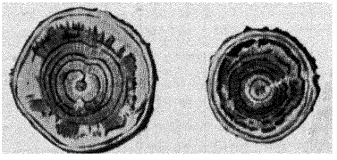


Fig. 31. *Verticillium* wilt. Cross-section of an infected cherry trunk, showing the brown stain in the wood (after Van der Meer, 1925).

See further under the individual crops: cherry, p. 156; raspberry, p. 212; egg-plant, p. 284; potato, p. 328; strawberry, p. 360; tomato, p. 376; chrysanthemum, p. 434; dahlia, p. 446; lilac, p. 534; elm, p. 558.

Powdery Mildews (*Erysiphaceae*)

History. Powdery mildews have long been known as parasites of plants in all parts of the world and their oldest genus *Erisiphe* was established by Linnaeus in 1767. Many species were described by De Candolle between 1802 and 1815, but the first critical study of these fungi was made by Léveillé, who recognized in 1851 six genera, *Sphaerotheca*, *Podosphaera*, *Erysiphe*, *Phyllactinia*, *Microsphaera* and *Uncinula*. These were based on the number of spores and asci and on the structure of the thread-like or bristle-like appendages which cover the outside of the fructification. Léveillé did not realize the parasitic nature of powdery mildews and called them " false parasites." De Bary in 1863–70 was the first to demonstrate the true relationship between mildew and host. In 1900 there appeared an important monograph of the powdery mildews of the world by E. S. Salmon, later Professor at Wye College, Kent. Salmon's nomenclature is usually followed in English mycological literature but he took rather a wider view of the limits of species than commends itself to many European mycologists. In 1933 Blumer published a monograph of the European powdery mildews based largely on his studies in Switzerland. The account of the powdery mildews in this book is based on Blumer's work supplemented by the authors' own observations.

Symptoms. Powdery mildews are found on a great number of dicotyledonous plants and on grasses but on no other monocotyledons nor on gymnosperms or cryptogams. Blumer's monograph contains a list of 1,100 flowering plants known to be attacked by one or more of the powdery mildews in central Europe. All fungi of this family are obligate parasites and form a white powdery or mealy coating over the affected parts of the host plant. This may be in small or large patches or in a continuous layer over entire leaves or shoots. In some mildews the covering is thin, in others thick and close. The appearance also varies with age, usually becoming thicker and more felted in the later stages of the disease.

In some species the white mycelium of the early stages of an attack is soon replaced by dark mycelium so that, as in American gooseberry mildew, the affected parts become covered with a dark-brown or chocolate-coloured felt. Most powdery mildews sooner or later produce perithecia on the superficial mycelium. When young these are usually yellow, but when ripe they appear as minute black specks scattered over the surface.

Mildew attacks occur primarily on the leaves, especially on the upper surface, sometimes on the lower also. Apple mildew may attack the flowers and occasionally appears on the green

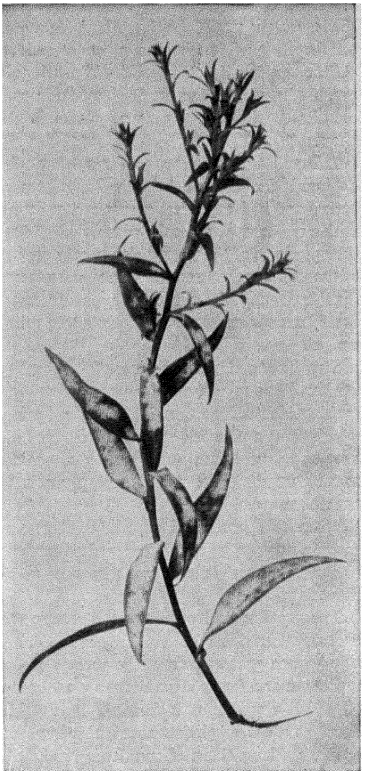


Fig. 32. Powdery mildew on Michaelmas daisy.

fruit. American gooseberry mildew occurs on the leaves, the tips of the shoots and commonly on the fruit. Sometimes a patch of mildew on the upper side of a leaf corresponds to a patch of rust infection on the lower side or is associated with insect galls or cracks in the tissues.

Affected parts of the host are often discoloured; for example, begonia leaves become brown and hydrangea leaves turn red or brown with a red margin under the patches of mildew. The presence of the fungus also checks growth of the part attacked and may thus lead to some malformation. The mildewed apple leaves are small, narrow and somewhat curled or rolled up.

Structure and development of a powdery mildew. The mycelium of a powdery mildew consists of rather stout, much-branched hyphae which form a network over the surface of the host and bear innumerable chains of conidia making up the white mealy coating visible to the naked eye.

From this superficial mycelium short absorbing hyphae, haustoria, are pushed into the underlying host cells to withdraw nourishment from them.

The rapid spread of mildews is due to the enormous numbers of conidia produced. A side branch grows up from the surface of the mycelium and forms a conidiophore at the tip of which a conidium is formed. Below this develops a second, below that a third and so on until a long chain of conidia is produced. The conidia are not exactly alike in all the species but usually they are delicate cylindrical or barrel-shaped bodies, individually colourless and appearing white when massed together. In investigations of some species Hammarlund has found that the conidiophores usually function for six days and each produces in that time from 10 to 33 conidia. In damp air the chains hold together longer than in dry air. The fungus spreads more rapidly in dry air and conidia produced then grow most quickly and infect the host most easily. In grass mildew, however, the conidia seem to be very little affected by the humidity and seem to be more effective when produced in moist air.

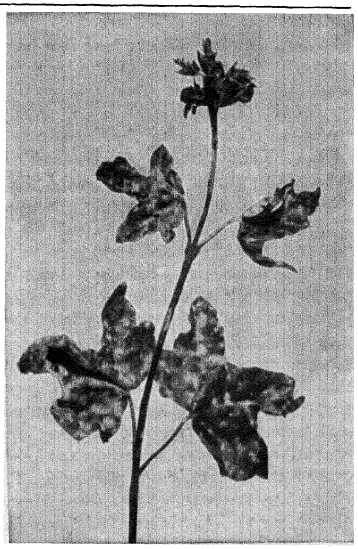


Fig. 33. Powdery mildew on maple leaves.

Temperature also has a great influence on conidial production; the optimum is about 77° F. Heavy rain destroys the conidiophores of powdery mildews and hampers dispersal of their conidia (Yarwood, 1936a).

Light favours the production of conidia and in darkness fewer and weaker conidia are formed. In *Erysiphe polygoni* on red clover Yarwood (1936b) found that each conidiophore typically produced a single mature conidium per day, which was liberated about noon. In cloudy weather this periodicity in conidial production was less obvious. Spores taken from mildewed plants in late morning and afternoon had a high germinative capacity, whereas those taken late at night or in the early morning germinated poorly. Inoculations made during the day gave a higher percentage of infection than those made at night. The conidia are shot off the conidiophores so violently as to be thrown twenty times their own length into the air. This is unimpressive when compared with the violent expulsion of ascospores, which may in some fungi be shot 2,000 times their own

length, but it is sufficient to ensure the conidia getting into the air stream instead of falling passively on the mycelium which bore them.

The perithecia are formed as a result of sexual reproduction. The time necessary for their development varies, but in Blumer's experiments some species had produced ripe perithecia only 10–12 days after infection. Some species of mildew commonly produce them in large numbers, in others they are rare. Thus, in vine mildew they are so seldom seen that the fungus had been known in Europe for half a century, during which period it had often been very destructive, before the perithecia were described about 1892. Perithecial formation seems to be influenced by several factors. The same species of mildew may produce many perithecia on one kind of host and only a very few on another. There are a number of hosts on which perithecia have never yet been seen so that it is not always certain which powdery mildew does occur on them. These conidial states are temporarily classified in the "imperfect" genus *Oidium*. It may also happen that the same fungus on the same host plant produces many perithecia in one region but few in another. Thus the very common oak mildew had not been seen to produce perithecia in England until 1945, but in the hot dry summer of 1947 they were fairly plentiful in south-eastern England. On the whole warmth favours perithecial formation and high atmospheric humidity checks it, though not if the soil is dry. A severe attack of the fungus is conducive to their production.

The number of asci formed inside each perithecium is surprisingly small as compared with, for example, the apple scab fungus which often has up to 100 in each perithecium. In the mildew genera *Sphaerotheca* and *Podosphaera* there is only one ascus per perithecium, in the other genera the number varies from 2 to over 30. The wall of the perithecium bears a number of appendages of very characteristic shape which have been used for purposes of classification ever since Léveillé's day. The primary appendages are at the base of the fructification and serve to anchor it. The secondary ones are stiff and found mostly in the lower half of the spherical perithecial wall, spreading out to form a ring round it. In several genera the tips of these appendages are branched more or less regularly in a manner characteristic of the genus (Fig. 34).

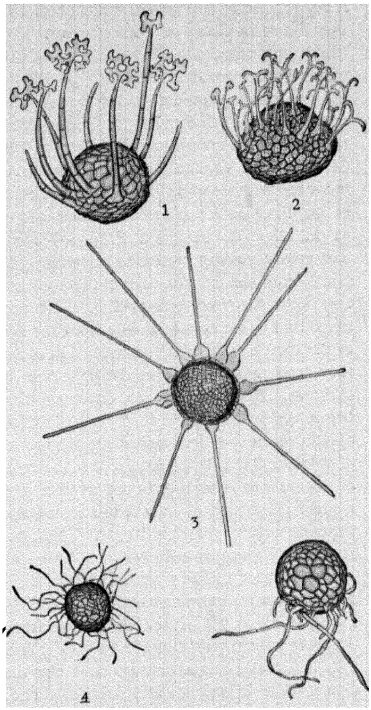


Fig. 34. Perithecia of 5 genera of powdery mildews, showing their characteristic appendages: 1. *Podosphaera*, 2. *Uncinula*, 3. *Phyllactinia*, 4. *Erysiphe*, 5. *Sphaerotheca* (greatly enlarged, after Blumer).

Germination of conidia and ascospores depends on a number of factors. Usually they germinate readily if the necessary moisture is present. If they are exposed to direct sunshine for 3–4 hours their viability is lost but they can survive for 10 days if stored dry (Hammarlund, 1925). Yarwood found

that though conidia of *Erysiphe polygoni* were soon killed by dry air at high temperatures they were able to germinate on glass in completely dry air if the temperature was favourable. With *Erysiphe graminis* on oats Grainger (1947) found the conidia germinated better on a dry surface in a saturated atmosphere than they did when placed in drops of water. They could not germinate, however, at relative atmospheric humidities below 90%. Blumer has seen conidia germinate and send out hyphae up to ten times their own length. The optimum temperature for germination is said to be from 64–77° F. and Voglino (1905) gives the maximum temperature as 95° F. and the minimum as 39° F. Conidia of some species die at 32° F., others can survive 2° F. for 6–8 hours. Temporary cooling seems to stimulate germination of the conidia. Neger (1902) found that light favoured germination. The germinating hyphae sprang mainly from the side of the conidium facing the source of light and they grew more quickly in the light than in darkness. At a certain age, however, they began to grow away from the light. The effect of light seems to differ according to the species of mildew. Germination requires a high degree of atmospheric humidity; in dry weather only a small proportion of infections succeed and their incubation period is longer. The latter is generally given as 2–8 days.

Overwintering takes place in various ways. Free ascospores germinate best at 50–68° F. but soon die at 95° F.; within the perithecia they may remain viable for two years (Voglino, 1905). Actually it is not always clear how the first mildew infection starts in spring and, moreover, in many species the attack is delayed until summer. During the last few years, however, overwintering by means of mycelium has been demonstrated in so many instances that one is apt to conclude this is the normal method. In many species perithecia are so rare that they can be of little practical significance from this standpoint. In most species the overwintering mycelium occurs between the scales of some of the host's buds and from it the newly developing leaves become infected. This hibernating mycelium is hard to detect but Appel has established that the vine mildew overwinters as mycelium in the branches and in buds formed during the summer. Rose, peach, apple, oak and hawthorn mildew have all been shown to overwinter in buds (Dillon Weston and Taylor, 1944). Blumer's investigations suggest that forget-me-not mildew may be transmitted with the seed. This is not surprising for the young fruits are often covered in mildew.

Host range. The mildew of one plant does not usually attack others, or only a few related species. Although several common mildews, especially *Erysiphe cichoracearum* and *Erysiphe polygoni*, are recorded on an enormous number of herbaceous plants it is probable that they exist in numerous physiological races, which look alike but are narrowly adapted to grow on one or a few hosts only. This host specialization has been demonstrated more fully in the grass mildew, *Erysiphe graminis*. On the other hand some races of mildew have a favourite host plant on which they particularly flourish and a number of secondary hosts more or less resistant to attack on which they are able to persist in a more or less impoverished condition. Within the same host species there may also exist strains or races which differ greatly in susceptibility to a particular mildew.

Susceptibility to mildew. Numerous experiments have shown that susceptibility or resistance of plants to mildews is controlled by inherited characters. It can also, however, be modified by environmental factors and by the age of the plants. Many species of mildew only attack fully developed parts of the plant. Others, including several important to gardeners, e.g. American gooseberry mildew, hawthorn and rose mildew, attack mainly comparatively young growth.

Nutritional factors may also affect susceptibility. Excessive unbalanced nitrogenous manuring is generally believed to increase it, especially when associated with a deficiency of potassium. Phosphoric acid tends to reduce susceptibility, but plants suffering from a combined deficiency of nitrogen and phosphoric acid are less susceptible to mildew.

Some plants may be more susceptible on an alkaline than on an acid soil.

In 1936 Lowig published the results of pot experiments with various grasses in which silicic acid combined with potassium and as an ingredient of Thomas meal was shown to increase their resistance to mildew. Lithium salts have a similar effect (see p. 38).

According to Rivera (1915) mildew attacks become more severe when turgidity is reduced, as by drying the soil or by suddenly raising the temperature. On the other hand, in an experiment of Blumer's with *Echium vulgare* infection was most successful when the leaves were well supplied with water and were turgid. Wounded plants are more susceptible than uninjured ones.

Often an apparent decrease in the plant's resistance may be traceable to improved conditions for the growth of the fungus, making it able to attack more easily. For example, plants not attacked by mildew in the open may be covered by it in greenhouses. It is generally agreed that still, warm air favours the powdery mildews. On the other hand, in hothouses plants near the doors may be the first to be attacked. There may be several reasons for this. Plants in that position are particularly exposed to infection. Also they are more exposed to draughts and hence to excessive transpiration. They may therefore lose their turgidity and their susceptibility may thus be increased.

Plants sometimes attempt to oppose the intrusion of haustoria into their cells by secreting a rubber-like mass around them, cutting them off from the cell contents. Attacked leaves are often shed early.

Spread of the mildew fungus. Even though conidia may only remain alive for a few days they play the chief part in the spread of powdery mildews. They are distributed mostly by the wind but insects may also carry them, while birds, other animals, tools or the clothes of workers may play a minor rôle.

Some perithecia may become detached from the mycelium; others are firmly fixed to it and to the tissues of the host plant. The appendages of some species may assist in scattering them, in that several perithecia may hang together and be carried away like fluff in the wind. Dispersal of the fungi in this way can be of little importance, however, in comparison with the immense numbers of conidia produced.

Control. (1) As already noted, host varieties often differ greatly in their susceptibility to mildew. Some roses are well known to be almost immune from it while others are so susceptible as to become unsightly unless repeatedly sprayed to control it. Further details about resistant varieties are given under the different crops.

(2) Environmental conditions play a great part in control. Mildews are most at home in a fairly high temperature. The conidia are shot off under dry conditions and are then most infective but they need humid conditions for germination. In enclosed situations where the air is usually warm it remains damp for a long time after rain or dew. Thus American gooseberry mildew, for example, is worse in enclosed gardens than where the bushes are exposed. It is well known that roses tend to be more attacked by rose mildew when they are trained against a wall, especially a south wall, than when they grow on a trellis, a pole or in some other free-standing situation. The high temperature on a south wall is obviously one of the reasons why mildew does so well there. An additional reason may be that plants there often lack water and are hence rendered more susceptible to infection.

Where mildew attacks are to be anticipated nitrogenous manuring should be kept well in hand and adequate amounts of potassium and phosphoric acid supplied.

(3) As a rule cutting off and destroying the shoots first attacked will not check the disease and often, as in gooseberries, it merely encourages growth of new shoots which will be particularly susceptible. Better results are obtained by cutting out and burning the diseased leaves and shoot tips at the end of the season, especially in mildews like that of apple which overwinter in the buds. Autumn pruning of infected tips is also effective in checking American gooseberry mildew. With herbaceous

perennials the tops and withered fallen leaves should be collected in autumn and burned. The remaining stumps can be picked off and destroyed in spring when they are quite withered and before the new shoots appear above ground.

(4) Direct control by sprays or dusts if necessary must almost always be repeated several times. Usually the best results are obtained when the treatment is begun before the attack has become really serious.

As the mildew mycelium is mostly on the surface of plants and only the haustoria penetrate the host cells, it is easier to kill than that of most other parasites which live mainly inside their host tissues.

Formalin (p. 595) kills the mildew it touches but has no protective after-effect. Salicylanilide (p. 599), white oil (p. 601) and petroleum emulsions (p. 598) probably have a more lasting effect. Washing-soda solution is recommended against American gooseberry mildew, and on some plants spraying with pure water destroys the mildew (Yarwood, 1939).

If it does not matter that a visible deposit is left on the plant various sulphur preparations are very effective against powdery mildews, e.g. lime sulphur 10:90 for winter spraying of vines or 2:100 or 3:100 for summer sprays of roses, hawthorn, etc., in nurseries. Various proprietory colloidal preparations containing sulphur are also effective and do not leave such a conspicuous deposit as lime sulphur. Dusting with flowers of sulphur or green sulphur is very commonly used for mildew control, so also is evaporation of sulphur in hothouses (p. 600). Very low concentrations of hydrogen sulphide are effective in killing powdery mildews (Yarwood, 1940).

Root Rot, Foot Rot and Damping Off

(*Rhizoctonia solani* Kühn = *Corticium solani* Bourd. & Galz., *Pythium de baryanum* Hesse, *Phytophthora* spp. and other fungi)

Damping off is a term applied to the rotting of young seedlings at or slightly above soil level so that they fall over and die. When the

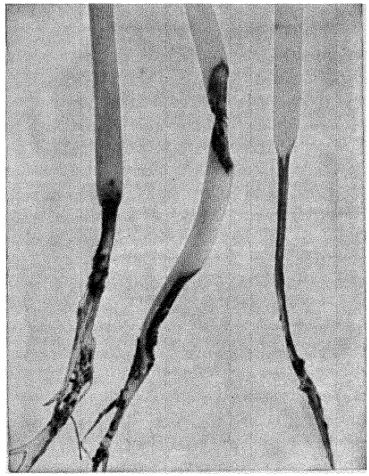


Fig. 35. *Corticium solani* lesions on stems of cabbage seedlings. That on the right shows "wire-stem" (*S. p. F.*).

stems of young plants, especially of the cabbage and beet families, shrivel and become dark and hard just above soil level the terms "wire stem" or "black-leg" are used. Foot rot is a similar condition in older plants. The same fungi that cause stem lesions and damping off are often responsible for root rots, especially in greenhouse soils.

The fungi usually involved in damping off and foot rot are *Corticium solani*, *Pythium de baryanum* and several species of *Phytophthora*, especially *P. parasitica* Dast. and *P. cryptogea* Pethybr. and Laff. *Thielaviopsis basicola* (Berk.) Ferraris, several species of *Fusarium* and *Cylindrocarpon radicicola* Wollenweber are also associated with rotting, especially of roots, but they seem to be usually only weak parasites following other species of fungi or attacking tissues weakened by other causes.

Corticium solani causes a clearly defined rot of shoots at about soil level. At first the lesions are grey and watery but they soon dry out, becoming sunken and light or dark brown. Because of its tendency to form flat rough brown or black sclerotia, especially on potato

tubers, this fungus is often called " black scurf " or " the dirt that won't wash off " but sclerotia are rarely to be found on damping off or foot-rot lesions. Though the name *Rhizoctonia* is that best known in plant pathological literature it strictly applies only to the sterile sclerotial state of the fungus and should be replaced by *Corticium solani*, the name applicable to the fertile, perfect state. This occurs quite commonly as a white powdery coating on full-grown potato stems a little above soil level and may also be found on other plants. It consists of a thin web of mycelium bearing scattered basidia each with four spores. The " propagation fungus " *Moniliopsis aderholdi* Ruhl, found especially on beet or boxes of young plants, like begonias and cyclamen, that have been kept warm and damp, is closely related to *R. solani* and perhaps only a form of the same species. Some authorities like Müller (1923) and Wellensiek (1925) prefer to regard it as a distinct fungus, others, like Simon Thomas (1925) and Schultz (1937) believe it to be only a strain of *R. solani*. Its light-brown mycelium grows like a fine spider's web over soil and plants and causes rapid decay.

Pythium de baryanum attacks very young seedlings. The stalk from soil level upwards, sometimes as far as the cotyledons, shrivels to a slender thread and the seedling falls over. It is best known as a parasite of cress but, like several other species of *Pythium*, it is able to attack many other plants.

Phytophthora parasitica and *P. cryptogaea* cause damping off like that due to *Pythium* but are more active and dangerous parasites. They are particularly well known as the agents of damping off and foot rot in tomatoes but can attack almost any herbaceous plants and tree seedlings.

Cylindrocarpon radicicola, *Fusarium* spp. and *Thielaviopsis basicola* are usually associated with a rather deep brown discoloration of the rotting tissue, especially in roots. They are often secondary to *Corticium* or other active parasites.

Olpidium brassicae (Woron.) Dangeard is a very minute fungus which occupies the root hairs and outermost cells of fine roots in cabbage, tomato, etc. When very abundant it may cause injury to the roots but is likely to be important only in very wet soils.

Some seed-borne fungi such as *Alternaria brassicae* and *Phoma lingam* (Tode) Desm. may cause rotting of seedlings but these are in rather a different category from the common soil organisms mentioned above. Species of *Alternaria* may, however, cause decay of cuttings. Neergaard (1936) has demonstrated that root rot in celery and carrot may be caused by the related black mould *Stemphylium radicinum*.

Distribution of foot rot and damping off fungi in soil. Experiments with about 100 soil samples from nurseries in the Copenhagen district, made by C. A. Jørgensen and A. Weber in 1926–7 at the Danish Plant Pathological Research Laboratories, showed almost all the nursery soils contained both *Corticium solani* and one or more species of *Pythium* and *Phytophthora*. The tests were made by sowing white cabbage and cress in the soils. The first-named was mainly attacked by *Corticium*, the other by *Pythium*.

In 1933 C. A. Jørgensen and G. Johansen investigated the ability of these fungi to spread through clay soil, gravel, leaf mould and ordinary field soil. They showed that leaf mould and field soil became thoroughly permeated by *Corticium*, but gravel and clay soil were much less so. This agrees with the general conception of *Corticium* as a fungus living saprophytically in organic débris in the soil. When gravel or clay were mixed with the field soil or leaf mould the fungus spread much less readily. *Pythium*, on the other hand, spread rapidly through soils of all kinds.

These experiments gave much the same results as those of Abdel Salam (1933) with *Corticium solani* isolated from lettuce. He found it gave a very high percentage of infection on lettuce grown in leaf mould and clay soil but a low percentage in sand. His " clay soil " apparently corresponded closely to Jorgensen's " field soil." In all his soil mixtures inoculation with *Pythium* gave almost 100% infection.

C. A. Jørgensen has also investigated the influence of soil reaction on *Corticium* and *Pythium* and has found that only a very alkaline reaction, i.e. up to pH 8.4, checked *Corticium* root rot. Soil reaction has a greater influence on *Pythium* root rot but liming alone is unable to prevent its attack altogether.

In Ceylon Park (1927) investigated the effect of soil moisture and atmospheric humidity on *Corticium* and found changes in it had little effect as long as it did not exceed levels suitable for seedling growth. When, however, the atmospheric humidity was increased from 41% relative humidity up to saturation point, *Corticium* attack did increase very rapidly.

Influence of temperature. Simon Thomas, working in Holland with strains of *Corticium solani* from Europe, America and Java, found he could divide them into two groups. One, including three races isolated from begonias, had its optimum temperature above 70° F. and would not grow at 40° F. A strain from cotton also belonged to this group, and so did *Moniliopsis aderholdi*. The other group, isolated from potato, beet and turnip in temperate climates, had its optimum temperature below 70° F. and grew slightly even at 40° F. In Germany Wollenweber stated *Corticium solani* had its optimum temperature at 77° F. and *Moniliopsis aderholdi* at 84° F. C. Müller (1923) found that *C. solani* grew between 38° F. and 90° F. with an optimum at 75° F. The sclerotia germinated between 48° F. and 86° F., with an optimum at 73° F. Abdel-Salam found a strain of *C. solani* isolated from lettuce grew actively at temperatures down to 46.5° F. whereas one from tomato had its optimum temperature at 77° F. The general conclusion appears to be that numerous races of *C. solani* exist, adapted to flourish at the temperatures in which they are found in nature.

Hemmi (1923), working with cress seedlings, found *Pythium* most destructive at 68–86° F. and *Corticium* at 61–75° F. Bewley (1929), in reference to tomato root rot, stated that the optimum temperature for *Phytophthora parasitica* is 80° F., that for *P. cryptogea* 75° F. and that for *C. solani* 64° F. None of these fungi caused appreciable damage below 50° F.

Host range and specialization. If several species of plants are sown in a soil infected with different types of root rot fungi some of them will be attacked by one fungus and others by another. As control measures are not the same for all soil-borne fungi it is important to know which plants each is most likely to attack. Much information on this point has accumulated from examination of diseased plants from nurseries. The problem has also been investigated directly by C. A. Jørgensen, O. Nielsen and A. Weber, who have grown many different plants in soil infected either by *Corticium* or *Pythium* and have also worked with various races of these fungi on different hosts. There is also much information available in the literature on this subject, and the following table of susceptibility is drawn up from all these sources. Peltier (1916), for example, has given a list of 165 host plants of *Corticium* in U.S.A. Many of these are garden plants affected only with root rot.

Various research workers have noted that *Corticium* strains in general are most pathogenic to the host from which they were isolated or on its close relatives. Results of the inoculation experiments of C. A. Jørgensen, O. Nielsen and A. Weber with races of *Corticium* from various hosts may be summarized as follows:

1. *Corticium solani* from cabbage and cauliflower destroyed 90–100% of cabbage, radish and stock seedlings but did not kill tomatoes.

2. *Corticium solani* from potato tubers had little power to attack wallflower, cabbage, cress or stock, and even less to infect cucumber or tomato. Usually it was completely unable to infect these hosts.

3. *Corticium solani* from sweet pea caused heavy infection of cucumber, cabbage, stock and radish, but not of tomatoes.

4. *Corticium solani* from begonia leaves heavily infected cabbage.

5. *Corticium solani* from hydrangea cuttings caused only slight infection on radish or stock and none on cucumber or tomato.

6. *Corticium solani* from turnip seedlings had no effect on cabbage, cucumber or tomato.

7. *Rhizoctonia tuliparum*, a distinct species causing rotting of tulip bulbs, infected neither cucumber, cabbage, radish nor tomato. Dennis (1941) found that strains of *C. solani* isolated from potato tubers were unable to attack swede roots though a strain able to do so occurs in swede fields. W. C. Moore (1943) found a strain isolated from radish cankers could attack radish and turnip but not potato tubers, carrots or parsnips. Wellensiek (1925) found that *C. solani* isolated from tomato foot rot was entirely pathogenic to potatoes and a strain from potatoes caused severe injury to tomatoes. Evidently there are at least two sharply differentiated groups of *Corticium* isolates, one able to attack potato and often tomato, the other unable to attack these plants but parasitic on members of the cabbage family. Members of this group seem sometimes able to attack hosts belonging to very different families.

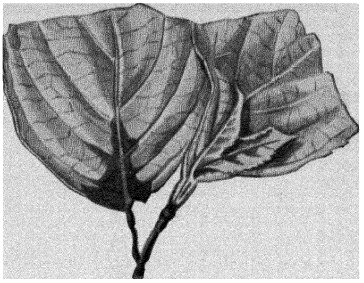


Fig. 36. Rotting of hydrangea cutting by *Corticium solani*.

Races of *Pythium* isolated from cabbage and cress were strongly pathogenic to both cress and spinach. There seems to be little evidence of host specialization in *Pythium* and *Phytophthora* species.

Susceptibility of Horticultural Crops to Foot Rot and Damping Off Fungi

Pythium and Phytophthora	*Corticium solani*
Cucumber	Potato (sprouts)
Antirrhinum	Antirrhinum
Begonia	Begonia cuttings
Celosia	Celosia
	Wallflower
Dianthus (cuttings and seedlings)	
	Hydrangea cuttings
Cabbage	Cabbage
Cress	Cress
Kochia	Kochia
Sweet Pea	Sweet Pea
	Stock
Nemesia	Nemesia
Pelargonium cuttings	
Parsley	Parsley
Pine seedlings	Pine seedlings
	Pteris (fern)
	Radish
Beetroot	Beetroot
Lettuce	Lettuce
	Celery
Tomato	
Verbena	Verbena
Viola	
Culinary pea	

Prevention. To avoid trouble from damping off of some kind the seed must be free from infection. As a precautionary measure it is well to use one of the standard seed disinfectants. Shaking up with cuprous oxide or zinc oxide at the rate of one level teaspoonful per pound of seeds has been found very effective as a seed protectant during germination (Ogilvie and Hickman, 1938).

Cuttings should be taken only from healthy plants.

All pots and seed boxes must be free from infection. In general thorough washing may be sufficient to ensure this but if an outbreak of damping off has occurred it is best to steam all pots thoroughly. Chemical disinfectants may be used but, especially with earthenware pots, there is a risk of poisonous amounts of the disinfectant remaining which may injure seedlings. Cardboard pots which are destroyed after use are, of course, free from any chance of contamination unless they have been stacked on infected staging or have lain on a heap of infected soil. When an outbreak has recurred all staging should be well scrubbed with disinfectant.

The usual source of trouble lies in the use of infected soil. Unfortunately the fungi concerned are so widespread that it is difficult to obtain soil completely free from them. Even soil fresh from a field is likely to contain strains of *Corticium solani*, though, as a rule, soil on which a particular crop or its close relatives have not been grown for several years is unlikely to contain strains of *C. solani* actively parasitic to that crop. It may, however, contain *Pythium* and *Phytophthora*.

Where there is only slight infection of the soil, seedlings may escape attack if they are well grown. Plants making rapid but sturdy growth are less likely to succumb than those with weak, soft, forced growth. Though different strains of the parasites differ widely in their temperature requirements, it may be said as a general rule that damping off is unlikely to be serious at temperatures below 54° F. Soil moisture seems to play little part in *Corticium* attack but it is much encouraged by a moist atmosphere (see p. 70). *Pythium* and *Phytophthora* are water moulds, however, and likely to be favoured by soil moisture. One should, therefore, not only avoid unnecessary watering but also prevent too much dampness at night by watering early in the day, keeping the house well ventilated and avoiding low night temperatures. Bewley (1929) states that loosening the surface soil, which leads to drying of the top layers, may check *Phytophthora* attacks but not those of *Corticium*. To avoid weak growth and encourage ventilation it is important not to sow seed thickly and not to crowd plants at later stages of growth.

The composts usually prepared for cuttings and seedlings are likely to suit *Corticium solani* particularly well. Where trouble has been experienced some relief may be obtained by mixing gravel with the compost or covering the surface with a thin layer of gravel or sand. It may even be better to use in seed boxes a field soil mixed with gravel instead of a compost.

Finally, in all large nurseries where seedlings are raised in considerable numbers routine soil sterilization should be done as a precautionary measure. The best and most effective method is by steaming. Excellent steam sterilizers capable of dealing with a few tons of soil, sand or leaf mould at a time are on the market. Sterilizing with dry heat is less effective and more difficult to carry out thoroughly.

Those who are unable to steam their potting soil may have recourse to chemical disinfectants. Unfortunately the fungi concerned differ somewhat in their susceptibility to chemicals. C. A. Jørgensen has examined the action of various preparations against *Corticium* and *Pythium* and has published the results of 265 experiments.

The following were very effective against *Pythium*:

1. 8 oz. commercial formalin in 2 gallons water applied per square yard. Sow 3 weeks later.

2. 8 oz. raw carbolic acid in 2 gallons water per square yard, sow 4 weeks later.

3. 4 oz. Kerol in 9 gallons water per square yard, sow 4 weeks later.

The following were very effective against *Corticium*:

1. 2½ oz. Uspulun per square yard, of top soil, dissolved in enough water to drench the soil thoroughly. Plant 2 weeks later.

2. ½–1 oz. corrosive sublimate plus 8 oz. saltpetre dissolved in 10½ gallons of water per 8 square yards, plant one week later.

Uspulun was very effective but is now off the market, though other organo-mercury disinfectants may be available.

The corrosive sublimate solution was first used at Lyngby by C. A. Jørgensen, who has tested it thoroughly. Where the soil has been thoroughly soaked with it, it has given excellent results against *Corticium solani*, but has sometimes checked seed germination and has checked the rooting of cuttings. It should be done at least two weeks before the soil is to be used. Addition of the saltpetre often seems to overcome the injurious effect, which seems to be most marked on very alkaline soils of pH over 8.0. In addition the sublimate treatment checks weeds and also the growth of blue-green algae which are apt to make the surface soil of seed boxes wet and crust-like.

Seeds of various plants, including cabbage and stock, germinated much more slowly and on the whole less effectively when watered with the sublimate saltpetre solution just after sowing than when watered with water alone. Stock could be safely watered with sublimate saltpetre immediately after transplanting, even if it had been done some weeks earlier. Shoot cuttings of begonia tolerated it very well but leaf cuttings would not root in it. The solution should not, therefore, be used on soil for hydrangea or begonia cuttings.

Sublimate saltpetre can be used where *Corticium* foot rot is expected or against *Moniliopsis aderholdi*. For example, where the latter has been seen to appear in patches we would advocate watering the patches and the adjacent plants with sublimate saltpetre solution to check its further spread. Even with begonia and hydrangea it is better to do this and to kill the plants in the immediate neighbourhood rather than to let the patches spread unchecked.

The experiments indicate that cucumber, cabbage, celery, spinach and tomato as well as antirrhinum, sweet pea and stock may all be sown in soil treated with sublimate saltpetre solution 1–2 weeks earlier. Cucumber, spinach and tomato, however, are often attacked by *Pythium* or *Phytophthora* and sublimate saltpetre is less effective against them than against *Corticium solani*.

Corrosive sublimate is a very deadly poison and care should be taken not to dissolve it in any utensil from which drinking or household water may be taken, to keep the undissolved salt out of the reach of children and irresponsible persons and to pour all unused solution down a drain as soon as disinfection has been completed.

Cheshunt mixture has been widely advocated in England for checking damping off in cucumbers and tomatoes. We have never heard of any injurious results from the use of this solution, nor, unfortunately, has it been effective in our experience. In the experiments carried out in Denmark it had practically no effect on *Pythium* on cress. Possibly its action is specific to *Phytophthora*.

Dusting with sulphur has some effect against *Moniliopsis aderholdi* mycelium growing over the surface of soil and plants but has little or no effect on true damping off or root rot.

Brassicol is used as a dust to stop damping off outbreaks that have just begun. It is applied either alone or mixed with sand (⅛–¼ oz. in 17 pints of sand) which is then spread in a thin layer over the affected patch of soil and its surroundings. Plants should be shaded for some days after this treatment.

Quick rooting by cuttings counteracts foot rot and root rot and to assist this some of the proprietary hormone mixtures marketed for the purpose may be used. Or, the cuttings may be placed for 24 hours in 1 pM of permanganate of potash. This has been tried with good results with chrysanthemum, hydrangea, Christmas rose, pelargonium and begonia cuttings.

Because *Pythium* and *Phytophthora* are water moulds, able to thrive on decaying plant remains in water, they may be introduced to a

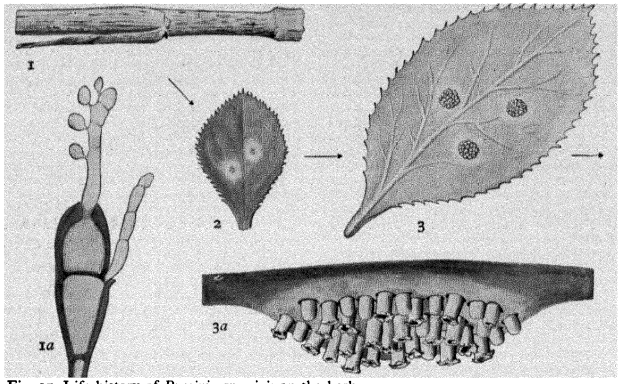


Fig. 37. Life history of *Puccinia graminis* on the barberry.
1. Overwintered cereal straw covered with teleutosori.
1a. Germinated teleutospore with its 2 promycelia, the upper bearing its 4 basidiospores (× 500).
2. Patches of spermogonia on upper side of a barberry leaf, each patch surrounded by a yellow zone.
3. Patches of æcidia on the under side of a barberry leaf.
3a. Patch of æcidia (× 10).

glasshouse in the water supply, especially if this comes from surface wells or tanks. All tanks in the houses should be thoroughly cleaned out and disinfected at regular intervals.

Rust Fungi

Attacks of rust, especially on cereals, have been familiar to farmers from very ancient times and are mentioned by most of the ancient Greek and Roman writers on natural history. The name is very appropriate to this group of fungi because the most abundant spore form in many species contains a bright orange pigment. Millions of these spores make the affected plants look as though dusted with a rusty powder. Colourless, yellow, brown or black-coloured spores also occur in most species. Rust fungi are classified as basidiomycetes but their basidium is a peculiar one which appears for only a short time during the germination of the spore form common to all rusts, the teleutospore. All are obligate parasites and many species have a peculiarly complicated life history in which four different types of spore follow one another in regular succession. In some species all these spore forms develop on the same host plant, in others two appear on one kind of host plant and the other two on a host plant belonging to an entirely different family. This strange condition, called heteroecism, is well illustrated in the life history of the common black rust of cereals, in which it was first demonstrated.

Black rust of cereals (*Puccinia graminis* Pers.) is so called because of the conspicuous black stripes formed on the culms by the final spore stage produced in late autumn. Under a microscope these stripes are seen to be composed of tightly packed two-celled bodies with thick brown walls. These are the resting spores or teleutospores, which carry the fungus over winter. In spring each cell of the teleutospore germinates to form a short four-celled hypha, the basidium, on which four basidiospores are produced. These are shot off into the air but are unable to infect a cereal or grass leaf. Only if they fall on a leaf of barberry (*Berberis vulgaris* or its close allies) can they develop any further. If they do so they penetrate the barberry leaf and form thickened, circular, orange patches of tissue. Closely packed on the upper side of these patches there next appear a large number of very minute yellow pustules, just visible to the naked eye. These

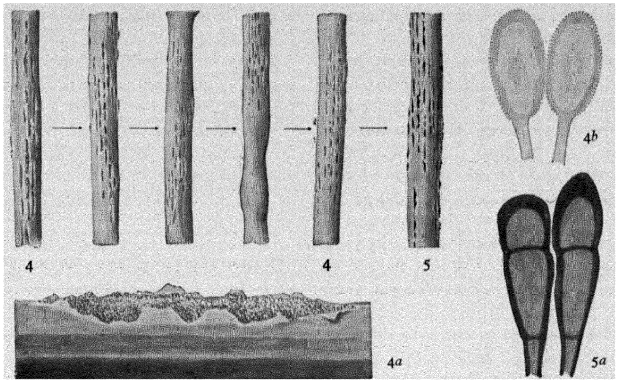


Fig. 38. Life history of *Puccinia graminis* on the cereal.
4. Brown uredosori on culms of a cereal or grass, successive generations of uredospores arise throughout the summer.
4*a*. Uredosorus (× 10).
4*b*. 2 Uredospores (× 500).
5. Black teleutosori formed on the culm in autumn.
5*a*. 2 Teleutospores (× 500).

are the spermogonia, which exude tiny drops of nectar containing large numbers of male cells (spermatia). Insects attracted by the nectar spread these spermatia from pustule to pustule and bring about a kind of fertilization. As a result the second spore form appears, usually in May or June, on the lower side of the same orange patches on the barberry leaves. These are the bright orange aecidiospores, borne in short chains, some 10,000 at a time, inside minute white cup-shaped fructifications, the aecidia. No further development takes place on the barberry and the aecidiospores cannot infect another barberry leaf. They can, however, infect a leaf of the same kind of cereal as bore the teleutospore from which the barberry was originally infected. Black rust exists in many specialized races, some attacking only wheat, others only barley, oats, rye or various grasses. Black rust from one kind of cereal cannot be made to infect another. An aecidiospore which has fallen on the appropriate cereal leaf germinates, penetrates the leaf and forms a mycelium running between the cells. In about eight or ten days a yellow patch appears, the skin of the leaf is burst open and a patch of one-celled bright orange summer spores, uredospores, appears. These are blown about by wind and can infect another leaf of the same kind of cereal. They are produced in incalculable numbers in successive generations and are the spore form responsible for the common name "rust." Uredospores are produced in such numbers that some are almost certain to fall on any susceptible plant in the district. Infection is favoured by damp air and by vigorous growth of the host, and rust is hence most destructive in low-lying situations and on crops that have received a great deal of nitrogenous manure. At the end of the season when the host begins to fade the final black teleutospore stage appears again and the life cycle of the rust fungus is completed.

From the above account it will be seen that the barberry plays a critical part in the life of the rust. In the first place, in climates where the winter is too severe for uredospore generations to be produced all through the winter, the reappearance of black rust in spring is dependent on the presence of a barberry bush for the basidiospores to infect. Secondly, because sexual reproduction takes place on the barberry, there is an opportunity for

hybridization of different varieties and races of the rust and the production of new ones. There is always a risk that some of these new hybrid races may be able to infect varieties of the host plant immune to the old races of rust. Because of this essential rôle of the barberry, cultivation of susceptible species of *Berberis* was prohibited in Denmark by a law of March 27th, 1903, except in botanical gardens and reserves. This led to a decline in the amount of damage from black rust until a new generation of growers tended to observe the old law less strictly. Then in 1937 and 1938 severe black rust infestations led to its being enforced again. The prohibited species of barberry are *Berberis vulgaris* and its varieties, *B. canadensis*, *B. crataegina*, *B. koreana*, *B. ilicifolia* and *B. wilsonae*. Species of *Mahonia* are also susceptible but the common *Mahonia* rust is a different fungus which cannot attack cereals or grasses.

Since 1903 a number of other small-leaved species of barberry not susceptible to rust have become popular. The most important of these is *B. thunbergi*. Black rust is not usually a menace to cereal cultivation in the British Isles, except, perhaps, in Pembrokeshire, where it seems often to be prevalent. There is no law prohibiting barberry cultivation in this country and it is not unusual in some districts to find barberry bushes in the hedges of cornfields. Often the aecidia they bear belong to a race of black rust infecting only wild grasses, but it is obviously unwise to continue using the common barberry as a hedge plant.

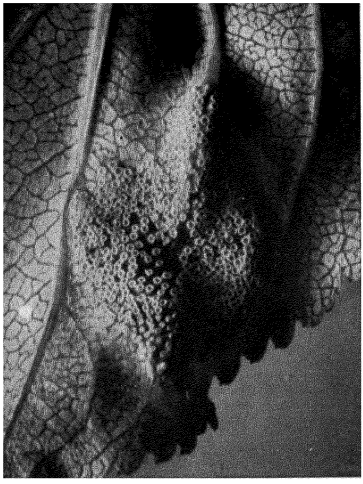


Fig. 39. Aecidia of crown rust of oats and grasses on the under side of a buckthorn leaf (enlarged) (see turf, p. 554).

Not all rusts have this complicated life history. Some have all four stages on the same host plant, e.g. mint rust, rose rust. Others omit one or two stages and many produce only teleutospores, as for example hollyhock rust. It will be readily understood that in the case of heteroecious rusts the connection between aecidial and teleutospore stages can only be demonstrated by experimental cross-inoculation. This has been done for nearly all British aecidial stages but in other parts of the world there remain many as yet uninvestigated aecidial fungi which have to be classified temporarily in the imperfect genus *Aecidium*. Similarly a few rusts are as yet known only in the uredo stage.

In temperate climates overwintering is usually done by the teleutospores, but some species pass the winter as mycelium in the tissues of the host; examples are birch rust, several rusts of junipers and the rust of St. Brigid anemone. In districts where a rust is unable to survive the winter it may still recur every season because of infection by wind-blown uredospores from a considerable distance. Sometimes, as in barley rust in England, it survives the winter in the uredospore stage. Similarly on plants like chrysanthemums which are moved into greenhouses for the winter rust may survive for many years in the uredospore state alone. In tropical climates with well-defined wet and dry seasons it is often the summer drought rather than the winter that rusts find it difficult to survive. In North India the cereal rusts survive the summer in the hills and invade the low-country crops from thence each winter. Uredospores of carnation rust can survive six months' drought.

Rusts are classified mainly by the kind of

teleutospore they produce. Two-celled teleutospores forming a single layer in the pustules, as in black rust, are characteristic of the genus *Puccinia*. One-celled teleutospores belong to *Uromyces* (example, carnation rust); those of *Phragmidium* are many-celled (example, rose rust). Two-celled teleutospores stuck together to form large orange horn-like bodies occur in *Gymnosporangium* (example, juniper rusts). Other rust genera, like *Melampsora* and *Coleosporium*, form their teleutospores within the tissue of the host. Typical aecidia, like those of black rust, are often called " cluster cups," as for example in gooseberry rust. Less circumscribed aecidia sometimes form large powdery orange masses where they have forced open blisters in the skin of a stem. These are sometimes called " blister rusts " as on the branches of five-needled pines.

Rust pustules are often attacked by parasitic fungi (*Darluca* and *Tuberculina*) and by the larvae of certain midges. These tiny maggots are hard to see because the spores they have eaten show through their transparent bodies and make them the same colour as the rust. None of these natural enemies plays any appreciable part in controlling rust attacks.

Occasionally attempts have been made to use rusts to check the growth of introduced weeds, especially of the common field thistle in North America, but with little success. In parts of Asia nettle stalks swollen by the attack of rust are eaten as a delicacy.

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) Mass.)

History. Mlle Libert first described this fungus from Belgium in 1837 under the name *Peziza sclerotiorum*. When the German mycologist Fuckel later erected the genus *Sclerotinia* he referred her species to it as *S. libertiana*, a name often used in the older literature. The first specific epithet for the species must, however, be used, so that the correct name for the fungus is *Sclerotinia sclerotiorum*. Our knowledge of its parasitic activity dates from the work of de Bary, 1886. It will attack a large number of herbaceous plants and occurs in all parts of the world. *S. sclerotiorum* may

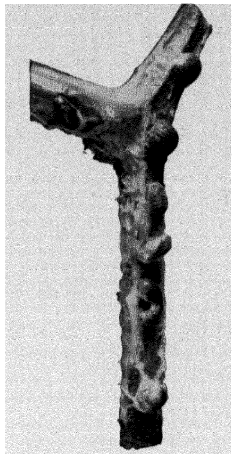


Fig. 40. Mycelium and sclerotia of *Sclerotinia sclerotiorum* on cucumber stem.

occur in association with grey mould (*Botrytis*) and the two fungi often cause somewhat similar diseases but there is no connection between them. *S. sclerotiorum* has no conidial stage and the perfect state of *B. cinerea* is another species of *Sclerotinia*.

S. sclerotiorum may attack growing plants both out of doors and under glass and is often destructive to vegetables in storage. Besides many dicotyledonous seedlings the following host plants are often attacked:

FLOWERING PLANTS

Antirrhinum (flower infection)	Iris
Campanula	Lupin
Dahlia	Nicotinia
Delphinium	Petunia
Forsythia	Pyrethrum
Helianthus	Zinnia

VEGETABLES

Artichoke	Caraway
Beetroot	Celeriac
Cabbage	Celery
Capsicum	Cucumber
Carrot	Dill

VEGETABLES—*continued*

Jerusalem artichoke
Lettuce
Onion
Parsley
Pea
Potato
Tomato

FRUIT

Apricot and peach (ascospore infection of the flowers in California)
Orange
Melon

WEEDS

Chicory
Prickly lettuce (*Lactuca serriola*)
Stinging nettle

Symptoms and progress of the disease. When growing plants like lettuce are attacked they become in the course of two or three days flabby as though cooked. In the older parts of woodier plants like tomato stalks the soft ground tissue decays while the wood and bast remain.

In the field the most striking feature of this fungus is its abundant white woolly mycelium,

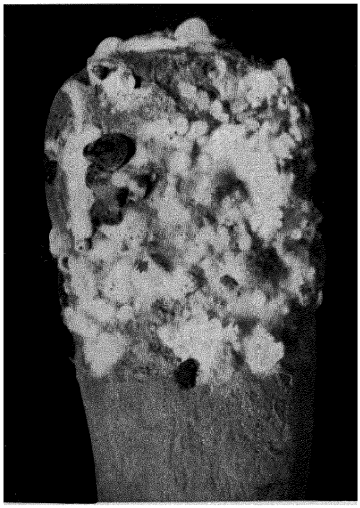


Fig. 41. Carrot rotted by *Sclerotinia sclerotiorum*.

to be found in hollow parts of the plants and, if the air is damp, growing over their surface as well. It may grow short distances over the soil from plant to plant and in stored vegetables it may cover the whole contents of the store.

Aided by the mucilaginous coating of their walls the hyphae adhere firmly to the host plant and bore a way into the tissue by mechanical pressure (Boyle, 1921). Once inside they exude poisonous substances which kill the surrounding cells. The mycelium permeates these and kills the tissue beyond and so on. In young plants like lettuce, where the cells are full of nutrients, the fungus spreads very rapidly. If there are wounded or dead parts on the plant, like withered leaves or faded flowers, these are still more easily invaded. Damp corners of leaves are also often infection courts. Birds eating the seeds in ripening sunflower heads may both wound the tissues and bring *Sclerotinia* infection with them.

The ordinary mycelium of *S. sclerotiorum* is very long-lived in soil; for example, it survives the summer in the dirt of vegetable stores that have not been thoroughly cleaned. The sclerotia or resting bodies are much more resistant, however. They originate as little white tuber-like lumps on the mycelium, often covered with watery drops like dew. As they mature they become hard and dry with a black crust. When formed in hollow stalks they are often small, but on stored vegetables they may be up to one or one and a half inches long. The " basket " of a ripened sunflower head may be completely filled with a large net-like sclerotium. Young sclerotia may be found on mycelium that is only three or four days old.

Sclerotia may remain alive in a dormant condition for two or three years but they may also germinate after a short time. Often they produce the same white vegetative mycelium, but in some circumstances (especially when food is short?) they bear the cup-shaped fructifications. A single sclerotium may yield from one to thirty-five of these, each with a long slender stalk terminating in a shallow yellowish-brown cup (apothecium) about ¼ in. across. The disc inside the cup is closely packed with asci, each containing eight colourless, one-celled spores. A single cup may shoot up to 30 millions of these spores into the air, any one of which, if it lands on damp soil or on a susceptible plant, may germinate and

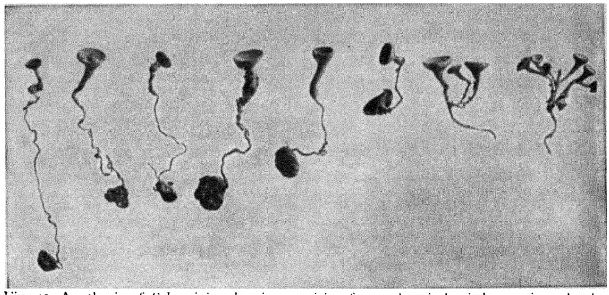


Fig. 42. Apothecia of *Sclerotinia sclerotiorum* arising from sclerotia buried at various depths in the soil. Natural size (*S. p. F.*).

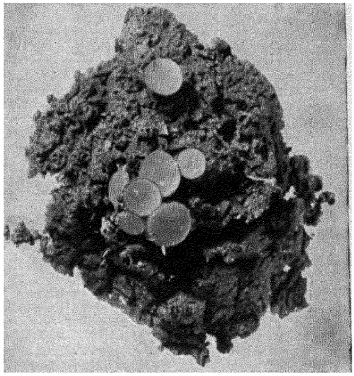


Fig. 43. Apothecia of the clover rot fungus, *Sclerotinia trifoliorum*, seen from above (× 2) (*S. p. F.*).

produce a new mycelium. The length of the stalk to each cup depends on the depth to which the sclerotium is buried. Even those three inches below ground may still be able to produce spores at soil level.

The chief requirement for activity of this fungus is moisture. It thrives in cellars, on overcrowded or weedy crops, in ill-ventilated frames and glasshouses and in wet weather. At 83–86° F. its growth ceases, it does best at 68–77° F. but can still grow slowly at 50° F. and even frost does not kill the mycelium.

Control. To control *Sclerotinia* rot crops must be kept as dry and well ventilated as possible. With espaliered melon newly infected on the stalks it may be sufficient to wash them with corrosive sublimate saltpetre solution. All suspected plants that may carry sclerotia in their tissues should be burned.

Cellars and similar places used for vegetable storage should be thoroughly cleaned in spring, all soil should be removed from them and sulphur burnt there (see p. 580). Soil removed from such places should be regarded with suspicion and not scattered on or dug into a vegetable plot. Soil that has borne an infected crop should be regarded as dangerous for at least the next three years. Lettuce and sunflower are particularly liable to contaminate the soil with sclerotia of this fungus. Also, as highly susceptible crops, they should not be grown on land suspected to be infected.

Disinfection of the soil may be effective if done thoroughly. The soil should be moist at the time, for perfectly dry sclerotia are most resistant to chemicals. Carbon disulphide and corrosive sublimate are ineffective. In North America (Poole, 1922; Krout, 1923) formalin is often recommended at the rate of 14 oz. in 4½–8 gallons of water per square yard. After watering the soil of glasshouses with formalin at the rate of 3½ oz. in 4½ gallons per square yard we still found some sclerotia able to germinate. In Germany brassicol powder mixed with the soil at the rate of 1¾ oz. per square yard has been recommended. It is effective against *S. trifoliorum* on clover but we have as yet insufficient experience of its use against *S. scleriotiorum*.

BACTERIA

Bacteria are often classed as the lowest group of plants. They form a very numerous and varied group characterized by the minute size of their cells which multiply by splitting in half. Each half then forms a separate plant, grows, divides again and so on indefinitely as long as conditions remain favourable. Some bacteria can form a thick-walled resting spore within their cell walls but the majority of species parasitic to plants cannot do this. Whether or not bacterial cells contain a nucleus or a body functioning as one is a hotly disputed question still unsettled. Because of their multiplication by repeated division the bacteria are sometimes called schizomycetes or fission fungi, but their relationship to true fungi is very remote. Like the latter, however, they are always devoid of green colouring matter and most of them are dependent on organic matter produced by other living organisms.

The bacteria that play a part in breaking down dead animals and plants perform an essential service to green plants. Others are able to absorb and fix some of the free nitrogen in the air and thus increase the nitrogen content of soil. A few specialized forms perform similar nitrogen fixation inside modified parts of some flowering plants; examples are the root nodules of all members of the pea family and the leaf galls of *Ardisia*. Bacteria of this type are living with green plants in a mutually advantageous relationship (symbiosis). Finally, a comparatively small number of species are dangerous parasites of man, animals or plants.

Bacteria swarm in soil and are blown about with dust, scattered by raindrops or transported great distances adhering to commercial articles. Many of them are motile and can swim about for themselves in drops of water. Moisture in some form is essential for bacterial attack on plants. Some species penetrate fresh wounds, others enter by the water pores of leaves (e.g. black rot of cabbage), through nectaries or through cracks in the epidermis. Often the symptoms of a bacterial disease are quite distinctive and easily recognized; examples are the brown veins of cabbage black rot, the yellow mucilage in hyacinth yellows, the smell and leakage of bacterial soft rot. Actively parasitic bacteria may also be followed by putrefactive forms causing soft, evil-smelling rots in the dead tissues.

Because of their minute size attempts to classify bacteria by their appearance under the microscope have not been very satisfactory. More elaborate classifications have been based on their chemical activities, and ability to utilize various substances as food, but there is still no general agreement on the classification and nomenclature of the species parasitic to plants. These are all rod-shaped and are known in various classifications under the generic names *Bacterium*, *Pseudomonas*, *Phytomonas*, *Xanthomonas*, *Erwinia*, *Corynebacterium*, etc. The confusion is not eased by the fact that the same bacterium can sometimes attack many different plants but has often been described under different names from different hosts. Also, as in fungi, biological races occur and the virulence of even a single race may vary, be impaired, or lost in culture on artificial media and restored by passage through a susceptible host. All the plant parasitic forms can be grown in artificial culture. Actinomycetes (see potato scab) have fine hyphae which readily break up into rod-shaped cells very like bacteria and some authorities believe them to be remotely related to the tubercle bacilli.

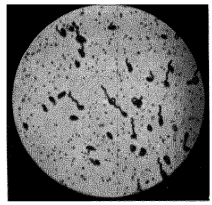


Fig. 44. Bacteria of hyacinth yellows (× 800) (*Photo van Slogteren*).

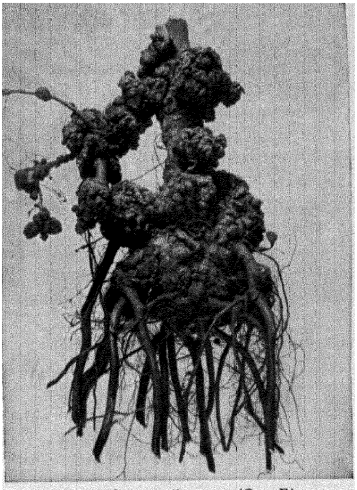


Fig. 45. Crown gall on pear (*S. p. F.*).

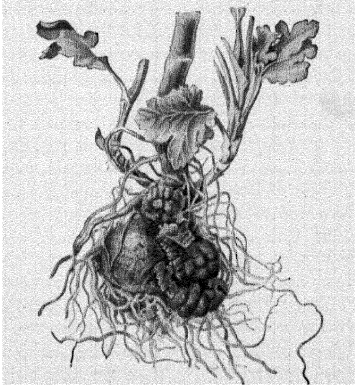


Fig. 46. Chrysanthemum with crown gall.

Crown Gall (*Pseudomonas tumefaciens* (E. F. Sm. & Towns.) Stevens)

The curious bacterial disease known as crown gall occurs on a large number of plants throughout the world. In it the cells of the host are stimulated to grow and multiply so as to form hard irregular galls. They may occur on almost any part of a plant, but because those found round about the junction of root and stem in fruit trees were the first to be studied the disorder is generally known as crown gall, even when it occurs on other organs.

Host plants. Upwards of 70 different kinds of plants, belonging to 39 families, are known to be attacked by crown gall, and bacteria from a gall on one plant are able to induce gall formation on many others. Wormald (1945) has shown, however, that different races of the crown gall bacterium do exist in England. He distinguished a chrysanthemum type which does not produce galls on apple trees, a dock type, infecting raspberry, apple and tomato but not chrysanthemum, a group of strains producing small galls on tomato only and others feebly parasitic to apple. No doubt many other more or less specialized races exist. Crown gall on chrysanthemum, raspberry, rose, tomato, vine and apple is dealt with under each of those hosts. Amongst British economic plants it is known to occur also on:

Almond
Apricot
Asparagus sprengeri
Begonia
Beetroot
Blackberry
Cydonia japonica
Dahlia
Elm
Euonymus
Gooseberry
Hawthorn
Hollyhock
Hop
Loganberry
Lupin
Marigold
Melon
Myrobolan
Pear
Pelargonium
Philadelphus
Phlox
Poplar
Privet
Runner bean
Swede
Sugar-beet
Vegetable marrow
Walnut
Willow

It also occurs on wild plants. Wormald isolated the organism from galls on wild bramble and dock (*Rumex crispus*) and found the common scentless mayweed (*Matricaria inodora*) was susceptible to infection by his chrysanthemum strain. Palm found crown gall on sheep's sorrel (*Rumex acetosella*) in

Sweden, and in North America it has occurred on wild mallow (*Malva rotundifolia*).

Bacteria in relation to the gall. As early as 1880–90 Italian botanists thought crown gall might be a bacterial disease, but it was American plant pathologists, especially Erwin F. Smith, who first gave a convincing demonstration of its origin. At first great difficulties were encountered in isolating the responsible bacteria in pure culture as they are apt to be lacking in the inner, older gall tissue. Experience has shown that they are more easily obtained from the outer tissues and from the soil adhering to the galls.

The size of the gall produced depends partly on the strain of bacterium used and partly on the host plant. When growth is active the galls may become quite large, but if the plant is about to flower or fruit the development of galls on its stems may be greatly retarded. Similarly, on actively growing plants the galls may become apparent only 2–4 weeks after inoculation while on slow-growing ones they may not be visible for months.

Stimulation to growth and multiplication of the affected cells is presumably due to chemical substances secreted by the bacteria. It has long been known that similar outgrowths can be induced to form by injecting plant tissues with appropriate chemicals such as lactic acid. The galls are composed mainly of enlarged thin-walled cells but scattered among them there may also occur some resembling wood fibres and vessels. The whole tissue is abnormal, however, and may quickly decay, though in perennial woody plants it may become hard and persistent. Galls may subsequently be produced from parts of the plant far away from the point of inoculation. Gall tissue has a high degree of independence. It may become larger than the normal part of the plant, as often occurs in sugar beet, and if grafted to other plants it may survive and grow on them. Thus C. O. Jensen grafted gall tissue from fodder beet on to red beet and found the yellowish tissue of the former united with the latter and grew to quite a large gall. Even the small galls produced by treating slices of parsley and cabbage tissue with 1% lactic acid solution can be grafted in this way (Bittmann, 1925). Ø. Winge (1927) has shown that while normal beet cells have a chromosome number 18, those of galls induced by inoculation with a pure culture of *Pseudomonas tumefaciens* have 36 or occasionally 72 or more chromosomes, i.e. they are tetraploid and occasionally octoploid.

Recently Braun (1943, 1947) has demonstrated that *Pseudomonas tumefaciens* may bring about permanent alterations in normal plant cells in a very short space of time. Once this transformation of normal cells into tumour cells has been accomplished the latter continue to develop independently of the bacteria. If they are grown in artificial tissue culture free from bacteria and then re-implanted into a healthy host of the same species they again give rise to typical crown gall tumours.

Obviously copious gall production on the roots of a plant may be very injurious to it but often plants with quite large galls on the crown appear otherwise perfectly healthy. Other kinds of irregular outgrowth on stems and roots are liable to be mistaken for crown gall, and there often occur, especially on herbaceous plants, leafy galls due to quite a different bacterium, *Corynebacterium fascians* (Tilf.) Lacey. These are fairly common on, for example, strawberry and sweet pea. In North America there occurs another infectious disorder of fruit trees, hairy root, due to a third kind of bacterium.

Growth of the bacteria. *Pseudomonas tumefaciens* can grow in artificial culture at temperatures from 32–98° F. but thrives best at 77–86° F. It cannot survive temperatures over 122° F. At favourable temperatures it can spread through soil at the rate of about half an inch a day, as, for example, from the surface of gall tissue, from which bacteria are continually being emitted (Banfield, 1934). In commercial practice infection is spread by the sale of recently infected plants in which gall formation has not yet become apparent. The bacteria can evidently overwinter easily in the soil and Banfield showed that in unsterilized soil they could survive at least 14 months under nursery conditions in the absence of any host

plant. In old nurseries the soil is no doubt very generally infected, and the practice of grafting stocks at or near soil level makes infection very easy. As a rule the bacteria cannot penetrate unwounded tissues.

Prevention. Crown gall is seldom prevalent or serious in England, even on nursery stocks, and most information about preventive measures comes from North America (Waite and Siegler, 1926) and Germany (Stapp and Müller, 1938; Hornbostel, 1939). Research on its control has not yet been called for in Denmark.

In California search has been made for resistant species of *Prunus* that can be used as stocks, and *Prunus mume* has been recommended as a suitable stock for apricot.

Damage to the roots must be carefully avoided during grafting. Clean cuts do not cause much damage but, for example, the danger of infection is greatly increased if shoots are torn off the parent plant instead of being cut off cleanly.

Fungicides containing mercury have been used with good effect against crown gall. In the older experiments Uspulun and Tillantin were used; nowadays the modern organo-mercury bulb and tuber steeps may be tried. To disinfect beet, watering with a solution of 2 oz. of the fungicide in 17 pints of water (about $\frac{1}{2}\%$ solution) per square yard has been used. A similar solution may be used for dipping the roots, or a disinfectant clay gruel or " slurry " may be used, using a good rich clay of which about 4 oz. in 2 gallons of water makes a suitable thickness. At East Malling Wormald found immersing apple root stocks in a mixture of 2–4 oz. Uspulun and $3\frac{1}{2}$ gallons soil in $1\frac{1}{2}$ gallons water gave considerable control of crown gall.

Seed beet and transplanted beet may be disinfected 10–14 days before planting out. Samples should be taken from the year-old seedlings. If they seem quite healthy nothing need be done before planting out, but if many are infected all the plants should be dug up in spring, the affected ones sorted out and destroyed. The healthy ones should then be dipped in a disinfectant clay gruel so that they are covered with it up to the neck. The disinfection encourages root development but, on the whole, it is desirable to transplant susceptible plants as few times as possible as in doing so it is impossible to avoid damaging some roots and so providing openings for infection.

If the roots are to be cleaned up they should be dipped in the fungicidal solution ($\frac{1}{2}\%$ solution) for ten minutes. They should then be kept for some days in a cellar with damp straw over their roots, or should be set temporarily in disinfected soil. Before finally planting out the roots should be dipped in disinfectant clay gruel.

At root grafting the plants should first be soaked for ten minutes in fungicidal solution (9 oz. in 22 gallons of water) and after the grafting is done they should be dipped in the same solution. Grafting table and grafting knife should also be disinfected at frequent intervals. Before planting out the roots should be dipped in disinfectant clay gruel.

If individual trees in a fruit plantation are found to be diseased they may be dug out, roots and all, and burned. If the galls are to be cut away to try to save the tree the knife should be disinfected frequently by dipping in methylated spirits and passing it through a flame. All wounds should be smeared with tar or with the clay gruel. There is, however, little or no information about the effectiveness of such treatment.

VIRUS

The Latin word " virus " (=poison) has always been used in pathology for poisonous or infectious matter; nowadays it implies more specifically infectious matter composed of particles too small to be visible under a microscope in the same way as fungi and bacteria. Of late years purified nucleoproteins have been isolated from the juices of plants infected with certain virus diseases and there is much evidence to suggest that these substances are the viruses themselves. Some of them, like the virus of tomato bushy stunt, can be obtained in a crystalline form. Others, like



Fig. 47. Raspberry leaf with the virus disease " Mosaic No. 1 " (*Photo Harris*).

that of tomato and tobacco mosaic, have been prepared as " liquid crystals," in which rod-like particles are so arranged as to give the liquid some of the optical properties of a crystal.

Though the study of viruses is a very new one virus diseases of plants have existed from time immemorial. " Parrot " tulips, with striped flowers, due to virus infection, were known and valued in the sixteenth century, and there exist pictures of potato plants clearly infected with mosaic disease dating from the end of the eighteenth century. A mottling of leaves due to patchiness in distribution of chlorophyll is a symptom common to the majority of virus diseases, though the " mosaic " pattern formed is often characteristic of the particular disease. Streaking or " breaking " of flower colour is another frequent consequence of virus infection, as is the development of dead spots and streaks of tissue in leaves and stems. Often the phloem of virus-infected plants is damaged and starch may accumulate in the leaves causing them to roll upwards at the edges and become stiff. Most virus infections cause more or less stunting of growth but a few lead to excessive growth either of the whole plant or of particular organs and may cause great distortion of the foliage.

Viruses are responsible for some of the most destructive plant diseases known and are especially to be feared because once a plant has become infected by one of them it is almost always impossible to cure it. Virus diseases are known to attack nearly all the families of flowering plants, though few are met with on forest trees and none on conifers or other gymnosperms. A few attack fruit trees, especially in North America. Of European herbaceous crops only asparagus and flax seem to be entirely free from them. Some crops are liable to infection by more than one virus; potato and tobacco are each attacked by about twenty different ones, causing more or less distinct diseases. Many of these were overlooked or misunderstood until recent advances in virus studies directed attention to them. It is likely too that intensive cultivation and more extensive interchange of plants and plant products in modern times have rendered virus diseases more prevalent than formerly.

Pasteur mooted the possibility of an infectious substance invisible under even the best microscopes in connection with his work on rabies. Then in the nineties of the last century the Russian Iwanofski and the Dutchman Beijerinck demonstrated that juice from mosaic-diseased tobacco plants remained infective even after it had been filtered through porcelain with pores small enough to remove any bacteria present in it. By an extensive use of this principle, using collodion membranes in which it is possible to calculate the pore size, it has been possible to estimate the size of virus particles. Because of their minute size this is most conveniently expressed in millimicrons or millionths of a millimetre (1000 $m\mu$ = 1 μ used in measurement of microscopic objects). The following table gives some idea of the comparative size of plant and animal virus particles and of protein molecules.

Diameter of	about
Egg albumen molecule ...	4 $m\mu$
Foot-and-mouth disease virus particles	8–12 „
Virus particle in tobacco and tomato mosaic	15 „
Same particle length	150–300 „
Infantile paralysis virus particle	50 „
Length of a bacterium of tuberculosis	1000–3000 „

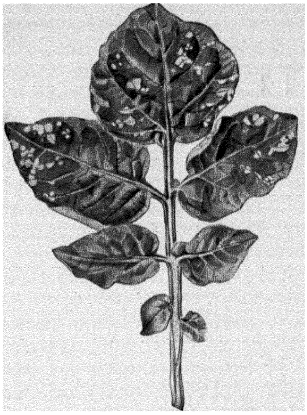


Fig. 48. Aucuba mosaic of potato.

In purified preparations of tobacco mosaic the particles are always united to form rods of any length up to 1000 $m\mu$ but it is not known whether this is the case also in the plant. Within very recent years it has been possible to photograph dried purified virus preparations with the electron microscope. Magnifications obtained in this way far exceed any possible with light in the ordinary microscope, but it is not clear how far the particles photographed in such preparations correspond to those present in living infected cells.

A natural consequence of the infinitesimal size of virus particles is that juice from an infected plant can often be very greatly diluted and still contain enough particles in a single drop to bring about infection. The degree of dilution at which infection is no longer obtained, the dilution end point, is fairly constant and characteristic of a particular virus. Thus tobacco mosaic virus has a dilution end point of 1:1,000,000 and *solanum* virus 2 one of about 1:1,000. No conclusions regarding the number of particles present can safely be drawn from such data, however, for it is not known how many particles are needed to bring about infection with a particular virus.

Viruses differ greatly in their resistance to chemicals; that of tobacco and tomato mosaic is unaffected by ether, chloroform, carbon tetrachloride, strong mineral acids and by germicidal concentrations of corrosive sublimate but is destroyed by 80% alcohol or 40% formalin. Most other viruses are much less resistant to alcohol and formalin or carbolic acid. Washing with soap and water easily removes virus or virus-infected sap. Light has little effect on viruses nor has freezing, but each virus has a characteristic reaction to heat, becoming permanently inactivated at a particular temperature. This thermal inactivation point is so constant as to be of value in identification of viruses. Attempts have been made to use heating to destroy some viruses within the host in the few cases where their thermal inactivation point is below the temperature at which injury occurs to the host cells.

The longevity of viruses in extracted juice and in dried leaves also differs greatly from one to another. Dried tobacco leaves have been found to contain active tobacco mosaic virus after 24 years. Curing tobacco leaves does not destroy it and tobacco on sale in England has been shown to contain active virus. Hence smoking and chewing tobacco may be an important source of infection to tomato crops since the virus of tobacco mosaic is also the cause of tomato mosaic. In sterile filtered juice this virus can also remain infective for several years, but most plant viruses are destroyed in a few days or even hours in extracted juice. Some, like the virus of potato leaf roll, cannot be recovered from extracted juice at all. Viruses of this latter description cannot be transmitted to healthy plants by mechanical inoculation with infected sap.

Those which can survive for a longer or shorter period in extracted sap are usually easily transmitted in this way.

Some viruses have a very wide host range, notably those of tobacco mosaic, cucumber mosaic and tomato spotted wilt; others can infect only a single species of host or a few nearly related species. Cross inoculations on suitable differential hosts are a useful method of identifying plant viruses.

It is important to bear in mind the distinction between the virus responsible for a particular disease and the disease as it manifests itself on the infected plant. But because virus particles are invisible in the host tissues, and it was long before any virus was isolated in a reasonably pure state, the first known viruses were named after the diseases they caused, as for example tobacco mosaic, potato leaf roll and so on. The large number of viruses now recognized and the wide host range of some of them renders a more formal nomenclature necessary. For convenience we here follow the system of Dr. Kenneth Smith in which the virus receives the generic name of one of its hosts, usually that on which it was first recognized or most fully studied, followed by a serial number. Thus the virus of tobacco mosaic is called *Nicotiana* Virus 1 on whatever host it appears. In this system 15 *Nicotiana* Viruses are recognized, 18 *Solanum* Viruses and so on. A few diseases are due to simultaneous infection with more than one virus and in any case, because a single virus may cause quite different symptoms on different hosts or even on different varieties of the same host, it is necessary to have a common name for the disease caused as well as the formal name of the virus concerned. Kenneth Smith's system provides an artificial standard nomenclature but gives no indication of the mutual relationships of the viruses listed. Various attempts at a more natural classification have been made especially in America, but none are satisfactory. All have the fatal weakness of being based on the symptoms caused rather than on the properties of the viruses themselves. A rational classification of viruses can only be expected when the physical, chemical and serological properties of all plant viruses have been much more fully investigated than has been possible hitherto.

Inter-relationships of virus and host. When a plant becomes infected by a particular virus disease symptoms appear only on the subsequent growth. The already-formed leaves remain apparently unchanged, but the virus permeates every part of the plant, though it tends to be most abundant in the actively growing parts. Infection only takes place when the virus is brought into direct contact with living protoplasm, i.e. it must be introduced into living cells by a wound of some kind. The wound need not be large; with highly contagious viruses like *Nicotiana* Virus 1 it may be sufficient to break open a hair cell on the leaf. Viruses of this kind are easily spread by leaves rubbing against one another, by persons fingering an infected leaf and then touching a healthy one or by clothing brushing against the different plants. Experiments by Caldwell (1930, 1931), in which portions of a tomato stem were killed by steam or chloroform, have shown that whereas the water and dissolved salts in the ascending sap passes through the dead zone unchecked along the wood vessels, movement of virus does not take place through the dead tissue. This and other evidence suggests that at least some viruses travel along the phloem strands at a velocity in some instances of 1–1½ inches an hour.

Not all viruses can be transmitted by mechanical inoculation, i.e. by contagion. Many can only be spread from infected plants to healthy ones by the agency of sucking insects. For a few viruses no natural means of transmission have yet been found, and their existence can then only be demonstrated by grafting a scion of an apparently diseased plant on to a healthy one and noting if the new growth on the latter becomes diseased.

The agents that spread viruses. *Nicotiana* Virus 1, the cause of mosaic disease in tobacco and tomato, is remarkable in that, though it is extremely contagious and easily spread by contact on knives, fingers, clothes, chewing tobacco, etc., no insect is known to be able to transmit it. Probably the most active agent

in spreading other viruses in temperate climates is the ubiquitous green peach aphis (*Myzus persicae*). This is known to be able to transmit 25 different viruses. Some 23 other species of aphis are known to transmit one or more plant viruses. In warm climates a large number of viruses are spread by leaf hoppers and a few by white-fly. Tomato spotted wilt virus is peculiar in being transmitted only by thrips.

The relationship between insect and virus is not as a rule a matter of simple mechanical transference of infected sap from one leaf to another. Apparently the virus taken up when the insect is feeding on an infected plant has to find its way into the salivary glands. It may do this gradually over a long period; for instance, a peach aphis that has fed on a leaf-roll-infected potato plant remains for several days able to infect healthy potatoes on which it subsequently feeds. With a few viruses, notably *Solanum* Virus 2, the cause of potato leaf drop streak, a starved aphis fed on an infected plant for a few minutes is more infective than an unstarved aphis or a starved one which has fed for a long time. These facts have an important bearing on the time and rate of virus spread in the field. The virus of tomato spotted wilt can be transmitted only by thrips which have fed on an infected plant while in their larval stage, the adult insects are apparently incapable of picking up the virus. Because of the intimate relationship between so many viruses and insects, especially aphides, the influence of climatic factors on the multiplication and spread of sucking insects is of great importance. So too are local conditions such as shelter or proximity to plants on which aphides pass the winter.

Soil infection is usually of little importance with viruses. Wheat mosaic virus is said to persist in soil, and to enter wheat seedlings through the roots. Tomato mosaic virus may persist for a long time in infected plant fragments in soil. Seedlings may then become infected if their cotyledons or lower leaves brush against some of this infected débris (Johnson, 1937). Virus infection of tomatoes does not normally occur through the roots.

Seed-borne infection is usual with mosaic diseases of leguminous plants. Bean seeds 32 years old have been found still to yield mosaic-infected seedlings (Pierce, 1929). Lettuce mosaic is also seed transmitted, so perhaps is cucumber mosaic. In the case of tomato mosaic indications are that while the embryos of seed from the fruit of diseased plants are probably free from the virus it is present in or on the seed coat. Because of the very resistant and highly contagious character of this virus there is an obvious risk of virus dried on the seed coat occasionally being picked up by the germinating seedling. Reddick (1931) has suggested that bean mosaic may be transmitted by pollen.

Overwintering. Because viruses permeate all parts of the infected plant overwintering presents no problems in the case of perennial hosts. Potato viruses pass the winter in the tubers. Viruses with a wide host range may pass the interval between crops by infecting susceptible weeds. Viruses like that of tomato spotted wilt pass the winter in this country largely in perennial ornamental greenhouse plants such as winter cherry, arum lily, chrysanthemum, hippeastrum, etc.

Reliability and variability of symptoms. Apparently identical symptoms may be produced in a given host by quite different viruses —for example, the two kinds of streak disease in tomato. Symptoms closely simulating some virus diseases, such as potato leaf roll, may be due to non-infectious causes. A particularly complicated situation is found with the potato viruses. Here, for example, *Solanum* Virus 1 causes a fatal disease (top necrosis) in varieties like King Edward but causes only a mild form of mosaic mottling on the leaves of others like Majestic. Symptoms alone cannot, therefore, be relied upon for identification of the virus present in a diseased plant. On the other hand apparently perfectly healthy King Edward plants contain a virus which causes extreme distortion when introduced by grafting into the variety Arran Victory. This condition in which a plant remains infected with a virus without showing any symptoms is called " carrying " a virus, and carrier varieties in

potato, strawberry, and raspberry are a serious problem in virus control.

True carriers show no disease symptoms under any external conditions. Sometimes, however, the symptoms of virus diseases become modified or suppressed by changes in temperature, nutrition or age of the plant. Thus fern-leaf disease of tomato, caused by infection with the virus of cucumber mosaic, develops only at rather low temperatures. In certain leaf ring-spot diseases of tobacco the symptoms disappear altogether with age so that the plant appears healthy again though its leaves are still full of virus.

3 THE SOURCE AND DEVELOPMENT OF PLANT DISEASES

In practice, a disease can seldom be said to have only a single cause; even if one particular night's frost can be specified as the immediate cause of the trouble a subsidiary factor may also be involved, as for example late maturing of the buds because of an attack of mildew or a deficiency of potash. Often several factors co-operate to cause disease, e.g. over-liming, loose soil, drought and boron deficiency. In such a case the progress of the disease will be modified if rain comes or if borax is supplied in time. With parasitic diseases, however, there are certain contributing factors which it is helpful to be able to recognize.

The source of infection is primarily another diseased plant where, if bacteria are involved they have multiplied in millions, or the parasitic fungus has produced masses of spores or, in the case of virus diseases, the diseased cells form a reservoir of virus. Dead plant parts exposed to the air no doubt dry out but this does not necessarily destroy the parasite; alternate wetting and drying or freezing and thawing is necessary for germination of some fungal resting spores. Often, as the vegetative parts of the host die, the parasite takes up its abode inside tubers or seeds. A single disease may have many possible sources of infection. For example, mosaic disease of greenhouse tomatoes may come from other plants already in the house, from weeds outside it, or from the tobacco chewed or smoked by the gardener.

Where the same crop is grown in succession soil infection is often the most important source of disease. Decaying remnants of diseased plants often fill the soil with spores and resting bodies of various kinds which are very long-lived and hard to destroy. The fungus that causes black scurf and foot rot and also the honey toadstool that attacks trees can themselves spread actively through soil and other root-infecting fungi spread along roots. The black scurf fungus can remain alive for 6–7 months in air-dry soil. In a composted soil the fungi of lettuce, downy mildew, potato blight, tomato leaf mould and spinach leaf spot lost their capacity to infect by the second year, but those of tomato canker and celery leaf spot were still active in the second year (K. Röder, 1941). Spores of the cabbage club root fungus are estimated to remain infective in soil for at least 4–5 years and those of potato wart disease for 12 years. When dust is blown from infected soil or water drains from it during floods the infective spores are transported with it, as they may also be on soil adhering to transplanted plants or on tools, boots, etc. Some infective resting-spores are not destroyed by passage through the intestines of domestic animals, so that their manure forms a possible source of infection. Infection in manure, however, often comes from decaying fodder and other refuse thrown on to the manure heap.

When a disease increases or decreases over a period of years without any obvious cause one must take into consideration the many factors that may influence the amount of infectious matter remaining in the soil. For

example, it may be increased by the presence of susceptible weeds between successive crops, as in club root of cabbages, etc., or it may be reduced by liming, cultivation or by competition with other soil organisms, harmless to the crop, which crowd out the parasite (antagonism).

Channels of infection may be short, as when cabbage is planted in soil infested with the club root fungus or when lettuce is sown where melons have left behind a crop of *Sclerotinia* sclerotia. In the latter instance the hyphae are able to grow from the sclerotium to the young lettuce. Rain splashes throw earth containing bacteria up on to young leaves and scatter the conidia of apple scab from leaves to fruit. Air currents lift bacteria and fungus spores and the wind may then carry them over long distances. Dust has been blown from the Sahara to Stege in Denmark in three days and there is evidence that other things come with it. Spore traps exposed from aeroplanes at a height of 10,000 feet have caught many mould spores as well as those of rust and smut fungi. Many of these have been shown to be still alive at that altitude.

Animals, especially insects, carry a great deal of infectious matter around with them; e.g., ants carry grey mould spores, flies and wasps carry *Monilia*, aphides carry the conidia of apple scab; a few insects perform this transport of fungi deliberately as when bark beetles plant their tiny fungus gardens in their galleries within the bark of trees. Insect carriers of viruses are discussed on p. 86.

Man, however, does more than all other animals to disseminate diseased plants. At least a couple of hundred diseases are known to be spread by infected seed. In the past timber containing Dutch elm disease, diseased fruit trees, pine trees with blister rust in their bark, gooseberry bushes with American gooseberry mildew and potatoes with wart disease have been sent from one continent to another. Spores of the cabbage club root fungus are introduced to seed beds with manure and the fungus is then scattered far and wide when slightly infected seedlings are sold. One has often to reckon with more than one such means of transporting a single disease.

Conditions of infection. Happily we know of many cases where seed or soil contains infectious matter but thanks to good growing conditions the plants escape infection. For infection to take place certain conditions must be fulfilled. Some of these depend on the hereditary constitution of the plant (see " immunity " and " power of resistance "), others on that of the parasite (see " physiologic races "). Conditions of cultivation may affect both the parasite and the host. These include especially the local climate, the weather during the particular season, and what we call the microclimate, i.e. the temperature and humidity in the immediate surroundings of the plant. Firing, watering, shading and ventilation are all decisive factors as regards the incidence of disease in our hothouses. Even out of doors the microclimate may be modified by providing shelter, thinning crops and encouraging air drainage.

The various kinds of powdery mildew generally produce their most infective conidia in a dry atmosphere. Most other fungi require moist air to produce their spores or other infective bodies in large numbers. Many actually require a drop of liquid water on the spot at which they are to penetrate the host plant. The importance of dew in this connection is often under-estimated. If one scratches the bark of trees with a knife in dry weather and in wet or during spring and autumn the difference in texture is very noticeable and one may fairly assume that the difference is equally apparent to the hyphae of fungi that attack bark. If all spores germinated as soon as their requirements for warmth, moisture, and oxygen were satisfied, the majority would do so in the wrong situations and therefore in vain. It has been proved, however, for anthracnose of bean and for *Monilia* that soluble salts and carbon dioxide excreted by the host stimulate the spores to grow.

Compactness of the soil, its moisture content, temperature, nutrient content and reaction (pH) are decisive factors in many cases.

Most fungi infect plants most readily in damp soil but some require much oxygen and are therefore most active in dry, light soils with many air spaces (example, potato scab).

A successful attack (infection) takes place when all the conditions necessary for infection have been fulfilled. After the act of penetration of the host has taken place a certain time elapses, sometimes short, sometimes long,

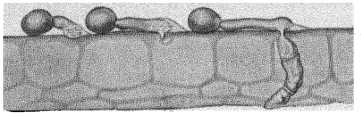


Fig. 49. Conidia of grey mould germinating on the surface of a tomato fruit. On left a conidium has germinated and formed a germ tube which has applied itself closely to the host cell. Centre, the germ tube has put out a peg-like protuberance penetrating the wall of the host cell. Right, penetration is complete and a hypha is growing into the tomato tissue. × 500 (after Blackmann and Welsford).

before the disease becomes apparent. This is called the incubation period. In practice the actual outbreak of the disease has usually to be dated from the moment when an expert can see the discoloration or other characteristic symptom with the naked eye or with a hand lens. A disease can sometimes be diagnosed earlier if a microscope is used.

In diseases of trees the incubation period may be very long. Thus in peach yellows it is two years. With the polypore fungi that attack the wood of tree trunks years may elapse before the appearance of fructifications (bracket fungi) or the stress of a sudden storm discloses the damage that has been done. In the case of bacterial leaf spots the incubation period may be only two days under very moist conditions or, more often, 7–8 days.

Even though the incubation period itself may not be unchangeable but varies somewhat according to temperature, vigour of the plant, etc., it is often important to be sure how much or how little we do know about it. In legal cases the question often arises whether a consignment of plants or produce was infected before it changed hands and whether the purchaser could have detected symptoms of disease at the time he took delivery. When attempting to check a disease it is important to know how soon after infection fresh spores or other infective bodies may be expected to be produced. For vine wilt an incubation calendar has been worked out which is very widely used, by which a vine grower can observe rainfall and temperature and then work out from a plotted curve which will be the last day for effective spraying.

A single rust spore from far away, carried on the wind, may start an outbreak of rust. More often, however, outbreaks arise from mass attacks by many spores at once. Attacks are often more successful when delivered by combined forces in this way, as has been demonstrated by inoculating seedlings with different known numbers of spores.

Symptoms. In general a disease is recognized by its symptoms but these must be examined as critically as possible before a pronouncement is made. In attacks by fungi microscopic examination of the spores is a valuable aid to diagnosis. When the fungus is not fructifying it can often be isolated in pure culture on artificial media. It may then fructify or the appearance of the culture may be characteristic of the species. Bacteria exhibit characteristic chemical reactions in pure culture.

Symptoms of disease are seldom entirely dependable. Thus in cucumbers mosaic-mottled leaves may be due to virus infection or to copper poisoning; dry brown patches on leaves may be due to hail, sunburn, chlorine poisoning, spray damage, deficiency of calcium or manganese, bacteria, fungi or insects. Diagnosis by symptoms may usefully be checked by observation of the progress of the disease, and its prevalence in the district, by microscopic examination, isolation of the parasite in pure culture and by soil or plant analysis.

Epidemics. The quantity of cluster cup rust on a barberry bush in May and June (see p. 74) depends on the number of teleutospores on cereals or grasses in the vicinity and on the weather. The bush cannot produce more cluster cups during the rest of the year. If spores from a cluster cup produced by the

oat race of the fungus fall on an oat leaf, the rust fungus produces its summer uredo spores there after 8–10 days which in turn will be able to infect another oat leaf. Eight days later there will be a second crop of summer spores, eight days after that a third crop and so on, as long as there is enough moisture and warmth and fresh green oat leaves are available. Thus the barberry becomes infected, whether lightly or heavily, only once each year, but an epidemic may be started on the oats and spread over the whole field, throughout the whole country and even across a continent. (Strictly the term " epidemic " applies to human diseases, from the Greek epi=upon, demos=people. The corresponding term for plant diseases is " epiphytotic," but for convenience the familiar word epidemic is commonly used.)

If then there is (1) a parasite able to infect and produce new crops of infective bodies several times during a short period, (2) a sufficient number of susceptible plants in a limited area and (3) favourable climatic and other environmental conditions, an epidemic may be expected. To a certain extent it is already possible to predict epidemics of plant diseases (see potato blight and apple scab) but usually there are too many unpredictable factors involved. Thus an importer or breeder may introduce a new host variety on which an epidemic breaks out before experience of its susceptibility has led to its rejection. The fungus may suddenly produce a new physiological race capable of attacking different host varieties or the latter may be weakened by infection with other parasites. On the other hand hyperparasites may destroy the fungus, changes in horticultural practice (manuring, watering, time of sowing) may make the host more resistant or enable it to escape infection. Burning of infected rubbish or destruction of susceptible wild plants may remove the sources of infection.

Attempts have been made to increase resistance of plants to parasites by feeding them with small quantities of certain chemicals (chemotherapy) but without great success. The effect of lithium in increasing resistance to powdery mildew has already been mentioned (p. 38) while lead and zinc make plants more sensitive to them. Claims have been made that boron manuring increases resistance to certain diseases but that is only the case when the plants were previously deficient in boron. Boron does not increase the resistance of normal plants to disease.

RESISTANCE AND IMMUNITY TO PLANT DISEASES

In a particular garden cabbages may be attacked by club root but adjacent rows of beetroot will remain completely free from that disease. Nor do they induce the club root fungus to attack them; this fungus and beetroot are completely indifferent to one another. Where a parasite is able to attack a plant but the latter is able to exclude it no disease is produced, the plant is resistant to the disease caused by that particular fungus. In some cases the parasite is actually able to enter the tissues of the host but their resistance to it is sufficient to prevent any appreciable damage. Resistance to disease may depend on many factors, many of which are still very imperfectly understood. It may, however, be useful to summarize what is known about them. Resistance to physiological disorders necessarily depends on quite different factors from resistance to parasites but the same terminology is in use for both types of " resistance." In physiological disorders, especially nutritional deficiencies, the same degree of apparent " resistance " may be due to quite different causes in different plants. For example, of two species neither of which is prone to suffer from manganese deficiency the first may have an unusually high capacity for absorbing the little manganese available to it, the other may have an unusually low manganese requirement.

Age and environment. The stage of development or age of the plant often decides whether or not it is susceptible to a parasite. Thus, damping off occurs in young plants, while many bracket fungi (polypores) attack only old trees. But within the susceptible age class environmental conditions, especially nutrition, soil reaction, light, temperature and humidity,

decide, for example, whether the seedlings are to be hard or soft, or slow or quick growing, and whether maturity and old age set in early or late. Excessive supplies of nitrogen encourage many diseases but check others. The effect of high temperatures in increasing resistance of the tomato plant to *Verticillium* wilt is well known. The germination capacity of wheat and barley seed and its liability to suffer from seedling blight, caused by *Gibberella saubinettii*, may be affected by its protein content, which depends on the condition under which the seed crop was grown. Low protein content is correlated with a high degree of resistance (Bayles, 1936).

Inherited resistance. Many attempts have been made to discover what it is in the constitution of individual plants or varieties that renders them resistant to particular parasites, under conditions where other varieties become diseased. The results have shown that this kind of constitutional resistance may be of many kinds.

Passive resistance. By this we mean the condition in which the parasite is unable to establish itself at all in the host tissues. This may be due to mechanical or chemical causes. Mechanical resistance to infection may lie in the construction of the epidermal cells, especially when they are covered with a thick layer of wax. The restriction of potato blight to the fruit in tomato plants, though it readily invades leaves and haulms of the potato, may be due to differences in construction of the epidermal cells in the two plants. Often, however, when resistance to infection has at first been ascribed to mechanical causes closer investigation has shown that chemical factors under the control of the living cells are in fact involved. Red-scaled varieties of onion are never attacked by *Colletotrichum*, though white-scaled varieties are susceptible to it. Careful research by Walker *et al.* (1929) has shown that the resistance is due to a water-soluble phenolic compound, protocatechuic acid, which is a molecular constitutent of the colouring matter quercetin found in the red scales. Other types of resistance may depend on the absence of some substances essential for the life of the parasite. Hollyhock rust attacks the common green form of *Lavatera arborea* but not the variety with variegated leaves, unless a shoot of the latter is grafted on to the green-leaved form. The interaction of scion and stock is not always to the same effect. Susceptibility of rose stocks to crown gall is affected by the kind of scion grafted on to them, but in vines stocks of the American kinds resistant to *Phylloxera* remain resistant even if scions of the susceptible European kinds are grafted on to them. In apples resistant stocks may increase the resistance of the scions to apple scab. Tomato is susceptible to leaf mould (*Cladosporium fulvum*) and potato is not, but even when a tomato scion is flourishing on a potato stock it remains completely susceptible to infection by leaf mould.

Active resistance, in which a parasite gains entrance to a plant but its spread through the tissues is checked in the early stages, is very general. When certain fungi and bacteria attack a plum-tree leaf it reacts by laying down a ring of cork cells round the point of attack. The patch of tissue within the ring dies and ultimately drops out, giving the well-known symptom called " shot-hole disease." In other cases the cells secrete rubber, resins, latex or anthocyanin in attempts to localize the invasion. According to Dufrenoy (1936) in many resistant plants, when a few cells have been killed by the invasion of a fungus the surrounding ones have their contents broken down into poisonous phenolic compounds which render them unsuitable for growth of the invading hyphae. This happens in various leaf-spot diseases in the so-called hypersensitive type of resistance to rust fungi. In the latter case the rust spore germinates, the hypha enters the host, often through a stoma, the cells adjacent to it are killed, and it is unable to proceed further. Hence in practice the plant is resistant to the rust though a few minute yellow or brown specks of dead tissue (sub-infections) may be observed on it. In potato varieties immune to wart disease the fungus enters the tissues but after 48 hours its growth ceases.

True humoral immunity such as is found in men and animals that have survived a

disease, and whose blood contains antibodies protecting them against reinfection by it, does not occur in plants. In the case of virus diseases, however, it has been found possible to prevent infection of a plant with a destructive strain of the virus by inoculating it beforehand with a mild, harmless strain of the same virus. This has been done especially with mild strains of *Solanum* Virus 1 but the discovery has not so far proved of practical value. From pelargonium attacked by crown gall there has been obtained a substance (bacteriophage) that destroys the crown gall bacteria (Chester, 1933). Bacteriophage, however, appears to be an infectious principle analogous to a virus and not to the antibodies produced in the blood of animals.

Acquired resistance and tolerance. The degree of resistance shown by a particular variety to a specific parasite is not necessarily fixed and unalterable. As regards potato scab, each potato variety shows a characteristic degree of resistance but their relative resistance is not the same at all times and places. This is probably because the degree of resistance depends on a number of factors, including:

(1) The structure of the lenticels.
(2) The thickness of the cork layer in the skin of the tuber.
(3) The capacity for regeneration of the cork layer.
(4) Possibly properties of the living protoplasm.

One or several of these may be predominant in a particular variety and some, at least, may be modified by external conditions of growth.

The poisonous grass darnel (*Lolium temulentum*), a rare cornfield weed in Britain, is almost always completely permeated by a fungus, though it shows no sign of disease and it is impossible to distinguish infected from non-infected plants without microscopic examination. This type of resistance, where the parasite is allowed to develop freely without the host either suffering injury or gaining apparent benefit from its presence, is known as " tolerance." A somewhat similar condition is found in the valuable pasture plant perennial rye grass.

Tolerance is not far removed from the type of association of fungus and flowering plant found in orchids and forest trees where each partner is believed to derive some benefit from the association (symbiosis). In associations of this kind, however, the fungus is usually restricted to particular organs of the plant, especially to specialized roots (mycorrhizas). A similar relationship between flowering plant and bacteria is seen in the root nodules of members of the pea family. Orchid seed cannot germinate unless the appropriate fungus is present, or, in artificial culture, unless it is supplied with sugars. If, however, the fungus becomes too luxuriant it may destroy the seedlings. In mycorrhizal symbiosis, then, we may envisage a perpetual struggle between the two partners, neither of which gains the victory under normal circumstances.

Hypersensitivity. The type of active resistance in which a parasite is checked by death of the surrounding cells has already been mentioned. In other cases death of the entire plant soon after infection may be advantageous to the host species as a whole by preventing the perpetuation of diseased stock. When *Solanum* Virus 1 is inoculated to certain potato varieties such as Epicure and King Edward it kills them within a few days by a disease called top necrosis. These plants therefore cannot produce tubers which would carry the virus over to the next year. The individual perishes but the health of the community is saved.

In some cases we can scarcely speak of resistance; plants merely avoid infection in various ways, especially by their susceptible phase of growth not coinciding with an active stage in the parasite's life. Thus needle rust (*Chrysomyxa*) of spruce can only infect the early shoots (Ferdinandsen and Jørgensen, 1938). Early potato varieties escape blight by being mature and dug before it begins to spread.

The Role of Plant Breeders

If resistance to disease is an inherited character in plants then in nature those that are resistant will survive and spread at the expense of the non-resistant. Clear proof that disease resistance is inherited was first given by Prof. Biffen at Cambridge in 1905, but the selection of resistant types had been a horticultural practice much earlier than this.

Researches into heredity have shown that disease resistance may depend on one or more inherited factors (genes) which may be either dominant over, or recessive to, susceptibility. The hereditary constitution has to be worked out separately for each disease. Thus in the case of *Fusarium* wilt peas are found to be definitely either resistant or not, whereas in tomatoes several grades of resistance are found, and among cabbages both types of reaction are found.

Physiological races of the parasites (see p. 59) make the plant breeder's work still more complicated. One variety of bean will be completely resistant to race A of the anthracnose fungus, fairly resistant to race B, very sensitive to race C and so on. Another variety will be sensitive to A but resistant to B. At least 30 physiological races of the anthracnose fungus are known to exist and, even when resistance to all these has been secured in a single variety, it is desirable to add also resistance to bean rust, bacterial blight, *Sclerotinia*, and so on. It is, therefore, unreasonable to expect that plant breeders shall in a few years be able to produce plants resistant to all possible species and races of parasites, while preserving or even improving the commercial quality of the crop. In the case of important crops, breeding should be undertaken in each country to produce varieties resistant to the physiological races present locally. As an ideal, varieties resistant to many physiological races (total resistance) are naturally to be preferred to those resistant only to a few physiological races (specialized resistance). As fungi are constantly producing new physiological races the plant breeder's work can probably never be completed. It has subsequently been shown that in the German famine winter 1916–17 a new race of wheat rust had made its appearance for which the breeders were not ready.

The breeder's first step usually consists of selection of the most resistant forms from the varieties already in existence. By crossing it is then possible to combine disease resistance with other desirable qualities such as quality and high yield. If disease resistance depends on one dominant gene results are quickly obtainable. If many genes are involved and many grades of resistance are found a prolonged period of crossing, testing and selection will be called for, but it may be possible in time to obtain offspring more resistant than either parent.

Breeders and plant pathologists must work together to obtain the best results, not by protecting the experimental plants but by exposing them to the greatest possible amount of infection. The degree of resistance is not always the same in seedlings and in mature plants, but usually it is possible to eliminate all susceptible types by exposing the seedlings to infection. In tests of this kind it is important to ensure that environmental conditions are the most favourable to the parasite. Thus, if cabbage seed is sown in soil permeated by *Fusarium conglutinans* and kept at a temperature of 72° F the fungus kills off the most susceptible plants. If the temperature is raised to 79° F. it kills still more until only the most resistant survive. In this way it has been possible to select and introduce into commerce the most resistant cabbage varieties.

4 THE ECONOMIC IMPORTANCE OF PLANT DISEASES

The annual value of Danish garden and nursery products has been estimated at 80–90 million Kroner. Such a valuation, based on market and auction prices, can only be a rough guess because a great many such products never go to market or auction at all. Moreover the yields of all crops vary greatly from year to year and prices fluctuate still more, partly because the demand for a product varies from time to time and partly because the value of home-grown produce depends partly on the degree to which it is replaceable by imported produce; for example, an abundant supply of bananas or oranges may reduce the demand for apples and hence the money value of the apple crop.

Attempts to express the annual loss from plant diseases in terms of money are still more unreliable than estimates of the total money value of production. A local outbreak ruining a crop in one district may lead to a rise in its price in the unaffected districts so that the overall money value of the crop in the country as a whole remains unchanged. Destruction of outdoor crops may enhance the value of glasshouse produce. Hence a loss of about 10% of the yield of a crop may not be tantamount to a loss of 10% of the receipts, and estimates of loss on a basis of the cash lost to the growers may largely conceal the real damage in reduction of yield due to a disease.

As far as Denmark is concerned the best-documented loss from a plant disease is probably due to potato blight. From the results of over 1,400 spraying experiments carried out in all parts of the country over a great many years it has been found that on an average about 15% of each year's crop is lost by failure to spray the potato crop. At present about one-third of Denmark's potato crop is sprayed, either once or twice. The following estimate of costs is based on pre-war values when the value of 1 Kg. of potatoes in the ground was estimated at 3 Kr. and the cost of spraying 1 hectare once (including the price of 20 Kg. copper sulphate) was 24 Kr. and spraying twice 48 Kr., i.e. an average spraying cost for the country of 36 Kr. per hectare.

PER HECTARE (=2.47 *acres*)	*Not sprayed*	*Sprayed*
Yield of tubers in 100 Kg.	166	196
Yield increase from spraying	—	30
Value of crop in Kroner	498	588
Increased value of sprayed crop in Kroner	—	90
Cost of spraying in Kroner	—	36
Net profit from spraying in Kroner	—	54

The yields quoted in the above table correspond to 6.6 tons from the unsprayed crop and 7.8 tons from the sprayed crop per acre, an increase from spraying of 24 cwt. per acre.

In 1938 Denmark's total potato crop covered 79,200 hectares, the total yield 13.9 million 100 Kg. bags, giving an average yield of 17,600 Kg. per hectare (about 7 tons per acre). On this basis, when, as usually happens, one-third of the Danish crop is sprayed there is a yield of 80 million Kg. more tubers than would be obtained if no spraying were done, while if the whole crop were sprayed the increase in yield would be 240 million Kg. The corresponding money values after the cost of spraying has been deducted are 1,400,000 Kr. and 4,200,000 Kr. respectively. Moreover these are low estimates, for, even though the increased yield would perhaps tend to depress the price of potatoes, the spraying gives the following additional advantages:

(1) it increases the keeping quality of the tubers; (2) it leads to less grading and hence a higher average price for the crop; (3) better seed potatoes; (4) a higher percentage of starch and thus a better price for factory potatoes. As mentioned above, the calculations are based on the assumption that half the sprayed area receives one spray and the other half two sprays. Double spraying throughout would still further increase the average yield.

Similar losses from potato blight probably occur in most years in the South and West of England. The actual reduction in yield due to this disease depends largely, however, on the stage of growth reached by the plants when blight breaks out. In England it is generally considered that North of the Humber blight usually comes too late for the increased yield due to spraying to be economically worth while. In the North of England and in Scotland losses from blight occur rather among the stored crop and are to be controlled not by spraying the growing crop but by burning down the haulm before lifting.

Direct losses in value of crop in terms of money depend largely on the basis of estimation. Amongst those worked out on the soundest basis are the German calculations made up to 1935 and 1936. Fungus diseases, mostly scab and brown rot, were estimated to reduce the value of the German fruit crop by 10% or 40 million Reich Marks yearly. Diseases and pests together destroyed 20% of the vegetable crop to the value of about 70 million R.M.s yearly. Germany's entire annual agricultural and horticultural produce was valued at 13 to 15 thousand million R.M.s, of which pests and diseases were estimated to destroy about 15%, worth about 2 thousand million R.M.s.

Expense of plant protection. The most obvious expense is, of course, the cost of the chemicals used. Before the war Denmark imported about 1,200 tons of copper sulphate, most of which was used to fight plant diseases and weeds. About 500 tons of sulphur were also imported but no doubt much of this was used for other purposes. Fungicides and insecticides were also imported to the value of 1.5 million Kr. annually for agricultural and horticultural use.

Labour costs, especially for spraying, may also be very considerable and the implements employed are often expensive. Other expenses may also be incurred in the struggle against disease, as for example in growing virus-free seed potatoes.

In Denmark the state bears the expense of instruction in plant pathology and disease prevention. In 1938–9 a grant of 33,000 Kr. was made for dealing with infectious plant diseases. In the same year 90,000 Kr. was also allotted from the national exchequer for experiments in plant pathology of which 7,000 was earmarked for chemical control, but otherwise the experiment stations provide part of the expenses of control experiments themselves.

Indirect losses of many kinds are often also caused by plant diseases. Often the cultivation of valuable specialities has to be abandoned or fresh stocks introduced on to clean land at considerable expense. Presence of infected material may necessitate changes in practice, as for example in seed production where it may be necessary to keep the second year's growth well removed from the first. Diseases that develop or spread rapidly during transport and storage of plant products may necessitate very costly regrading. Quarantine regulations imposed by most countries in an endeavour to keep out plant diseases lead to increased expenses in connection with certificates of health and certificates of origin where export trade is concerned. Moreover the presence of a dangerous disease in a country may limit the field of its export trade in the crop concerned—for example, potato wart disease.

The economic importance of plant diseases can be greatly restricted by quick and effective action. This is especially so when a new disease is introduced into a country and there is still a chance of stamping it out.

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II

DISEASES OF TREE AND BUSH FRUIT

2 DISEASES OF TREE AND BUSH FRUIT

APPLE

Apple Scab (*Venturia inaequalis* (Cooke) Aderh. = *Fusicladium dendriticum* (Wallr.) Fuck.)

History. The apple scab fungus has been known for at least a century. Probably it was this that Fries described in Sweden in 1819 as *Spilocaea pomi*. It was certainly well described by Wallroth from Thuringia in 1833 as *Cladosporium dendriticum*. Fuckel in 1869 first distinguished apple scab (*Fusicladium dendriticum* (Wallr.) Fuck. from pear scab *F. pirinum* (Lib.) Fuck.

Rostrup described the disease in Denmark in 1875, and in subsequent years it was found to occur there fairly generally.

In 1894–7 Aderhold demonstrated the connection between the conidial stage of *F. dendriticum* and the perithecial ascospore stage *Venturia inaequalis* (Cooke) Aderh.

Symptoms. Apple scab may occur on flowers, leaves, fruit and twigs.

Infection of the flowers is usually first seen when it has developed sufficiently to make the flowers fade. Then larger or smaller blackish spots are found on the flower stalks. The mycelium of the fungus occurs within the discoloured tissue and conidia appear on the surface of the spots.

The attack on the leaves may vary somewhat in appearance, especially in the distinctness of the spots. Some of the leaves that first open are seen to have dark spots at their tips, having been infected by spores which landed on them at the green tip stage of the opening bud. Leaves attacked so early often fall while quite young and scabbed leaves in general tend to fall prematurely.

If infection occurs early the spots may appear all over the leaves and on both surfaces. If it takes place at many different points on the leaf at the same time a number of small spots arise that soon join up and cover the surface of the leaf with a dark, almost olive-green coating of mycelium and spores.

If only a relatively small number of spores falls on the leaf, spots are formed, but only those closest together amalgamate while the others remain separate. Under quite a low-power lens it is then easy to see that the mycelium on the surface of these spots forks repeatedly and that the edge of the spot consists of a fringe of fine radiating hyphae (Fig. 50). Often the surface of the leaf becomes arched in the centre of the spots on the upper leaf surface. Leaves with many such spots then look pock-marked from the under-side. On the upper surface the spots often appear whitish at the centre, for the fungus causes the cuticle to separate from the surface of the leaf, leaving a narrow cavity filled with air. The fungus seems to die out at the centre of such spots but at their outer edge there remains a dark

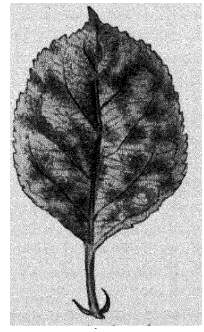


Fig. 50. Apple scab on upper surface of leaf.

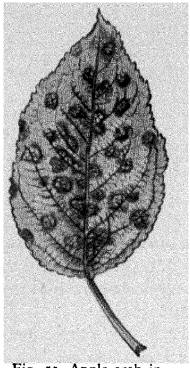


Fig. 51. Apple scab in the blister stage, upper surface of leaf.

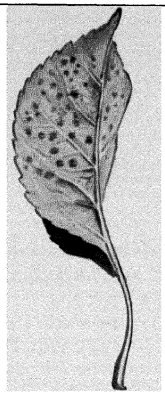


Fig. 52. Apple scab, small spots on under-side of leaf.

fringe of living mycelium growing steadily outwards (Fig. 51).

Spots with a raised whitish centre may also be found on the under-side of the leaves but are far more frequent on the upper surface, where the spots are, as a rule, very distinct. Spots on the under-side of leaves often have an ill-defined character and seem shallower than those on the upper surface; they seem to be masked by the covering of hairs.

Sometimes the spots on the upper surface are seen to be chiefly along the midrib. On leaves that occupy a horizontal position with one half curved upwards many small spots sometimes appear late in the season on the up-turned portion of the lower surface (Fig. 52). The conidia have fallen on to such leaves from infected branches above and have infected those parts of both surfaces which were more exposed to them.

Often the spots are far more numerous on the upper surface than on the under-side of the leaf, but the opposite condition may occur. Even in autumn we have seen trees the leaves of which bore many small spots on their lower surface while few had them on the upper. The latter were especially leaves at the tips of long shoots. We think the reason why the under-sides of the leaves were infected in those instances may have been that infection occurred in periods of little rainfall but heavy dew. The dew dried quickly from the smooth upper surface whereas the hairy under-surface remained moist long enough for infection to take place.

Keitt and Jones (1926) have found that the upper surface of apple leaves becomes immune to infection before the lower surface and that the spots that arose after the leaves had acquired a considerable degree of resistance, especially those on the under-side, were of the less well-defined type. Wilson (1928) has noted that by far the greater number of spots that appear in the later part of the growing season belong to this ill-defined type on the under-side of the leaf. According to Folsom (1926) when the leaves are fully developed not only are they less susceptible to infection but conidial production on the spots is reduced. Hockey (1929) finds the leaves are more susceptible just before and during flowering than at any other time.

The fungus continues to live in the fallen leaves and during the winter it forms in their tissues numerous small blackish perithecia, scarcely visible to the naked eye.

Apple scab spots on the fruit, like those on the leaves, may be of various kinds. If infection takes place early large parts of the surface are covered by an olive-green or nearly black coating of mycelium and conidia. The attacked parts grow very slowly, and therefore if one side of the fruit is attacked and not the other,

the fruit becomes irregular and malformed. Because the diseased skin cannot keep pace with the growth of the underlying flesh the infected areas usually crack (Fig. 53 and Plate 7, Fig. E).

Johnstone (1931) found that fruit with a waxy surface are most easily infected at the eye or on the under-side, where drops of water collect. Fruit with a downy or mat surface becomes infected all over.



Fig. 53. Apple scab on the fruit of Gul Richard. The deeply cracked scabs on the left open the way for *Sclerotinia fructigena* and other fruit-rotting fungi.

While the spots are young the remnants of the skin through which the fungus has burst can still be seen lying on the dark mycelium. In older spots this upraised skin only remains at the margin, while the centre of the spots has often become covered with a layer of cork. In these old spots the fungus has often died out at the centre but is still living at the edges where it continues growing outwards so that the spots slowly become bigger (Fig. 54).

In autumn an apple often bears one large spot and numerous small ones. The big one is due to infection in early summer while the small spots are due to infection in later summer when the fruit has been wetted by rain or dew, which often does not dry up till late in the day in August or September. Prevention of these numerous small spots may call for spraying or dusting quite late in the year.

Scab spots may spread considerably during storage and some—storage scabs—may even appear which were not visible when the fruit was picked. These are circular, saucer-shaped, black and shining, and are due to late infection just before the fruit was picked.

The attack on the shoots begins in summer as a dark fungus coating on still unripe shoots into which the fungus gradually grows. When the shoots have finished growing the attacked parts are found to bear a number of small, round, raised blisters in and under which the fungus is to be found. The blisters may burst the same autumn but usually do not do so until the following spring when mycelium and conidia appear as a black layer lining the cracks (Fig. 55).

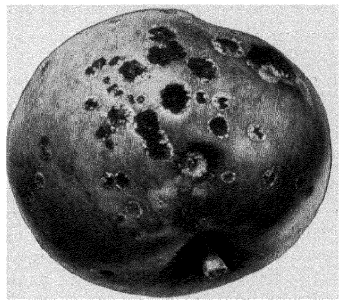


Fig. 54. Apple scab on Bismarck fruit. Note loose cuticle over the edge of the spots.

Apple varieties differ considerably in the susceptibility of their shoots to scab infection. Cox's Orange Pippin and Graastener are particularly susceptible while Bismarck is seldom attacked on the twigs, though its fruit is very susceptible to scab.

Associated with the blisters on young twigs are the bud scale infections discovered by

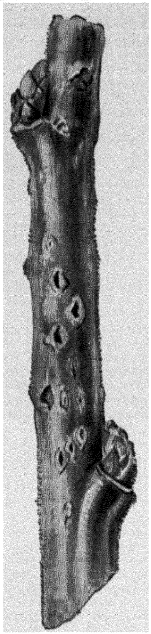


Fig. 55. Apple scab pustules on the twig (× 3).

Salmon and Ware (1931). Here, too, blisters appear containing mycelium and conidia below the ruptured epidermis. These lesions may be a very important source of infection for the opening leaves when the buds are bursting. Infected bud scales remain attached to the twig for quite a long time.

Life history of the fungus. Perithecia of the apple scab fungus form on the fallen infected leaves during winter and ascospores are shot out of them in the following spring. This is the perfect stage from which the name *Venturia inaequalis* is derived. On all the other centres of infection only conidia of the *Fusicladium dendriticum* stage of the fungus are found.

In addition to the fallen leaves the fungus overwinters in blisters on the twigs and bud scales, though lesions on bud scales have not yet been seen in Denmark. This question of the means of overwintering is of the greatest practical importance in connection with the outbreaks of scab in the following year and has been much investigated. We, too, have worked on this problem.

When an ascospore or conidium of the scab fungus falls on an apple leaf it germinates if sufficient moisture is present. Opinions differ regarding the precise method by which entry is gained to the leaf. According to Young (1927) the germinating hypha may enter at any point; it need not find a crack in the cuticle first. Other workers maintain that a small crack in the cuticle is necessary before the hypha can enter, but these cracks are generally plentiful. As long as the leaf remains green the mycelium is restricted to a layer just under the epidermal cells and produces numerous conidia on their outer surface. Not until the leaves have died and fallen does the mycelium penetrate deeper into the tissues where during winter it produces the perithecia and asci (Fig. 56). The perithecia (Whetzel, 1916; Wilson, 1928) are formed just under the epidermis, through which they break when ripe. They are formed under either surface of the leaf, mostly under whichever happens to be uppermost as the leaf lies on the ground. In complete darkness they are abnormal. They seldom form more than one-third of an inch from the leaf spots present in autumn and usually develop just below the edge in spots of the well-defined type. They may be formed under any part of the ill-defined spots. It

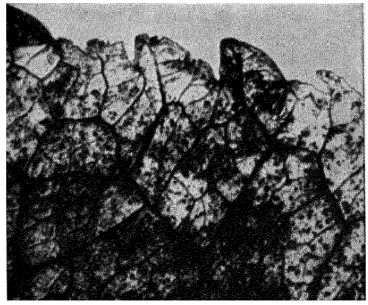


Fig. 56. Overwintered apple leaf infected with scab. The black dots are perithecia (× 10).

seems as though late infections which have borne few conidia are most able to form perithecia. Infected leaves do not infect other fallen leaves during the winter. Wiesmann (1934) states that perithecia are most abundant after early leaf fall and that their numbers differ on different varieties of apple.

They may be extraordinarily numerous. On 0.15 of a square inch (1 cm.²) of the upper surface of an apple leaf in Denmark we have found 150 perithecia, each containing 60–70 asci which in turn each have eight spores. Therefore in that small area of leaf there were about 80,000 spores, yet it was not very heavily infected.

From a similar area Wallace (1913) in New York State has noted that 5,630 spores were shot out in the course of 45 minutes, and it must be remembered that this ejection of spores may go on over a very long period. According to Wallace, once a perithecium has begun to discharge its spores it will be emptied in 24 hours or less if kept damp, so that new perithecia must be constantly ripening. In air analysis in Ohio, Keitt and Jones (1925) found 7,000 ascospores per cubic metre (about $1\frac{1}{3}$ cubic yards) about a fortnight before the apple trees blossomed.

This enormous production of ascospores on overwintered scabby leaves makes it important to destroy these leaves before the spring. Wiesmann (1935), by exposing spore traps in the orchard, has found that burying the leaves over quite a small area round the traps reduced the number of ascospores in the air to $\frac{1}{5}$.

The time the perithecia ripens depends on a number of factors. There seems to be a difference in time of ripening in different apple varieties, and Keitt and Wilson (1926) considered that ascospores ripened earlier in leaves that fell early in autumn than in those that fell late. When the perithecia are ripe the spores, which are quite light, are shot as much as $\frac{1}{4}$ inch into the air, and if there is any wind blowing they may be carried far afield.

In most cases the perithecia begin to ripen in spring when the buds begin to open and show green at the tip, but ejection of spores is most abundant a little later, depending mainly on the weather. For scab control it is important to know when the first heavy discharge of spores from ripe perithecia is to be expected, for the tree should then be protected by a spray or dust. It is known that once the perithecia are ripe the spores will be shot out as soon as the leaves bearing them are wet enough; dew, or even a light shower, is insufficient (Pierstorff, 1932), if the leaves have become thoroughly dried for a week or more previously; they must be completely wetted right through.

Ripening of ascospores takes place rather slowly at temperatures below 52° F. or over 75° F.; temperatures from 61 to 72° F. are most favourable. Drought checks development of perithecia (Keitt and Jones, 1926). The fungus can remain alive for a long time in dry leaves, but its development stops even if kept warm. Too high a temperature also checks perithecial development, but once development is completed warmth encourages ripening of the ascospores (Winkelmann, 1937). According to Holz (1939) ripening of the ascospores in Altenland is closely related to temperature after 1st March. Temperatures earlier in the year are of little importance.

In our own experiments at Lyngby we found the same pieces of leaf, about 1.86 square inches, examined for a whole month, ejected their ascospores more powerfully every time they had been thoroughly dried and wetted again than when kept continuously damp. In practice this means that showery weather, when the showers are heavy enough to wet the leaves right through, with spring temperatures rather high for Denmark (61–72° F.), favours ripening of perithecia and ejection of their spores.

By analysing the atmosphere Frey and Keitt (1925) found that ejection of ascospores from ripe perithecia began soon after rain had begun to fall and continued while it rained as long as any ripe ascospores were left. Active spore discharge continued at least three hours and at most fifteen hours.

Wallace (1913) found that spores may be ejected five minutes after dry leaves have been soaked with water.

In U.S.A. the incubation period for ascospore infections has been estimated as

at least 7–8 days by Adams (1925) and Young (1927). Winkelmann and Holz (1936) in Europe found the optimum temperature is 66° F. and the incubation period is then 9–14 days for ascospores and 8–10 days for conidia. Temperature has a great effect on the rate of growth. Keitt and Jones found that the time a leaf has to be kept damp in order for an ascospore that has fallen on it to germinate and penetrate becomes shorter as the temperature rises, up to a certain limit. Thus at 32.8° F. the leaf must remain damp for 13–18 hours, at 48° F. for 9–11 hours, at 59° F. for 8½ hours, at 68° F. for 4–6 hours and at 78.8° F. for 8–10 hours The germinative capacity of the spores is reduced if they are wetted and then become dry. Once the germ tube has penetrated the epidermis of the leaf it is able to obtain enough moisture from the tissues for continued growth even if the outer surface becomes dry. Infections that have progressed as far as this usually proceed and form conidia. These data show how much moisture and warmth influence the progress of infection in spring. Short, damp, cold periods do not allow the fungus to gain entry into the leaves, but long, damp, cold periods do so and so does any damp, warm period, short as well as long. This agrees closely with practical experience that, like so many other fungus diseases, it is worst in damp, mild weather. In our climate outdoor temperatures are seldom too high for the fungus.

In U.S.A. a close watch is kept on development of the perithecia and when ripe ascospores are found in the fallen leaves an immediate spray warning is broadcast on the radio, with the weather forecast, so that fruit growers may hasten to apply a protective spray or dust before the expected ascospores are ejected.

Where rain can be predicted with certainty one or two days in advance this method is very valuable. In Ohio in 1925–6 the weather was predicted with certainty three days in advance throughout the critical period of ascospore discharge. In Denmark it is, unfortunately, impossible to tell three days ahead when rain is going to fall. It is therefore useless to undertake laborious annual studies on ripening of ascospores without this meteorological support. We did it for four years and found the first ripe perithecia at the green tip stage of the apple buds and the most violent ascospore discharge from overwintered leaves when rain came between the pink bud stage and actual blossoming. If the spring has been dry, spore discharge from fallen leaves may still take place six weeks after blossoming.

The second important means of overwintering is the infected shoots. Here the fungus lives deeper inside the plant tissue than on either the fruit or the living leaves. The mycelium occurs under blisters in the bark which mostly burst in spring at the same time as conidia are formed on the mycelium. The fungus is difficult to kill here for as long as the blisters are closed the mycelium cannot be reached by sprays and even after they have cracked open it is difficult for sprays to penetrate and reach the hyphae and new-formed spores. Even if the spray deposit penetrates into the blisters it cannot reach and kill all the mycelium. Yet a strong enough spray can damage the conidia that are formed and probably too some of the mycelium concerned, and this checks conidial production immediately after spraying.

Pear scab often kills the infected shoots but this is seldom the case with apple scab. Generally the scab fungus has died out in twigs more than two years old. Shoot infections vary greatly on different varieties and in different seasons and localities. The importance of shoot infections lies in the fact that conidia are to be found in the scab blisters from the beginning of spring and these conidia are so situated as to be easily carried, especially in wet weather, on to the leaves and flowers of the bursting buds on the same tree, but they are not dispersed far and wide like the ascospores. As Dillon Weston and Pethybridge (1933) have demonstrated, the young leaves may become infected by direct contact while the buds are bursting, with scab blisters immediately around the buds. Thus varietal susceptibility to infection of the bud scales may have great influence on the spread of scab during the summer.

Shoots may become infected during almost the entire growing period. Infection takes place only in the youngest parts of the shoot, for once a cork layer has developed under the skin the tissues cannot be attacked. Quick-growing shoots, like water shoots, take a long time to produce a cork layer and therefore remain susceptible for a long time.

The first scab spots seen on the green parts of the tree are derived from the fungus on overwintered leaves or twigs. Conidia then develop on the surface of these spots and during the summer they rapidly spread infection further if conditions are favourable. The incubation period may be 13–14 days, usually longer but occasionally shorter, so in wet summers we can reckon on 1–2 generations of the fungus per month.

According to Dillon Weston and Pethybridge and Marsh and Walker (1932) conidia spread most during wet weather. When the mycelium is dry the conidia adhere firmly to the conidiophores. They are only found in the air in rainy weather and especially when a strong wind accompanies the rain. Thus one can often see most scab blisters on the side of shoots exposed to the wind. Conidia are not spread as far as ascospores. Far fewer conidia were captured on spore traps set up only 1½ yards from trees with diseased shoots than on traps set right at the outer edge of the crown. Conidia may also adhere to insects, mites and other animals and be spread about in that way.

Conidia show little resistance to drying. They seem to germinate less regularly than ascospores. If either conidia or ascospores fall on a hairy surface this may hinder infection by keeping the spores away from direct contact with the epidermis. Even if there is enough moisture between the hairs to allow the spores to germinate the germ tube has to be very long to reach the tissues and is exposed to danger of drought before it gets so far.

Leaves and shoots are only susceptible to infection when young. If they remain very hairy until about ripe it is difficult for them to become infected. On the other hand leaves that quickly develop a smooth, shiny surface that dries quickly are very resistant to scab. In autumn we often see leaves severely attacked in the tips of shoots while the older leaves lower down are healthy. In the autumn conidia spread naturally from spots on diseased leaves or fruit to old as well as young leaves, but only the latter could become infected. How long leaves and shoots remain susceptible cannot be definitely stated; it depends on variety, weather conditions and nutrition. Moore (1930a), in Kent, has noted in manurial trials that far more scab occurred on unmanured than on manured plots. As a rule it can be stated that the quicker the various parts of a tree ripen the sooner they will become scab resistant. In damp, cold weather the young organs are delayed in development and, therefore, remain longer exposed to infection than in warm weather. Heavy rain checks infection, for spores and conidia are washed away before they have time to germinate and infect.

Apparently more conidia form on the scab spots early in summer than later on, and on spots on fairly young leaves as compared with those on older leaves. Conidia formed on young leaves germinate better than those from older spots.

Fruit may become infected at any time during the whole summer and up till the time of picking. In 1934 at several places in Denmark striking outbreaks of small scab spots appeared at picking time, at the end of October, on apples like Bismarck, leaves of which were very little attacked. The summer had been dry but September and especially October were wet. It seemed as though there was a marked spread of conidia from scab spots formed earlier in the year, but by that time the leaves were too mature to become infected.

Importance. Apple scab is of immense economic importance, partly because it is prevalent nearly every year and partly because it can be injurious in so many different ways.

When one consults the surveys of horticultural plant diseases published annually since 1916 by the Danish State Plant Pathological Experiment Station one finds that during the first 28 years apple scab was mentioned as mild only in 1917, 1921, 1925, 1929, 1933 and 1941. In all other years it is recorded as from fairly

serious to exceedingly virulent. That is, its incidence has only been below normal once in four years, actually rather regularly every fourth year, and during the remaining years it was of great economic importance. According to this cycle 1937 should have been a mild scab year but in fact it was one of severe scab. No such regular rhythm in scab incidence can be deduced from the English records. According to Pethybridge (1934) 1931–32 were bad scab years, while Moore, W. C. (1943, 1948), recorded the disease as below normal in 1933–4, 1940, 1942–3 and 1946, and above normal in 1937 and 1941.

Local differences in climate both in Denmark and in Britain are responsible for marked differences in intensity of scab attack in different parts of the country. In some years the disease is most conspicuous early in the season; in others, as in 1938 in England, it develops later on.

This is, then, a fungus so general in its occurrence that apple growers must always be on their guard against it. It is impossible to assess accurately the amount of damage it does. Fruit growers consider mainly the direct damage to the fruit and realize how great this may be, but it does far more damage than that.

The fungus is injurious in the following ways: (1) It reduces fruit set by attacking the flower stalks. (2) It reduces the yield of marketable fruit because apples attacked early do not attain their full size and may drop prematurely. (3) It reduces the value of the fruit partly by spoiling its appearance and (4) partly by impairing its keeping quality. Many fungi that cause rots of stored apples gain entrance partly or mainly through scab, e.g. brown rot, pink mould, blue mould and many minor rots. In the storage experiments at Blangstedgaard, 1912–22, it was demonstrated in many cases that in consignments attacked by scab there remained only half as many sound apples as in consignments of scab-free fruit of the same variety. The results were the same whether the apples were stored in cellars, fruit stores or cold stores. (5) It attacks the shoots, in virulent cases killing them outright and always leaving small wounds open to infection by the canker fungus. (6) Finally it attacks the leaves. The damage to them may be serious enough to make the trees unthrifty. Carbon assimilation by the leaves is reduced because the diseased areas either do not assimilate or do so with difficulty. Moreover the diseased leaves are often smaller than healthy ones and tend to fall early. As a result of the decreased assimilation the shoots often fail to ripen properly in autumn, are destroyed by frost the following winter and form courts of entry for the canker fungus. Trees severely attacked by scab every year are nearly always heavily cankered as well (see also p. 113).

Varietal susceptibility. It is difficult to make dependable statements about varietal susceptibility because the information available comes mainly from casual observations at different times and places, and not from strictly comparable observations on one experimental area. However, when we find a variety consistently reported as susceptible from different districts over a series of years, it is fair to conclude that it is so. Relative susceptibility is much more difficult to establish.

The problem of varietal susceptibility is further complicated by the existence of different physiologic races of the apple scab fungus, as demonstrated by Palmiter in U.S.A. (1934), Wiesmann (1931) in Switzerland and Rudolff and Schmidt (1934, 1935) in Germany. These races have been differentiated partly by their differing capacities for infection and partly by their appearance and rate of growth in pure culture on artificial media. Inoculation experiments with conidia of different races have shown that conidia from a particular apple variety infect leaves of that variety much more readily than leaves of other varieties. The latter are, however, infected and to varying degrees. In one experiment White Transparent was shown to be completely resistant to conidia of 7 out of 11 cultures tested, originally isolated from 11 different apple varieties, but it was invariably severely infected by the 4 remaining cultures. These 4 races, however, could not infect all other varieties.

Races of the apple scab fungus were found to differ in the size of their conidia, rate of

growth and optimum acidity for growth, which suggests that some races may attack mainly the sweet varieties.

In experiments at East Malling, described by M. H. Moore (1936), it appeared that the apple stocks may affect susceptibility of the scion to scab. Attack on fruit, leaves and shoots was not completely studied but on the whole the attack was least severe on trees worked on East Malling stocks III, IV, XIII and XV and most severe on I, IX, and XVI, though varieties differed somewhat in reaction on the different stocks. The influence of the stock on scab seemed to be affected somewhat by the weather. Soil conditions, too, may possibly affect varietal susceptibility to scab. In manurial experiments scab was worst on the unmanured trees.

In published statements regarding susceptibility to scab it is often impossible to tell whether scab on leaves or fruit or on both is meant. As the fruit scabs are the most conspicuous, statements generally refer to them, but it appears that there is usually a close correspondence between susceptibility of leaves and fruit. The position is different with attacks on the shoots; a variety may be susceptible to scab on leaves and fruit but have resistant shoots, e.g. Bismarck. The scab lesions on shoots are so inconspicuous that unless they are specifically mentioned it is safe to assume they have not been taken into account in assessing varietal susceptibility.

No apple variety is immune from scab. Those cited below as fairly resistant may all be severely attacked if weather conditions are particularly favourable to the fungus and opportunities for infection are plentiful.

FAIRLY RESISTANT

Belle de Boskoop	Koldemosegaard
Bodil Neergaard	Reinet
Bramley's Seedling	Lane's Prince Albert
Coulon	Pederstrup
Filippa	Skovfogedæble
Guldborg	White Transparent
Høve Reinette	Tyrrestrup

SUSCEPTIBLE

Beauty of Kent	Hawthornden
Bismarck	Husmoder
Boiken	Ildrød Pigeon
Cellini	Keswick Codlin
Charlamowsky	Laxton's Superb
Cox's Orange Pippin	Lord Suffield
Cox's Pomona	Maglemer
Graastener	Mølleskov
Yellow Richard	Signe Tillisch

Cooke (1943) found the following varieties to suffer little from scab on the fruit at Shinwell, Berkshire: Barnock Beauty, Byford Wonder, Cairn Pippin, Cellini Pippin, Charles Ross, Chelmsford Wonder, Ecklenville Seedling, Encore, Emperor Alexander, Fearn's Pippin, George Carpenter, Golden Noble, Herring's Pippin, Houblon, Mère de Ménage, Monarch, Norfolk Beauty, Queen Caroline, Reinette du Canada, Ribston Pippin, Rival, Warner's King and Wealthy.

Cox's Orange Pippin, Graastener and Lord Suffield are particularly prone to be attacked on the shoots. Blisters appear on the young twigs of the two first-named while on Lord Suffield the bark is ruptured over comparatively large patches of twig and falls off in flakes.

Control. One should try to prevent ascospores and conidia of the scab fungus from landing on the green parts of the apple tree and try also to prevent their being able to penetrate the tissues if they do land there. One must, then, try to destroy all possible infective material which may carry the fungus over winter, and apply protective sprays or dusts to prevent infection of the plant tissues. One can also try to avoid very susceptible varieties, but as none are highly resistant to scab one should not usually allow this to be a prime consideration when deciding which varieties to grow.

Destruction of infective material means, principally, getting rid of fallen, scabby leaves. In Denmark this is usually done by digging or ploughing them into the soil. Care should be taken that no leaves survive the winter under rows of adjacent bush fruit or in hedgerows. Digging or ploughing in autumn should

not be undertaken until all the leaves have fallen.

Diseased shoot tips may also be sources of infection. This is best prevented by cutting out the affected twigs. It may be advisable to cut back every year those varieties most liable to scab lesions on the shoots, e.g. Cox's Orange Pippin.

A concentrated spray before the buds open may be of some service and for this purpose a 6% copper sulphate spray as the buds begin to break has been recommended. Such a practice cannot replace the regular spring spray programme which should be carried out in any case.

Copper and sulphur-containing fungicides are still the main protectants against apple scab, especially Bordeaux mixture, lime sulphur, proprietary sulphur preparations and sulphur dusts. As these treatments are preventive they are best applied just before the trees are exposed to infection. Their efficacy is greatly reduced if they are not used until after the trees have been bombarded with ascospores and conidia. It is therefore important to spray or dust immediately before rain.

Usually it pays to do one's utmost to hinder the early infections, for if they are unchecked the disease may rapidly attain disastrous proportions in a favourable summer. Unfortunately it is quite impossible to make the early treatments so effective that the fungus is unable to spread at all during the summer. Hence one must always be ready to spray or dust against a late attack. In years when there has been a dry spring and summer, scab is often negligible during most of the season, but if a wet or even very dewy autumn follows, many small scab spots may appear, especially on the fruit.

The number of times it is necessary to spray or dust depends on: (1) how much infective material there is, (2) the weather, and (3) the varieties of apple grown.

In well-kept orchards where a rational disease programme has been followed for several years it may be so successful that hardly any scab spots can be found even on the ripe fruit. In such orchards, especially if the nearest scabby trees are some way off, the routine spray programme forms a kind of insurance against fresh outbreaks that may originate from the few lesions that may have been overlooked. In an orchard that has attained this degree of health one can maintain the trees in good condition with far fewer sprayings or dustings than when one is trying to clean up a neglected, infected orchard, where there is a large quantity of infective material left in spring, or where scabby trees exist in neighbouring gardens.

More treatments against scab will usually be necessary in wet seasons than in dry seasons.

If some varieties are found to be more scabby than others they should be sprayed more often that year, or at least in subsequent years, than the less susceptible varieties.

Under conditions particularly favourable to scab it is best to make two treatments between bud burst and blossoming. After the first spray new parts of leaves and buds emerge and should be protected by a second pre-blossom spray. That is, we should treat the trees the *first* time as soon as there is prospect of rain after the buds have burst, if it can be done before the rain comes, otherwise when the buds are in tight clusters (see p. 584). The *second* treatment should come when rain is again forecast or when the earliest varieties are about to bloom. When there are many trees to deal with the earliest varieties should be sprayed first. The standard English spray schedule includes three applications of lime sulphur or Bordeaux mixture, the first at the green cluster stage of the buds, second at pink bud stage and the third when 80% of the petals have fallen.

As all sprays, especially those after blossoming, are likely to cause some scorching of leaves and russetting of fruit, no more should be applied than are absolutely necessary for efficient scab control, but in wet seasons it may be necessary to spray susceptible varieties 5 or 6 times more.

Although it is always best to spray or dust before rain with its attendant release of infective spores, something can be achieved by treating the trees after ascospores and conidia

have landed on them and even after infection has begun. Hamilton's investigations (1931) showed that sprays, especially lime sulphur—lead arsenate, but also to some extent Bordeaux mixture, have an effect on scab even after the fungus has made considerable development. The sprays were more active when applied during or immediately after inoculation than when first applied some time later.

The sprays mentioned may also damage the conidia already formed on the spots and check the future production of conidia.

The usual spray recommended in England is lime sulphur 1:40 at green cluster, 1:60 at pink bud and 1:100 at petal fall and subsequently. M. H. Moore (1930b) used lime sulphur 1:30 for Cox's Orange Pippin before flowering and 1:100 after flowering. Lime sulphur is generally preferred to Bordeaux mixture because it is easier to prepare, gives some control of red spider and on most varieties causes less russetting of the fruit. Stirling Castle and Lane's Prince Albert are very susceptible to injury by lime sulphur and should receive it at the rate of 1:100, if at all, before blossoming and not at all after blossoming. For such varieties 8:12:100 Bordeaux mixture may be used. It may also be useful in clearing up scabby neglected trees, but copper sprays should not be used on sensitive varieties like Cox's Orange Pippin, Lord Derby or Worcester Pearmain. Lead arsenate is often added to the pre-blossom sprays to control caterpillars and nicotine to the pink bud and post-blossom sprays to control capsid and aphids. Goodwin *et al.* (1934) found that addition of $\frac{3}{4}\%$ cottonseed oil to a 4:6:100 Bordeaux spray reduced the tendency to scorching without impairing its fungicidal activity.

See also the spray programme on p. 584 and discussion of sprays on pp. 582 and 586.

Mildew (*Podosphaera leucotricha* (Ell. & Everh.) Salm.)

Symptoms and development. When the apple buds open some are delayed and when their leaves do emerge they are covered with a mealy white coating, generally first visible

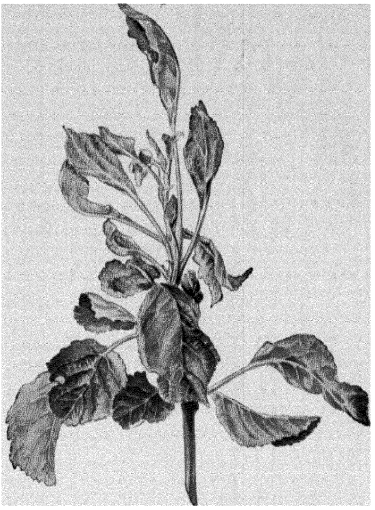


Fig. 57. Apple mildew. Small, inrolled apical leaves covered with a white coating of mildew.

on the under-surface. Soon all the leaves, petioles, and shoots emerging from the infected bud are covered with it, as well, eventually, as the flowers which are malformed with narrow thickened petals and set no fruit. The infected leaves are narrow with their edges rolled inwards and point upwards unlike normal leaves. Gradually they take on somewhat of a reddish or brownish hue and often fall prematurely (Fig. 57). These are the primary infections derived from diseased buds.

Secondary infections may develop on other leaves in the course of the summer. Occasionally the green fruit too may be infected, and be covered with a thin white mealy layer, which causes the skin to become rough and corky. In virulent attacks the fruit may crack. Mildewed apple fruit has not been seen in Denmark, but has been reported from Sweden (Eriksson, 1909) and Germany (Lüstner, 1923). It is reported occasionally in England. Mildew was seen on pear fruit from Fejø in 1937 and was seen again on pear trees there in 1939. The shoots were covered with a white, felt-

like, fungus layer that became somewhat discoloured (see Fig. 122).

The black perithecia of the fungus develop embedded in the white mycelium on the twigs. They occur in England and have been found on both apple and pear in Sweden (Hammarlund, 1942), but apparently are not general in Europe. In North America so many perithecia occur on the tips of the shoots as to form quite a dark coating (Fischer, 1920). In Europe the fungus overwinters principally as mycelium in the buds. The climatic requirements of apple mildew are a little obscure. It was very serious in Germany in the warm, dry summer of 1921, but also in the cold, damp summer of 1923 (Lüstner). In Denmark it seems to like warm conditions, for it is most prevalent on espaliers. In England it was exceptionally bad in 1938 when a warm, dry March and April were followed by a dull, wet summer (Moore, W. C., 1943).

Woodward (1927) gives 50–59° F. as the optimum temperature for germination of conidia, but 68–72° F. as best for hyphal growth. Berwith (1936), on the contrary, found the optimum temperature for germination of conidia lay between 66 and 72° F. while none germinated if the atmospheric humidity fell below 90%.

Infection takes place most easily on fairly young leaves and on summer shoots; the leaves quickly become immune. Under favourable conditions an infection becomes visible 48 hours after inoculation.

In inoculation experiments in England Woodward obtained infection on apple, pear and quince with the same fungus. It is also said to attack medlar and was observed on *Pyrus floribunda* at Long Ashton in April 1937 (Moore, W. C., 1943). Some apple varieties are more often attacked than others, notably Bismarck, Boiken, Cox's Orange Pippin, Graastener, Säfstaholm and, in especially warm summers, Bodil and Neergaard. Mildew attacks equally young trees in nurseries and old ones in orchards. In nursery trees Wormald and Harris (1937) found more severe attacks on East Malling stock XVI than on numbers XI, IX and XII.

Importance. The disease seems to be more serious in Germany than in Denmark. Lüstner thought it the most serious fungus disease of apples after apple scab.

In 1927 Jørstad wrote that apple mildew had become much worse during the past few years. In Sogn it was destructive on Graastener. In Denmark it was widespread in 1939 but has not been prevalent since.

Control. As overwintering takes place mainly in the bud, and especially in the terminal bud, trees that have been attacked should be severely pruned in winter so that all new shoots are topped. As soon as primary infections are seen in spring the diseased tips should be cut off and burnt. Often only a few survive the winter pruning, and it is quite possible to remove them all, whereas if they are left the disease may become really serious. W. C. Moore (1948) attributes a recrudescence of apple mildew in England since 1945 partly to a change in pruning practice, by which many current season's laterals are left unpruned. Infected buds that would be removed by the old practice of spur pruning are then left on the trees.

Winter sprays have no effect on apple mildew as they cannot reach the mycelium well hidden in the buds. Summer sprays or dusts are effective, however. Lime sulphur 2: 100 or sulphur dust seem to control its spread in Denmark and are recommended in other countries. Sprays cannot prevent the primary infections originating from diseased buds but they hinder establishment of secondary infections.

Spraying or dusting against apple mildew should be done before flowering and again just after, and if necessary at intervals during the summer.

In Norway Jørstad (1937) recommends a lime sulphur spray 2½: 100 with addition of a spreader, either 5 lb. of wheat flour or 1 of gelatine per 100 gallons. In severe outbreaks the first spray is required as the leaf buds open. One or two further sprays are required before flowering and another after flowering, but then only with lime sulphur 2: 100. If the first spray is not applied in time the mildew may gain the upper hand in spite of subsequent sprays.

In U.S.A. Stewart (1915) recommended spraying with lime sulphur 2: 100 plus 6 lb. of iron sulphate per 100 gallons. From Wageningen, Holland, comes a recommendation to spray with lime sulphur 4: 100 when the buds are well swollen.

Potash deficiency favours mildew so potash manuring may be necessary.

Canker (*Nectria galligena* Bres. = *Cylindrocarpon mali* (All.) Woll.)

Symptoms. The canker fungus causes wounds on trunks and branches of apple and pear, less often on other trees such as cherry, beech and willow, and may also cause eye rot of apples and pears. It never attacks the roots.

Apple canker starts at a wound or from a piece of dead tissue such as a bud, a spur, a dead pruning stub or a frosted shoot. It is first visible as a small, slightly sunken patch of bark distinguished from the surrounding healthy tissue by its brown colour and tendency to crack. As it grows it becomes elliptical, increasing lengthwise to the branch rather than laterally. On older branches a ring of callus forms round the wound in each growing season but the fungus breaks through this and spreads further. The canker then comes to be situated on a swollen piece of branch and surrounded by a number of more or less concentric ridges. It often remains restricted for years to one side of the branch which then functions more or less normally. On young branches, however, the canker often completely girdles the bark and the portion above the wound then dies (Fig. 58). In old cankers the dead central tissues crack and fall out exposing the wood. Closed cankers arise when the surrounding belt of callus grows sufficiently to meet over the wound. As the fungus penetrates further each year and a fresh layer of callus forms, the site of the canker comes to be occupied by an irregular swelling. On the Pacific coast of U.S.A. apple cankers do not become ringed by callus. This is perhaps due to their rapid growth; they may grow as much as 2 feet in a year there.

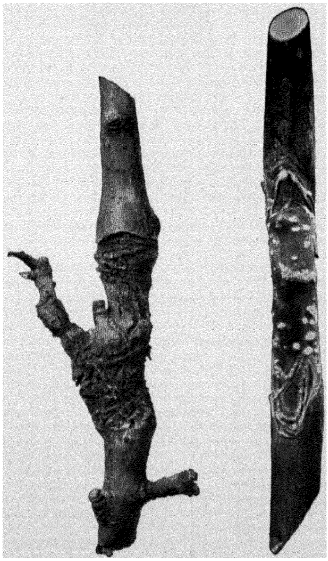


Fig. 58. Old canker on apple branch.

Fig. 59. Apple canker with conidial pustules.

Soon after the canker has become visible conidial pustules of the fungus may appear on it. This stage was formerly called *Cylindrocarpon mali*, *Fusarium mali* All. or *F. willkommii* Lind. (Fig. 59). The pustules are whitish or cream-coloured and become slimy with spores in wet weather. Older ridged and cracked cankers bear in their fissures the tiny, globular or egg-shaped, blood-red perithecia. These are about $\frac{1}{50}$ inch across and are easily confused with eggs of red spiders, which are commonly laid in cracks of the bark. The eggs are slightly smaller than the perithecia and not such a dark red.

Nectria galligena also causes eye rot of apple and pear fruit while still on the tree. The spots are sharply defined, brown and sunken, and occur mainly towards the tip of the fruit, usually starting at the eye. When infection occurs before the fruit has ceased to grow it assumes a characteristically flattened appearance because of the early death of the eye and surrounding tissue. On the brown spots there appear numerous whitish or pinkish conidial

pustules. Infected apples may become mummified and remain on the tree, in which case the red perithecia of the fungus may appear on them during the winter.

Leaf infection by the canker fungus was observed on pear trees in England in 1924. Conidial fructifications appeared on blackened lesions at the base of the leaf stalks, causing the leaves to wilt and die, and in the following February cankers were initiated round the leaf scars.

The conidia are responsible for spread of the fungus during the summer months. Ascospores are ejected from the perithecia all the year round, in England most freely in February and least in September (Marsh and Munson, 1939). They die after drying for about 5 days.

Development. The fungus cannot normally penetrate healthy uninjured tissue. Moritz (1930), however, succeeded in inducing it to do so when the branches were kept very moist so that the lenticels were abnormally developed and infection occurred through them.

In England, Wiltshire (1921, 1922) found that many infections took place in spring and autumn through tiny cracks at scars made by leaf fall. Small amounts of water often collect in the angles of the scars and assist germination of the conidia. During breaking of the buds in spring their growth may cause fresh cracks in the old leaf scar, through which infection can take place. It may also occur through scab lesions. Wiltshire (1922) found mycelium of the canker fungus in scab wounds even in the spring.

Often the stub of a branch is seen in the middle of the canker, often due to an imperfectly ripened shoot having been killed by frost during the winter. The fungus enters the killed twig and spreads thence into the main branch. Storm damage, hail, pruning wounds and insect injuries may all open a way for the canker fungus.

Nectria galligena is most abundant on apples but quite common also as a cause of cankers on pear. Rostrup found it on cherries (Lind, 1913) but it has not been found on plum. It is also a cause of canker on ash and may occur on beech, on which, however, an allied species, *Nectria ditissima* Tul., is more common.

Importance. Canker is a very serious disease of apples and pears and may kill susceptible varieties unless they receive protection against it. On the other hand cankers which

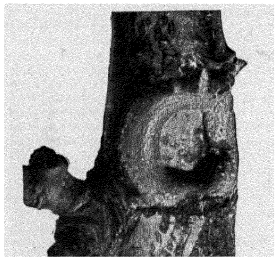


Fig. 60. Apple canker cut out and healed by callus formation.

Variety	*Yards of Dieback in Branch Tips*			*Number of Cankers*		
	Unsprayed	*Bordeaux*	*Lime Sulphur*	*Unsprayed*	*Bordeaux*	*Lime Sulphur*
Wealthy	2.7	0.1	5.7	10	1	10
Mølleskov	21.8	3.1	9.1	35	8	21
Lane's Prince Albert..	25.1	2.4	2.4	57	3	0
Elmelund	33.8	1.5	3.8	90	3	9
Bellefleur de France ..	50.1	1.7	18.5	100	7	50
Dynaes	185.3	5.4	35.9	290	17	78
MacIntosh	1210.0	127.2	579.9	870	160	370
Average	218.4	20.3	92.8	207	28	77

only partially girdle branches may increase their fruiting capacity in the same way as artificial ring barking.

Canker seems to be less prevalent in Denmark than formerly. Improvements in orchard management both as regards disease control and manurial treatment have reduced the number of wounds available as infection courts and led to better-ripened wood, so that fewer twigs have been killed by frost.

Eye rot of the fruit due to this fungus is seldom of great economic importance. In England Dillon Weston (1927a) recorded attack on about 25% of a crop of Worcester Pearmain, a particularly susceptible variety. In Denmark the disease has been noted chiefly on pears (Amanlis, Conseiller de la Cour, Count Moltke and Tongres).

Varietal susceptibility. Varieties differ greatly in susceptibility to canker, so much so that highly susceptible varieties may go out of cultivation on that account. However, it appears that in Denmark the disease can be kept under control on almost any variety by good cultivation and a rational disease control programme. Thus old varieties such as Cellini, once abandoned on account of susceptibility to canker and other diseases, are now being grown again. In private gardens trees are apt to be more neglected and varietal susceptibility becomes of more importance. The following notes on susceptibility of apple varieties may therefore be useful.

Apples

VERY SUSCEPTIBLE

Cellini	Ildrød Pigeon
Cox's Orange Pippin	MacIntosh
Cox's Pomona	Signe Tillisch
Flaskæble	White Transparent
Graastener	Winter Gold Pearmain
Hawthornden	

VERY RESISTANT

Bismarck	Høve Reinette
Bodil Neergaard	Koldemosegaard
Boiken	Reinette
Bøghs Citronæble	Manks Codlin
Casseler Reinette	Pederstrup
Dumelow	Red Pineapple
Filippa	Venus
Frogmore	

Many pear varieties are so resistant that the disease is of no importance to them under ordinary conditions. The most susceptible varieties commonly grown are: Amanlis, Bonne Louise, Clara Frijs, Autumn Bergamot and Kejserinde.

Control. Canker control is partly direct and partly by indirect methods. The former includes cutting out the cankers. Thin cankered branches are cut out altogether and burnt. On thick branches and trunks the cankers are cut out to remove all discoloured wood and bark and leave the edges of the wound smooth and clean. A chisel or gouge may be needed for this as well as a knife. The edges of the wound in the wood should be left absolutely perpendicular so that callus can easily grow over them (Fig. 60). Untreated wounds heal well but are open to reinfection. Hence all wounds should be treated with white lead paint, as recommended for silver leaf (p. 192) or asphalt paint. Grafting wax, coal tar and special canker preparations may also be used. In 1934 Appelts Canker mixture and Barkolit were tried at the experiment station at Lyngby with good results. Coal tar is in general use, often with good results, but it may stop callus formation, especially on stone fruits, where its use is not recommended. White lead paint seems to be coming more and more into favour for the purpose. All mummified apples should also be removed and burnt.

More important than direct control by cutting out the cankers is trying to prevent infection by the fungus. Everything that may hinder ripening of the wood should be avoided, e.g. drought, excessive or late nitrogenous manuring, damage to foliage by pests and diseases, caterpillars, red spiders and scab, also spray injury. Where growth is too vigorous grassing down the orchard may be helpful.

The effect of a disease control programme on canker may be illustrated by the results of some experiments. In 1929 a number of trees were removed from a spraying experiment at Blangstedgaard, where unsprayed trees had been compared with others sprayed with either Bordeaux mixture or lime sulphur, supplemented by lead arsenate and nicotine. The

opportunity was taken to measure the length of dieback and to count the number of cankers per 100 Kg. (about 45 lb.) of branch (see table on p. 114).

Although the effect of the spraying varied somewhat it gave excellent control of canker on all varieties, especially Bordeaux mixture, which also gave the best control of scab on the leaves. There is no direct relationship between susceptibility to scab and susceptibility to canker but heavy scab infection increases the opportunities for canker infection. Control of insect pests also probably played a part in making the spraying effective against canker. In a smaller spraying experiment at Lyngby half of 96 apple trees (24 varieties) received two summer sprays from 1927–30, with Bordeaux mixture or lime sulphur, with or without lead arsenate and nicotine according to circumstances. A number of dead cankered branches were removed, but in spite of this there was still a marked difference in 1930 between sprayed and unsprayed trees of varieties susceptible to canker.

VARIETY	NUMBER OF CANKERS	
	Unsprayed	*Sprayed*
Cox's Pomona	28	2
Signe Tillisch	30	4
Taarnborg Graastener	31	1

With pears it is very important to keep down scab on the twigs, for the canker fungus gains ready access through these lesions.

In England the canker fungus has been shown to penetrate through leaf scars in spring and autumn, and two autumn sprays and one in spring have been found to prevent this. They cannot be recommended for general use without further trial and should be unnecessary in orchards where trees are well cared for. A spray of Bordeaux and oil emulsion in March and another immediately after leaf fall may be worth while on nursery stock. Aphis damage may also form wounds and should be prevented as far as possible. No additional control measures for eye rot of the fruit have been suggested.

In England and Wales sale of trees substantially affected by canker is prohibited by the Sale of Diseased Plants Orders of 1927 and 1936.

Surface Canker (*Pezicula corticola* (Jørgens.) Nannf. = *Neofabraea corticola* Jørgens. = *Myxosporium corticolum* Edgert.)

This disease was examined in Denmark and described in detail in 1930 by C. A. Jørgensen.

Symptoms. The disease starts as discoloured, sunken patches on the bark. Branches less than a finger in thickness are seldom attacked; the lesions usually begin to develop round the base of dead spurs, in angles of the branches, or at the edge of cracks in the bark.

The fungus spreads very slowly from the point of infection during the first summer and not until the beginning of August or later does its growth rate increase. Then in the course of a month or two the bark lesions (see Fig. 61)



Fig. 61. Surface canker on branches of apple (left and centre) and pear (right) (after C. A. Jørgensen).

may attain a length of 5–6 inches and may extend half way round the circumference of the branch. Diseased areas on the trunk and thicker branches are usually elliptical (Fig. 62), or round, and seldom more than 3–4 inches in diameter.

Later in the autumn, when the lesion ceases to extend, the boundary between diseased and healthy bark is usually very clear cut, and often defined by a crack. The diseased bark becomes

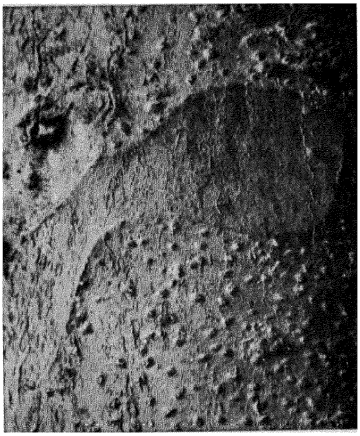


Fig. 62. Apple surface canker enlarged to show *Myxosporium* pycnidia. A strip of healthy bark crosses the centre of the photograph (after C. A. Jørgensen).

slightly sunken, is reddish grey or reddish brown, and innumerable small pustules appear on its surface. These contain the conidia of the *Myxosporium* stage of the fungus which ooze out in whitish masses when ripe.

Often individual cankers cease growth at the end of the first year, but sometimes they extend farther in the following year. When that happens they do not spread outwards evenly all round but from a few points on the edge of the old canker, so that the new growth has a semicircular shape. New infections usually take place in the neighbourhood of old ones; new patches of diseased bark appear and extend, often to the edges of the old ones. In America the disease is called "superficial bark canker." It is characteristic that it never penetrates deeply into the bark but the scars may be very long and narrow. They may run for two or three feet down one side of a stem yet be no more than 1½ inches broad.

Cause. On the branches of apple trees showing the symptoms described above C. A. Jørgensen found the fungus *Myxosporium corticolum* and later on its perfect stage which he called *Neofabraea corticola*. The connection between the two types of fructification was proved by single-spore cultures. In Sweden Bjørling (1943) has described a similar disease, attributed to *Myxosporium mali* Bres. The *Myxosporium* stage has been known in England since 1920 (Gilchrist, 1923) but the perfect state has yet to be collected there.

Importance. The disease has not been shown to be responsible for serious damage. A layer of cork forms sooner or later along the line between healthy and diseased bark and prevents further penetration by the fungus. The dead bark soon becomes covered with cracks, and the branches increase in thickness and gradually throw it off, often in the course of a year or two.

Apple varieties apparently differ slightly in susceptibility to surface canker, for Cox's Orange Pippin was not attacked by it in an orchard where Cox's Pomona, Graastener, Lord Suffield and Queen were all affected.

Myxosporium canker has long been known in America but is not considered serious there either. Gilchrist described a serious outbreak involving the deaths of some trees in England. The symptoms differed somewhat, however, from those found in Denmark and in North America, so that other parasites may have complicated the situation in that instance.

Control. As the disease is conspicuous rather than dangerous we think it unnecessary to prescribe special means of control. We find, however, that trees receiving a tar-oil winter spray are more susceptible to it than those not so sprayed. If it did become troublesome in an orchard, therefore, it would be well to cut out winter spraying, at least of the most susceptible varieties.

Other Minor Cankers

Neofabraea perennans Kienholtz=*Gloeosporium perennans* Zeller and Childs, is the cause of a disease called perennial canker in North America. It also occurs in Denmark (Fig. 63) and has recently been recognized in England (Wilkinson, 1943, 1945). It penetrates the branches through wounds such as those due to frost or insects. The cankers are often somewhat sunken with cracked and frayed bark and may develop round buds or spurs.

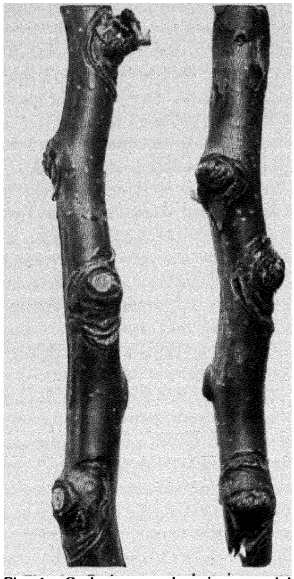


Fig. 63. Cankers on apple twigs, caused by *Gloeosporium perennans* (*S. p. F.*).

Similar lesions may, however, be due to frost alone, or represent the initial stages of common apple canker. The disease was called perennial canker because in North America the lesions were found to increase in size for six or seven years, each season's growth being limited by a ring of callus. Subsequent investigations have shown that this only happens when the callus layer is injured by low temperature or woolly aphis attack. In England the fungus causes mainly a dieback of summer-pruned trees and the cankers do not grow more than one year unless woolly aphis is present.

N. perennans also causes a slow circular brown rot round the lenticels on apple fruit, which was especially prevalent in Denmark in the winter of 1940–1. The rot spread most rapidly at temperatures from 50 to 60° F.

Summarizing his observations on the species of *Gloeosporium* on apple in England, Wilkinson (1945) concluded that whereas *G. perennans* is an active wound parasite of apple branches, *G. album* and *G. fructigenum* are non-parasitic to branches. The first two species produce indistinguishable rots of the fruit (see p. 124).

Neofabraea malicorticis Jack.=*Gloeosporium malicorticis* Cord. causes an apple disease called anthracnose in North America, especially in Oregon and Washington states. It causes dark elliptical sunken cankers up to a foot long, which may girdle and kill small branches. It also causes a fruit spot like *G. perennans*.

Control. With all these minor cankers control measures consist mainly in maintaining vigorous normal growth and avoiding production of poorly ripened wood susceptible to frost. Scab and sucking insects must be controlled and mechanical damage to the fruit avoided as far as possible. Diseased branches should be cut out and burnt as soon as detected.

Phacidiella discolor (Mout. & Sacc.) Potebn.

Rostrup found the conidial stage of this fungus on apples in Denmark as long ago as 1902 and called it *Pyrenochaeta furfuracea* (Fr.) Rostr. He described it thus: "The apples develop a black skin in which numerous small flat crowded warts appear. In a damp atmosphere these soon develop into spherical pycnidia up to $\frac{1}{25}$ inch across, covered with fine grey hairs and each with a small, black, glistening, conical mouth, making the whole surface of the fruit rough." To this picture we may add that the apples may become almost covered with a close, flat, whitish mycelium (Fig. 64).

Fruit rotting by this fungus usually comes late in the season and has never yet been of economic importance. It has also been found on pear and quince fruit.

Phacidiella discolor can also cause small brown spots on apple leaves. We have isolated it from such spots and inoculated the isolate to apple fruit, in which it produced the typical black rot. It also causes a form of canker on apple, pear and quince branches (Fig. 65) (Brooks, 1928). This is the only phase of the disease so far recognized in England. The cankers are like those of *Myxosporium corticola* but are particularly liable to peeling off of the

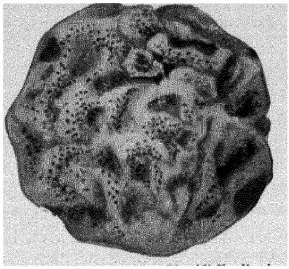


Fig. 64. Apple rotted by *Phacidiella discolor*.



Fig. 65. Apple branch with canker caused by *Phacidiella discolor* (*S. p. F.*).

surface layers of bark. The conidial fructifications appear on the dead bark as small round dark-grey or olivaceous pycnidia about $\frac{1}{25}$ inch across, each containing several cavities from which the spores exude in creamy masses. This stage has received the names *Fuckelia conspicua* Marchal and *Phacidiopycnis malorum* Potebn.

Control measures are the same as for surface canker.

Yet another minor canker, caused by *Pezicula pruinosa* Farlow, with the conidial state *Sphaeronema pruinosum* Peck, has been briefly described by W. C. Moore (1947).

Leaf spots. Moore (1939) described a peculiar angular leaf spot about $\frac{1}{12}$ inch across associated with *Phyllosticta angulata* Wenzl. These spots were at first pale yellowish and inconspicuous but later turned light grey on the upper surface of the leaf and deep brown below. Further investigation showed them to be due to the frog-hopper *Cercopsis sanguinea* Geoff. feeding on the leaves. The *Phyllosticta* appears later on the tissue killed by the insect (Moore, 1940).

Dennis and Wakefield (1946), described small circular whitish spots with a narrow brown margin on apple leaves from Scotland. On their upper surface these spots bore tiny black pycnidia just visible to the naked eye. Two distinct fungi were found on the spots, *Phyllosticta mali* Prill and Del. and *Coniothyrium pirinum* (Sacc.) Sheldon. The same species were found on apple leaf spots in Virginia by Crabill in 1911, and *C. pirinum* is apparently the same as *C. tirolense* Bubak and *C. piricola* Potebnia, found respectively on pear leaves in the Tyrol and apple leaves in Russia. Rostrup found *P. mali* on apple leaves in Denmark. *Phyllosticta briardi* Sacc. is a third pycnidial fungus associated with leaf spots of apple in Italy.

Crabill made inoculation experiments with his fungi and found that neither of them could attack uninjured leaves. Probably, like *P. angulata*, they are secondary invaders following insect damage or apple scab spots.

Another kind of leaf spot, found by Crabill in Virginia and Salmon in England (1907), is caused by *Sphaeropsis malorum* Peck. This is a true parasite and produces large irregular dark-brown spots dotted with tiny black pycnidia. When the spots are numerous the infected leaves may shrivel and fall. The same fungus causes one of the minor rots of stored apples (see p. 125).

Bud Rot (*Fusarium lateritium* Nees *var. fructigenum* (Fr.) Wr.)

This disease is probably rather common in England but it is inconspicuous and is usually overlooked. It may be recognized by death of the flower buds, less often the leaf buds, just

as they are about to burst in spring. Usually there is no fungus visible but occasionally pink conidial pustules appear on the bud scales, and later in the season wefts of pink mycelium can be found between the scales. A similar disease occurs on pear buds in Holland (Aderhold, 1901). According to Wollenweder and Reinking *F. lateritium* is the conidial state of *Gibberella baccata* (Wallr.) Sacc. and is common on twigs of deciduous trees and shrubs as well as on pear, quince and rose fruits. They regard the variety *fructigenum* as indistinguishable from this species.

Dillon Weston (1927b), who studied bud rot in the orchards of the Isle of Ely, found Emneth Early and Lord Grosvenor most susceptible, Grenadier somewhat less so, Lane's Prince Albert and Lord Derby moderately affected and Bramley's Seedling, Newton Wonder and Worcester Pearmain only slightly. The disease appears to be most prevalent in the spring following a wet summer and autumn. Control measures have not been worked out but it is obviously advisable to cut out and burn diseased twigs.

Blossom Wilt (*Sclerotinia laxa* Aderh. & Ruhl. f. *mali* (Worm.) Harrison = *Monilia cinerea* Bon.)

Symptoms and development. About a fortnight after the trees have come into blossom whole trusses of flowers begin to wither (Fig. 66). From the infected flowers the fungus grows down into and kills the spur. Often it penetrates into the branch to form a canker. Sometimes the branch is girdled so that the whole portion above the canker dies. The dead blossoms remain hanging and in damp weather the grey pustules of the *Monilia* stage appear on them in great numbers. In the following winter and spring *Monilia* pustules break out on the bark of the dead spurs and cankers and conidia borne on them form the source of infection for the next season's blossom. Moisture, as rain or dew, is necessary for spread of the fungus.

Importance. The damage done by this disease depends largely on the variety of apple. On the most susceptible varieties most of the fruiting spurs may be killed in seasons favourable to the fungus (Fig. 67).

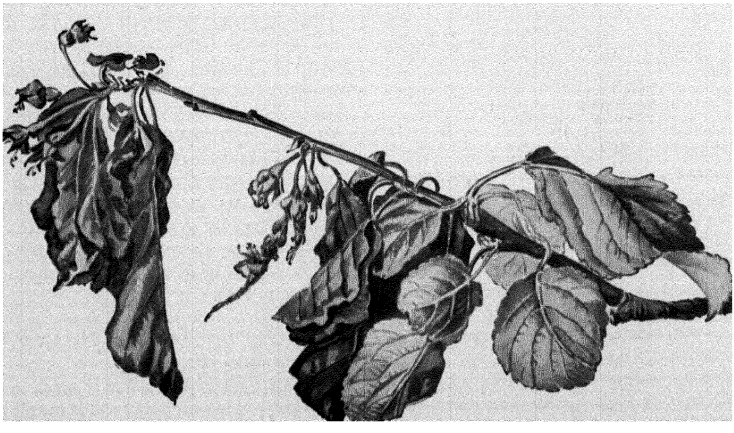


Fig. 66. Wilt of apple blossom, caused by *Sclerotinia laxa* f. *mali*.

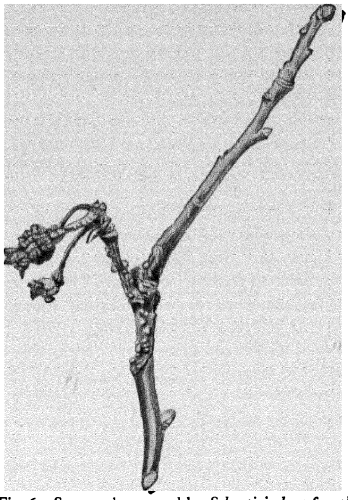


Fig. 67. Spur canker caused by *Sclerotinia laxa* f. *mali*. Note conidial pustules on overwintered twig.

VERY SUSCEPTIBLE VARIETIES

Charlamowsky	Lord Derby
Cox's Pomona	Maglemer
Queen Louise	Pederstrup
Ecklinville's Seedling	Red Astrakan
Filippa	Säfstaholm
James Grieve	White Transparent
Keswick Codlin	Wealthy

SUSCEPTIBLE

Belle de Boskoop	Golden Noble
Bramley's Seedling	Høve Reinet
Casseler Reinet	Lane's Prince Albert
Cellini	Lord Grosvenor
Cox's Orange Pippin	Manks Codlin
Flaskeæble	Venus

Brown rot of apples is usually due not to *S. laxa* but to *S. fructigena*. Wormald (1945) has, however, discovered that *S. laxa f. mali* can occasionally cause a black rot of apple fruit.

Control. All wilted and cankered spurs and branches should be cut out and burnt as soon as possible. If this is done in time it may be possible to prevent the fungus from spreading down to form cankers on the branches. The ordinary tar-oil winter spray helps to destroy the pustules on the bark. Where the disease has been very prevalent a white oil—Bordeaux spray (1 part oil to 9 parts Bordeaux) is recommended, or a 1:50 lime sulphur spray when the buds are showing green tips.

Brown Rot and Spur Canker (*Sclerotinia fructigena* Aderh. & Ruhl. = *Monilia fructigena* Pers.)

Symptoms and development. Conidia of the fungus appear in summer on yellowish cushion-like pustules and one can often see clearly that infection has spread around a single diseased fruit or spur. The conidia are scattered by rain splashes and on the bodies of insects, sometimes by wind. The fungus also spreads from fruit to fruit in a cluster by direct contact and sometimes grows down the stalk into the spur and forms a canker. It does not spread through the branch in subsequent years but in the following summer rather large yellowish conidial pustules appear on the dead wood. These are a potent source of infection of the young fruit. The same fungus attacks stone fruits and is known to be able to penetrate them through the unbroken skin. Wounds are, however, the chief infection courts. Cracks in the skin due to scab, spray damage, heavy rain, hail, frost or insects, especially wasps and boring larvae like those of the Codling moth and apple sawfly, are important while the fruit is still on the tree. Bruises and wounds, left when the stalk is pulled out by careless pickers, are important centres of infection in stored fruit. The riper the fruit the more susceptible it is to brown rot.

A brown spot appears on the infected apple, and spreads rapidly until in a few days the whole fruit may be rotten. Conidial pustules break out in a ring round the point of infection and as the rot spreads further concentric rings of pustules appear. At first they are white, but soon turn yellowish with the spores (Fig. 68). Conidial production may be in full swing only 7–8 days after infection and if conditions favour spread of the disease it soon attains

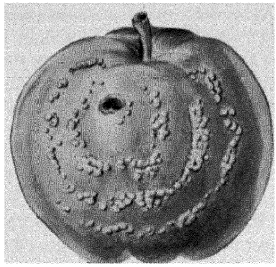


Fig. 68. Brown rot of apple, infection occurred at a maggot-hole.

epidemic proportions. At first the brown flesh does not shrivel but when the whole fruit has become decayed it gradually shrinks and becomes mummified (Fig. 69). Most of them fall to the ground and rot but a few may persist in the top soil for some years, and after at least two winters they may bear the cup-shaped fructifications of the perfect *Sclerotinia* stage. These are very rare in Denmark and have yet to be found in Britain. Far more dangerous are the mummified fruits that remain hanging on the trees and bear fresh conidia in large numbers in the following summer. The fungus has been found still alive in a mummified fruit ten years old.

Few conidia are produced on apples that rot in storage and sometimes they turn black and shiny instead of brown. Usually the fungus does not break out of these black apples but in moist conditions the mycelium may emerge and penetrate the surrounding fruit. Black apples are not due to a special form of fungus but to the conditions of storage (Plate 7, Fig. D).

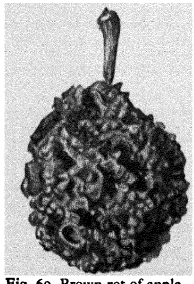


Fig. 69. Brown rot of apple, overwintered mummified fruit.

Pears, quinces and *Cydonia japonica* may also be rotted by *S. fructigena* but, like apples, are very seldom attacked by *S. laxa* or its form *mali*.

Plums are rotted by both *S. fructigena* and *S. laxa*, though the former is more prevalent in Denmark. Mummified plums are particularly liable to remain hanging on the tree and are important sources of infection. Cherries are also attacked by both fungi, chiefly by *S. laxa* while they are still green and by *S. fructigena* when they ripen, especially if they become cracked after rain. Sweet cherries are particularly susceptible to *S. fructigena* and so are Myrobolans. Peaches and apricots are rotted by both species; the peaches fall, the apricots generally remain hanging as mummies. In severe outbreaks in Germany 98% of the fruit has been destroyed by infection through cracks at the tip and side of the fruit. Apricot varieties differ somewhat in their tendency to crack in this way (Maier, W., 1942).

In North America, Australia and New Zealand, stone fruit is rotted by an allied but distinct species, *S. fructicola*, so far found in Europe only on imported fruit.

Conditions for infection. Pustules of *S. fructigena* may be found in winter but the conidia are then unable to germinate. The fungus needs a warm temperature and hence does not become infective until late in the spring. It also needs moisture, so that it tends to be more prevalent on low ground than on high, and is assisted in its spread by wounds of all kinds.

Importance. Brown rot may be very destructive in wet summers and losses from it continue after picking. Spur cankers are especially important as a method of overwintering and a source of infection in the following season. No variety of apple, pear or stone fruit is immune from brown rot but some seem particularly susceptible to it, see table following.

VARIETAL SUSCEPTIBILITY

VERY SUSCEPTIBLE

Apple

Alexander	Gyllenkrok
Cellini	Laxton's Superb
Cox's Pomona	Oudrupgaard
Filippa	Signe Tillisch
Frogmore	

SUSCEPTIBLE

Belle de Boskoop	Graastener
Bismarck	Høve Reinet
Bøghs Citronapple	Keswick Codlin
Charlamowsky	Lane's Prince Albert
Cox's Orange Pippin	Lord Suffield
Queen Louise	Red Pineapple
Flaskeæble	Säfstaholm
Golden Noble	White Transparent

VERY SUSCEPTIBLE

Pear	*Plum*
Clara Frijs	Jefferson
Greypear	Victoria

SUSCEPTIBLE

Pear	*Plum*
Amanlis	Althanns Reine Claude
Bonne Louise	Bavays Reine Claude
Colorée de Juillet	Hungarian Prune
Giffard	
Grev Moltke	
Herrepear	
Kejserinde	

Control. Diseased fruit should be removed as soon as possible, partly to prevent their functioning as sources of infection, partly to stop the fungus from growing down the stalk and killing the spur. Cankered spurs and branches should be cut and burnt.

Brown rot is reduced by a regular spray programme for control of scab and insect pests. Scab cracks and apple sawfly holes are important infection courts for *S. fructigena*. Clusters of fruit are more liable to rot than single apples and this should be borne in mind when the crop is being thinned.

Care should be taken to avoid damage at picking and bruised fruit should be used at once. Storage at low temperatures reduces the rate of development of brown rot. It is well to cool fruit before sending it away.

Dry Eye Rot (*Botrytis cinerea* Pers.)

Symptoms. The common grey mould, *Botrytis cinerea*, sometimes causes a rot of ripe apples. Usually there is only one infection point on each fruit but the rot spreads fairly quickly through the flesh. It commonly begins at the eye and under damp conditions it breaks out to cover the fruit with a loose grey mould (Fig. 70).

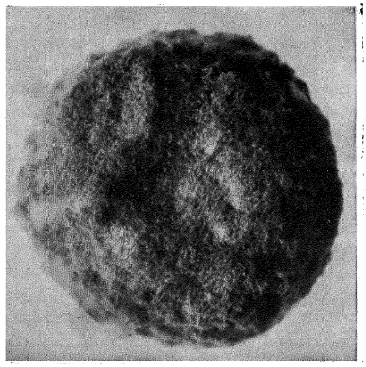


Fig. 70. Apple rotted by grey mould (*S. p. F.*).

Cause. There is no specialized strain of *B. cinerea* peculiar to apples. Wormald has shown that they may be rotted by strains of the fungus collected on many different plants (see p. 60). The conidia germinate on and penetrate through wounds but mycelium from a diseased fruit can grow directly from it into an adjacent healthy one. According to Heald and Sprague (1926) it may also penetrate the lenticels to cause small pale spots with a dark ring round them. In very wet seasons it may possibly also cause spotting and blotching of the leaves (Moore, W. C., 1948).

Importance. Eye rot caused by *B. cinerea* may occur on the tree (see Wilkinson) in which case care is necessary to distinguish it from eye rot caused by the canker fungus *Nectria galligena* (see p. 113). It is more familiar, however, as a rot of stored fruit.

This may occur at temperatures down to 32° F. but is more rapid at high temperatures. As a rule it is less destructive than blue mould (*Penicillium*). Horne and Colhoun (1939) have found that apples from different orchards may differ in susceptibility to *Botrytis* rot.

Control. It is important to avoid bruising the fruit, which should be stored in a cool place. In America Cooley and Crenshaw (1931) obtained some control of *Botrytis* rot in stored pears by wrapping them in paper soaked in 2.5% copper sulphate solution allowed to dry. Oiled paper such as is used to prevent scald can also be impregnated with this solution. If pears are damp when wrapped in these papers they are damaged, but otherwise no harm results from their use. A similar control of *Botrytis* on apples is to be anticipated but we have no experience of the method, which should be tried out on a small scale before general adoption.

Bitter Rot (*Gloeosporium* spp.)

Symptoms. *Gloeosporium album* Osterw. causes well-defined brown, firm, slightly sunken spots on stored apples. At the centre is a dark speck, next comes a light-brown zone and outside is a broad dark-brown marginal ring (Fig. 71). In time small conidial pustules appear on the spots. At first they are greyish yellow or yellowish brown and hairy, later they become tiny glistening white heaps of conidia (Fig. 72, and Plate 5, Fig. B). Usually there are several spots on each apple but they slowly enlarge until they meet and the entire fruit is destroyed. Though the disease is usually found on stored fruit it sometimes occurs on apples still on the tree. The fungus may also attack pears and quinces.

Cause. The fungus *Gloeosporium album* penetrates the fruit through lenticels, small wounds, scab spots or bitter pit lesions. In England Ogilvie (1935) has found that when the apples are ripening *Gloeosporium album* conidia are produced on old mummified fruits and on old pruning scars. The fungus seems only able to attack dead wood or to penetrate from it very slowly into living tissue adjacent. Probably most of the fruit infection takes place in the orchard but overwintering of the fungus on shelves and boxes in the storeroom has perhaps also to be reckoned with.

Wilkinson (1939) found that apples from trees heavily manured with nitrogen were much more heavily infected with *Gloeosporium album* than those from trees which received little nitrogen.

Importance. *G. album* may cause considerable loss but is normally less destructive than *Penicillium*.

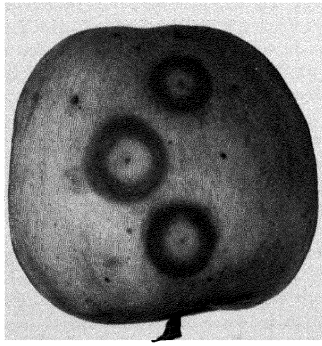


Fig. 71. *Gloeosporium album*, young spots on Pederstrup apple.



Fig. 72. *Gloeosporium album*, older spots covered with conidial pustules on Høve Reinet apple (*S. p. F.*).

Glomerella cingulata (Stonem.) Spauld. & v. Schrenk = *Gloeosporium fructigenum* Berk. causes a very similar rot of apples. Its pustules, however, differ from those of G. *album* in bearing pinkish spore masses that in moist weather ooze out into worm-like tendrils (Plate 5, Fig. D). It is not confined to apples but rots also pears, cherries, quinces, peaches and grapes.

G. perennans (see p. 117) produces a rot indistinguishable from that of *G. album* except by microscopic examination of the spores.

Black Rot (*Physalospora obtusa* (Schwein.) Cooke. = *Sphaeropsis malorum* Peck)

This fungus causes a leaf spot of apples (p. 119) and a mild form of canker, called New York canker, because of its prevalence in that state, but is best known as the cause of a black rot of apples both on the tree and in store. Lesions first appear as small brown spots anywhere on the surface of the fruit, though most often at the eye end. Frequently they start round insect punctures. The spot increases in size and darkens until it becomes black. In a few weeks after infection numerous black pycnidia, the *Sphaeropsis* stage of the fungus, appear on the rotten areas. Eventually the whole fruit decays and shrivels to a black mummy, which may remain hanging on the tree for a year or more. The perfect perithecial stage develops in the bark of old cankers but is rarely seen.

Black rot is best known in North America where it is controlled by a 4:4:50 Bordeaux spray about mid-July, followed by a second two weeks later (Wolf, 1913). Usually the winter spray programme against apple scab is sufficient to control this disease as well. Dennis (1943) has described a very similar black rot of apples caused by the rare fungus *Strasseria carpophila* Bres. & Sacc.

Fish-eye Rot (*Corticium centrifugum* (Lév.) Bres.)

This is a storage rot common on apples in North America and found by Colhoun (1938) in Northern Ireland.

The spots are light brown and slightly sunken. Often they are centred on scab lesions, in which case there is a dark centre surrounded by a tan-coloured zone with dark-brown ring at the outer edge. The whole somewhat resembles the eye of a fish, hence the common name of the disease. It is thought that infection occurs in the orchard when spores of the *Corticium* alight on a patch of dead or moribund tissue, such as a scab spot, lenticel, stalk or calyx lobe. The disease then develops later when the fruit is stored. The fungus is unable to penetrate sound epidermis.

Phytophthora Rot (*Phytophtora cactorum* (Leb. & Cohn) Schroet. and *P. syringae* Kleb.)

This is a brown fruit rot of apples and pears, somewhat resembling ordinary brown rot but lacking the characteristic *Monilia* pustules. On apples the affected areas may be confused with bruise marks but show no flattening or depression of the surface. The colour of the rotten skin varies with the variety, being grey-brown in Allington Pippin, chestnut brown in Monarch and dark brown in Lane's Prince Albert. On pears the rotting areas are dark brown and well defined. The rotting flesh is brown, with brown streaks along the vascular strands, and is firmer and tougher than in ordinary brown rot (Wormald, 1919; Ogilvie, 1931).

Cause. Phytophthora rot may be caused by two species of water mould, *P. cactorum* and *P. syringae*. Infection comes usually from the soil, hence the disease is usually seen on windfalls and on fruit hanging on branches close to the ground. The fungi are probably splashed on to these during heavy rain. Rots caused by these two species cannot usually be distinguished without microscopic examination and cultural studies. On pears, however, *P. cactorum* produces numerous glistening granules, resembling hoar frost, under moist conditions. These are formed by clusters of sporangia projecting from the skin. Sporangia are seldom formed by *P. syringae*. Sound fruits readily become infected by contact with diseased ones.

Control. The disease is usually troublesome only in wet seasons or in districts with a high rainfall. Control measures include propping

the lower branches well clear of the ground and collecting and destroying windfalls. Trees known to suffer from the disease may be sprayed with Bordeaux mixture.

Sooty Mould

Symptoms. Apples with sooty mould look dirty in late summer and when picked. Especially around the stalk or over one side of the fruit there is a more or less continuous dark or nearly black fungus coating that can be rubbed off by wiping the surface vigorously with a damp cloth.

Cause. The dark coating is caused by various imperfect fungi, known collectively as "sooty mould." In the tropics there is a rich flora of sooty moulds which grow on the surface of living leaves, somewhat as powdery mildews do in temperate climates. These are often restricted to individual host plants. The sooty mould of North and West Europe, however, is a mixture of mould fungi which occurs indifferently on conifers and deciduous trees and shrubs. They all have in common the capacity to grow in a very strong sugar solution, such as is deposited on the surface of plants infested by sucking insects like aphides and scale insects, which excrete the so-called "honey dew," rich in sugar. Plants infested with these insects are more greasy and sticky to the touch than others. The fungi grow exclusively on the honey dew and never penetrate the underlying plant tissue.

The honey dew with its accompanying sooty mould occurs not only where the insects are feeding but drips over other parts of the plant.

Importance. Sooty mould is often only noticed when the apples are being graded. As the fungi do not penetrate the fruit they make no difference to its quality, provided it is peeled before being eaten, but they render it unattractive and hence less saleable.

Control. Sooty mould can be avoided by keeping the trees free from aphides. When it is present on a crop it may be worth while to remove it. This is most easily done by washing the apples in tepid water with a little vinegar and salt. They may also be wiped clean with a damp cloth. In either case the work is tedious and it may impair the keeping quality of the fruit.

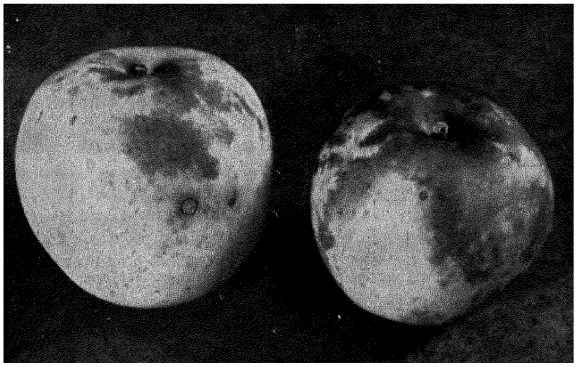


Fig. 73. Sooty mould on apple.

Fly Speck (*Leptothyrium pomi* (Mont. & Fr.) Sacc.) and Sooty Blotch (*Gloeodes pomigena* (Schw.) Colby)

The fly speck fungus produces groups of round black dots on apples and sometimes on pears and plums. The groups are about $\frac{1}{8}$ to $\frac{1}{4}$ inch across and under a lens their component black dots are clearly visible. These are the tiny pycnidia of the fungus and superficially resemble fly excrement, hence the common

name of the disease. The fungus is only on the skin and can be scraped off, but when plentiful it may spoil the appearance of the fruit.

Associated with *Leptothyrium pomi* is usually the fungus of sooty blotch, *Gloeodes pomigena*. This causes greenish or brown blotches, up to $\frac{1}{4}$ inch across, though several may join up to cover a large area. The blotches bear a thin film of hyphae amongst which are numerous black specks easily visible under a lens. The fungus continues to develop during storage and then becomes more conspicuous.

Both these troubles are worst on the fruit from unsprayed trees and are kept in check by the routine summer sprays for scab control. They may be troublesome in wet, dull summers, and it may then be advisable to treat infected fruit before it is sold or stored. Wormald (1937b) recommended dipping apples and plums in 5% chloride of lime for one minute, then laying them aside wet for ten minutes, rinsing in running water and drying in the open air. The solution is made up by mixing $\frac{1}{6}$ lb. of chloride of lime with a gallon of water, leaving to settle and pouring off the clear liquid for use. Experience in Denmark suggests that even after bleaching in this way it may be necessary to wipe the fruit with a cloth to get it clean.

Blue Mould (*Penicillium expansum* Thom)

Symptoms and cause. This is a disease of stored apples, which causes them to turn light brown, soft, watery and rotten and become covered by a dusty blue-green layer of mould. The latter starts as small grey-green pustules but these may ultimately link up to form an almost continuous layer over the fruit (Plate 7, Figs. A and B). The species of *Penicillium* usually concerned is *P. expansum* Thom.

The conidia are very tiny, dry and light. If one blows at an apple covered with blue mould one sees a cloud of dust rise, formed of innumerable conidia. The least touch or draught in the storeroom sets free many conidia and the disease therefore spreads very easily.

If the conidia fall into water they do not get wet but float on the top like dust, hence they can be blown into the air again.

Blue mould usually enters apples through wounds, but may sometimes penetrate through the lenticels. The conidia cannot germinate at temperatures near freezing point, but once the fruit has been infected the mycelium can go on growing even at 32° F., though much more slowly than at higher temperatures. Its optimum temperature for growth is 77° F. (Ames, 1915).

If conditions are favourable for the fungus the apples may rot completely in a fortnight. Only ripe or nearly ripe apples are attacked, hence the disease is scarcely ever seen while the fruit is on the tree.

Mycelium of the fungus can grow directly from one diseased apple into a healthy one touching it in store. Horne and Colhoun (1939) state that the same variety of apple from different nurseries may show considerable differences in susceptibility.

Importance. In all apple-growing countries blue mould is considered one of the most destructive storage diseases. Various species of *Penicillium* also cause rot in other kinds of fruit, such as oranges and lemons, and are well known as moulds of jam. As the disease develops gradually during storage and especially on fruit bruised in packing and transit, the loss usually falls rather on the fruiterer and consumer than on the grower, if the latter sells his apples soon after they are picked.

Control. The fungus is so ubiquitous and the conidia so easily dispersed that it is almost impossible to prevent some of them from landing on stored apples sooner or later. By disinfecting storerooms and packing materials, and quickly destroying affected fruit, one can try to limit the chances of infection.

The principal emphasis should be laid on avoiding injury to the apples. In this connection a sound spray programme to prevent scab lesions and attacks by other fungi or insects is important. Care in picking, grading and packing will reduce bruising and wounding. The fruit should be stored in as cool a place as possible, to retard fungus growth. If the fruit is destined for cold storage it should go there immediately after picking. Storage

for only one week at room temperature in a warm August may allow the fungus to gain access to the fruit to such an extent that losses will be severe in spite of subsequent cold storage. Storerooms and boxes should be thoroughly cleaned (see p. 580).

Pink Rot (*Trichothecium roseum* Fr.)

This is a disease of the stored fruit which starts as a pale pink bloom over the surface of scab spots. The fungus is a very common mould on all kinds of decaying vegetable matter and cannot penetrate healthy apples. The conidia germinate on and penetrate scab lesions and other wounds. Under favourable conditions of humidity the fungus can spread into the underlying flesh so that a rotting zone surrounds the scab spot (Plate 7, Fig. F). It does not penetrate deeply into the flesh but gives it a bitter flavour. The close connection between pink rot and scab is due partly to the rough cracked surface of the scab spot and partly to its capacity for retaining moisture. Pink rot is favoured by conditions which cause the fruit to "sweat."

Importance. This fungus may considerably reduce the keeping quality of scabby fruit.

Control. The important thing is to avoid scab. When scabby fruit has to be stored it should be kept as cold and dry as possible without causing it to shrivel.

Other Minor Fungus Fruit Rots

Colhoun (1938) found a light-brown rot affecting the stalk end of stored apples in Northern Ireland to be caused by the pycnidial fungus *Phoma mali* Schulz. & Sacc. The rot spreads rapidly through the ripe fruit and pycnidia appear beneath the skin 4/5 weeks after infection. The disease may cause considerable loss towards the end of the storage season. Wormald (1942) observed two unnamed species of *Phoma* causing stalk-end rot of apples in England. A very similar rot of stored fruit is caused by *Phomopsis mali* Roberts.

Fusarium lateritium var. *fructigenum* (Fr.) Wr. and other species of *Fusarium* cause a small brown circular rot which gradually involves the whole fruit. In the later stages of decay whitish tufts of hyphae emerge through the lenticels.

The pin moulds *Rhizopus nigricans*, *Mucor racemosus* Fres. and *M. piriformus* Fischer may cause soft light-brown watery rots with an alcoholic odour.

Apple Rust (*Gymnosporangium juniperinum* (L.) Mart.)

Symptoms. The first symptom is the appearance of yellow spots on the apple leaves, bearing on their upper surface the spermogonia of the fungus, like black specks. Later the æcidia or cluster cups appear on the lower surface of the spots. These cups are wide and shallow and when the spores are ripe the wall of each cup splits into numerous white fibres which bend outwards to expose the spores. Orange or reddish spots may also appear on the apples themselves, especially towards the eye.

The aecidiospores are able to infect only the common juniper (*Juniperus communis*) on which the fungus causes spindle-shaped swellings of trunk and branches. From these the irregular masses of chocolate-coloured teleutospores emerge in spring. When they are ripe they swell up in damp weather to form large jelly-like clumps that turn yellow-brown as the basidiospores develop on the teleutospores. When the basidiospores have been shed the jelly-like masses dry up and fall, leaving brown scars on the bark.

Cause and development. The rust of apple and juniper is caused by a fungus whose alternation between these hosts was demonstrated by A. S. Ørsted in 1866. The cluster cup stage was formerly called *Rœstelia penicillata* Fr. and the juniper stage *Gymnosporangium tremelloides*. This fungus shows a high degree of specialization and Eriksson distinguished five physiologic races of which four infect mountain ash and only the fifth, which he called *G. tremelloides* f. sp. *mali*, infected apple. The annual life cycle of the fungus is thus from juniper to apple in spring and from apple back to juniper in summer. It can survive from year to year in the juniper branches but nevertheless they are often killed in the course of a year. This is a peculiarity of this rust, for other



Fig. 75. Apple leaf covered with the yellow spots of mosaic disease.

varieties. The symptoms are expressed differently by different varieties and may sometimes be completely lacking though the virus is still present. The spots on the leaves are small, yellow or cream-coloured. Apple mosaic has been recorded several times in England and has been transmitted by grafting in the variety Lord Lambourne (Wallace, Swarbrick and Ogilvie, 1944) (Fig. 75).

Another virus disease, " false sting," causing scars like capsid injury on apple fruit, has been reported from Nova Scotia (Hockey J. F., 1943). Wallace, Swarbrick and Ogilvie have observed a similar condition on the cider apple Sweet Alford in England.

Rubbery Wood, Flat Limbs and Chat Fruit

During the last few years a peculiar condition has been noted in the varieties Lord Lambourne and Graastener in Denmark, and in Lord Lambourne, Miller's Seedling, Dartmouth Crab and James Grieve in England. Some of the main branches become so soft and flexible that they can be bent without snapping, as though made of a rubbery substance. The weight of a few fruit is sufficient to bend such branches to the ground. Usually they taper very rapidly from base to tip. Anatomical investigations have shown that this peculiar flexibility is due to most of the wood remaining unlignified (Beakbone and Thompson, 1945). There is some evidence that rubbery wood is due to a virus infection.

Flat limb is a condition observed in Lord Lambourne in which deep furrows and ridges occur along the branches which sometimes strangely resemble the bones of some large animal (Fig. 76). It was described by Hockey (1943) on the variety Gravenstein in Nova Scotia. He regarded it as a manifestation of incompatibility between scion and stock.



Fig. 76. Flat limb of apple.

A third abnormality in Lord Lambourne has been called chat fruit, because the tree bears fruit only a quarter the normal size. It has developed when Lord Lambourne was grafted on to Redcoat Grieve (Crane, 1945).

Shedding of Blossom and Young Fruit

It is usually desirable that not all the blossoms on a fruit tree should set fruit; if they did the fruit would be too small unless extensively thinned by hand.

Some flowers fall immediately after blossom-

ing, before any fruit has set. Usually these have not been fertilized. Weather conditions during blossoming play a great part in the success of fertilization, directly and indirectly. Fertilization is less easy during rain and in very dry air. Indirectly weather influences fertilization by its effect on insects. In bad weather few insects visit the flowers; honey bees, for example, do not fly so far afield as in good weather. Frost during or just prior to blossoming may destroy the pistils and thus render flowers infertile (p. 140).

Self-sterility of varieties is an important cause of poor cropping. Trees which blossom freely year after year but set little or no fruit usually lack a suitable fertilizing variety in their neighbourhood.

If the gardener knows the names of his fruit trees and knows which varieties are able to fertilize them it is advisable to plant one of the latter close by. Where circumstances make this impossible it may suffice to graft some branches with a fertilizing variety. Until these have grown sufficiently it may be helpful to obtain flowering branches from a suitable variety and hang them in pails of water in or near the blossoming tree. Without cross-fertilization small fruit may be formed, but usually soon falls off. A few varieties, however, can bear unfertilized fruit, for example the apple Graastener and some pear varieties. In such cases copious fertilization may result in deformed fruit.

Fruit may also fall even though fertilized. Thus, much fruit often falls from apple trees in June and early July. This is called the "June drop" but it is often most abundant in July.

This fruit fall is often due to nutritional factors. It seems that active carbon assimilation by the leaves is more important for satisfactory fruiting than a supply of nitrogen, phosphorus or potassium. The poor fruit crop which often follows very heavy blossoming can be attributed directly to a smaller leaf surface on the flowering spurs as compared with those that do not flower. Hence the fruit may be under-nourished with carbohydrates, even though the trees have been liberally manured.

The young leaves and green fruit compete for water and nutrients but the leaves have the prior claim. When transpiration is very active, or the reserves of nutrients in the tree are used up and are not being replaced fast enough, the fruit suffers first. As a result it lacks a strong enough tissue binding it to the tree. The June drop is often great when dry weather occurs in June and July. Many fruit growers try to avoid this by watering their trees in dry seasons. If, however, a very heavy fall of rain comes suddenly at the end of a dry period, it too may result in a large fall of fruit, especially of apples.

Control. To ensure a good fruit set and avoid a heavy fall of young fruit the grower should arrange to have hives of bees in or near his orchard and should try to maintain favourable conditions of growth, keeping the soil in good order, the trees well manured, and watered if the weather is dry. He should also have a regular pest and disease control programme. Another frequent cause of fruit fall is attack by insect larvae, but this falls outside the scope of the present book.

Experiments in the United States (Gardner *et al.*, 1939) have shown that shedding of immature fruit can sometimes be prevented by spraying the trees with growth substances. Apples show considerable varietal differences in response to these sprays, but experiments at East Malling showed that an α-naphthalene acetic acid spray at the strength of ten parts per million gave good results with Beauty of Bath, Worcester Pearmain and Miller's Seedling. Similarly the usual heavy drop of Cox's Orange Pippin fruit in July was largely controlled by applying this spray on 1st July. The varieties Edward VII, Allington Pippin and Bramley's Seedling seem to give little response to the spray (Vyvyan and Barlow, 1947).

Leaf Scorch (Potassium Deficiency)

The first symptoms of potassium deficiency may be development of large numbers of fruit buds, early and abundant blossoming, or the appearance of a narrow reddish edge to the green leaves. Usually the leaf edges turn brown and shrivel, the leaves remain small and

few, the shoots are short and in extreme cases the flower trusses wither soon after opening and the branches die back (see coloured plates 1, 2 and 3). The fruit is poor, pale and woody. When potassium deficiency is suspected an analysis of the orchard soil should be obtained as similar scorching may be due to drought, strong winds, especially off the sea, waterlogged soil, or to a shallow soil (Wallace and Proebsting, 1933; Warne, 1937). If phosphorus is abundant at the same time chlorosis may set in (p. 42).

At East Malling potassium-deficient apple trees were found to be particularly prone to spray damage and somewhat more susceptible to mildew, but a good supply of potassium does not protect trees against scab or canker. Aphides seem to prefer trees suffering from potassium deficiency.

Magnesium Deficiency

Of late years considerable attention has been paid to magnesium deficiency in apple trees, both in England and in North America, especially on acid sandy soils.

The first symptom is the appearance of pale-green patches between the veins of older leaves on some shoots. These faded areas turn brown and die and may sometimes link up round the edge of the leaf, somewhat as in potassium deficiency. As the season advances the symptoms appear on younger leaves higher up the shoots. In extreme cases they drop, leaving conspicuous stretches of bare wood (Southwick, 1943).

Magnesium deficiency symptoms tend to be accentuated by liberal potassium manuring. Kidson *et al.*, 1940.

Control. Where magnesium deficiency is suspected steps should be taken to have affected shoots analysed. Wallace (1939) has shown that this affords a useful means of diagnosing the condition. When it has been confirmed the deficiency is most easily corrected by manuring with magnesium salts, such as Epsom salts, at the rate of about 1 cwt. per acre, or with magnesian limestone, at the rate of 500–1,000 lb. per acre. The latter may be expected to be rather slow acting.

Nitrogen Deficiency

Apple trees more often suffer from excess of nitrogen than from too little. Nitrogen deficiency may, however, occur in grass orchards or when too vigorous an undercrop is grown. In manurial trials at Blangstedgaard and Long Ashton apple trees suffering from nitrogen starvation have been distinguished by weak growth, poor blossoming, late leafing and early leaf fall. The leaves showing yellowish-green or reddish-yellow coloration are mostly those towards the tips of shoots. The bases of the shoots bear few leaves. The fruit is small and highly coloured. Trees with plenty of nitrogen have large leaves, long, soft shoots and large, poorly coloured fruit. With excess nitrogen the bark may become thick and soft and very liable to injury in winter. The branches are spindly and swollen.

Boron Deficiency

In parts of the United States, Canada, especially British Columbia, New Zealand and Australia, boron deficiency is responsible for malformed apple fruit, variously described under the names drought spot, internal cork and corky core. Affected fruit may be dimpled and irregular, or may show only brown corky spots in the flesh rather like those of bitter pit (p. 134). The disease appears to be much less prevalent in Europe but has been described by Jamalainen in Finland and by various workers in parts of Switzerland. Boron deficiency in apples has been long overlooked in England and probably occurs there only in a mild form. Levy (1947) has described an occurrence of small dark-brown spots in the outermost $\frac{1}{4}$ inch of the flesh of Northern Spy apples. The spots were not visible from the outside and were not in contact with the skin. The number of apples affected with this disease was reduced by treating the trees with boric acid solution. As comparatively small quantities of boric acid or borax may be injurious to apples, growers should not embark on a scheme of boron manuring until the presence of boron deficiency in their orchards has been confirmed at a research station.

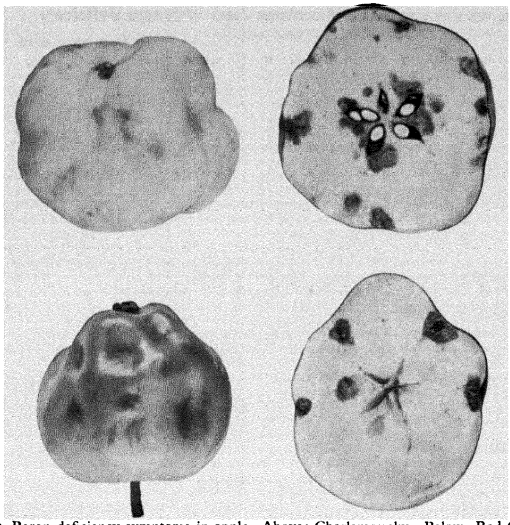


Fig. 77. Boron deficiency symptoms in apple. Above: Charlamowsky. Below: Red Canella (after Jamalainen).

Bitter Pit

Symptoms. Bitter pit typically starts just under the peel as brown spots $\frac{1}{8}$–$\frac{1}{6}$ inch across in the flesh. As the brown tissue shrinks the overlying skin is pulled inwards so that small shallow pits appear in the peel corresponding to the outermost spots in the flesh. The skin in the pits may be darker than the rest. The brown spots may increase slightly in size and several may join together. In some varieties they may be located in a zone round the middle of the fruit. Usually they are more numerous towards the eye end (Plate 8, Figs. B, D and F).

Bitter pit mostly appears during storage, but in some varieties it may develop on the unpicked fruit at any time after it is half green. Then dark spots show through the skin, followed by the pitting. Bitter pit on the tree is not easy to distinguish from some forms of corking due to boron deficiency (see p. 133). The brown flesh has a bitter taste, but does not usually make the fruit uneatable.

Cause of bitter pit. Bitter pit occurs wherever apples are grown on a commercial scale but its precise cause is still not fully understood. Occasionally it appears also on pear and quince. It occurs even in the irrigated orchards of North America and Australia, and is a serious problem to these fruit exporting countries, for apples apparently normal when despatched may be affected with bitter pit when delivered.

It was first described in Germany, under the names *Stippen* and *Stippigkeit*, where Jäger thought it to be caused by rapid evaporation. In 1892 Cobb in New South Wales coined the name bitter pit. In Denmark it is troublesome in many orchards and enforces early picking and consumption of the fruit. In England its occurrence is seasonal and erratic, being worst in years of drought or fluctuating rainfall.

Palm (1924) and Atanasoff (1933) suggested that bitter pit was due to a virus. This view is generally discredited today. Claims that it is due to boron deficiency or can be prevented by boron manuring appear to be due to confusion with corky core or drought spot. Levy

and Roach found that it was not reduced by injecting the trees with boron compounds, and Wallace and Jones (1940) found no evidence that it was connected with a deficiency of any of the known essential minor elements. In Denmark also manurial experiments with manganese and boron have given negative results. Many lines of evidence indicate that bitter pit is a physiological disorder bound up with disturbances in the water relations of the fruit. It is a general experience that the trouble is most prevalent in large apples. Correlated with this is its occurrence especially on young trees and in light crops. Heavy applications of farmyard manure or of nitrogenous and potassic fertilizers tend to encourage it.

From the earliest investigations it has been noted that marked variations in water supply towards the time of fruit ripening were dangerous. Excessive warmth is also harmful. It is not, however, a dry soil in itself that is responsible for bitter pit. In Australia the disease is more prevalent in hollows than on well-drained slopes. In irrigated American orchards bitter pit is worst when a sparing water supply in early summer is succeeded by heavy watering towards picking time. It is encouraged by a soil with low humus content and low water-retaining capacity, and in Denmark it has been noticeably reduced by mulching the soil with straw or seaweed. McAlpine noticed the beneficial effect of green manuring as early as 1915. Grass orchards usually yield smaller fruit with less bitter pit. Soil reaction seems to play no definite rôle in the disease. American experiments have shown that excessive foliage production encourages bitter pit and when the trouble develops on the tree it may perhaps be due to the leaves withdrawing water from the ripening fruit in periods of temporary water shortage. Bitter pit seems, indeed, to have features in common with blossom-end rot of tomato (Smock, 1941).

The effect of the time of picking on bitter pit development in storage has been thoroughly investigated. The factor involved is the degree of ripeness of the fruit, which is partly a question of the conversion of starch into sugars and can be estimated by an iodine test (Bigelow *et al.*, 1905; Carne *et al.*, 1929). Most workers have found least bitter pit developed after late picking, but somewhat fluctuating results have been secured at Long Ashton. In a picking experiment with Venus in Denmark we found most bitter pit in the late harvested fruit. Cold storage reduces the tendency to develop bitter pit but varietal peculiarities have to be taken into account. Microscopic examination shows that the diseased spots contain starch that has not been transformed into sugar in the usual way. Probably when a water shortage arises these patches of cells poor in sugar were unable to secure water in competition with the rest of the flesh and this may be why they died and turned brown.

Bitter pit has been observed in the following apple varieties in Denmark; those particularly prone to it are printed in bold type.

In England, Newton Wonder is particularly susceptible both before and after picking.

Allington	James Grieve
Arreskov	Keswick Codlin
Baldwin	Lane's Prince Albert
Beauty of Kent	Lord Lambourne
(Springrove)	Lord Suffield
Belle de Boskoop	Manks Codlin
Bismarck	Marie Grejsen
Blenheim	Marselisborg Som-
Bramley's Seedling	meræble
Charles Ross	Mølleskov
Coulon	Nonnetit Bastard
Cox's Orange Pippin	Northern Spy
Early Victoria	Pederstrup
Ecklinville's Seedling	Purpurrød Cousinot
Elmelund	Ribston Pippin
Frogmore	Rødt Ananasaeble
Graastener	Signe Tillisch
Guldspir	Sønderskov
Hans Mathiesen	**Tyrrestrup**
Husmoder (Bellefleur)	**Venus**
Hvid Astrakan	Warner's King
Høve Reinet	Wealthy
Ildrød Pigeon	

Control. With young trees and susceptible varieties it is advisable when pruning and grafting to aim at a large crop of medium-sized fruit. Manuring should be sparingly

done and nitrogenous manures should be withheld until really necessary. Mulching with straw or seaweed or growth of a cover crop is advisable.

Fruit should not be picked unripe; the correct time for picking must be determined on the spot. Cold storage helps to reduce bitter pit in the harvested crop. At the first sign of its appearance steps should be taken to use the consignment as soon as possible.

Addition of manganese sulphate to the Bordeaux spray or possibly also to a lime sulphur spray may be tried, at the rate of about 2 lb. per 100 gallons.

This treatment has sometimes given beneficial results in Denmark, but not always.

Older trees which consistently yield fruit with bitter pit should be reworked with a less susceptible variety.

Lenticel Spotting

These spots have been most studied in North America where they are known as Jonathan spot, because of their prevalence on that variety. They appear during storage and four types have been distinguished, as Jonathan spots A, B, C and D. It is not yet clear whether or not these are well-defined distinct diseases caused by different storage conditions.

Spot A is a very small sharply defined spot round the lenticels and shows no tendency to expand (Fig. 78).

Spot B forms a ring round the lenticel (Fig. 79). The ring is due to a colour difference between the central and marginal parts of the spot. It eventually darkens throughout and thus resembles spot C.

Spot C is a dark round spot which is not always centred on a lenticel. The spots are numerous and gradually increase in size until they run together (Plate 7, Fig. C). The tendency for spot formation to occur round the lenticels seems most pronounced when the fruit ripens quickly at a high temperature. If the apples are removed from the storeroom the spots seem to fade and turn brown.

We have given the name Jonathan spot D to spots found on Bellefleur (Husmoder) from Blangstedgaard in 1937. These were small, brown, slightly sunken and mostly about $\frac{1}{25}$ inch across, occasionally up to $\frac{1}{12}$ inch. They did not penetrate the flesh (Fig. 80). In England a form of lenticel spotting is characteristic of the variety Allington Pippin. In Australia a very detailed account of these and other non-parasitic blemishes of apples has been published by Carne (1948).

Cause. In most cases the real cause of the spotting is unknown. The discoloration is due to colour changes in the skin. The cells of the flesh immediately below the spots have a lower acid content than the others. This may partly explain the change in colour as the colouring matter anthocyanin changes from red to blue-black when acidity is reduced.

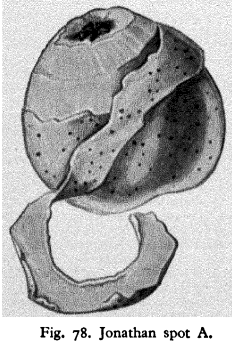


Fig. 78. Jonathan spot A.

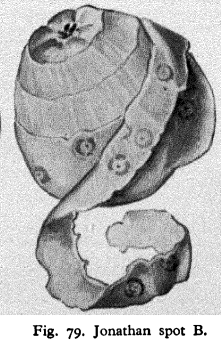


Fig. 79. Jonathan spot B.

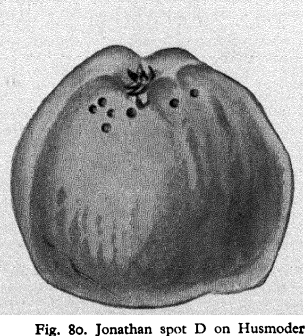


Fig. 80. Jonathan spot D on Husmoder (Bellefleur).

Apples placed in cold storage immediately after picking, when they had a high acid content, developed less Jonathan spot than those whose acidity had been reduced by storage at a high temperature.

Importance. In North America these spots may be very serious, especially on highly coloured varieties like Jonathan. In other varieties they occur mostly on the coloured side of the fruit. Wealthy is another susceptible variety. We have seen the condition mainly in storage experiments at Blangstedgaard, occasionally elsewhere. No parasite has ever been demonstrated as the primary cause of the spots, though fungi frequently penetrate the lenticels within the spots and start secondary rots. At Blangstedgaard we have seen Jonathan spot on Beauty of Kent, Bismarck, Bramley's Seedling, Phillippa, Graastener and other varieties, both green and coloured. On late varieties such as Bramley's Seedling and Laxton's Superb a form of lenticel spotting develops when they are stored near ripe Worcester Pearmain or James Grieve. In such cases the trouble seems to be due to ethylene gas given off by the ripe fruit (Kidd and West, 1938).

Control. Various storage methods, such as improved ventilation and the use of oiled paper wraps, or specially impregnated paper designed to keep up the acidity of the fruit, have been tried abroad and at Blangstedgaard, but have not given consistent results. On the whole, storage in carbon dioxide chambers has given the best results, whereas low-temperature storage and wrapping in oiled paper has several times proved unsuccessful (Esbjerg, 1941).

Manurial treatment seems to have some influence on the occurrence of this disease, but the relationship is not yet fully understood. At Blangstedgaard large apples have proved more liable to spotting than small ones (Esbjerg, 1938). Owing to the risk of spotting by ethylene it is inadvisable to store different apple varieties together. Ethylene can be removed from the air of an apple store by the use of brominated activated carbon (Smock, 1944).

Glassiness (Water Core)

Symptoms. Glassiness is not noticeable, either in apples still on the tree or in storage, until large portions of the flesh have become glassy and waterlogged, extending from the skin right through to the core. In the early stages, however, the glassy tissue occurs only around the vascular bundles surrounding the core. In later stages of the disease the whole flesh is waterlogged, the walls surrounding the compartments of the core crack and are covered with hair-like outgrowths which ultimately turn brown. The glassy flesh has a flat, sweet, or rather musty flavour. Glassiness never appears until the fruit is nearly ripe. When it is only feebly developed it may disappear during storage but in severe instances breakdown occurs and the glassy flesh soon turns brown (see Plate 4, Figs. A, B and C).

Cause. Brooks and Fisher (1926b) and Fisher and Harley (1930) in North America have investigated the development of water core or glassiness in apples. They found that apples freely exposed to sunlight showed a strong tendency to water core whilst those that were shaded were usually entirely free from it, except in very late pickings. Sunburned apples were extremely susceptible to water core.

They also showed that some of the changes taking place in the glassy apples resembled those occurring during normal ripening, but they occur much more quickly in glassy fruit. Under the influence of high temperature premature conversion of starch into sugar occurred, bringing about an abnormally high osmotic pressure in the cells, which led to the tissues becoming gorged with water. Glassy tissue has a water content about 20% higher than normal apple fruit flesh. The exposed side of apples had a higher sap concentration than the shaded side and was more susceptible to water core.

The prevalence of glassiness varies with the season. In Denmark it seems to be most plentiful when in August there are high day temperatures and cold dewy nights. In America the number of glassy apples on 13-year-old Delicious trees was proportional

to the number of leaves per apple. Heavy nitrogenous manuring favoured glassiness (Harley, 1938).

A Husmoder tree in a Zealand garden has been observed to bear normal apples when the crop was heavy and glassy ones in the following year when the crop was light.

Importance. Glassiness is not really a storage disease nor is it usually very important. In 1934, however, at least 75% of the fruit on Colouns Reinet was glassy at one place in Lolland.

In America, Brooks and Fisher (1926a) found that glassiness may disappear in the course of 3–5 months' storage and that apples recover from it nearly as well in cold storage as at ordinary room temperature. Small apples recover more readily than large ones and there is also a difference in varietal capacity for recovery. Plagge, Maney and Picker (1935), however, consider that apples recover better in ordinary storage than at low temperatures.

Varietal susceptibility. Glassiness has been observed in the following varieties in Denmark: Beauty of Kent, Bodil Neergaard, Bramley's Seedling, Charles Ross, Coulons Reinet, Graastener, Guldborgæble, Husmoder, White Astrakhan, Høve Reinet, Lane's Prince Albert, Mølleskov, Rival, Roundway Magnum Bonum, White Transparent, Venus and Warner's King. To these must be added Morsø Glassæble, which is always glassy. In England the condition is associated mainly with Rival.

Control. No practical method of preventing glassiness is known. Affected fruit should be picked as soon as it is recognized and used quickly. It is of poor flavour and keeping quality but is still usable.

Hollow or "Telescoped" Fruit

Symptoms. At picking or during storage some apples may be found to have a hole straight through the core so that one can look right through them from the eye to the stalk (Fig. 81). As a rule there is a more or less distinct furrow down each side of the split. If the fruit is cut through along the line of the furrows the flesh is found to be continuous along only a narrow strip on each side while the whole core and its surrounding tissue is grey or black and often soiled by droppings of earwigs or other insects (Fig. 82). Various fungi, such as species of *Fusarium*, may gain a foothold and set up a rot in the core.

Cause. The most probably explanation is that at some time during the summer the apples were exposed to a period of wet weather which caused sudden rapid growth. The woody tissue in the core was unable to keep pace with this and split. Hollow apples are usually big ones. In 1933 the condition was most prevalent where the apples were lop-sided because of very rich manuring.

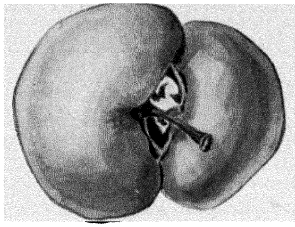


Fig. 81. Hollow fruit, Belle de Boskoop.

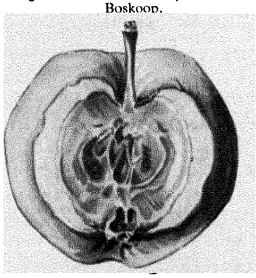


Fig. 82. Same fruit as Fig. 81 cut open.

Importance. The condition occurs every year in Denmark, especially in Belle de Boskoop, but we have also seen it in Bramley's Seedling, Cox's Pomona and Coulon. In England it occurs in Warner's King (Wormald and Harris, 1937). As a rule only a few apples are affected, but on trees bearing a small crop of large fruit as many as 30% may be "telescoped."

Control. As the fruit cracks most easily

when rain follows drought it may possibly be prevented by watering the ground before it becomes really dry. The disease is of too little importance to have stimulated research either into its cause or methods of prevention.

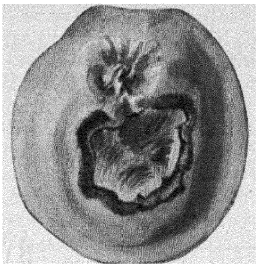


Fig. 83. Sun scald on apple (cf. Fig. 84).

Sun Scald

Symptoms and cause. Damage to the "sunny side" of apples occurs sometimes in warm summers and is attributed to sun scald. A superficial grey spot becomes distinguishable from the healthy tissue and then becomes brown and sunken. The spots may be as big as peas or may be up to an inch across (Fig. 83). The large spots not infrequently occur on Pederstrup, the smaller ones on Bramley's Seedling. Ware (1932) described a similar condition on Bramley's Seedling and Allington Pippin in Kent, after an abnormally hot August. In North America Brooks and Fisher (1926a) described a somewhat similar condition which appeared when high temperatures succeeded a relatively cool period. It was also favoured by drought and was less prevalent on small, poorly developed apples than on large or normal-sized fruit. Apples exposed to the sun throughout their growth were less affected than those hitherto fairly well protected from it. Brooks found that the sunny side of an apple may have a temperature 9–18° higher than the shady side and up to 23.5° F. higher than the air on the shady side. Hot sunshine may also cause brown spotting between the lateral veins of the leaves.

Control. Very little can be done to prevent sun scald. Possibly watering in dry weather may be helpful as drought apparently makes apples more liable to suffer in this way.

Heat Damage

When apples are exposed to high tempera-

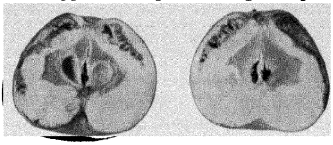


Fig. 84. Heat damage on apple (after Banga).

tures after a period of low temperatures, patches of tissue may die below the skin. These dead patches dry up, causing cavities to appear in the flesh so that the surface of the fruit becomes uneven. The damage is less visible from the outside than in sun scald but the two conditions probably merge into one another. (Fig. 84). We have never seen typical heat damage but it has been described in Holland by Banga (1938). Affected apples may also become glassy.

Frost-blistered Leaves

In some years a number of apple leaves are found to have blisters on their upper surface. The epidermis separates from the underlying tissue and cracks so that the green flesh is exposed like a powdery mass (Fig. 85). Growth of the leaves is checked, but they do not fall.

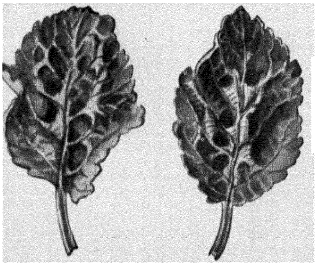


Fig. 85. Apple leaves damaged by frost.

The damage is due to low temperatures when the leaves are small and the blistered appearance is due to the growth of the lower surface of the leaves being checked by the cold. The leaves surrounding the flower trusses

are most exposed, as they are the first to expand.

It may be noted that the apple variety Oudrupgaard's Høstæble always has blistered leaves.

Frosted Blossom

Late spring frosts are a well-known hazard in orchards. The affect of frost on apple blossom has been studied in Wisconsin by Roberts (1946) who found that damage was largely to the pistils and ovules. Frost also loosened the epidermis on the receptacle of the blossom but the injury tended to heal very quickly. The buds showed a marked decrease in hardiness as they approached the full bloom stage of blossoming.

Frost Stripes on the Fruit

Corky, scurfy patches on the skin of apples may be due to many causes, including capsid injury and spray damage. Occasionally they are due to frost and then differ considerably in appearance on the various apple varieties. According to W. C. Moore (1943) the most familiar is a corky ring round the eye, as in Bramley's Seedling, but the corky area may form a band round the middle in Worcester Pearmain and Lane's Prince Albert; it may occur more or less all over the fruit or only on the cheek, as in Cox's Orange Pippin. In 1933 and 1938 Danish apples were often marked with corky stripes extending from the

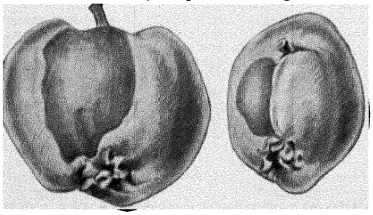


Fig. 86. Corky stripes due to frosting of the young fruit.

stalk to the eye (Fig. 86) which were attributed to frost damage. According to Clinton (1914) and Gardner (1927) the damage is done by frosts when the fruit is quite small or even during blossoming. Banga (1938) has described corky patches round the eye due to frost soon after fruit set.

Frost Damage to Ripe Fruit

Symptoms. Internal discoloration of apple flesh by frost takes several distinct forms (see Plate 5, Figs. A, C and E). In slight cases the flesh is not discoloured but the vascular bundles are more or less distinctly stained brown. In cross-sections of the fruit they appear as brown specks, in longitudinal sections as brown lines. When the fruit is thinly peeled the bundles may show up as a brown network. There may also be a brown stain round the core. Lightly frozen apples become floury and have a slightly yeasty flavour.

With severe frosting the inner flesh becomes glassy, watery and eventually brown, often with a yellow or greenish tint, and similarly discoloured streaks radiate towards the peel. The flesh becomes mealy.

External symptoms consist of discoloration in the outermost layers of cells. One form of frost damage results in discoloured patches of skin with ill-defined edges, like bruises. Another is confined to red apples or to the red side of the fruit. It consists of irregular, often oblong, curved, yellow-brown patches with irregular reddish-brown edges. The patches may remain unchanged in storage or may become sunken slightly, turn darker brown and increase in size.

The symptoms are said to be due to injury to the cells surrounding those originally killed by the frost. The tissue beneath the patches may die so that holes form, surrounded by dead, brown, corky tissue.

Symptoms of frost damage do not appear as long as the apples remain frozen, but develop during thawing and gradually grow more distinct. The shape of a frozen apple is easily altered by pressure from the surrounding fruit.

Cause. Ice formation in apples may begin at temperatures between 29.5 and 27.7° F., depending, among other things, on the sugar and salts content of the fruit. Apples that are completely undisturbed may be cooled several degrees below these temperatures without ice forming in them and without receiving any damage.

Importance. Frost damage to the ripe fruit

may be important in three different circumstances:

(1) frost in autumn before the fruit is picked;

(2) frost during transport;

(3) frost in the storeroom either because it is not well insulated or because the temperature of a cool storage room has accidentally been allowed to fall too low. Frost damage is not as a rule of great importance in Denmark or in England; it is a much greater problem in U.S.A.

Varieties differ in their reaction to frost. For example, in the winter of 1936–7 we saw Cox's Orange Pippin and Pederstrup damaged when many other varieties were not. In one instance the damage was done by a single night's frost while the fruit was still on the trees; in another it occurred in store.

Control. Care should be taken not to store apples where they are likely to be frosted and to ensure strict control of temperature in cool storage chambers. One fruit grower has found he could make his fruit storeroom, formerly penetrated by severe frost, frostproof by spraying it several times with water during frost. If apples have become frozen they should not be disturbed until after they have thawed. The damage is usually much less if they are left untouched to thaw slowly at a temperature between 32 and 40° F. If the freezing has been very severe, however, slow thawing will doubtless be of no avail.

If the fruit freezes on the trees it should not be picked while frozen. If the weather immediately after the frost is cloudy and cold the damage is likely to be less than if it is warm and sunny.

Frost Damage to Apple Trees

The severe winters 1940–2 caused much damage to apple trees in Denmark. Many trees were killed during the first winter, others seemed much damaged in the autumn of 1940 but recovered better than was expected, while others again which appeared little affected at first gradually showed dieback in more and more branches. Even after the mild winters 1943–4 some more of their branches died. The frost-weakened trees developed numerous cracks in their bark, which later fell off in sheets leaving the wood bare. Belle de Boskoop, Bramley's Seedling and Cox's Orange Pippin were particularly affected. More damage was no doubt done to late varieties like Cassler Reinet and Koldemosegaard, but these are little grown so that the damage to them was less noticeable.

The importance of well-ripened wood was noticed at many places. Thus almost everywhere the trees most damaged were those that had borne most fruit in 1939; those that had little or no fruit that year were undamaged. The damage was also clearly worst in low-lying areas. Often the lowest branches were most damaged, perhaps because they were most shaded and unable to store as much food as the others. Damage done to fruit trees at East Malling in the winter of 1939–40 has been described by Modlibowska and Field (1942). No trees or branches were killed outright, but bark was cracked and loosened and year-old twigs and spurs were injured. Apples that had received a heavy potash manuring suffered more than those with less potash. Fastening down the loosened bark with small nails in two rows on each side of the longitudinal cracks and sealing the cracks with vaseline led to more rapid and satisfactory healing.

Low-temperature Breakdown in Fruit

In English-speaking countries this trouble has been referred to under many names: flesh collapse, internal breakdown, Jonathan scald, physiological decay, soft scald and soggy breakdown. Some of these may lead to confusion with another trouble called internal breakdown or brown heart.

The disease is not due to frost, but appears when fruit is stored at low temperatures. Its appearance varies according to variety and treatment.

In the early stages the fruit looks quite normal from the outside in that there is no discoloration of the skin, for the tissue just below the peel is quite healthy. The apples have a rather pale look, however, and feel somewhat soft so that an experienced packer

can detect the damaged ones at quite an early stage. When they are cut through a faint yellowish-red tint appears in the flesh. As the disease progresses the flesh turns brown in a ring surrounding the core and ultimately the skin too turns brown (see Plate 6, Figs. B and C).

Before there is any visible sign of disease the initiation of the trouble can be detected by an alcoholic flavour which permeates the whole flesh. Visible symptoms appear soon after. According to Osterwalder and Kessler the brown tissue may contain 60 mg. or more of acetaldehyde per Kg. of fruit. Small brown spots may appear in the peel which gradually enlarge. In severe cases a complete ring of soft brown, soggy tissue surrounds the core which remains white and sharply separated from the brown flesh.

When fruit is cut immediately after being removed from the cold storage the broken-down tissue is found to be damp, hence the name "soggy breakdown," used in North America. When stored at higher temperatures this tissue soon dries and merges with the sound flesh so that the fruit appears over-ripe. The apples look almost as though baked, and when stored for long periods the damaged parts become shrivelled, corky and cracked. If other damage is present in the flesh, e.g. core rot, a white zone always remains between the two types of injury. During prolonged storage the individual fruits affected become worse but their number does not increase.

Another form of the trouble, which we call "soft low-temperature breakdown," occurs as elliptical light-brown patches $\frac{1}{8}$ to $\frac{1}{2}$ inch across, typically curved and of varying length and $\frac{1}{16}$ to $\frac{1}{8}$ inch deep. They often surround or nearly envelop patches of healthy tissue which remain as islands or peninsulas (Fig. 87). In the early stages only the skin is discoloured and the trouble then resembles scald, but the underlying tissue soon becomes soft. The chief difference between this type of cold injury and that described first is in the external appearance. In North America it has been called "soft scald" but we dislike this term as the trouble has no connection with true scald. The trouble may appear early in storage. In American Jonathans it appears between 15th November and 1st January. In 1937 we observed soft low-temperature breakdown about 1st April on Ellison's Orange and on fruit from a seedling. On the latter the damage first appeared soon after Christmas in fruit stored at 35–37° F.

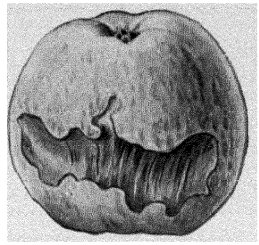


Fig. 87. "Soft low-temperature breakdown" on Ellison's Orange.

Cause. Low-temperature breakdown occurs when apples are stored at temperatures too low for them to carry out their normal storage development.

Plagge and Maney (1935, 1937) thought soft low-temperature breakdown dependent on the stage reached in conversion of nutrients in the fruit before it entered the store. At 33.8° F. (+1° C.) the risk of soft low-temperature breakdown is greatest if the fruit is respiring actively. If they are stored before or after the period of rapid respiration they are much less likely to develop the trouble. At 36.5° F. the apples are said to be less liable to this particular form of the injury, but we have seen it in Denmark in fruit stored at that temperature.

Sometimes the damage is worse if the apples are placed directly into cold storage and less if they experience delayed chilling.

Various orchard conditions also influence the amount of injury that develops in cold storage. Conditions making for spongy-fleshed fruit increase its susceptibility to damage by low temperatures. Heavy rain in the months immediately preceding picking, overmanuring, especially with nitrogen, poor cropping of vigorous trees, severe pruning and poor growth

all increase susceptibility to the trouble. Apples are easily damaged if they are picked when over-ripe or under-ripe. Some varieties are much more susceptible than others. In Denmark we have seen serious damage to Belle de Boskoop, Pederstrup and Sønderskov, amongst others. Of American varieties listed as susceptible only Jonathan and Wealthy are grown in Denmark.

At Blangstedgaard Pederstrup apples from dwarf trees, grafted on East Malling stocks IV and V, suffered when stored at 33.8° F., while fruit from trees on a wild stock were practically unharmed. Baldwin, Fameuse and MacIntosh have been resistant at Blangstedgaard. In American experience an atmospheric humidity exceeding 80% in the store is unfavourable. Increasing the percentage of carbon dioxide seems to increase resistance.

Importance. Low-temperature breakdown was noticed particularly after cool-storage plants had been established in Denmark. Soft low-temperature breakdown seems to be rather rare, but the other type of injury is not unimportant in cool-storage houses.

Control. One should try to avoid orchard conditions conducive to low-temperature breakdown and to pick the fruit at the correct stage of ripening.

The limiting temperature usually lies somewhere between 35.5 and 41° F. but may vary greatly from year to year. As for various reasons it may be desirable to store the apples at a lower temperature than 41° F., Banga (1938) recommends one of the following procedures:

(1) Keeping the apples at 35–37° F., but storing a sample of at least 50 fruit from the same consignment at 33.8° F. and examining it at regular intervals. When low-temperature breakdown is seen in this sample it may be expected to appear some weeks later in the rest of the consignment which should therefore be used before the damage has time to break out.

(2) Storing a sample of a particularly susceptible variety at a low temperature. From the time it takes to show signs of low-temperature breakdown one can judge whether the season has been one to produce fruit of high or low susceptibility and when injury appears in the susceptible variety the temperature of the storage chambers can be raised. Westeren's Wijnzuur is used for this purpose in Holland.

Storing in an atmosphere rich in carbon dioxide may sometimes check low-temperature breakdown, but not always. Gas storage of Bramley's Seedling has been recommended in England. In America successful results have been obtained by a few days' storage at a comparatively high CO_2 concentration, followed by storage at 32° F.

Brown Heart and Core Flush

There are three types of browning round apple cores: general browning, *Fusarium* core rot and brown heart.

General browning is only a form of over-ripeness which first manifests itself by the flesh turning brown near the core. Gradually the brown discoloration increases in intensity and extent until the whole apple is brown and unusable. As warmth accelerates ripening, general browning can be delayed by cool storage.

Fusarium core rot is a brown rot commencing at the core, which is stained red by the mycelium of a species of *Fusarium*. The fungus may be present before the fruit is picked but the rot is not seen unless the apples are cut. During storage it may spread outwards until the whole fruit is rotten. Possibly more than one species of *Fusarium* may cause this trouble. The disease seems to be more troublesome in Norway than in Denmark. No control measures are known. Core rot is also sometimes associated with a species of *Alternaria*.

Brown heart is a more complicated phenomenon.

Symptoms. In slight attacks, small, sharply defined spots, often very numerous, arise inside the flesh. They commonly start in the main vascular bundles surrounding the core. When the discoloration only affects the flesh next to the core the trouble is called " core flush " (Kidd and West, 1936, 1937), but it is often difficult to distinguish between the various forms of core rot. In serious cases a large part of the

interior of the apple is affected while the outer tissue remains healthy (Plate 6, Fig. A). When the affected flesh dies it easily dries and partially collapses, leaving cavities lined with brown cells. The apple then has a peculiar springiness but only in very extreme cases is it possible to see from the outside that the flesh is affected. When the cause of the trouble is removed spread of the injury stops. Core rot can be demonstrated by X-ray photographs of apples (Esbjerg, 1938).

Cause and importance. According to Kidd and West's investigations between 1923 and 1936, brown heart is due to the combined effect of excessive carbon dioxide and insufficient oxygen in the storeroom.

The amount of CO_2 required to produce the disease varies with temperature, ripeness of the fruit, variety and even individual apples. Esbjerg (1938) found the number of fruit browned at the core varied greatly with the variety stored. Thus after storage to 24th January, at 38.3° F. with outside ventilation, Beauty of Kent, Cox's Pomona and Philippa had no brown heart, Bellefleur had 60%, Bismark on wild stock had 97% and Bismarck on stock IV had 90% of the fruit affected. Stored under similar conditions, but at 34.7° F., Philippa, Beauty of Kent and Cox's Pomona were still undamaged, while the three others had 17%, 7%, 50% of the fruit diseased respectively. Not until storage was prolonged to 24th February did Cox's Pomona become affected, with 7% at 38.3° F. and 27% at 34.7° F. Bodil Neergaard is particularly prone to brown heart if kept long in store (its flesh may also go brown between the core and the peel in an ordinary storeroom). The damage may take place any time during storage but appears in its most characteristic form just before the apples ripen and go soft.

Brown heart appears where ventilation is bad, as in apple cargoes between Australia and England, where the cases are tightly packed in the ship's hold.

Control consists in preventing accumulation of carbon dioxide to an excessive extent. Susceptible varieties should not be kept very long.

Scald

This disease is known in fruit stores all over the world.

Symptoms. The disease may show itself as large or small, connected or scattered blotches, varying in colour from cinnamon to dark chocolate. The skin may become somewhat wrinkled. In mild cases the apple merely develops a brown tint and the skin remains smooth but its appearance is spoilt and its market value impaired. Generally the discoloration is quite superficial and affects only the 5 or 6 outermost layers of the flesh (Fig. 88, and Plate 8, Figs. A, C and E).

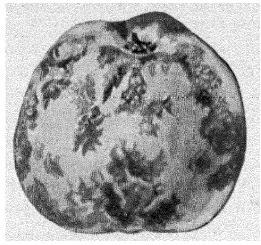


Fig. 88. Scald on Bramley, sharply defined but irregular patches.

Scald may appear in the storeroom, but often it first appears on cold stored fruit when they reach the market. Then, when kept at ordinary room temperature, they may develop a brown tinge about 12 hours after removal from store. Typical brown discolorations of the skin may appear in 24 hours, and in a few days the fruit may be so seriously affected as to be unsaleable. In Western Europe the brown skin appears as a rule before the apples reach the consumer. In colder climates, such as some of the North American states, the damage does not become visible until the consumer has bought the apples and placed them in a warm room. In later stages of scald the skin cracks, moulds like *Penicillium* enter and cause rotting of the flesh.

On Belle de Boskoop at Blangstedgaard we have seen an injury which we provisionally call "deep scald." In its most characteristic form the fruit bears only a few slightly sunken spots $\frac{1}{4}$–$\frac{1}{2}$ inch across.

Cause. Brooks, Cooley and Fisher (1919, 1923a), find that scald is due to the production by ripening fruit of volatile esters which poison the tissues if they are allowed to accumulate either in the flesh or in the surrounding air. Typical scald can be artificially produced in a few days by exposing apples to the vapours of ethyl acetate, amyl acetate or methyl butyrate. Scald is therefore a kind of self-poisoning of the fruit and is not due to accumulation of carbon dioxide (see brown heart, p. 143). Indeed, in American experiments accumulation of carbon dioxide to the extent of 1–6% of the air did not encourage scald, but appeared to check its development by delaying the ripening of the fruit. Similarly storage at 32° F. delays appearance of scald, especially if the fruit was packed in prime condition. Its development is accelerated by increase in temperature up to about 59–68° F.

The tendency of apples to scald varies from year to year, but the factors responsible for this variation are not clear.

Conclusions based on experiments with American varieties grown in America cannot always be applied elsewhere, and it is necessary for experiments to be conducted wherever fruit storage is being undertaken. It may even be necessary to work out the reaction of each variety under the local conditions.

In North America fruit packed green is much more susceptible than that allowed to ripen fully on the tree, and scald develops most on the green side of partly coloured apples. Very red varieties are found to be most resistant, while yellow ones are more resistant than green or than those changing from green to yellow. On the whole a poorly coloured fruit is more susceptible than a well-coloured one. At Blangstedgaard, however, severe scald is often seen on red apples. In America fruit from young trees is attacked more than that from older ones. In fruit from irrigated districts that borne by well-watered trees is affected more than that from lightly watered ones, irrespective of the size of the apples.

Importance. Scald has been known in North America since the beginning of this century, and in 1920 it was said to cause more loss in stored apples than all other diseases combined. Since then control measures have been perfected to such a degree that scald is no longer troublesome there.

Little attention has been paid to the disease in Denmark. It was first mentioned by Esbjerg in describing storage experiments with fruit at Blangstedgaard in the years 1918–22. Latterly, as fruit storage has developed, scald has become better known and often causes considerable loss.

Varietal susceptibility. Most of the literature on this subject relates to American varieties not grown in Europe.

At Blangstedgaard and elsewhere in Denmark scald has been seen on Alfriston, Beauty of Kent, Belle de Boskoop, Boiken, Bramley's Seedling, Graastener, Bellefleur de France, Jonathan, Lane's Prince Albert, Mølleskov, Newton Wonder, Nonnetitte and Philippa. Annie Elizabeth is said to be susceptible in England. This list of varieties will doubtless be greatly extended for probably most kinds of apple may develop scald.

Control. At Blangstedgaard (Esbjerg, 1941) storage in carbon dioxide chambers and in cold stores has always given less scald than storage in ordinary warehouses. In Rhode Island, Pieniazet and Christopher (1945) found that storing apples for 3–10 days at 32° F. in an atmosphere containing 30 or 60% carbon dioxide before they were placed in the ordinary cold-storage room gave very good control of scald in the variety Rhode Island Greening. Brooks, Cooley and Fisher (1923b, 1924) found that wrapping the fruit in oiled paper gave almost complete control. In Denmark this seems to hold good with some varieties only.

The method consists in wrapping the apples immediately they are picked in thin wrapping paper to which oil has been added, to the extent of 15–20% of the paper's original weight. A light, tasteless and scentless mineral oil should be used. Waxed paper is much less effective and ordinary wrapping paper is of little use. The oiled paper absorbs the odorous gases emitted by the fruit. Hence when apples are packed in barrels or hampers almost equally

good results have been obtained by scattered shredded oiled paper between the layers of unwrapped fruit. This method is quicker than wrapping. About 1 lb. of oiled shredded paper is used to an ordinary case of apples.

Circulation of air in the store is important to prevent the noxious vapours from accumulating around the apples. Unfortunately circulation of the air has given less satisfactory results in Denmark than in America. According to the American writers the critical period for development of scald is just after packing. Though the disease does not appear at once its foundation is laid at that time. Afterwards neither wrapping in oiled paper nor thorough circulation of air can repair the damage or prevent outbreak of scald in the store or after the apples are removed for sale. Banga (1938), considers danger of scald is reduced if the apples are allowed to lose a little of their moisture before going into cold store. The fruits should be picked when the stalk becomes loose, but while they are still hard. Kidd and West (1937) find that the earlier Bramley's Seedling is picked the more superficial scald it will have, but the longer it is kept at room temperature before storage the less scald will develop.

Over-ripeness (Mealy Breakdown)

Symptoms. This can hardly be called a disease as it is the natural outcome of senescence. The chief symptom is that the flesh instead of being firm and juicy becomes soft, dry and floury and ultimately turns brown.

In some varieties the change begins at the core, in others it starts with a softening or browning of the flesh under the peel.

Storage conditions may influence the onset of mealy breakdown (see general browning, p. 143), and it may also be affected or accelerated by the presence of bitter pit, sun scald, low-temperature breakdown or frost damage.

When over-ripening of the flesh has reached the peel large or small cracks develop in it (Plate 5, Fig. F).

According to Plagge, Maney and Pickett the trouble called "Jonathan breakdown" in American literature is a form of over-ripening.

Cause and importance. Over-ripeness occurs in apples stored too long or at too high a temperature, so that ripening is accelerated. Over-ripe apples are easily attacked by fungi such as *Penicillium* and *Botrytis*, especially in the later stages when the fruit has cracked. By that time they are quite worthless, so their destruction by fungi causes no direct loss, but they may form sources of infection for other apples.

Under uniform storage conditions early and semi-early varieties naturally develop mealy breakdown before late varieties.

When apples ripen very rapidly at high summer temperatures they may become floury and crack even while still on the tree. This certainly happens very seldom in Denmark but Banga has reported it in Holland.

Control. The storeroom must be kept as cool as possible without running a risk of low-temperature breakdown.

As very big fruit become over-ripe and floury most easily one should try to arrange the cropping so that the apples do not exceed a normal size.

A continual check should be kept on stored apples by taking periodic samples and cutting them across. If the least sign of over-ripeness is detected the rest of the consignment should be used quickly, as in the early stages it does not appreciably affect the quality of the fruit. Experienced persons can detect the onset of the condition by the rubbery feel of the fruit, without cutting it.

Summary of Apple Diseases

I. Roots

		page
A. Roots small:		
(1) Poorly developed	Potassium Deficiency	132
(2) Excessively fibrous	Nitrogen Deficiency	133
B. Normal size:		
(1) Roots dead, discoloured, rotten, smelling of alcohol	Suffocation	130
(2) Irregular lumps bearing bunches of fibrous roots	Crown Gall	129
(3) White or black " shoe-strings " under bark	*Armillaria* Root Rot	456
(4) Whitish fungus hyphae on roots, small hard bracket fungus at the soil level (very rare on apple)	*Fomes annosus* Butt Rot	508

II. Trunks, Branches, Twigs and Young Shoots

A. Shoots abnormally short:		
(1) Dead shoot tips associated with marginal leaf scorch	Potassium Deficiency	132
(2) Associated with narrow leaves, sometimes growing in rosettes	Iron Deficiency	36
(3) Scanty pale-green foliage	Nitrogen Deficiency	133
(4) Large leaves with dead spots on them, mainly on soils deficient in lime	Magnesium Deficiency	133
B. Shoots abnormally long and soft, bark may be thick and soft, frost damage may occur	Excess Nitrogen	133
C. Dead branches and twigs:		
(1) Large deep wounds, often with rings of callus	Canker	113
(2) Sunken bark, shoot tips frosted	Excess Nitrogen	133
(3) Brown bark with mucous drops oozing out (in North America)	Fire Blight	186
(4) Light and dark brown rings on bark, often round a dead spur. Grey or yellowish conidial pustules often present	Blossom Wilt	120
	Brown Rot	121
(5) Dead branches, small fungus pustules inside bark	Dieback	198
D. Lateral buds die or burst very late, especially lowest on shoots	Nitrogen Deficiency	133
E. Malformed branches:		
(1) Lumpy outgrowths bearing tufts of abortive roots	Burr Knots	130
(2) Smooth or rough swellings	Canker	113
(3) Flattened branches with longitudinal furrows, sometimes of a rubbery texture	Flat Limb	131
(4) Open wounds with rings of callus	Canker	113

		page
F. Damaged bark:		
(1) Bark dry and cracked across	Sun Scorch	27
(2) Bark dead and peeling off in sheets	Frost	141
	Canker (Papery Bark)	113
	Gloeosporium sp.	117
(3) Discoloured sunken patches, often round dead spur, often very long and narrow, ultimately conidial pustules present	Surface Canker	116
(4) Numerous small light-red wart-like conidial pustules	Coral Spot (*Nectria cinnabarina*)	555
(5) Sunken areas with concentric ridges	Canker	113
(6) Bark changed to a rusty powder (tan disease)	Excess Water	18
(7) Thick, soft bark	Excess Nitrogen	40
G. Brown stain in wood, small leathery bracket fungus, grey and velvety above, purple and smooth below, on dead wood	Silver Leaf	191
H. Damage to the current season's growth:		
(1) Blisters on bark, a blackish fungus layer inside	Scab	101
(2) Blisters on bark, yielding white conidial pustules in wet weather	Canker	113
(3) Blistered bark with NO fungus	Frost	141
(4) Scars, generally on one side of twig only	Hail Damage	20

III. Leaves

A. Superficial coatings of mould:		
(1) White and mealy coating	Mildew	111
(2) Dark or olive-green spots	Scab	101
(3) Sooty black coating, following aphides	Sooty Mould	126
B. Dead edges and blotches on the leaves:		
(1) Marginal scorching, spotting and discoloration after spraying or dusting	Chemical Injury	45
(2) Scorched edges	Potassium Deficiency	132
(3) Large dead patches between the veins	Sun Scorch	27
(4) Leaves wilt, die and remain hanging (only in North America)	Fire Blight	186
(5) Large leaves with dead spots between the veins	Magnesium Deficiency	133
C. Well-defined leaf spots:		
(1) Round brown spots or grey spots with brown edge, often bearing minute black dots	Leaf Spots	119
(2) Yellow spots with rust pustules underneath	Apple Rust	128
(3) Rather small yellow or cream-coloured spots	Mosaic	130
D. Discoloration of the whole leaf, often all leaves on the tree affected:		
(1) Slight silvery sheen to leaves, associated with brown stain in wood and often with papery bark	Silver Leaf	191
(2) Slight silvery sheen to leaves but wood and bark unaffected	False Silver Leaf	193
(3) Light-green, yellowish-green or reddish-yellow areas leaving main veins green	Chlorosis	36

(4) Yellowing or yellow-spotted leaves	Drought, or Irregular Water Supply	*page* 17
(5) Small light-green, yellowish or reddish leaves that fall early	Nitrogen Deficiency	133
E. Premature leaf fall	Spray Damage; Scab	45, 101
	Drought	17
F. Blisters and malformed leaves	Frost	139
IV. Flowers		
(1) White mealy coating to sepals	Mildew	111
(2) Dark spots on sepals and flower stalks, flowers fade and drop	Scab	101
(3) Whole flower trusses wilt and die, often spur killed also	Blossom Wilt	120
(4) Drops of slime ooze from wilted flowers (in North America)	Fire Blight	186
(5) Dropping of healthy blossoms		131
(6) Flowers few and late blooming	Phosphorus Deficiency	41
V. Fruit		
A. Young fruit falls in large numbers	June Drop	131
B. Peel damaged before ripening:		
(1) Russetting of the skin in large patches or all over the fruit, sometimes with reddish spots on the skin	Chemical Injury	45
(2) Scurfy streaks from eye to stalk or scurfy rings round the fruit or round the eye	Frost Damage	140
(3) Grey or brown sunken patch on side exposed to sun	Sun Scald	139
(4) Sunken scar, perhaps covering a pocket of dead flesh	Hail Damage	20
(5) Dark olivacious spots, turning black, often cracked	Scab	101
(6) Sooty " finger-prints " on the skin	Sooty Blotch	126
(7) Circular group of tiny black dots	Fly Speck	126
(8) Dark olivacious mould easily rubbed off	Sooty Mould	126
(9) White mealy coating	Mildew	111
(10) Orange spots with rust pustules (very rare)	Apple Rust	128
(11) Large fruit with poorly developed colouring	Excess Nitrogen	40
C. Diseases affecting the flesh of the fruit during the summer:		
(1) Fruit deformed with corky brown spots round the core and sometimes, also, under the skin	Boron Deficiency	133
(2) Dead tissue, often full of cavities, covering large areas under the skin	Heat Damage	139
(3) Small pits in the skin each covering a brown spot of flesh	Bitter Pit	134
(4) Glassy flesh	Glassiness	137
(5) Dead tissue through the core from stalk to eye	Telescoped Fruit	138

		page
(6) Soft brown rotting areas:		
(a) Large, quickly spreading patches with yellowish conidial pustules	Brown Rot	120
(b) Sunken spots, with small fungus pustules	*Glomerella*	125
	Gloeosporium spp.	124
(c) Light grey-brown patches with no conidial pustules	*Phytophthora*	125
(d) Small, rather dry, slow brown rot, mostly round the eye (see also *Botrytis*)	Eye Rot	123, 125
(7) Smooth, black rot	*Physalospora* Black Rot	125
D. Rots and diseases usually appearing during storage:		
(1) Rotting of the flesh associated with fungus:		
(a) Light-brown rot associated with green mould	*Penicillium* Rot	127
(b) Brown rot associated with loose grey mould	*Botrytis*	123
(c) Brown rot associated with fine rose-pink mould, especially round scab spots	Pink Mould	128
(d) Black rot with pycnidia in the skin	*Phacidiella*	118
	Physalospora	125
(e) Brown rot with whitish conidial pustules	*Gloeosporium*	124
(f) Smooth black apples without fungus pustules	*Sclerotinia*	121
(2) Superficial blemishes:		
(a) Small black spots, slowly broadening	Storage Scab	103
(b) Small dark spots, sharply defined, sometimes surrounded by a coloured ring	Jonathan Spot	136
(c) Small pale spots with brown edges round the lenticels	*Botrytis*	123
(d) Large, irregular, light or dark-brown blotches	Scald	144
(3) Discoloration of the flesh with no fungus associated:		
(a) Numerous dead brown spots under the skin	Bitter Pit	134
(b) Network of brown lines (dead vascular bundles) under the skin and sometimes round the core	Frost Damage	140
(c) Glassy patches of flesh	Glassiness	137
(d) Radiating strips of watery brown flesh	Frost Damage	140
(e) Mealy flesh with a yeast-like smell	Frost Damage	140
(f) Mealy flesh, sometimes turning brown, between the core and the peel	Over-ripening	146
(g) Brown rot starting round the core	Brown Heart	143
(h) Browning of the flesh spreading inwards to the core and outwards to the peel	Low-temperature Breakdown	141
(i) Superficial oblong sunken brown patches, soon turning soft and hollow and penetrating deeply into the flesh	Soft Low-temperature Breakdown	142

APRICOT (see also Peach)

Gummosis

Apricots are very liable to develop gummosis, even from small wounds (see p. 159). As far as possible one should avoid damaging the tree, especially the woody parts. Shoots that one will not want to retain at the autumn or spring pruning should be cut out during the summer if possible.

On the whole apricots suffer from the same diseases as peaches (see p. 178).

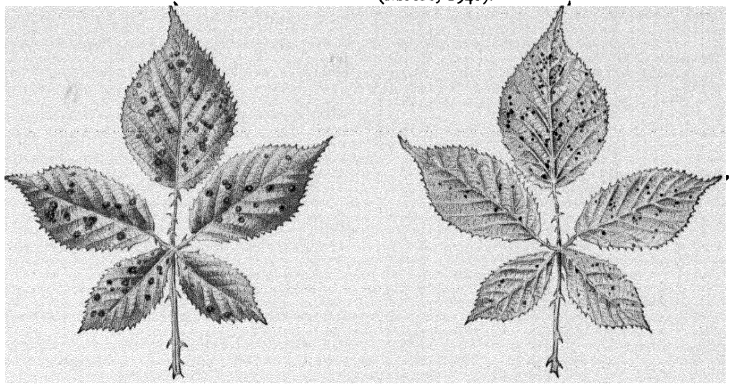


Fig. 89. Blackberry rust. (*a*) Upper-side of leaf with purple spots, white at the centre. (*b*) Under-side of leaf showing rust pustules.

BLACKBERRY (Bramble)

Rust (*Phragmidium violaceum* (Schultz) Wint.)

On the upper surface of the leaf the small spermogonia (male organs) appear first, followed on the lower surface by the powdery, orange-yellow, cluster cup stage. In this rust the latter occurs in rather irregular pustules, often arranged in rings. Later, small, round, cushion-like masses of yellow uredo spores appear on the under-side of the leaf and finally come black cushions of teleutospores. Conspicuous red or violet blotches, paler in the centre, on the upper surface of the leaf correspond to the uredo or teleuto pustules on the lower surface. Another rust, *Phragmidium rubi* (Pers.) Wint., distinguished by having usually six instead of four cells in its teleutospores, also occurs on bramble leaves especially near the sea. Instead of producing red blotches on the upper surface of the leaves over the spore pustules it causes small yellowish spots or none at all.

Stem rust (*Kuhneola uredinis* (Link) Arthur) forms long pustules with dusty yellow spores on blackberry stems, followed by small yellow pustules on the leaves. It is common on wild brambles and has been reported several times on the John Innes Thornless blackberries (Moore, 1948).

The only control measure we can suggest is raking up and burning or burying the fallen leaves in autumn to destroy the teleutospores. In severe outbreaks spraying with Bordeaux mixture or some other copper spray may be tried.

Leaf Spot (*Mycosphaerella rubi* Roark)

This disease appears as small round conspicuous spots with grey centres and dark purple borders scattered over the blackberry leaves. Each spot bears one or more minute black specks, the imperfect fructifications of the fungus, formerly known as *Septoria rubi* Westd. (see Fig. 137). Similar but elliptical spots up to $\frac{1}{4}$ inch long appear on the stems, where they closely resemble the lesions of

purple blotch. In severe attacks the leaves may become perforated, torn and fall prematurely.

Control measures include collecting and burning the dead leaves in autumn, for on them the perfect *Mycosphaerella* fructifications form during winter. Spraying as recommended against purple blotch may be necessary after a severe attack.

Purple Blotch (*Rhabdospora ramealis* var *macrospora* (Rob. & Desm.) Sacc.)

This fungus attacks the bramble stems and causes oblong brown patches with a reddish-violet margin (Fig. 90). Affected branches ultimately die.

Immediately after the fruit has been picked the affected branches should be cut out. A copper spray or dust applied once or twice before the fruit swells may check the disease. Late sprays are impracticable as they cause spotting of the fruit.

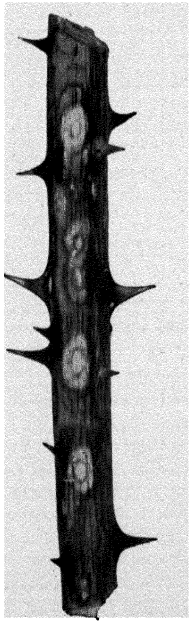


Fig. 90. Spots caused by *Rhabdospora* on blackberry stem, brown spots with purple edges.

Stamen Blight (*Hapalosphaeria deformans* Syd.)

A disease in which the anthers are destroyed and covered with the pycnidia of the fungus, from which ooze white masses of spores. Owing to inadequate pollination affected flowers set imperfect fruit. This disease occurs on cultivated dewberry, boysenberry and evergreen blackberry in North America and on wild bramble flowers in Britain (Noble and Gray, 1947), but has yet to be observed on cultivated blackberries in the British Isles.

Dwarf

A serious virus disease common on cultivated blackberries, cut-leaved blackberries, loganberries, phenomenal berries and youngberries in the South-east of England. Affected plants produce numerous small stunted shoots with distorted leaves, giving a witches' broom effect. They do not fruit and eventually die. Little is known about the disease and the only practicable control measure seems to be to root out and destroy all infected stools as soon as they are recognized. A mosaic disease of blackberries has also been reported several times (Moore, W. C., 1943).

CHERRY

Blossom Wilt (*Sclerotinia laxa* Aderh. & Ruhl. = *Monilia laxa* Ehrenb. = *Monilia cinerea* Bon.)

As long ago as 1900 the Russian mycologist Woronin demonstrated that two very similar fungi were associated with brown rot of fruit, those we now know as the conidial stages of *Sclerotinia laxa* and *S. fructigena*. The conidial stage of the first, characterized by the grey colour of its pustules, he called *Monilia cinerea* Woron., and when its ascospore stage was later discovered it was called *Sclerotinia cinerea*. In the meantime an apparently identical fungus had been discovered on peach and described as *Monilia laxa* Ehrenb. When the very rare ascospore stage of this was found it received

the name *Sclerotinia laxa*. This is indistinguishable from the cherry fungus and is the oldest name available for the perfect stage. It is therefore now in general use though it is only the conidial stage that is usually seen; indeed the ascospore stage of *S. laxa* has only once been found in Britain.

Blossom wilt due to this fungus is common and destructive on sour cherries, less so on sweet cherries, pears, plums, peaches, apricots, nectarines, *Prunus triloba*, *P. pseudocerasus* and *P. nana*. For a similar fungus attacking apple blossom see p. 120, and for the attack on fruit see the next column.

Symptoms and development. On cherry trees one often notices year after year that a number of blossoms wither without setting fruit (Fig. 91). On sweet cherry the fungus may

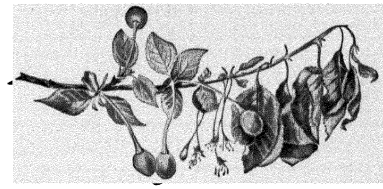


Fig. 91. Wither-tip of cherry, caused by *Sclerotinia laxa*.

grow down the stalk from a single infected flower and kill the spur so that the whole flower truss dies. In sour cherries the fungus spreads from the infected leaves into the main twig so that nearly all the flower-bearing wood is killed. Infection may occur through unopened flower buds which then remain conspicuously dead and shrivelled when the healthy blossom opens, or it may take place through the stigma of the open flower. In the latter case the damage first becomes visible 10–12 days later, when the shoots die back soon after the fruit has set. In severe outbreaks, especially on Morellos, the whole tree looks as though frosted or swept by fire. Infection of the young wood by *S. laxa* is one of the many causes of gummosis. Apparently the quicker the shoot is killed the less abundant is the gum production.

Occasionally young leaves also become attacked and the fungus then spreads down through the leaf stalk into the shoot, causing it to wither. This wither-tip phase of the disease is much more prevalent on plums than on cherries. Small grey conidial pustules are produced on all the dead tissues and these survive the winter. Hence there are plenty of spores to infect the flower buds next spring.

On pears *S. laxa* usually causes only blossom wilt and death of the spurs. On plum it may almost completely destroy a season's blossom and be responsible for much wither-tip owing to the infection of young leaves, especially those attacked by the leaf-curling aphis *Anuraphis helichrysi* Kalt.

The same fungus is a common cause of brown rot and mummified fruit in both

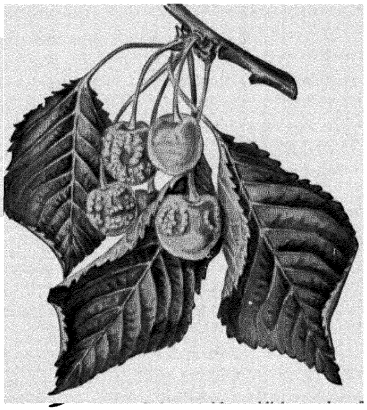


Fig. 92. Brown rot of cherry with conidial pustules of *Sclerotinia fructigena*.

cherries and plums. Mummified fruit hanging on the trees produce conidial pustules the following spring and are an important source of blossom wilt infection. Similar mummies that have fallen and remained on or near the surface of the soil may very rarely produce clusters of small brown-stalked cup-shaped apothecia. These have never been seen in Denmark; possibly the orchards have a high soil reaction which does not favour development of the fungus. Brown rot may also be caused by *Sclerotinia fructigena* (Fig. 92) (see p. 121).

A moist atmosphere encourages blossom wilt and the disease is seldom seen in the drier parts of America where orchards have to receive artificial irrigation.

Varietal susceptibility to blossom wilt has not been fully investigated. Sour cherries are particularly susceptible to it, especially Morello and van der Nat. Ostheimer also suffers greatly but in experiments at Weihenstephan the variety Montmorency Stark appeared to be immune (Elssmann, E., 1939). Of plum varieties Yellow Apricot is most susceptible.

Control. As the conidia are abundantly produced on the dead flowers and spurs these should be cut out and burned as soon as they are seen. All mummified fruit should also be collected and burned and, especially on plum, the withered tips should be cut out and destroyed.

The ordinary tar oil or caustic soda and soap winter washes help to destroy the conidial pustules and by controlling aphides they help to reduce the amount of wither-tip in plums.

In England a 6:6:100 Bordeaux spray just before blossoming is recommended on cherries. Confirmatory results have been obtained with this spray in Denmark, where the recommendation is to apply one spray about 12 days before blossoming and another 2–5 days before (i.e. when the flower buds form a tight cluster with the petals visible). On plum trees a 1:50 lime sulphur spray may be used at a similar time.

In France peach and apricot are sprayed with Bordeaux mixture when the buds swell, again when the flower buds show pink and a third time when the petals are shed from the set fruit, but the trees may easily be scorched, especially by this last spray. In Italy a 1:20 lime sulphur spray is applied in November and in February, but this is not very effective against *S. laxa* and a strong Bordeaux spray just before flowering is recommended as well. A similar spray towards the end of the winter has been tried in Germany.

Little attempt has been made to prevent brown rot by direct application of sprays to the developing fruit. Dusting with a fungicide 10 days before picking has given good results in America (Heald, 1926), but these were not confirmed in Germany (Klebahn, 1930). Careful picking to avoid bruising or breaking the skin will help to reduce loss from brown rot during transit and storage. The stalks should be left on the larger varieties of plum, otherwise the fungus may enter by the wound where the stalk was removed. Wrapping peaches in paper may also help to reduce bruising and spread of brown rot.

Witches' Broom (*Taphrina cerasi* (Fuck.) Sadeb.)

Symptoms. This disease is most frequent on sweet cherries, but may also occur on sour cherries and other species of *Prunus*. Large broom-like tufts of branches develop in the crown of the tree (Fig. 93). These may be so open as to be easily overlooked in winter but are easily distinguished at blossoming because they bear few flowers and become leafy earlier than the normal branches. Because of their weight the brooms are usually somewhat pendulous. At the base there is a common stem which may be much thicker than the branch to which it is attached. From this stem spring long, slender branches that may grow out in all directions and are themselves irregularly branched, so that small tufts of twigs occur within the main broom. If the broom has originated from an infected terminal bud on an upwardly directed branch it does not hang down, but stands straight up at the tip of the branch.

Leaves on the witches' broom are often smaller and more rounded than normal ones. They are also thicker and soon become reddish and covered on their under-side by a whitish bloom of asci. As they wither they give off a smell of cumarin like that of sweet woodruff or of new-mown hay. They gradually turn brown and fall.

Cause. Witches' broom of cherry is due to infection by the fungus *Taphrina cerasi*, also sometimes called *Exoascus cerasi*. Ascospores from the diseased leaves fall on to buds and germinate to give a mycelium which penetrates the branch, stimulating it to abnormal growth year after year. Once a branch has

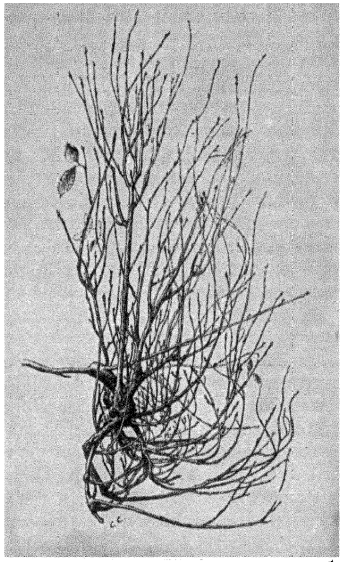


Fig. 93. Witches' broom of cherry (after Rostrup)

become infected it will always remain so and the leaves on the broom will be diseased every year.

Importance. As witches' brooms never bear fruit their presence means a loss of crop, but they are seldom prevalent enough to be important in Denmark. At one time they were apparently very prevalent in Germany. The disease occurs throughout Europe, in North America and in Japan. In England it has been reported mainly from the southern counties.

Control. The brooms must be cut out, far enough back to remove the base from which they spring. Cutting out should preferably be done in winter or at blossoming, i.e. before the asci appear. Where they have been very abundant a 3:3:50 Bordeaux spray may be given as a precautionary measure when the leaves are unfolding.

Leaf curl (*Taphrina minor* Sadeb.) is a rather similar disease in which the affected leaves do not occur on brooms but are scattered along otherwise normal branches. It is occasionally observed in Kent and other southern English counties. Control measures are similar to those for witches' brooms, by cutting out infected branches below the lowest curled leaf.

Cherry Scab (*Venturia cerasi* Aderh. = *Fusicladium cerasi* (Rabenh.) Erikss.)

Cherry scab is much less important than apple and pear scab, but it may sometimes be destructive and seems to have been on the increase during late years. Fruit, leaves and shoots may all be attacked.

Spots on the fruit are the most general symptom of the disease. They are small, round, grey or olive green and appear on the ripening fruit, which ceases to grow and shrivels up (Fig. 94). If the fruit is attacked when still young it is often shed.

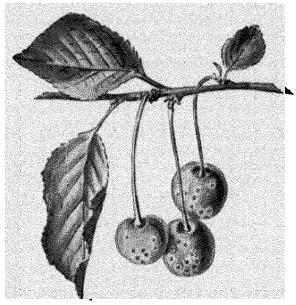


Fig. 94. Cherry scab on the fruit.

The spots on the leaves are very minute, round and dark olive, usually on the upper surface of the leaf. In very severe outbreaks the leaves are killed, but there may be an extensive attack on the fruit without any associated leaf spots.

Spots on the buds are rare; they are small, brownish, irregular or oval.

Sour cherries are more susceptible than sweet cherries.

In Denmark this disease was particularly noticed in 1916, 1927–30 and 1943. In the latter year it appeared for the first time on

the Morello cherries at East Malling, Kent (Moore, M. H., 1944), and also on Carnation, Coe's Late Carnation and Kentish Red sour cherries. Only a few spots were seen on sweet cherries, possibly because they ripened earlier. The disease appeared again at East Malling in 1945 and also in Essex (Moore, W. C., 1948).

A similar fungus causes scab on plums, see p. 196.

Control of cherry and plum scab may be secured by spraying with 8:8:100 Bordeaux mixture before flowering and with 4:8:100 after flowering or with 1:50 lime sulphur solution both before and after flowering. Loewel advocated a spring spray with 6% carbolineum added to a 2–4% Bordeaux spray.

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

Verticillium infection may be responsible for sudden death of stone fruit trees, especially cherry. In Denmark it has also occurred on Myrobolan and in England on plum (Keyworth, 1944). In Holland, where much damage has been done by it to cherry orchards, especially in Gelderland, the disease has been studied by Van der Meer (1925). It generally appears 4–6 years after planting, first on single trees, later on several. In that province potatoes are commonly grown between the trees in certain years.

Symptoms. The disease becomes apparent in July or August, when the older leaves turn yellow. The discoloration spreads along the branches until it may involve the leaves on the current season's growth. These may turn reddish violet with dry brown curled margins and brown patches between the veins. The yellow leaves wilt, hang down and are shed, starting at the base of the branches. Those towards the tips may hang on longer, even when quite dead and shrivelled. A larger or smaller number of branches dies and there may or may not be an exudation of gum from these. The whole tree may die either that year or in the next two or three years, but it may survive. In the latter case it retains a sickly appearance because of the large number of dead branches, and its growth will usually be weakened.

If a wilted branch is cut off at the base a brown area will be seen in the wood, often spreading radially. It usually has an irregular edge with wedge-shaped protuberances between two annual rings (see Fig. 31). Instead of the brown central patch there may be brown rings, arcs or dots scattered through the wood. The brown spots between two annual rings lie in the inner part of that particular zone of wood. If the branch is cut through at several points higher up the brown central patch will be seen to disappear usually between 8 and 16 inches from the base, but the brown spots may extend even into branches that are only two years old.

A brown patch will also be found in the wood of the trunk which, at the base, resembles that in the branches but about halfway up the trunk becomes more compact, darker, and almost extends outwards as far as the bark. At the top of the trunk the irregular patch and brown spots reappear. Similar brown stains occur in the wood of the main roots. *Verticillium alboatrum* or its sclerotial form *V. dahliæ* can be isolated from the stained wood. Inoculation experiments on one-year-old cherry seedlings undertaken in May and June with *Verticillium alboatrum* and its variety *caespitosum* isolated from cherry, and with *V. alboatrum* from potato, reproduced the disease. In one instance the fungus spread two feet along the wood in 19 days. Keyworth (1943) found evidence that *V. alboatrum* from decaying infected hop bines could infect Victoria plum.

Inoculation experiments with trunk, root and soil at various times between 15th February and 22nd September showed that one-year-old cherry seedlings could be infected by root and trunk inoculation in May and June and by inoculation of the soil before the middle of May. Individual infected seedlings died in the first year, but some survived and grew away from the disease after a few years.

Control. Potatoes are not infrequently attacked by *Verticillium* and hence are a dangerous crop to interplant between cherries.

Soil from tomato and cucumber houses may also be infected and should not be introduced into cherry orchards (see also p. 60).

Bitter Rot (*Glomerella cingulata* (Stonem) Spauld. & v. Schrenk = *Gloeosporium fructigenum* Berk)

In July 1937 we found a virulent outbreak of bitter rot on sour cherries at Charlottenlund and later we also observed it elsewhere. First brown spots appeared on the fruit and spread until they covered the surface. Finally the whole fruit became shrivelled. Scattered over the surface were minute black fructifications from which oozed light-red masses of conidia. These were the imperfect stage, *Gloeosporium fructigenum*, of the fungus *Glomerella cingulata*.

G. Johansen in 1937 isolated the fungus in pure culture and showed that it could reproduce bitter rot on apples of the varieties Beauty of Kent and Cox's Pomona 10–12 days after inoculation.

A similar disease is known in Switzerland, where to control it a Bordeaux, lime sulphur or proprietary sulphur spray is recommended after leaf-fall, again after 14 days and a third time 4–5 weeks later.

Leaf Scorch (*Gnomonia erythrostoma* (Pers.) Auersw. = *Libertina stipata* (Lib.) v. Höhn)

Symptoms. Shortly after the leaves unfold, indistinct pale spots appear on them, which in the course of the summer change to large irregular brown patches, often with a yellow margin. Diseased leaves eventually curl up, wither and die (Fig. 95). The fungus penetrates the leaf stalk and prevents the formation of the absciss layer which brings about leaf fall. Hence the diseased leaves remain hanging on the tree all winter and are most easily recognized at that time.

Cause. On the under-side of the leaf spots fructifications occur containing elongated hook-shaped spores which, however, do not germinate and are believed by some to be male bodies which may or may not be functional. On the dead leaves the ascus fructifications develop during autumn and winter. These are minute perithecia, each with a long neck protruding from the leaf surface and easily visible under a hand lens. It is from these that the fungus received its name "erythrostoma," red-beaked. Ripe ascospores are ejected from the perithecia when the buds break in spring.

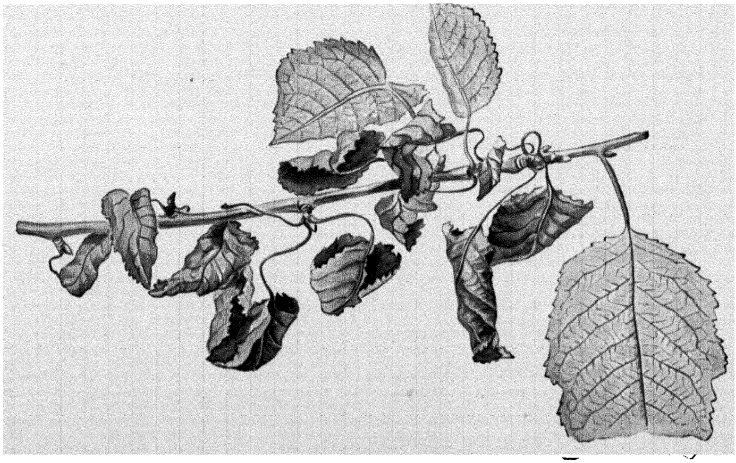


Fig. 95. Cherry leaf scorch.



Fig. 96. Cherry branch in June. Among the new leaves are the remains of those killed by leaf scorch the previous summer.

Importance. The fungus is not yet of economic importance in Denmark as it has so far been noted there only on bird cherries. In Sweden, however, it has done considerable damage in Bigarreaux and hybrid cherries. Serious outbreaks have also occurred on cultivated cherries at Altenland near Hamburg and in Switzerland. Leaf scorch is not often reported in England, but does occur on Waterloo, Early Amber and Frogmore Bigarreau and also in wild cherries, according to Wormald.

Control. Experience gained during the serious outbreak near Hamburg about 1880 showed that picking off and burning all the diseased leaves in winter controlled the disease in two years. Extensive infection from wild bird cherries has been observed in Sweden. Where hand picking is impracticable a Bordeaux spray while the leaves are unfolding and again after petal fall may be tried.

Chlorosis (Manganese Deficiency)

In the Sittingbourne district of Kent investigation of acute chlorosis of cherry trees, often accompanied by poor cropping and dieback, has shown that it was due to a deficiency of available manganese (Thompson and Roberts, 1945). Spraying with 5% manganese sulphate solution in February cures the condition for the following summer but has to be repeated each year. In the orchards multiple deficiencies of iron, manganese and zinc have been detected. Leaf symptoms are not of much value in diagnosing complex troubles of this kind, and when chlorosis of cherries does not respond to winter spraying with manganese



Fig. 97. Gummosis of cherry.

or iron salts alone advice should be sought from a Horticultural Research Station or an advisory chemist.

Gummosis

Exudations of gum frequently appear as hard, more or less transparent, brown drops or crusts on stone fruit twigs and branches, especially on cherries (Fig. 97). These are an indication that all is not well with the tree. It is true that some trees apparently suffer from gummosis for several years and look none the worse for it, but more often the affected branch or even the whole tree dies in a few years.

When gum is produced in trees the inner bark and cambium turn first yellow and then brown. There is also a tendency to gum production in the newer, outer layers of wood. The leaves of affected branches may be small and weak and when gum production is extensive they begin to drop and wilt. This withering may occur suddenly; leaves turgid in the morning may be limp by afternoon and never regain their turgidity. Gum develops by a chemical change in the cell wall and hinders the supply of water to the leaves by stopping up the water-conducting vessels, especially those last formed. The gum is not soluble in water. As dissolution of the tissues proceeds small pockets of gum arise, which gradually enlarge until they reach the surface. There is then a wound from which drops of gum are exuded.

Gum formation may also start near a wound caused by mechanical injury, or in tissues damaged by bacteria or fungi (see bacterial canker, p. 199; blossom wilt, p. 152; *Verticillium* wilt, p. 156; silver leaf, p. 191; dieback, p. 198).

Gummosis may also be a reaction to frost damage and often appears immediately after a hard winter and on trees with poorly ripened wood. Late frosts are particularly destructive. The damage may also first appear a considerable time after the frost.

In Holland gummosis is worst in wet years. Van der Broek (1937) thinks the wild cherries used as stocks are too vigorous. Their roots absorb more nutrients than the scion can use. There is therefore an abnormal accumulation of nutrients in trunk and branches which leads to gummosis.

In Denmark it is a fairly general practice to lime the soil to prevent gummosis and this may sometimes have a good effect. Indiscriminate liming is undesirable, however, because bacterial canker is worst on soils rich in lime (see p. 201). In various quarters it has been asserted that watering with common salt solution is a remedy for gummosis. Deichmann in " Haven," 1914, reported gummosis in a ten-year-old tree was cured by watering the soil with a solution of 1 lb. cooking salt in 1 gallon water over the whole area between a distance of 2 ft. 8 in. from the trunk and the outer limit of the branches. It is doubtful if this would always be effective, but it is possible that the lack of or unavailability of some soil nutrients may be one of the causes of gummosis.

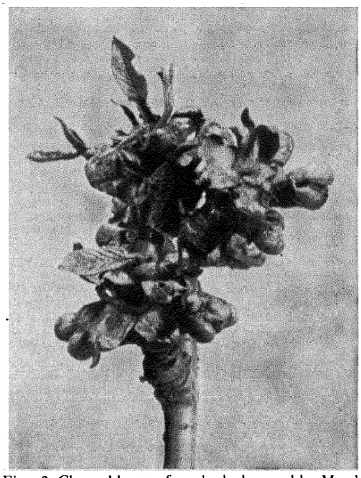


Fig. 98. Cherry blossom from buds damaged by March frosts. Only shrivelled tissue remains among the expanded bud scales (*S. p. F.*).

Frost Damage

In May 1931 we noticed in several places a somewhat strange result of frost damage in the previous March. The winter had been mild and March was a bright, sunny month,

which may have led the buds to be unusually forward in development when the frost came, though there was no sign of their bursting. On sweet cherries the damage showed itself by there being no flowers in the buds when the outer scales opened. Instead there were inside only small, brown, scorched, flower rudiments about $\frac{1}{12}$ inch across (Fig. 98). In sour cherries the bud scales only opened slightly, and the damage inside was like that on sweet cherries. It was, however, more general on sweet than on sour cherries. As usual with frost damage it was worst on trees on low ground. At several places the flowers were destroyed on the lower branches while over the top of the crown flowering was normal. Similar damage has been seen in subsequent years, but not to such an extent as in 1931.

Summary of Cherry Diseases

I. Trunk, Branches and Twigs

A. Current season's growth:		*page*
(1) Dark oblong patches of sunken bark round dying buds	Bacterial Canker	199
(2) Small dry brown spots with red margins	Shot-hole	197
(3) Death of the flowering spurs	Blossom Wilt	152
B. Older bark:		
(1) Sunken patches of bark, brown at the edge in section	Bacterial Canker	199
(2) Gummosis:		
(a) With brown edges to the diseased bark as seen in section	Bacterial Canker	199
(b) Associated with a brown stain in the wood	*Verticillium* Wilt	156
(c) Not associated with dead tissue	Non-parasitic Gummosis	159
(3) Bark peeling off exposing powdery, rusty surfaces	Tan Disease	19
C. Dead branches	Dieback	198
D. Tufts of twigs bearing green leaves at blossom time	Witches' Broom	154

II. Leaves

A. Thickened pale reddish leaves with a musty smell when withered:		
(1) All the leaves on a dense tuft of twigs affected	Witches' Broom	154
(2) Affected leaves scattered over a normal branch	Leaf Curl	155
B. Leaf spots:		
(1) Small round grey or blackish spots	Cherry Scab	155
(2) Small round spots that eventually fall out:		
(a) Edges of the spots water-soaked and translucent	Bacterial Canker	199
(b) Edges of the spots dry and not translucent	Shot-hole	197
(3) Pale yellowish-brown patches	Leaf Scorch	157
(4) Large light-brown scorched patches	Spray Damage	45
C. Wilted or dead leaves:		
(1) Small, dull or flabby leaves	Gummosis	159
(2) Rolled leaves, eventually turning yellow	Bacterial Canker	199
(3) Yellow wilted leaves, eventually shed, or reddish-violet leaves, curled up with scorched edges	*Verticillium* Wilt	156

		page
(4) Curled dead leaves hanging on the trees throughout the winter	Leaf Scorch	157
D. Coating of mould:		
(1) White	Mildew	195
(2) Black	Sooty Mould	126
E. Chlorosis	Manganese Deficiency	158
III. Flowers		
A. Buds more or less opened, flower parts turned brown	Frost	159
B. Flowers and flower buds all wilted together	Blossom Wilt	152
IV. Fruit		
A. Small patches on the skin:		
(1) Round grey or blackish patches	Cherry Scab	155
(2) Sunken dark-brown patches	Shot-hole	197
B. Cracks	Cracked Fruit	205
C. Brown rot, leading to mummified fruit:		
(1) Conidial pustules yellowish	*Sclerotinia fructigena*	121
(2) Conidial pustules grey	*Sclerotinia laxa*	152
(3) Tiny black fungus pustules bearing light-red masses of conidia	*Gloeosporium*	157
D. Shedding of undersized fruit:		
(1) Skin marked with round grey or blackish spots, the kernel developed in the stone	Cherry Scab	155
(2) No spots, kernel shrivelled in the stone	Shedding of Unripe Fruit	131

COB NUT AND HAZEL

Frost Damage

Danish observations indicate that hazel catkins are easily damaged by frost. The varieties show great differences in their reaction to frost.

Nut Drop (*Sclerotinia fructigena* Aderh. & Ruhl.)

The common brown rot fungus of apples and plums can also attack cob nuts and filberts. Brown patches appear on the husk and spread into the shell of the nut often giving it a striped appearance. The diseased nuts contain no kernel and are shed in August. Wormald found that only wounded nuts could be artificially infected with a spore suspension of *S. fructigena* and that natural infection nearly always occurred on nuts punctured by the nut weevil (*Balaninus nucum*).

In damp conditions the familiar yellowish conidial cushions appear on the dead tissue.

G. Johansen (1937) was able to infect hazel and apple with *S. fructigena* from Myrobalan fruits, and Buchwald (1943) has shown that the strain attacking hazel can also rot plums. For an account of the disease on Kentish cobs see Wormald (1944) and M. H. Moore (1947). For control measures see apple, p. 121.

Hazel Mildew (*Phyllactinia suffulta* (Rebent.) Sacc.)

This fungus, also known as *P. corylea* (Pers.) Karst., forms rather a sparse powdery coating on hazel leaves. Its tiny round perithecia, at first yellow, then black, are usually present in large numbers. When very prevalent, mildew may cause premature defoliation, even on wild hazel bushes. Mildews

referred to the same species occur on alder, beech, birch and hornbeam leaves, but the investigations of Voglino (1905) and Hammarlund (1925) indicate each host probably has a physiological race of the fungus peculiar to it.

No control measures have been worked out for hazel mildew, but the usual sulphur fungicides employed against other powdery mildews would probably be effective in this case also.

CURRANT, RED AND BLACK

Rusts of Currant

Leaves of currant bushes are liable to infection by three different rust fungi. Bushes in country gardens, especially in low-lying situations where sedges are plentiful in neighbouring ditches, are often attacked by the cluster cup stage of *Puccinia pringsheimiana* Kleb. (see under gooseberry, p. 172). This is also common on the leaves of wild currant bushes, for example in the Norfolk Broads, and may occur on the fruit.

In Europe the true currant rust, *Puccinia ribis* DC., occurs, especially on redcurrant leaves. This causes yellow spots in the centre of which is a dark-brown pustule of teleutospores, sometimes surrounded by a ring of smaller pustules (Fig. 99). Rostrup (1902) has counted over 1,300 pustules on a single leaf. The fungus may also infect the berries, which are then yellow and almost transparent with pustules of spores on their upper surface.

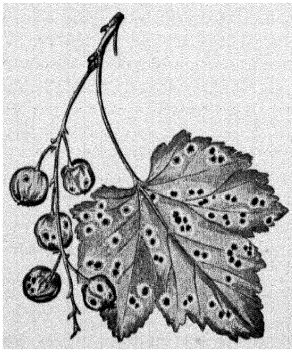


Fig. 99. Redcurrant rust, *Puccinia ribis* (after Rostrup).

Puccinia ribis has a very curious history in Britain. It was found in a Morayshire garden by Keith in 1894, but was not rediscovered until 1946, when it was seen by Dr. E. G. Gray on very old redcurrant bushes at Coulmony in the same county (1947). This rust is believed to over-winter as mycelium in the currant buds.

Much more important than either of these is another rust, *Cronatium ribicola* J. C. Fischer, which has its uredo and teleuto stages on various species of *Ribes* and its æcidial stage on five-needled pines. This fungus forms yellowish uredo pustules on the under-side of the currant leaves, followed late in the season

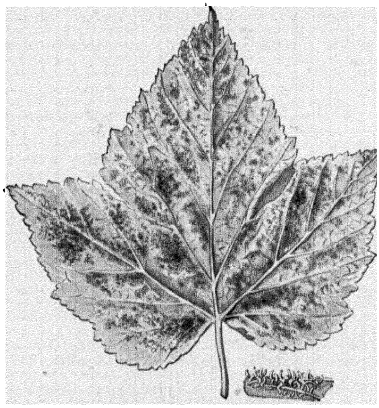


Fig. 100. *Cronartium ribicola* on under-side of currant leaf.

by a dense felt-like yellow-brown coating on the entire lower leaf surface (Fig. 100). This is made up of minute waxy hair-like bodies, about $\frac{1}{12}$ inch long, each composed of numerous, colourless teleutospores arranged in rows stuck together. These germinate while still on the leaf and produce basidiospores capable only of infecting five-needled pines. On the latter, for example, Weymouth pine (*Pinus strobus*), white pine (*P. monticola*) and other species such as *P. cembra* and *P. lambertiana*, the rust

causes yellow spots on the needles and on the bark of the short shoots. From these the mycelium grows back into the twigs where small spermogonia are formed in the bark. In the following and subsequent years the diseased branches swell and bear large yellow blisters containing æcidiospores, hence the name blister rust applied to this phase of the disease (Fig. 101). The blisters burst, scattering the spores, which are able to infect currant bushes in the vicinity. Later cankers and resin exudations develop on the swollen

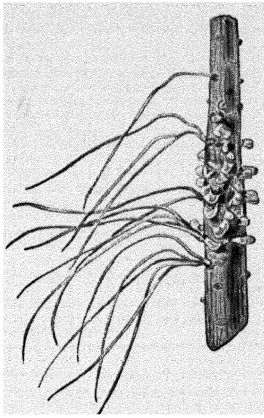


Fig. 101. Blister rust on Weymouth pine.

branches. Ultimately the branches or even the whole tree is killed.

According to observations in North America blackcurrants may become infected by æcidiospores from pines over 90 miles away. The uredospores, of which there may be up to seven generations in a season, then spread the disease from currant to currant. On the other hand, the thin-walled basidiospores produced on the currant leaves at the end of the season are only infective over short distances. Pines usually remain uninfected if more than 100 yards from rusted currants. The fungus may overwinter on fallen blackcurrant leaves and re-infect blackcurrants by uredospores in the spring.

History. Blister rust was first found in Russia and East Prussia about 1860; during the 70's of the last century it was observed all over Eastern, Central and Northern Europe, including Denmark (Rostrup, 1877). It occurs on wild *Pinus cembra* in the Alps, in Russia and in Siberia, but does little damage to that species so that it was probably long overlooked.

In 1887 Klebahn demonstrated the connection between rust on currants and blister rust of five-needled pines.

Blister rust was apparently introduced into North America in 1909 on young Weymouth pines imported from Holsten. Further importation of pines from Europe was prohibited, but too late to prevent the disease from becoming established in North America. The result has been a disastrous outbreak of disease which threatens to destroy all the valuable native Weymouth pine forests of the Eastern United States and, more recently, also the Western white pine in the Pacific coast states and British Columbia. The existence of blister rust makes it impossible to establish plantations of Weymouth pine or its allies in Britain or in Western Europe.

In currants the disease is of minor importance, but it may cause premature defoliation, especially in blackcurrants.

Control. The most effective control measure is to keep currants well away from five-needled pines. In North America maintenance of a belt of 300 yards free from any cultivated or wild species of *Ribes* is recommended round Weymouth pines. Young five-needled pines may be infected without showing symptoms when they are sold.

To prevent overwintering of the rust on fallen currant leaves these should be swept up and burned or buried.

In plantations where currants become infected year after year some protection may be given by a Bordeaux spray, as recommended for leaf spot (p. 173). This should be applied immediately after flowering and again after the fruit has been picked. In nurseries an earlier spraying is practicable. According to Tubeuf (1914) care must be taken to spray

the under-side of the leaves as infection takes place through the stomata there.

Individual pines may sometimes be saved if the diseased branches are cut off 10 inches or more below the swellings as soon as the disease is observed. The cut surfaces should be covered with oil paint.

Dieback (*Botrytis cinerea* Pers. ex Fr. and other fungi)

Redcurrants, blackcurrants and gooseberries are all liable to suffer from invasion of the branches by the common grey mould *Botrytis cinerea*. This causes dieback and under favourable circumstances it may kill the whole bush. Infected branches often show a characteristic scorching of the leaves. This starts at the edge and penetrates inwards in triangular patches between the veins. In damp weather the grey mould appears on the under-side of these patches and may also break out through cracks in the back of dying branches.

Diseased branches should be cut off and burned as soon as possible and heavily attacked bushes should be removed altogether (see also under gooseberry, p. 174).

A weak parasite of currant and gooseberry branches is the pycnidial fungus *Cytosporina ribis* Magn. This produces very numerous minute black pustules within the bark, from which yellowish tendrils of spores are exuded in moist air.

The common coral spot fungus, *Nectria cinnabarina* (Fr.) Fr., also often occurs as a weak parasite of currants. It usually establishes itself on dead stubs left in pruning and spreads from them into the living branches. Wood infected by this fungus becomes covered with small, hard, bright pink conidial pustules, followed by dense clusters of minute red egg-like perithecia (see also p. 555).

Black pustule (*Plowrightia ribesia* (Fr.) Sacc.) is an ascomycete which produces black oval warts about $\frac{1}{8}$ inch long on branches, especially of redcurrant. These warts contain numerous perithecia, the apertures of which appear as minute pimples on the black surface. Infection takes place in the same ways as with coral spot. Control of both these diseases consists in avoiding leaving long stubs when pruning and in cutting out and burning diseased branches.

Collar Rot (*Fomes ribis* (Schum.) Fr.)

Old currant and gooseberry bushes are sometimes attacked by the woody bracket fungus *Fomes ribis*. This forms hoof-like or shelf-like fructifications, yellow-brown and velvety above, with concentric ridges, grey-brown and covered with minute pores on the under-surface. They occur mainly at the collar, just above soil level and are perennial, increasing in diameter until they may be 5 or 6 inches across.

The fungus enters through wounds and does not produce its fruit bodies until 3–4 years after infection. It is only a feeble parasite and infected bushes may survive for many years. They should, however, be dug out and destroyed and replaced by young ones.

A form of this fungus is common in the South of England as a parasite of old spindle trees (*Euonymus europaeus*).

The common toadstool *Collybia velutipes* also sometimes parasitises old redcurrant bushes. It is easily recognized by its bright yellow cap and dark-brown or blackish velvety stalk. Fructifications of this fungus are very common on dead wood and tree stumps during most winters and it usually becomes parasitic only on very old and useless bushes.

The honey toadstool, *Armillaria mellea* (Fr.) Quel. (see p. 456), may occasionally cause root rot and collar rot of currant, and silver leaf (*Stereum purpureum* (Fr.) Fr.) (see p. 191) has been seen on red and black currant and gooseberry in the South of England (Moore, W. C., 1943).

Reversion (*Ribes* Virus I)

Reversion, first described on blackcurrants in England in 1917 (Hatton and Amos), was recognized in Denmark on blackcurrants in 1924 and on redcurrants and *Ribes sanguinea* since 1928. It is probably the most serious disease of blackcurrant.

The early stages are difficult to recognize. In spring its presence may be betrayed by a

yellowish colour of the leaves. Lateral buds that should give flowers grow out into shoots instead. By June or July it is easily recognizable. Leaves of healthy blackcurrant bushes have 5–7 pairs of side veins and fine toothed margins. Leaves of " reverted " bushes have

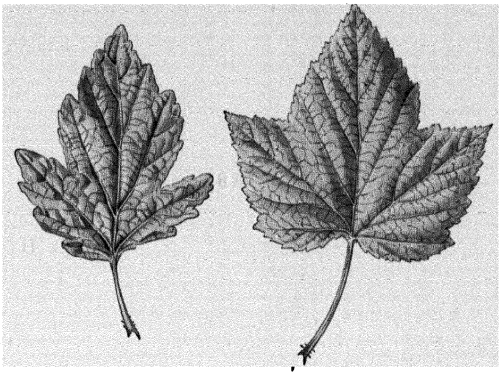


Fig. 102. Left: leaf from reverted blackcurrant bush. Right: leaf from healthy bush.

mostly less than 5 side veins; they become elongated and the marginal teeth are larger and rounded. The shape may somewhat resemble a maple leaf (Fig. 102) (Amos and Hatton, 1927). The fruiting capacity of the bush is greatly reduced. Reversion has been shown to be transmissible by grafting and there is some evidence that the natural agent of transmission is the big bud mite (*Eriophyes sibis*) (Amos *et al.*, 1927).

Leaves like those of reverted bushes may be produced on damaged shoots or on bushes cut back hard, but the bushes grow away from such a condition, whereas in true reversion they become progressively worse.

All reverted bushes should be destroyed, even if only a single branch is affected. Cuttings should only be taken from perfectly normal bushes. Big bud mite should be controlled by spraying with lime sulphur 1 : 20 just before the flowers open. In the past this disease has been spread largely by the use of infected cuttings, and to check this the Ministry of Agriculture in England and Department of Agriculture for Scotland operate a scheme for inspection and certification of healthy blackcurrant bushes.

Other diseases. Variegated leaves (panachure) with conspicuous bright yellow spots are not uncommon on red- and blackcurrants. They seem to be of little importance. In North America a mosaic disease has been reported on *Ribes americana*, *R. floridum* and gooseberry.

Leaf Scorch (Potassium Deficiency)

Leaves of currant or gooseberry bushes lacking potassium develop scorching round the edges. In severe cases this is followed by defoliation. Leaf scorch due to potassium deficiency is easily corrected by manuring with sulphate of potash at the rate of about 4 cwt. per acre. If this is applied early, for example in March, good results may be obtained in the same year (Fig. 103). If nitrogen was lacking as well as potassium there will be little or no response unless sulphate of ammonia or other nitrogenous fertilizer is applied at the same time. Liquid manure or fowl manure may be used, but may not induce a response until the following season.

Though leaf scorch is often due to potassium deficiency a very similar condition may arise

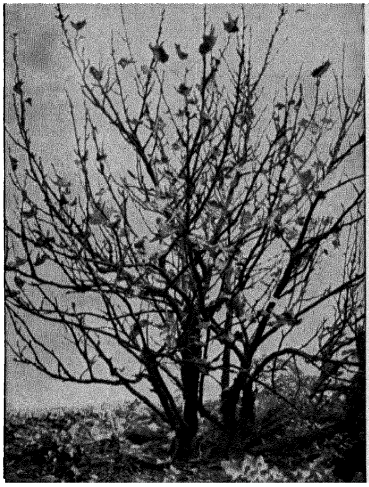


Fig. 103(*a*). Currant bush defoliated by potassium deficiency (*S. p. F.*).

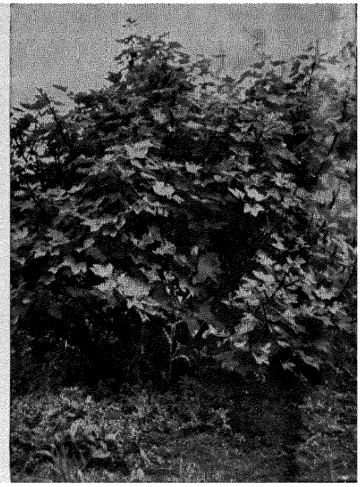


Fig. 103(*b*). Bush in same plantation manured with potassium sulphate (*S. p. F.*).

from several other causes. Application of fertilizers containing chlorine may lead to a broad light-brown marginal scorching. Similar damage may be due to cold winds and perhaps to insect attack.

Sterility and Running Off

It sometimes happens that currant bushes which have flowered well set little fruit or the berries fall off when quite small (" running off "). If this occurs only in particular seasons it is usually due to frost at flowering time, or to defective pollination.

If it occurs on the same bushes year after year they should be examined during flowering to see if the stigmas are normally developed. Some bushes have been found, especially on Funen (H. Gram, 1926), which were always barren and had either poorly developed stigmas or none at all. These bushes were mostly obtained from one old nursery on Funen, now closed. They were often very vigorous as none of their energy was used in fruit production.

Any such bushes should be cleared away and cuttings should be taken only from those known to bear well.

In blackcurrants Wellington, Hatton and Amos found the varieties most prone to sterility were those like French Black (Merveille de la Gironde) and Boskoop Giant, on which the stigmas are raised above the anthers. In Baldwin's Black the stigmas are level with or below the anthers, so that self-pollination can take place and sterility is seldom troublesome (Fig. 104). By growing bushes of cross-pollinated varieties in warm, sunny, sheltered places insects may be encouraged to visit them, especially if beehives are kept nearby.

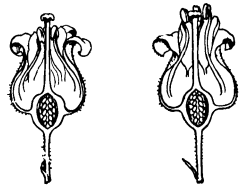


Fig. 104. Blackcurrant blossoms in longitudinal section. On left anthers below level of stigma; on right anthers and stigma on same level (after Wellington).

Summary of Currant Diseases

I. Branches and Shoots

		page
A. Dead branches:		
(1) Grey mould appears in cracks of bark under damp conditions	*Botrytis* Dieback	174
(2) Coral pink pustules on dead bark	Coral Spot	555
(3) Black warts on dead bark	Black Pustule	164
B. Decrepit bushes with hard bracket fungus at the base	Collar Rot	164
C. Dead branches with brown lines in the wood	*Verticillium* Wilt	60

II. Leaves

A. Marginal leaf scorch:		
(1) Scorched edges typically narrow and brown	Potassium Deficiency	165
(2) Scorched edges broad and light brown	Chlorine Poisoning	166
B. Large wedge-shaped brown areas between the veins	*Botrytis* Dieback	174
C. Centre of leaf yellowish-red (redcurrant) or purple (blackcurrant)	Magnesium Deficiency	38
D. Leaves with a white mealy coating, later brown and felted	American Mildew	168
E. Leaves narrow with less than five side veins especially in blackcurrant	Reversion	164
F. Leaf Spots:		
(1) Yellow blotches	Variegation	165
(2) Yellow spots with dark brown rust pustules	*Puccinia ribis*	162
(3) Thickened orange patches with cluster cups	*Puccinia pringsheimiana*	172
(4) Yellow spots followed by yellow-brown hairy felt on lower surface	*Cronartium ribicola*	162
(5) Numerous brown blotches between veins	Phosphorus Deficiency	41
(6) Small brown spots, turning white with a brown edge	*Septoria* Leaf Spot	174
(7) Dark brown spots, sometimes causing defoliation	Leaf Spot	173

III. Fruit

A. Barren bushes and shedding of fruit	Sterility and Running Off	166
B. Rust pustules:		
(1) Orange	*Puccinia pringsheimiana*	172
(2) Pale yellow	*P. ribis*	162
C. Mealy white coating, turning to a brown felt	American mildew	168

ELDER

Bark Fungus (*Corticium sambuci* Fr.)

Conspicuous white patches frequently occur on the bark of old elder bushes, particularly on the main stem near the ground. Sometimes these are so extensive that the bark appears to have been whitewashed. Though related to *C. solani* the fungus concerned is not a parasite and does no harm to the bushes. The honey toadstool (*Armillaria mellea*) has been observed to kill elder in England (Moore, W. C., 1943).

Gloeosporium Fruit Rot (*Gloeosporium fructigenum* Berk. var. *sambuci* Müll-Thur.)

A serious fungus disease of the fruit was found in 1932 in a large elder plantation at Lyngby. The ripening fruit shrivelled and became dotted with minute, slightly raised, fructifications. These produced such large masses of pale reddish conidia as to colour the whole surface of the fruit.

Investigation showed that the fungus was a form of *Gloeosporium fructigenum*. Müller Thurgau (1922) had described an earlier outbreak of the disease in Switzerland and had shown by inoculation experiments that the fungus on elder could not attack apples, the host of the typical race of *G. fructigenum*. He therefore regarded it as a physiological race peculiar to elder. G. Johansen (1937), however, succeeded in infecting apples with a *Gloeosporium* culture isolated from elderberries.

The disease had been seen at Lyngby before 1932 and has occurred subsequently. There have also been outbreaks in other places, though it does not seem to be of general occurrence. Where it arises no dead elderberries should be allowed to remain hanging on the bushes through the winter and the ground should be turned over to bury fallen berries, so that none remain above ground in the spring.

Spraying experiments at Lyngby have shown that Bordeaux mixture may check the fungus, but only if applied after the fruits have finished swelling. If this is done they will still be somewhat spotted with copper sulphate and lime when gathered, and it may be necessary to wash them in 2% acetic acid before they are used.

FIG

Canker (*Phomopsis cinerescens* (Sacc.) Trav.)

Fig cankers arise at pruning wounds and around the stumps of twigs removed in pruning. They are due to infection by the fungus *Phomopsis cinerescens* and are usually dotted with the minute pustules formed by its fructifications. Old cankers often bear concentric ridges marking stages in their development. Another fungus, *Gibberella moricola* (see mulberry, p. 177) is sometimes associated with the *Phomopsis* on dead fig twigs.

Fig canker may be controlled by cutting out and burning the diseased branches, taking care to cut back flush with the branches when pruning and protecting pruning wounds with white lead paint (see p. 192).

Mosaic

This is a virus disease which causes yellowish-green scattered blotches on fig leaves or smaller light-green spots with narrow reddish edges, mostly along the veins. In severe cases leaves may be deformed and fall early and the fruit may be spotted.

Fig mosaic seems to be fairly common in England, though it was not reported before 1935 (Ainsworth, G. C.). It is impossible to cure infected trees so care should be taken to propagate only from healthy ones.

GOOSEBERRY

American Mildew (*Sphaerotheca mors-uvae* (Schw.) Berk.)

History. American mildew was described by Schweinitz in North America in 1834. In 1900 it suddenly appeared on old-established gooseberry bushes in three gardens on the north coast of Ireland. There was no proof of its introduction from America. In 1902 it was found in other places in Ireland, but it did not appear in England until 1906.

About the year 1900 severe outbreaks of the disease were seen in Russia at two widely separated localities, where it was said to have

been present for about 10 years. In 1902 it was found at several places in the Baltic states, in Central and Little Russia and in the then Russian Poland. In 1903–8 it was found in Posen and in East and West Prussia. In Sweden it was first noticed in 1901, in Norway 1904, in Holland 1907, and had been found in all the Dutch provinces by 1909.

The first report of the disease in Denmark was from Tikøb and Staunsholt, North Zeeland, in 1904 (Rostrup, 1904). It had appeared at Tikøb in 1902 on bushes planted in 1901. These were of English varieties and the disease had probably been present in the country in nurseries since about 1900. Eriksson thinks it was introduced into Sweden in 1900 in plants from a Danish nursery. In 1905 it was found in six places in Zeeland, one on Funen and two in Jylland. In 1907 it was found at over 100 places and in 1908 at about 200 places all over Denmark. By that time it had spread to more places where gooseberries were grown in Europe.

Symptoms. The first sign of an attack is a white floury coating on leaves, tips of shoots and young fruit (Fig. 105). It is always young plants that are affected, never those that have finished growing. Mildewed leaves remain small and stunted, shoot tips are apt to branch and ripen badly and berries do not attain their normal size.

The white coating is easily rubbed off. After a few weeks it becomes chocolate brown on the shoots and berries and then forms a tough felted layer, which adheres firmly to the surface (Fig. 106). Small, dark, spherical perithecia develop in this felt.

In addition to gooseberries the fungus attacks red- and blackcurrants and other species of Ribes. In 1918 it was very prevalent on redcurrants in Denmark. It has very occasionally been found on raspberry in Sweden.

Cause and development. This disease is caused by the mildew fungus *Sphaerotheca mors-uvae*. This grows over the surface of the host, thrusting only its sucking organs (haustoria) into the underlying epidermal cells.

The life history and control of American gooseberry mildew have been very fully investi-

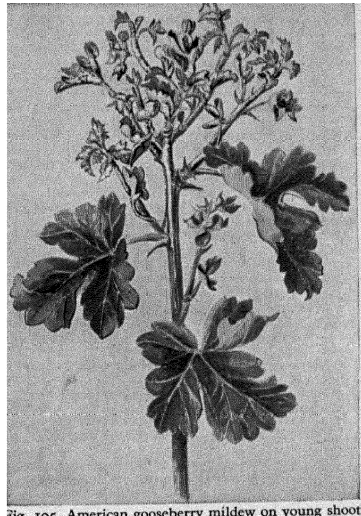


Fig. 105. American gooseberry mildew on young shoot.

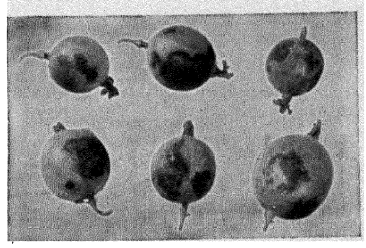


Fig. 106. American gooseberry mildew on the fruit.

gated. In England Salmon (1914, 1915) found that perithecia began to develop on the berries at the end of May or beginning of June. Perithecia collected in August burst and ejected their spores after a few hours under damp conditions. Those collected in November and kept dry in a laboratory until February or March burst then and ejected spores after a period varying from 1½ hours to several days under damp conditions. On the other hand perithecia collected out of doors at various places in the South of England in February or later apparently were dead. Salmon thought

these had not attained the degree of maturity necessary for them to survive the winter. Only those perithecia that have ripened by August are dangerous, and they are apt to fall off the berries and, to a lesser degree, off the shoots. According to Salmon occurrence of diseased berries on other branches is due to infection from perithecia that have over-wintered in budscales and similar places.

From his researches in Sweden Hammerlund (1915) reached somewhat different conclusions. He collected perithecial material at fortnightly intervals from October to May and tried to germinate the ascospores in the laboratory. This did not happen before February and then only with a few spores. The germinative capacity gradually increased, but not until May did 5–8% of the spores germinate. Possibly the low percentage germination was due to unsuitable laboratory technique. Hammerlund also observed that the brown mycelium on the shoot tips in May sent out thin-walled white mycelium like that found on the leaves in spring and summer and which seems able to bear conidia.



Fig. 107. American gooseberry mildew on the shoot tips at the end of the season.

Overwintering may, therefore, take place as perithecia or as mycelium. It may occur on berries lying on the ground, in the felted mycelium covering the tips of shoots (Fig. 107) and by perithecia lying loose in cracks and irregularities of the bark.

Reinfection in spring may commence on the highest shoots of the bush, but more often on berries and shoot tips near the ground or towards the inside of the bush, especially when the previous season's infection was heavy.

The history of the disease in Europe suggests that it has a capacity for very rapid spread. It has often been possible to show, however, that the first outbreak in a given locality originated on plants brought from previously infected localities. In Denmark it could be stated with confidence that about one quarter of the first hundred outbreaks detected in 1907 were due to infection from newly bought plants and that these originated at comparatively few nurseries. The fungus has many independent methods of dispersal. The conidia are light and may be spread by the wind. So, too, may the ascospores but they are much less numerous and probably not important as agents of dispersal by wind. Animals, especially insects, but certainly birds also, tools, market baskets, packing material, etc., may all play a part in carrying the conidia, and perhaps even perithecia, from diseased to healthy bushes.

Importance of the disease. The greatest loss from American mildew is due to infection of the berries which then appear unappetizing, are under-developed and have an insipid flavour. The young white stage found on unripe berries is easily washed off so that the fruit is still fit for cooking. Once the mycelium has turned brown it is difficult to wipe it off and the berries, though not poisonous, are unfit for use. As all the fruit on a plantation may be attacked the sale of ripe berries may be completely prevented.

Infected shoot tips cease growth prematurely and mildewed leaves remain small. Young infected bushes may be killed but usually it is only the shoot tips that die, to be replaced by young growth that becomes

infected in its turn. Attacks for several successive years lead to a progressive reduction in vigour of the bushes and to excessive branching. In Denmark during the 'twenties of the present century the disease was regarded as of less importance than formerly, but in subsequent years it appears to have become more destructive and has attracted a good deal of attention. A similar increase in intensity during the 'thirties followed by a decline in the early 'forties appears to have occurred in England (Moore, W. C., 1943, 1948). Evidently climatic factors play an important rôle, not yet fully understood.

Control. Early attempts to eradicate the disease by destruction of all infected bushes proved a complete failure. So did attempts to limit its spread by prohibiting sale of bushes and berries from infected localities. Sale for planting in Great Britain of any bushes substantially affected with American gooseberry mildew is prohibited under the Sale of Diseased Plants Orders of 1927 to 1941. No gooseberry or currant bushes may be imported into the British Isles without an accompanying Health Certificate issued by the Phytopathological Service of the exporting country.

Equally ineffective were such practices as planting chervil or chives under the bushes, though, as the disease varies in intensity so much from year to year, a grower may easily be misled as to the effectiveness of these or other control measures for a year or two.

The disease cannot be controlled solely by modifying the conditions of growth but neither will a spray programme be effective if growing conditions favour the fungus unduly.

The following horticultural recommendations should be noted:

(1) The bushes should be free-standing, not overshadowed by trees or other bushes.

(2) The ground should be kept clean and free from weeds.

(3) There should be an ample supply of phosphatic manure, but not too much potash and nitrogen. Late applications of nitrogen are especially undesirable as they encourage rank summer growth which will be attacked by the fungus and will not ripen. Potash deficiency encourages mildew and is to be avoided.

(4) During autumn the soil under the bushes should be turned over to bury fallen leaves. If the pH value is below six lime should be given.

As the attack is mostly on young growing tissues little is to be gained by cutting out diseased tips during the summer, the new growth formed will only be very susceptible to infection. Cutting off and burning diseased tips after leaf fall is, however, an excellent practice, providing it is not left until the winter, by which time many of the perithecia will have fallen.

Newly purchased bushes may be disinfected by being dipped into one of the following winter spray mixtures. All but the roots should be immersed.

One of the following winter sprays should be used:

Copper sulphate 6%, formalin 2½% and lime sulphur 10:90. Of these lime sulphur is often preferred because it also gives some control of red spider. The spraying must be done thoroughly so that all the shoot tips are completely soaked. In England and Ireland a spray with 2 lb. caustic soda in 10 gallons of water during February is recommended, especially when washing soda is to be used as the summer spray.

Summer sprays may be applied as soon as the attack is observed. Where the disease is usually prevalent the first spray should be given as soon as the flower buds are visible. Alkaline burgundy mixture (see p. 593) is particularly effective when used just before or just after flowering. If applied later or during sunny weather it is liable to cause scorching. Bordeaux mixture seems to be ineffective.

For later sprays to prevent mildew on the berries formalin may be used (1 pint commercial formalin in 25 gallons water with 2¼ lb. of soft soap). Lime sulphur about 1:30 is very effective but scorches varieties like Early Sulphur, Golden Drop, Yellow Rough and Leveller. Ammonium polysulphide 1:200 with soft soap 1 lb. in 25 gallons was much used in the past as it leaves no deposit on the

fruit. It is also liable to damage the sulphur-shy varieties mentioned above. A sulphur dust at the rate of about 20 lb. per 100 mature bushes gives good control on varieties not sensitive to sulphur. A washing soda and soft soap spray gives good control and may be safely used on any variety. It leaves no noticeable deposit on the fruit. The strength used in Denmark is 2½ lb. washing soda + 2½ lb. soap in 10 gallons and may cause scorching if not carefully mixed or if done in sunny weather. In England a strength of 1½–2 lb. washing soda and ½–1 lb. soft soap in 10 gallons water is recommended. The amount of soap required depends on the hardness of the water. At least two sprays are required, one as soon as the flowers have set and a second three weeks later. More may be necessary if heavy rains wash off the spray deposit.

Some differences in varietal susceptibility exist. Wild American mountain gooseberries are only slightly susceptible but generally bear only small fruit. The Canadian variety Red Jacket has fairly large fruit and is not much attacked by American mildew. Triumphant Yellow is also fairly resistant. All other varieties in general cultivation are so susceptible as to require protective sprays wherever the disease is prevalent.

European Gooseberry Mildew (*Microsphaera grossulariae* (Wallr.) Lév.)

This mildew, known in Europe since 1819, is called European gooseberry mildew to distinguish it from the disease caused by *Sphaerotheca mors-uvae.*

European gooseberry mildew occurs as a loose, dusty, grey coating on the older leaves. Shoots and berries are seldom attacked. In Denmark it has only been observed in gooseberries, but it occurs occasionally on red- and blackcurrant leaves in other countries, including England. The perithecia are produced on the mycelium and are easily distinguished microscopically from those of *Sphaerotheca* by their branched appendages.

The disease causes little or no loss. It may be prevented in the same way as American mildew, but winter spraying is hardly called for as there is no mycelium on the overwintering shoots.

Gooseberry Cluster Cup Rust (*Puccinia pringsheimiana* Kleb.)

Symptoms. In the spring orange-red, more or less thickened, arched or deformed patches appear on gooseberry leaves and fruit, and sometimes on those of red- and blackcurrants. Occasionally they also occur on the young shoots. On the upper surface of these blotches the spermogonia occur and on their lower surface is a dense cluster of tiny yellow cups with delicately toothed edges (Plate 10, Fig. B).

The uredo and teleuto stages occur on leaves of various species of sedge (*Carex*). This fungus is very closely related to one, *P. caricis* Rebentisch, which has its cluster cup stage on the common stinging nettle, *Urtica dioica*, and some authorities regard gooseberry cluster cup as only a physiological form of this sedge and nettle rust (Eriksson, 1920; Klebahn, 1922).

Development. The teleutospores of the rust overwinter on the sedge and germinate in spring to form basidiospores which infect gooseberries. The cluster cup spores formed on the latter cannot infect another gooseberry but only a sedge. Several generations of uredo spores occur during the summer on the sedges, so that infection of them is usually very extensive and there is an abundant production of teleutospores to carry the fungus over to the following year.

Importance. The amount of infection on gooseberries varies greatly from year to year and from place to place. According to W. C. Moore (1943), 1921, 1925, 1931, 1933 and 1945 were bad gooseberry rust years in Wales. A dry March seems to be the prelude to a severe attack because germination of the teleutospores is then delayed and is liable to occur all at once later on, when the gooseberry leaves are fully expended. Gooseberries near moors or marshy ground are always exposed to infection whereas those in gardens with no sedges in the vicinity are seldom, if ever, infected. The disease is only important on gooseberries where infection of the berries may lead to considerable loss of crop.

Control. Extensive gooseberry plantations should not be made in the neighbourhood of moors or marshlands. Sedges should be eradicated from the neighbourhood of all gooseberries. Peaty soil which may contain living sedges should not be brought into the plantations. In districts where infection is to be expected a single Bordeaux or colloidal copper spray applied to the bushes about a fortnight before they flower gives effective control (Saunderson and Cairns, 1937). The variety Red Jacket is seldom severely attacked.

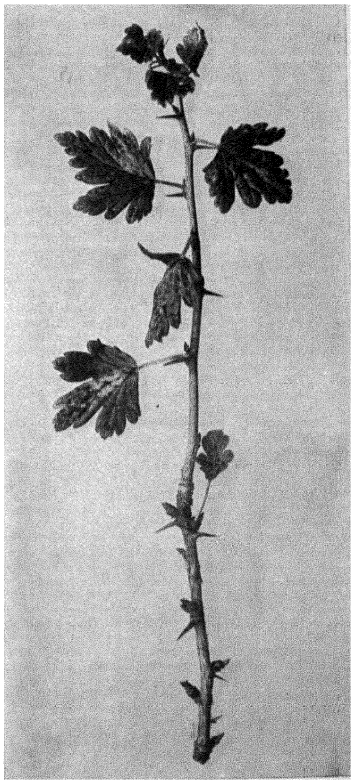


Fig. 108. Gooseberry leaf spot; the lower leaves have fallen.

Leaf Spot (*Pseudopeziza ribis* Kleb. = *Gloeosporium ribis* (Lib.) Mont. & Desm.)

Symptoms. The attack is most severe on the leaves, but all non-woody above-ground parts may be affected. Races of this fungus have been found on 25 species of *Ribes*, but they only cause important diseases of gooseberry, redcurrant and blackcurrant.

On the leaves the spots are small, dark brown, of an angular, irregular shape. They are first noticeable in June and later, as they become more numerous, several may coalesce to form large brown blotches. On the spots are minute brown *Gloeosporium* conidial pustules, visible under a lens, especially in damp weather when the spore masses become sticky and glisten. Diseased leaves turn yellow and soon fall off. The fungus attacks the oldest leaves first, so that defoliation proceeds from the base towards the tip of the shoots (Figs. 108, 109). Similar oblong spots appear on the petioles and fruit stalks, which may become girdled by them. Small black spots may occur on the fruit.

Cause and development. The conidial stage of the fungus occurs on the living leaves still attached to the bush. The ascus stage is a very minute discomycete, *Pseudopeziza ribis*, which develops on the fallen leaves and matures its spores in the following spring. It has been found on red- and blackcurrants and gooseberries (Klebahn, 1906, 1918; Blodgett, 1936). Various physiological races have been distinguished on different hosts and in different localities. All are pathogenic, but not equally so, and each most easily infects its own host plant. Blodgett (1935) found the ascospores germinate at 53° F. and the conidia at 68° F. Infection may occur on either leaf surface, but more easily on the lower.

Importance. The fungus does most damage by causing premature defoliation. Sometimes this occurs before the berries ripen and they are then underdeveloped and of poor flavour. In blackcurrant early defoliation may cause shrivelling of the fruit. Even when the fruit ripens normally the bushes are weakened so that their twigs ripen poorly and the formation

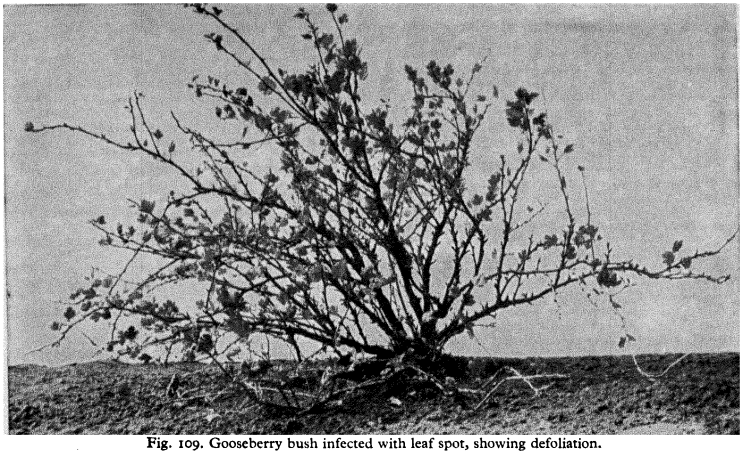


Fig. 109. Gooseberry bush infected with leaf spot, showing defoliation.

of flower buds for next year is unsatisfactory (Marsh and Maynard, 1930). Young plants in nurseries may be considerably weakened by leaf spot.

Varietal susceptibility. Among gooseberries May Duke, Walhalla, Whinham's Industry and Whitesmith are considered especially susceptible as well as a number of less valuable varieties. Achilles, Jaune Rouge and London are much less susceptible. Of blackcurrants, Baldwin, Boskoop Giant and Seabrook's Black are very susceptible, Climax, Goliath and Edina are less so. Redcurrant varieties derived from *Ribes rubrum* and *R. petraeum* (Red Spanish) are far less liable to leaf spot and defoliation than are those derived from *R. vulgare* (*sativum*).

Control. Fallen leaves should be raked up and burnt or buried in autumn. An 8:8:100 Bordeaux spray or equivalent copper fungicide should be applied just before flowering and again once or twice after the fruit has been picked. In nurseries the bushes should be kept covered with a fungicide throughout the summer, especially in wet seasons.

Bushes well supplied with nitrogen and phosphorus suffer less than lightly manured ones (Wallace, 1939).

Septoria Leaf Spot (*Mycosphaerella ribis* (Fuck.) Kleb. = *Septoria ribis* Desm.)

On gooseberry leaves and also on those of red- and blackcurrants this fungus causes small brownish angular spots. These are somewhat sunken and gradually turn grey in the middle, with a well defined dark red-brown margin. The grey centre is dotted with minute black pycnidia of the *Septoria* stage. The *Mycosphaerella* perithecia develop in spring on the fallen overwintered leaves. In Russia Vassilievsky (1924) found this disease causing small brown, rounded and slightly sunken spots on unripe blackcurrant berries. It may also occur on gooseberry fruit and cause it to fall.

In Britain *Septoria* leaf spot is probably widespread but of little importance. When it has become troublesome it can probably be controlled in the same way as *Gloeosporium* leaf spot (p. 173).

Dieback (*Botrytis cinerea* Fr.)

The grey mould fungus can attack any aboveground part of the gooseberry bush, but is most important as a cause of branch dieback. Leaves on affected branches wither quite suddenly; the branches dry up and often show a

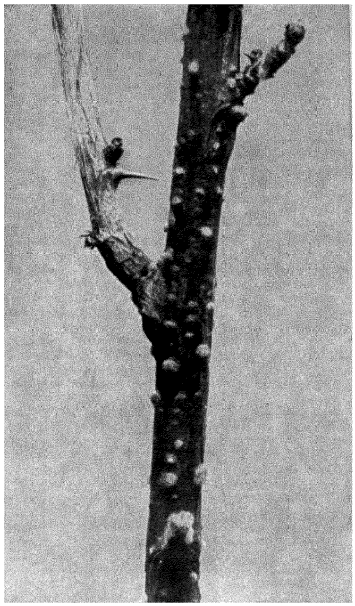


Fig. 110. Grey mould pustules on gooseberry stem.

brown stain in the wood, especially just below the bark. In damp weather tufts of grey mould appear in cracks of the bark (Fig. 110), and later on the usual small black sclerotia develop there. Sometimes the main stem is affected and the whole bush then dies. Branches weakened by other causes are particularly liable to attack. Thus, much *Botrytis* dieback was seen in Denmark after the severe winters 1940–2.

Shoot tips may also be infected, especially on young bushes; the leaves wither and the tips stand out bare and dead. In England it has been observed that such leaves first develop yellow then nearly white edges before withering completely. When the berries are attacked their skin turns grey or brownish, they rot and gradually become covered by grey mould (see also sun scald, below).

Marginal scorching of the leaves may also be due to damage to the roots, or to potassium deficiency and other bad growing conditions.

Control. Bushes killed by infection of the main stem should be pulled up and burnt as soon as possible. Individual dead twigs and branches should be cut out well back so as to remove all discoloured tissue, and should also be burnt.

Gooseberries should not be grown in enclosed damp or ill-drained situations. The soil should not be sour and the bushes should not be given heavy dressings of nitrogenous manures (see grey mould, p. 59).

Other Fungi Associated with Dieback

Several other fungi are commonly found on the branches of neglected and sickly gooseberry bushes. These include *Diplodina grossulariae* Sacc. & Bri., *D. oudemansii* All., *Leptosphaeria vagabunda* Sacc., and *Phoma suspecta* Mass.

The bark of affected branches becomes silvery and the fungus fructifications show up against it as small black specks. Rostrup (1902) considered *Leptosphaeria vagabunda* to be injurious in that it induced sickliness and dieback in branches and gradually spread through all the bushes in a large garden. In general it is difficult to decide if these fungi are parasitic or not, but none of them appears to be dangerous to vigorous, well-manured gooseberry bushes.

Reddening

It often happens that gooseberries turn red on one side while still far from ripe, even though they are of yellow or green fruited varieties. This is a response to unfavourable weather conditions such as too much sun, or strong or cold winds. It may reduce the market value of fruit, but does not impair their quality for stewing or jam making.

Sun Scald

In certain years many gooseberries turn pale, soften and soon fall off. They are then attacked by grey mould.

As far as we can ascertain this injury is a form of sun scald, possibly in association with drought.

The damage may be extensive; for example, the owner of a large gooseberry plantation in North Zeeland calculated that he lost nearly a ton of fruit in this way in 1934. There, as in other plantations, varieties with long green fruit suffered most.

As the damage seems to be worst on dry soil it may perhaps be restricted by judicious watering.

Summary of Gooseberry Diseases

I. Root and Crown

		page
Irregular nodules	Crown Gall	81

II. Branches and Shoots

A. Wilting shoots and dead branches:		
(1) Grey mould present under damp conditions	*Botrytis* Dieback	173
(2) Discoloured streaks in the wood	*Verticillium* Wilt	60
(3) Woody bracket fungus at the base	Collar Rot	164
B. Silvery bark covered with black dots	Branch Fungi	175
C. Loose, dusty grey mildew (seldom seen)	European Mildew	172
D. Dense floury white mildew, followed by a brown felt over tips of shoots	American Mildew	168
E. Orange-red swellings on green shoots	Cluster Cup Rust	172

III. Leaves

A. Leaf edges scorched:		
(1) Narrow light-brown scorching	Potassium Deficiency	165
(2) Broad reddish leaf edges (seldom seen)	Magnesium Deficiency	38
B. Loose dusty grey mildew	European Mildew	172
C. Floury white mildew, later turning brownish	American Mildew	168
D. Discoloured spots and patches:		
(1) Thickened orange-red patches	Cluster Cup Rust	172
(2) Small brown spots turning whitish at the centre	*Septoria* Leaf Spot	174
(3) Small brown spots, causing defoliation	Leaf Spot	173

IV. Fruits

A. Red or orange colouring:		
(1) Skin reddened on one side	Reddening	175
(2) Thick orange patches with cluster cups	Cluster Cup Rust	172
B. Soft berries:		
(1) Brown berries with grey mould	*Botrytis*	173
(2) Pale berries, often followed by growth of grey mould	Sun Scald	175
C. Very small fruit:		
(1) Berries dull, leaves scorched at the edge	Potassium Deficiency	165
(2) Berries small, leaves spotted and falling	Leaf Spot	173
D. Loose dusty grey mildew	European Mildew	172
E. Dense white floury mildew, soon replaced by a chocolate-brown felt	American Mildew	168

MULBERRY

Canker and Dieback (*Gibberella moricola* (Cesat. & de Not.) Sacc. = *Fusarium lateritium* Nees)

This fungus attacks young mulberry shoots, girdling them to cause small cankers and dieback. Reddish conidial pustules of the *Fusarium* stage appear on the dead bark. The perfect stage consists of clusters of blue-black spherical perithecia.

The disease is of little importance but cankered branches or twigs should be cut out and burnt. It was reported in England by Salmon and Wormald (1916).

Bacterial Blight (*Bacterium mori* Bayer & Lamb.)

Infected mulberry leaves bear small, angular, black spots, often surrounded by a yellowing zone. When they are numerous they may cause the leaves to turn yellow prematurely. On nursery stools similar black spots may appear also on the shoots.

Control measures have not been worked out, but it may be worth while to spray infected nursery stock with Bordeaux mixture.

The disease has been studied in England by Wormald (1924).

MYROBOLAN

Summary of Myrobolan Diseases

I. Root

		page
Tumours	Crown Gall	81

II. Branches and Trunk

A. Brown stain in the wood, often with gumming	*Verticillium* Wilt	156
B. Sunken dark patches on bark, bark brown when cut	Bacterial Canker	199

III. Leaves

A. Narrow leaves with scorched edges	Potassium Deficiency	42
B. Leaf spots:		
(1) Small brown spots, without translucent edges, later falling out	Shot-hole	197
(2) Small round dark spots with translucent edges, often falling out. Leaves eventually turning yellow	Bacterial Canker	199
(3) Wilted leaves, yellowing, sometimes reddening, withering and shrivelling	*Verticillium* Wilt	156

IV. Fruit

A. Brown rot with yellowish conidial pustules	*Sclerotinia fructigena*	121
B. Tough, elongated, pale fruit (Fig. 128)	Pocket Plums	195

This tree suffers from the same diseases as Plum. The more important are keyed out by symptoms in the above summary. Full descriptions will be found under Plum on pages 191–206.

PEACH

Leaf Curl (*Taphrina deformans* (Berk.) Tul.)

Symptoms. First signs of the disease are seen soon after the buds burst. The affected leaves are thickened, rolled and blistered over part or all of their surface. The blistered parts are fleshy and brittle but their colour varies somewhat from yellow to red according to the variety of peach or nectarine involved. In severe attacks the leaves are completely deformed (Fig. 111). After a time the affected parts become covered with a fine whitish bloom formed by the tightly packed asci of the fungus. Gradually the diseased leaves turn brown and fall, sometimes before they are quite dead. They are usually followed by a fresh set of leaves, which may be quite healthy.

Affected branches become thickened and stunted in growth; often they develop gummosis and may eventually die. Petals and stamens may be attacked, and in North America whitish, later reddish, slightly raised patches due to this fungus have been observed on the fruit. These patches crack, making it easy for rotting fungi to gain access to the flesh of the fruit. Klee (1940) has seen fruit attacked in this way in Germany.

Cause and development. Leaf curl is caused by the fungus *Taphrina deformans*. There has been considerable controversy over the way in which this parasite overwinters. Many authors believe it passes the winter as mycelium in the branches and grows into the young leaves as the buds burst, stimulating the cells of the infected tissue so that they grow abnormally and cause the leaf to be twisted and blistered.

Others, e.g. Fitzpatrick (1934) in Ontario, think the fungus does not overwinter as mycelium but that the leaves are infected from the bud scales as they emerge. The source of infection would then be ascospores or conidia budded off ascospores that have overwintered on the bud scales and twigs and have been washed into the bursting buds by rain. The spores germinate to form a short hypha which can penetrate either surface of the leaf, it does not enter by way of the stomata. Hesler and Whetzel (1917) had earlier considered this the probable method of overwintering. Mix (1935) grew the fungus in artificial culture and obtained yeast-like conidia which survived drying for 315 days and heating to 203° F. but not to 212° F.

Importance. Peach leaf curl was first described by an English gardener in 1821, and is now known wherever the tree is grown, especially as an orchard crop. It may be very destructive. Pierce calculated that in the last century it caused an annual loss to the peach growers of the United States of over $3,000,000. The loss today is no doubt much less because

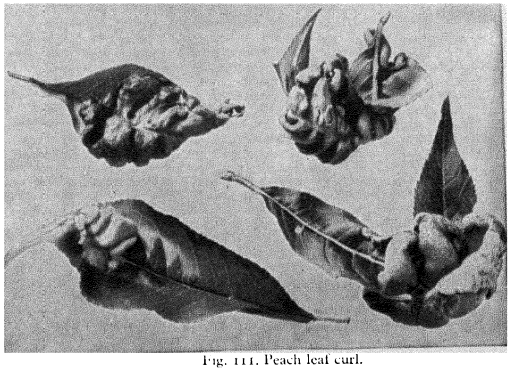


Fig. 111. Peach leaf curl.

of improved control measures, but the disease is far from eradicated. It has often been serious in Denmark. Damage is caused in various ways:

(1) Premature leaf fall and consequent production of a second set of leaves weakens the trees and reduces or entirely prevents the formation of fruit buds for the next year.

(2) Shedding of unripe fruit because of the loss of foliage.

(3) Occasional direct attacks on the fruit.

(4) Dieback of branches and, in extreme cases where all the leaves are lost several years in succession, death of the tree.

(5) Loss in the nursery through taking diseased buds for budding and by poor growth of the affected trees. The disease is most destructive after a damp autumn, for then the fungus has favourable growing conditions from the outset.

Control. On a small scale the blistered leaves should be picked and destroyed before the bloom begins to appear on their upper surface, i.e. before spores are produced. Generally, however, hand picking does not prevent a recurrence of the disease next year. Spraying must be resorted to just before the buds burst. When there has once been an attack the spraying must be repeated annually, for complete eradication is not to be expected and the efficacy of the spray varies somewhat from year to year.

In Danish experiments lime sulphur 4: 100 was quite effective. A 10:90 lime sulphur spray is quite harmless to the trees and is in general to be preferred because it also gives some control of aphis eggs and scale insects.

An 8: 8:100 Burgundy spray has also been recommended before the buds burst and a Bordeaux spray at leaf fall. Summer sprays with Bordeaux mixture or other copper fungicides seem not to suit the trees. All the sprays must be very thoroughly applied so that no part of the tree escapes. With espalier trees it may be very difficult to wet the bark next to the wall, but it can usually be done if the trees are well soaked with a powerful sprayer.

To some extent it is possible to avoid trouble by growing resistant varieties. Rivers Early is considered very susceptible, while Alexander, Hales Early and Waterloo are resistant. According to Cayley only varieties with glands on their leaf stalk are attacked by leaf curl. Mildew, on the contrary, attacks the varieties with no glands.

All possible steps should be taken to eradicate the disease from nurseries. Trees should not be sold from infected plantations until all peach trees in that part of the nursery have been thoroughly sprayed and have been observed to be free from leaf curl for at least one summer. Nor should buds be taken from them till then. No old diseased trees should be allowed to remain in the vicinity of nurseries. If newly planted trees are found to have peach leaf curl in their first summer it is reasonable to assume they were infected at the nursery.

Mildew (*Sphaerotheca pannosa* var. *persicae* Woronich.)

This fungus forms a thick mealy coating on young leaves and buds; on the fruit it produces pale, raised patches which later crack and are followed by rotting. Peach and apricot are both susceptible. It was at first believed that the fungus was the same as that attacking roses, but Woronichine (1914) proved that two different physiological races are involved. According to a verbal communication from Cayley, mildew occurs on those peach varieties which lack glands on their leaf stalks.

The fungus apparently overwinters in the leaf buds so in the spring following an attack a 10:90 lime sulphur spray may be tried, just before the buds begin to swell. During the summer, dusting with sulphur or other usual mildew remedies may be used (see p. 67). On indoor trees sulphur fungicides should not be attempted during or just after flowering.

Virus Diseases

In North America peaches suffer from six virus diseases so far unknown elsewhere. Several of these can be transmitted to other stone fruit, so that it is important to keep a strict check on importation of such trees from America.

In **peach yellows** the leaves become pale and brittle and curved like a claw. Here and there on the tree thin, narrow-leaved shoots occur. The fruit ripens prematurely. Infection can be transmitted by budding or grafting and by a species of leaf hopper, but neither by seed nor by sap inoculation. This is one of the few virus diseases that can be cured, at least in young trees. Potted infected trees kept at a temperature of 95° F. for 25 days were healthy in the following year and a similar result was obtained by plunging resting trees for 10 minutes in water at 122° F. (Kunkel, 1936).

In a rather similar disease, **little peach** ripening of the fruit is retarded. **Rosette** is characterized by a rosette-like arrangement of the leaves, which eventually became speckled. Infection transmitted by budding leads to death of the tree in, at most, 3 years.

In **red seam** there are irregular, prematurely ripened fruits, often with a red patch over the seam. In **phony peach** the trees gradually become dwarfed, with unnaturally vigorous foliage and worthless fruit. Here, curiously enough, infection takes place only through the roots and is easily transmitted by root grafting, but the incubation period is two years. Finally, a **peach mosaic** disease has also been recognized. A mosaic disease also occurs on stone fruit, including peach, apricot and plum, in Bohemia. A mosaic disease known as **line pattern mosaic**, in which pale yellow lines and sometimes yellow blotches appear on the leaves, was found on peaches in Lincolnshire and Kent in 1945 (Moore, W. C., 1948).

Spread of all these diseases has been checked by cutting down and burning diseased trees and by critical selection of bud wood and stock. Plum trees may be infected with peach yellows without showing any distinctive symptoms and may then remain for years a menace to surrounding peach orchards (Manns, T. F., 1934).

Other Peach Diseases

Grey mould (*Botrytis cinerea*) sometimes causes a rot of the fruit and a dieback of young twigs in trees under glass. Blossom wilt (see p. 152) occurs occasionally. For descriptions of the other diseases listed in the summary below, see under plum.

Summary of Diseases on Peach, Apricot and Nectarine

		page
I. Root		
Nodules	Crown Gall	81
II. Trunks and Branches		
A. On this year's shoots:		
(1) Dark oblong patches	Bacterial Canker	199
(2) Dry brown spots with red rims	Shot-hole	197
B. Dead branches	Dieback	198
	Leaf Curl	178
C. Gummosis	Leaf Curl	178
D. Dead branches with a brown stain in the wood, ultimately with grey and mauve fructifications	Silver Leaf	191
E. Stunted growth	Virus	179
III. Leaves		
A. All the leaves silvery	Silver Leaf	191
B. Thick yellow or red blisters	Leaf Curl	178
C. Yellow-spotted leaves or narrow leaves growing in close rosettes	Viruses	179

		page
D. Leaf spots:		
(1) Small brown spots with no translucent margin, often falling out	Shot-hole	197
(2) Small, dark, round spots with a translucent edge, often falling out	Bacterial Canker	199
(3) Whole leaf eventually yellow	Bacterial Canker	199
E. Thick, white, mealy coating	Mildew	179
IV. Flowers		
Flower trusses or buds withered	Blossom Blight	152
V. Fruits		
A. Small grey or greenish spots	Scab	155
B. White, mealy, raised patches, ultimately cracking	Mildew	179
C. Whitish raised patches, later turning reddish and cracking	Leaf Curl	178
D. Brown rot :		
(1) Rot covered with hairy grey mould	Grey Mould (*Botrytis*)	59
(2) Rot bearing yellow cushions of conidia	*Sclerotinia fructigena*	121
(3) Conidial cushions grey	*Sclerotinia laxa*	152

PEAR

Pear Scab (*Venturia pirina* Aderh. = *Fusicladium pirinum* (Lib.) Fuck.)

This fungus was first observed by Madame Libert, in Belgium in 1832, and was issued on diseased leaves in her exsiccati under the name *Helminthosporium pyrorum*. In 1840 it was recorded by Desmazières in France, but he later identified it with apple scab *Cladosporium dendriticum*, described by Wallroth in 1833. In 1869 Fuckel differentiated between apple and pear scab and called the latter *Fusicladium pirinum* (Lib.) Fuck. All these mycologists had been dealing with scab on leaves but in 1864 Peyl described the fungus on the fruit, under the name *Cladosporium polymorphum*. In Denmark pear scab on twigs and leaves was exhibited by Rostrup at the Seed Producers' Agricultural Exhibition in 1878. In *Gartner-Tidende*, 1886, he gave a full account of the disease on leaves, buds and fruit.

Finally in 1897 Aderhold showed that *Fusicladium pirinum* (Lib.) Fuck. had as its perfect stage the pyrenomycete *Venturia pirina* Aderh.

Pear scab is very like apple scab but is not due to the same fungus and the two parasites are confined each to its own host. Each species has two-celled ascospores, with one cell larger than the other. In pear scab the larger cell is uppermost in the ascus, in apple scab the lower cell is the larger.

Symptoms. Scab on the flowers is usually confined, as in apple blossom, to the flower



Fig. 112. Pear scab on the blossoms.

stalk and receptacle (Fig. 112), but Salmon and Ware (1924) have also seen the fungus on the pear petals themselves. Scab on pear leaves (Fig. 113) forms dark-brown velvety patches but is far less abundant than scab on apple leaves. Probably the glossy, quick-drying, upper surface of pear leaves is responsible for this. In Switzerland a form of pear scab occurs in autumn (Osterwald, 1923; Wiesmann, 1933) in which the leaves are shed before they assume the usual autumnal colouring. Such leaves bear hardly any of the usual conidia but bear numerous bodies resembling

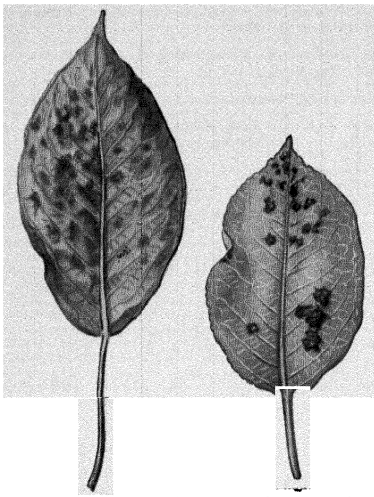


Fig. 113. Pear scab on the leaves. Left: upper surface. Right: lower surface.

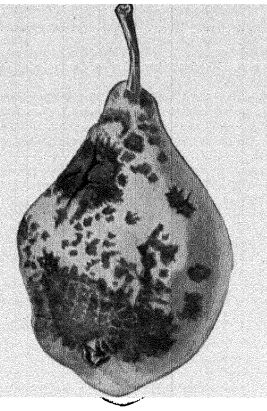


Fig. 115. Pear scab. The old cracked scabs open a way for *Sclerotinia fructigena* and other fruit-rotting fungi.

pycnidia. No perithecia develop on them and they can be of no importance in the overwintering of the fungus.

Pear scab on the fruit is often a very serious disease and leads to the fruit being deformed and split (Figs. 114 and 115).

Attacks on the shoots remain active apparently for several years and may, therefore, perhaps be better styled branch attacks. On the upper surface of the current season's growth there may be seen small blister-like swellings which burst either the same summer or the following spring, exposing the dark mycelium and conidia. Attacked shoots may die before they are a year old, but often both the apical bud and the shoot continue growing. Then, when the branch is two or three years old, longitudinal and transverse cracks appear in the bark so that it assumes a scaly appearance and the branch generally dies (Fig. 116). In the cracks of the bark are mycelium and spores, but conidial production generally dies down, the mycelium becomes compact and under favourable conditions perithecia may be produced in it.

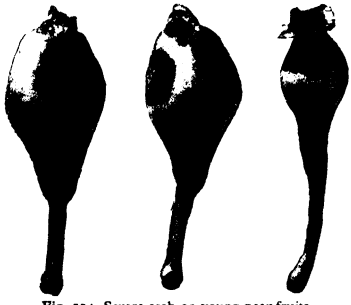


Fig. 114. Severe scab on young pear fruits.

Salmon and Ware (1924) found scab pustules on bud scales of pear. They point out that it appears not only on the current season's growth, but also on the fruiting spurs.

Development. Perithecia may develop on the fallen infected leaves during winter, but this is probably a less important mode of overwintering in pear scab than in apple scab. The ascospores ripen just after the buds burst in spring. Ewart (1910) has proved that conidia of pear scab are very resistant to cold, more so than those of apple scab, so that infection may also come from conidia borne

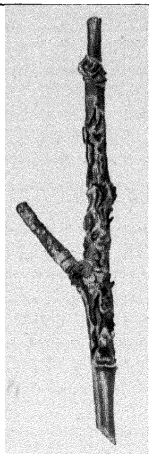


Fig. 116. Pear scab on a twig.

on scabby pears that have overwintered on the ground. The chief method of overwintering is, however, by the pustules on the previous summer's growth. Conidia produced in these infect the young leaves and fruit.

Importance. Pear scab is the worst fungus disease of pears, but the economic loss it occasions, though large, varies considerably from year to year, according to weather conditions. There is a close general correspondence between the ravages of apple and pear scab and they both cause the same kind of attack on the corresponding organs of apple and pear. Pear scab does more damage to the shoots and less to the leaves than apple scab.

Varietal susceptibility. Pear varieties differ greatly in their susceptibility to scab, though none is completely immune, and the degree of resistance of a variety may be modified by the environment. According to Wiesmann (1931) there are several physiologic races of pear scab, differing in host range and in growth in artificial culture. In inoculation experiments with the different races the percentage infection obtained varied from 26.5 to 0.5. The risk of severe infection of a particular pear variety is therefore greater when it is growing near other varieties susceptible to attack by the same strain of the fungus. The following list of susceptible and resistant varieties is based on orchard observations in Denmark, not on inoculation experiments; but compare, under apple scab, observations on the difficulty of grading susceptibility of varieties.

FAIRLY RESISTANT	SUSCEPTIBLE
Clapp's Favourite	Beurré d'Amanlis
Colorée de Juillet	Bonne Louise
Dobbelt Philip	Charneu
Eye Wood	Clara Frijs
Giffard	Doyenne du Comice
	Diel
	Josephine de Malines
	Kejserinde
	Napoleon I
	Nouveau Poiteau
	Seigneur
	William's Bon Chretien

In England Diels, Clapp's Favourite and Doyenne du Comice are especially liable to scab infection on the shoots. Bonne Louise is particularly prone to shoot infection in Denmark.

Control. The principles of pear scab control are the same as for apple scab, but it is more important to try to prevent infection of shoots and spurs.

As perithecia may develop on overwintered fallen pear leaves, these should be raked up and burnt or buried unless it is quite certain that there was no scab on them in the previous autumn. Diseased twigs and spurs should be cut out as far as possible and early copper sprays may check sporulation on the pustules that remain. A 6% copper sulphate spray or 8:8:100 Bordeaux mixture may be used just as the buds show green at the tip. But as the fungus penetrates well into the bark there is little prospect of killing the twig pustules completely by spraying.

Bordeaux mixture is to be preferred to lime sulphur and is generally safe on pears at the 8:8:100 strength, but if russetting of the

fruit occurs at that strength a 4:4:100 spray may be used. Where red spider is prevalent it may be preferable to use lime sulphur 2:100, but this may cause scorching of the leaves, especially if applied in sunny weather. For post-blossom sprays it is unsafe to use a stronger lime sulphur spray than 1:100, and this is inferior to Bordeaux mixture in controlling scab.

See spraying table (p. 584).

Dusting is less effective with pears than with apples, probably because of their smoother leaves.

Leaf Fleck (*Mycosphaerella sentina* (Fuck.) Schroet. = *Septoria piricola* Desm.)

Small circular or irregular brown spots on pear leaves, each ultimately becoming light grey at the centre, with a dark red-brown

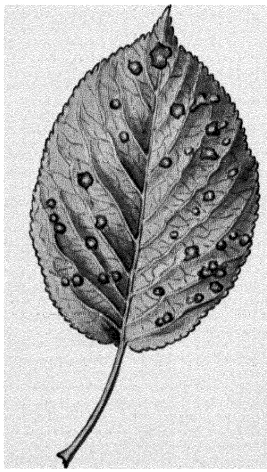


Fig. 117. Pear leaf fleck, grey spots with red-brown edges.

margin and dotted with minute black pycnidia, are caused by the *Septoria* stage of *Mycosphaerella sentina* (Fig. 117). The *Mycosphaerella* perithecia develop on the fallen leaves in spring.

In Denmark the disease is almost confined to Nouveau Poiteau or Tongres. It may somewhat restrict carbon assimilation and cause premature leaf fall, but has seldom been very destructive.

In America Stewart (1915) and Crosby (1929) consider the disease can be controlled by summer spraying with Bordeaux mixture or lime sulphur. In Denmark fairly severe attacks have occurred year after year on Nouveau Poiteau in spite of 3–4 summer sprays. No unsprayed controls have been available for comparison. Leaves fallen from infected trees should be raked up and burnt or buried in autumn.

Leaf Blister (*Taphrina bullata* (Berk.) Tul.)

Symptoms. This fungus, also sometimes called *Exoascus bullata*, forms blisters protruding from the upper surface of pear leaves. At first they are green, but later turn black



Fig. 118. Pear leaf blister.

on their upper surface, while the lower becomes covered by a white coating of asci. Leaf blister is seldom of much importance alone for usually only a few blisters occur on the diseased leaves. Briton Jones (1924) found pear leaves infected by leaf blister were particularly severely attacked by scab, perhaps

because the scab spores adhere easily to the blisters.

Control. The disease closely resembles peach leaf curl, and is controlled in the same way (see p. 178).

Rust (*Gymnosporangium sabinæ* (Dicks.) Wint.)

Symptoms. Red spots containing minute yellow spermogonia appear on the upper surface of pear leaves in spring. The spots enlarge to about ¾ inch and are elliptical, orange or nearly red with the spermogonia, now turned black, in the middle. They may have a narrow, bright red margin. Towards the end of the season the orange spots become thickened and starch accumulates in them, nourishing the fungus at the expense of the host. The under-surface becomes hard and spongy and brown wart-like bodies open at the tip appear on it. These are the aecidia, and were formerly known as *Roestelia cancellata*. At the tip they split into fibres which remain united by transverse bars to form a trellis-like structure through which the spores escape in autumn. Ultimately this disappears leaving only the warts, each with a pore at the tip. The pear blossom is very seldom attacked.

The other host of the fungus is the savin tree (*Juniperus sabina*). On this the affected branches are somewhat swollen and the fungus emerges from the swellings in spring in the form of yellow or orange tongue-like bodies. These may be up to ½ inch long in damp weather and have a gelatinous consistency. On their surface they bear the basidiospores. Later, during the rest of the season, only slight scars are visible on the bark where the tongues have been.

Cause. Different stages of the same fungus *Gymnosporangium sabinae* are responsible for this rust on both pear and savin. Micheli described the latter in 1729 as *Puccinia non ramosa, major pyramidata*. The rust on pear was described by Jaquin in 1773–8 under the name *Lycoperdon cancellatum*. In 1837 it was noted in France that the presence of savin was injurious to pears, but A. S. Ørsted in 1865 was the first to demonstrate the identity of the rust on the two hosts.

Pear trees may be infected each spring by basidiospores from the savin, and in autumn the latter is liable to be reinfected by *Roestelia* spores from the pear. But the fungus is perennial in the savin branches without killing them, so that once it has been infected a savin tree may remain a menace to neighbouring pears for many years.

Importance. The fungus is of no practical importance on the savin tree, but the attack on pear may be quite serious. Much of the upper surface of the leaves may be destroyed; they fall prematurely, thus weakening the tree, and in the worst cases the fruit too may be destroyed. For some ten or twelve years we have made observations at Lyngby on a diseased savin and neighbouring pear trees. The attack on the latter varies greatly in intensity from year to year. In a wet spring the teleutospores are developed in profusion on the savin and conditions are also favourable for spore germination on the pear leaves. The intensity of attack diminishes very rapidly with increasing distance from the infected savin; pear trees only about 10 yards away are much less damaged than those immediately adjacent to the savin. The importance of wind in spreading infection is very noticeable, and occasional infected pear leaves can be found at considerable distance from the savin. Where a severe rust outbreak has occurred on pears the savin should be sought within a radius of some 50 yards.

Prevention of pear rust is obviously to be accomplished by destroying infected savin bushes. There may be objections to this course in the case of large ornamental bushes and those on a neighbour's property. In such cases an attempt should at least be made to cut out and burn the infected branches, preferably before spore horns have formed on them. All branches on which swellings and small scars can be detected should be cut out before the month of April.

Summer sprays with lime sulphur or Bordeaux mixture do not appreciably prevent infection of the pear leaves.

Fire Blight (*Bacillus amylovorus* (Burr.) Trev.)

A disease was discovered in 1780 on pear twigs in New York State, to which the name fire blight was afterwards applied because of the scorched appearance of leaves and flowers. The disease was very destructive and speculation was rife as to its cause until Burrill in 1879 showed it to be due to bacterial attack. Bacterial diseases of plants had not previously been recognized, and Burrill had to face considerable criticism before his diagnosis was accepted and *Bacillus amylovorus* was recognized as the cause of fire blight. The organism is now known to attack many other rosaceous trees, including other species of pear, apple, quince, including *Cydonia japonica*, cherry, plum, almond, apricot, amelanchier, rowan, hawthorn and the flowers of strawberry.

Symptoms. Affected flowering spurs look as though scorched by fire and the brown leaves and blossoms remain hanging on the tree as in an attack of blossom wilt. Infection spreads through the bark, which turns brown and cracks here and there, to exude drops of slimy fluid swarming with bacteria. At the base of the spurs infection spreads into the branches, rings them, and in the course of a year or two may destroy a large tree. The bacteria overwinter in the infected bark and are exuded from it again in droplets in spring. Aphides, bees and other insects become contaminated from these slimy drops and transfer the bacteria to flowers. They are also scattered in rain splashes during storms. When green fruit are infected they become mummified.

Distribution. Fire blight is the most destructive disease of pear in North America and occurs also in Japan and New Zealand. It has been reported from Russia, Italy and Switzerland (1912, 1921, 1923), Baden (1913, 1915) and East Prussia, but none of these reports has been confirmed and they may well have been due to confusion with bacterial blossom wilt described below (Pethybridge, 1934). Great care is necessary to avoid introduction of the true fire blight from North America.

Control measures are based on ruthless cutting out and burning of infected branches (Day, 1928).

Bacterial Blossom Wilt (*Bacterium barkeri* (Berridge) Elliott & *Pseudomonas prunicola* Worm.)

This disease has much the same effect as typical blossom wilt due to *Sclerotinia laxa*. Whole flower trusses turn black and wither and the disease spreads into the spurs, killing them. Microscopic examination is necessary for reliable diagnosis. The dead tissue will then be found to be swarming with bacteria instead of containing fungus hyphae. In the

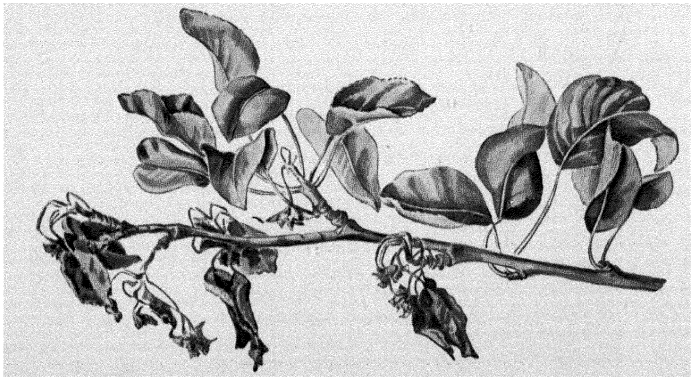


Fig. 119. Blossom wilt of pear caused by *Sclerotinia laxa*.

West of England *Bacterium barkeri* has been found to be the causal organism, but Wormald has found that *Pseudomonas prunicola*, one of the causes of wither-tip and dieback of stone fruits, can also cause both the death of pear flower spurs and a small blackish spot on pear fruit.

The only known control measure is to cut out and burn the wilted blossom trusses and dead spurs (Wormald and Montgomery, 1941; Wormald, 1946).

Stony Pit (cause uncertain, probably a virus)

It not infrequently happens that pears, especially Count Moltke in Denmark, become stony. They develop a dimpled surface because the places where stone tissue forms do not show the normal increase in size as the fruit grows. In virulent cases (lithiasis) the skin bursts above the stony areas and a white powder of loose stone cells is exposed (Fig. 120).

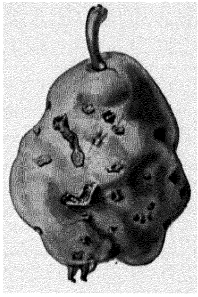


Fig. 120. Stony pit of pear.

Stone formation may occur at points of injury by sucking insects, bugs or others, or by hail. Most cases, however, are clearly due to some other cause. The trouble is especially frequent in gardens where the trees are under unfavourable conditions for growth, for example, where the earth is not kept loose beneath them and where shortage of water is liable to occur. The trouble is general on dwarf trees of Count Moltke, especially after the severe winter of 1940–2, apparently because the trees were weakened by the frost. The trouble is not general in well-kept orchards.

A similar condition is not uncommon in Pitmaston Duchess and Doyenne du Comice throughout the South of England, where it was unusually prevalent in 1944. Wallace, Ogilvie and Swarbrick (1944) think it a virus disease, possibly akin to that called stony pit in North America (Kienholz, 1939). Research is needed to settle this problem, for a very similar condition has been ascribed to boron deficiency (Kienholz, 1942).

Prevention. The soil should be kept loose under the trees. Covering it with peat moss litter or similar substance to check evaporation should also be tried. Trees especially exposed to drought should be kept watered. On sloping ground or near a path watering can be done through drain pipes inserted vertically into the soil around the tree. Watering should be done especially in early and mid-summer. In a dry August more restraint should be shown in watering. If the trees receive much water in August and heavy rains supervene the soil may become too wet for proper ripening of the wood that autumn. Count Moltke should preferably be worked only on a wild stock.

Wind Scorch

Where pear leaves are much exposed to high winds the leaves may suffer extensive browning and laceration. The condition is reminiscent of sun scorch, but distinguishable by the tattered appearance of the leaves. Pear leaves tend to turn brown or even black as soon as anything goes wrong with them. The trees should always be planted in as sheltered a situation as is available.

Sun Scorch

Symptoms. Large discoloured patches appear on the leaves, at first brown but soon turning black, and eventually the leaves are shed. Sometimes the damaged leaves occur mostly on the sunny side of the tree, in other instances they are scattered about it (Fig. 121).

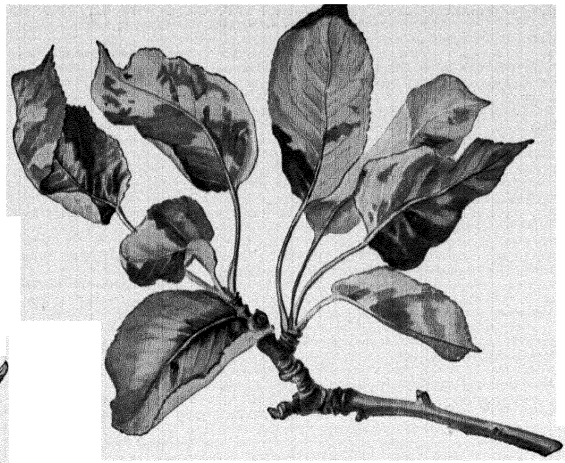


Fig. 121. Sun scorch on pear leaves.

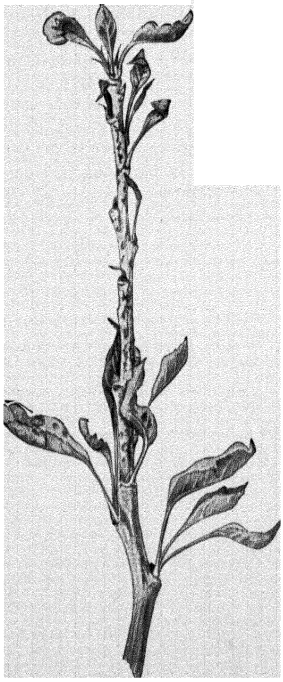


Fig. 122. Pear mildew (see apple, p. 111).

Cause. The damage is apparently due to excessive transpiration on warm days during dry weather.

Importance. We have seen trees severely sun scorched without suffering any permanent damage. In very severe cases, however, there is likely to be a reduction in the fruit crop that year and a loss of vigour in the tree.

In 1932 about a score of cases of sun scorch were reported within a short time of one another from different parts of Denmark. These and all later instances have all been on either Graapære (Grey Pear) and Count Moltke. Most occurred in July and August. Similar damage has been reported from Germany.

Prevention. The only action that can be suggested at present is to water susceptible varieties well in hot dry weather.

The other diseases of pear listed on p. 189 occur also on apple and are described under that host.

Silver leaf (see p. 191) rarely attacks pear trees but has occasionally been reported on them in England.

Summary of Pear Diseases

I. Roots

	page
See under apple	147

II. Trunk, Branches, Twigs and Spurs

A. Minute blisters on twigs, occasionally abundant enough to kill spurs	Scab	181
B. Dead spurs, ultimately bearing grey or yellowish conidial pustules	Blossom Wilt	120
C. Small sunken discoloured patches of bark, ultimately covered with grey or olivaceous pustules	Surface Canker	116
D. Deep cankers which may girdle and kill branches, bearing clustered dark red perithecia in winter and white pustules in summer	Canker	113
E. Shallow cankers, the bark cracking and peeling off in flakes, dotted with blackish pycnidia	*Physalospora* Canker	125
F. Brown bark oozing drops of slimy fluid (not in Europe)	Fire Blight	186
G. Dead branches	Dieback	198
H. Current season's shoots coated with a powdery felt and later bearing dark specks (perithecia)	Mildew	111

III. Leaves

A. Leaves narrow, small, yellowish green or turning brown between the veins	Iron Deficiency	36
B. Leaf spots:		
(1) Small, dark or olivaceous spots	Scab	181
(2) Small oblong, brown spots, turning whitish at the centre	Leaf fleck	184
(3) Small red-brown spots	Leaf Blight	207
(4) Large orange spots, *Roestelia* on the under-side in late summer	Pear Rust	185
(5) Dome-shaped blisters, first green, then black	Leaf Blisters	184
C. Scorched edges and discolorations:		
(1) Brown patches between veins, leaves slowly turn brown all over and fall	Sun Scorch	187
(2) Leaves scorched and shed after spraying	Spray Damage	49
(3) Torn leaves with dark brown markings	Hail Damage	20
(4) Scorched leaves hanging on the tree (not in Europe)	Fire Blight	186
D. Mealy white coating	Mildew	111

		page
IV. Flowers		
A. Spotted flower stalks, blossoms wither	Scab	181
B. Whole truss of blossoms wilts suddenly and spur killed :		
(1) Grey *Monilia* pustules on the dead flower stalk and spurs	Blossom Wilt (*Sclerotinia laxa*)	152
(2) No pustules	Bacterial Blossom Wilt	186
C. Flowering spurs appear as though scorched by fire (not in Europe)	Fire Blight	186
V. Fruits		
A. Young fruits fall in large numbers	Shedding of Fruit	131
B. Superficial damage:		
(1) White mealy coating	Mildew	111
(2) Russetting skin, after spraying	Spray Damage	49
(3) Small lumps with abundant stone cells in tissues	Dimpling	187
(4) Deformed fruits with yellow or orange spots	Pear Rust	185
(5) Blackish-brown spots, later associated with cracks	Scab	181
(6) Large ill-defined brownish stains in storage	Scald	144
(7) Small round brownish patches, often covered with very minute black dots	Fly speck	126
(8) Black coating easily rubbed off	Sooty Mould	126
(9) Small black scattered spots	Bacterial Fruit Spot	186
(10) Small clusters of brown cups with torn margins	Hawthorn Rust	441
C. Damage to flesh of fruit:		
(1) Deep cracks associated with superficial black blotches	Scab	181
(2) Quick brown rot bearing pustules of:		
(a) Blue-green mould	Blue Mould (*Penicillium*)	127
(b) Yellowish cushions	*Sclerotinia fructigena*	121
(c) Grey cushions (seldom seen on fruit)	*Sclerotinia laxa*	152
(d) Hairy grey mould	*Botrytis cinerea*	123
(e) Brown rot, often spreading from scab lesions with pinkish mould on surface	Pink Rot (*Trichothecium*)	128
(f) Sunken rot starting round the eye and bearing whitish pustules	Eye Rot (Canker Fungus)	113
(g) Rapid brown rot, especially of windfalls and fruit on low branches, without pustules but sometimes showing a white bloom in cracks	*Phytophthora*	125
(h) Black rot with numerous grey or olive pustules	*Phacidiella*	118
(3) Brown discolorations in the flesh:		
(i) Small dry spots	Stony Pit	187
(ii) Browning spreading outwards from the core	Core Rot	143
(iii) Browning of the flesh between skin and core	Low Temperature Breakdown	141

PLUM

True Silver Leaf (*Stereum purpureum* Pers.)

Symptoms. The first sign of silver leaf is the appearance of a silvery sheen on the leaves of one or more branches (Fig. 123). Gradually the condition spreads over the tree, and as the leaves age their metallic lustre becomes intensified and rusty patches appear on them, especially over the midrib. The leaf tissue may split and the edges of the holes and the leaf margins may become fringed with brown. In true silver leaf the branches gradually die and the whole tree may succumb. Sometimes, however, especially with apple trees, natural recovery may occur, especially in warm, dry summers.

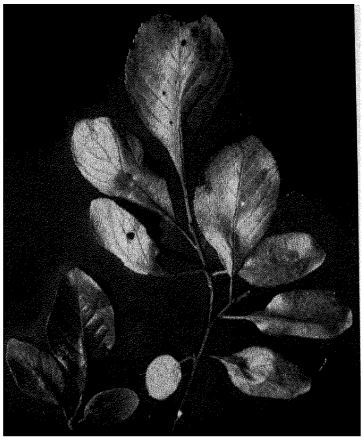


Fig. 123. Silver leaf of plum. Normal leaves on left.

After a branch has died, but often while the rest of the tree is still living, the fungus fructifies on the dead bark. When the tree is quite dead the fungus breaks out all over it, sometimes even on such parts of the roots as are not completely covered by soil. It forms a tough, thin, leathery crust over the bark, with a smooth purple surface. With age, however, the colour may fade to brown. The upper edge of the crust commonly bends over horizontally to form a small, thin, leathery bracket, smooth and purple on the lower surface but covered with an indistinctly zoned greyish felt on the upper side. The purple surface is covered by a layer of basidia.

When branches bearing silvery leaves are cut off a brown discoloration can be seen in the wood, but in the early stages of the attack it may be necessary to cut far down the bough before finding this stain. Microscopic examination shows that the cell walls are coloured brown, there is gum in the vessels and they are permeated by fungus mycelium. The fungus does not occur in the leaves; the silvery sheen there is due to the upper cuticle becoming detached as a response to the poisons liberated into the sap stream by the activity of the fungus in the wood. Some trees, like rhododendrons, may be killed by *Stereum purpureum* without showing silvering of the leaves. Sometimes, especially in top-grafted apple trees, infection by *S. purpureum* leads to the outer layers of the bark becoming detached and peeling off in papery scales. A similar condition may arise in the absence of infection, however, especially on heavy soils.

Cause. Prillieux described silver leaf of fruit trees in France in 1885 and it was noted in Germany by Sorauer in 1886 and Aderhold in 1895. None of these workers, however, recognized the connection between a fungus and silver leaf; they supposed it to be a non-parasitic disorder, perhaps associated with a lack of lime.

In 1902 Percival recorded the occurrence in England of *Stereum purpureum* on dead branches in association with silver leaf and demonstrated its connection with the disease by inoculation experiments. Most of our knowledge of the disease is based on the work of Professor F. T. Brooks and his colleagues at Cambridge, from 1911 onwards.

Development. The spores of the fungus drift about in the air, but can only gain access to living trees when they fall on open wounds due to pruning, breaking of branches, or other injuries. It is therefore easy for silver leaf to attack trees that have been cut back and

top-grafted, though false silver leaf is also liable to occur on them. Infection can take place in a week-old wound, seldom in one a month old and hardly ever in one three months old. There is also considerable difference between the ease with which wounds are infected in different months. Between September and May infection takes place easily, but in June, July and August it happens with difficulty or not at all. Probably during the summer the trees are able to secrete so much gum that the growth of the mycelium through the wood is prevented. If it is not stopped by a barrier of gum it grows quickly through the branches. Wormald (1936) has shown that in 10½ months it could grow about a yard along a branch. It does not spread through the soil from diseased to healthy trees, or only very rarely, when an infected root grows against a healthy one. No infection can be derived from the silvered leaves, because the fungus is never present in them.

Distribution and host range. *Stereum purpureum* occurs throughout Europe and North America, also in South Africa, Australia and New Zealand. It attacks especially woody members of the rose family such as apricot, nectarine, peach, plum, sloe, cherry, hawthorn and apple, also the ornamental *Prunus* species such as *P. triloba*, *P. mahaleb* and Portugal Laurel, occasionally mountain ash and rose, but rarely pear. Less often it attacks trees and bushes of many other families, including redcurrant, blackcurrant, gooseberry, ash, beech, birch, broom, elm, *Escallonia exoniensis*, horse-chestnut, laburnum, lilac, *Lonicera nitida*, *Pernettya*, *Philadelphus*, plane, poplar, rhododendron, walnut and willow. It is common on dead wood, fallen logs and cut stumps everywhere.

Economic importance. Silver leaf is important mainly on plums, especially on Victoria, on which it has been very destructive in England. It is apt to be worse the year after a heavy crop in which branches have broken under the weight of fruit. On apples it is less destructive, though some varieties, like Newton Wonder, Lord Suffield, Ecklinville, and Early Victoria, seem particularly susceptible.

In Denmark the disease has become more prominent in late years with the increase in fruit growing and was especially noticeable after the severe frosts of 1940–2.

Control. Silvered branches should not be cut out as soon as they appear, for there is always the possibility of natural recovery, especially in apple trees. When they begin to die, however, they should be cut back until no brown stain remains in the wood. Removal of infected branches should be done in summer. In England the Silver Leaf Order of 1923 requires the occupier of any premises on which plum or apple trees are growing to destroy by fire on the premises all the dead wood of each plum or apple tree before the 15th July of every year. Where the dead wood is in the trunk and extends to the ground the whole tree, including the roots, must be burnt. An occupier may also be required at any time to destroy wood of any kind on which fructifications of the silver leaf fungus are visible.

Stereum purpureum fructifying on dead wood of any kind is able to infect fruit trees. Brooks was able to infect Victoria plums in England from fructifications collected on a poplar stump at Lyngby in Denmark. It is, however, impossible to eradicate a fungus of this kind that grows on dead wood all over the countryside. As it only gains access to fruit trees through fresh wounds it is more practicable to restrict wounds as far as possible to the summer months and to treat them to prevent infection. All wound surfaces should be cut smooth and covered with grafting wax or white lead paint made up as follows: to 2 lb. of white lead paste (as bought) add 2 tablespoonfuls of linseed oil and 2 teaspoonfuls of paste drier. Mix, add 2 tablespoonfuls of turpentine and mix well. Coating wounds with tar does not protect them against *S. purpureum*. All wounds made in grafting should be coated with grafting wax. A well-thought-out pruning programme should obviate the necessity of cutting out large branches, and suitable thinning of heavy crops will prevent branches from being broken down by excessive crops.

In localities where the disease is very prevalent susceptible varieties such as Victoria, Czar and Jefferson should not be grown. Yellow Pershore, Rivers' Early Prolific, Blaisdon Red and Damascene seem to be resistant to silver leaf, and Monarch, Purple Egg Plum and Damsons are fairly so.

The stock seems to have some influence on ability of plums to recover from silver leaf. Wormald (1939) records a higher percentage of recoveries in Victoria grafted on common plum than in those on myrobolan. Vigorous trees well supplied with potassium and phosphorus tend to recover more readily than ones with weak growth.

False Silver Leaf

The symptoms of false and true silver leaf are the same in the early stages, namely a silvery sheen on the leaves. Both commonly occur on regrafted trees of plum or apple. With false silver leaf the branches do not die and there is no brown stain in the wood as can be found with true silver leaf, at least in the thicker branches.

Cause. False silver leaf seems to be due to an excessive supply of nourishment which causes the upper skin of the leaf to become slightly detached from the underlying tissue. The air filling this space under the skin is responsible for the silvery sheen. False silver leaf is mostly seen on regrafted or heavily pruned trees where there is an old powerful root system with a disproportionately small crown.

Importance. Trees recover naturally from false silver leaf. It may possibly have a slight temporary weakening effect, but on the whole the condition is of little importance. It is less likely to develop if some of the old top branches are left for a year or two after a tree has been regrafted. They can then be cut out a few at a time.

Brown Rot, Wither-tip and Spur Blight

Brown rot of plums is caused by either of the closely related fungi *Sclerotinia fructigena* Aderh. & Ruhl. and *S. laxa* Aderh. & Ruhl. The former produces yellowish or buff-coloured pustules on the rotting fruit, the latter has grey ones. In addition *S. laxa* causes the very destructive diseases known as blossom wilt, spur blight and wither-tip fully described under cherry on p. 152. Control measures are the same on plum and cherry (Fig. 124).

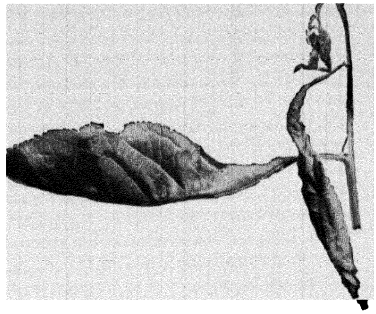


Fig. 124. Wither-tip of plum (*Photo Dept. Agric. Scotland*).

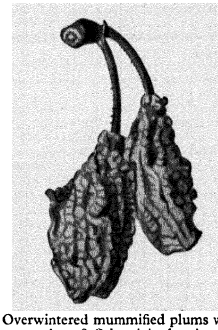


Fig. 125. Overwintered mummified plums with conidial pustules of *Sclerotinia fructigena*.

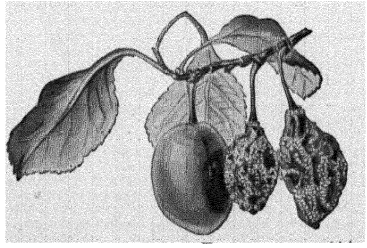


Fig. 126. Plums rotted by *Sclerotinia fructigena*, which spreads from fruit to fruit by contact.

Plum Rust (*Puccinia pruni-spinosae* Pers.)

The above name is that used for plum rust in England, but in most other countries mycologists prefer to split up the very large rust genus *Puccinia*, and the plum rust is then called *Tranzschelia pruni-spinosae* (Pers.) Diet. In its life history there is an alternation between plum and certain species of anemone. The uredo and teleuto or summer and winter spores are produced on plum leaves. Small, smooth, angular, yellow spots appear on the upper surface of the leaves and on the corresponding under-surface are the small pustules of spores, brown in the uredo stage, almost black in the teleuto stage. If the spots are very numerous the leaves may be shed prematurely.

The cluster cup or æcidial stage is on leaves of anemone, especially on cultivated St. Brigid anemone (*Anemone coronaria*) and *A. ranunculoides*, occasionally on wood anemone (*A. nemorosa*). Leaves of infected anemones stand more erect than those of normal plants and their lower surface is covered with æcidial cups. It occurred on *A. fulgens* in Cornwall in 1937–8 (Moore, W. C., 1943). The common rust on wood anemone, however, is not this species but the related *Puccinia fusca*, which has teleutospores only, and cannot infect plum.

Importance of the disease. Until recently, we have looked on the wild anemone as the main host for the cluster cup stage of this rust in Denmark, but of late years it has several times been found on St. Brigid anemone (*Anemone coronaria*), both on forced plants and on those growing out of doors. In England it is common on *A. coronaria* in the South-west and has been found sporadically elsewhere.

American workers, especially Dunegan (1938, 1939), have shown that æcidiospores from *A. coronaria* can infect apricot, peach, nectarine, cherry and plum. In Denmark the only stone fruit known to suffer from rust is the

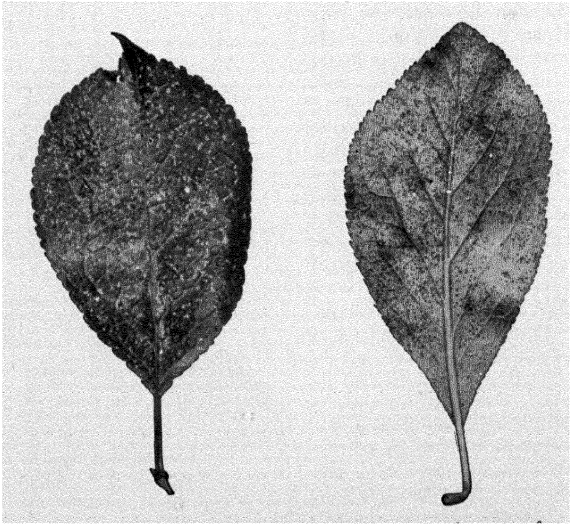


Fig. 127. Plum rust. Leaf on left shows the small yellow spots on the upper surface, that on right shows the brown spore pustules of the lower surface.

plum. In England also plum is the only tree usually attacked, but the rust has been seen on apricot, peach and nectarine (Moore, W. C., 1943). Even on plum it is prevalent only in the southern half of the country and it is very seldom seen in Scotland. In Denmark it was particularly virulent in the autumn of 1939 when instances of 90% leaf fall were reported as early as August. In Denmark the rust probably overwinters as mycelium in the root stocks of infected anemones as it is unlikely the uredospores can survive the winter there. In milder climates over wintered uredospores are probably an important source of infection to plum leaves. The teleutospores can, of course, infect only the anemone. After the hard winters in 1940–2 plum rust apparently disappeared from Denmark. In Norway another rust, *Thecopsora strobilina*, is common on plum leaves, in which it causes shot-hole disease. This fungus has its æcidial stage on the cones of the common spruce.

Control. Anemones should not be grown in the vicinity of plum orchards, and any infected anemone plants found should be destroyed. Wormald (1939) recommended spraying the plum foliage with 6:6:100 Bordeaux mixture when the fruit is about half grown, and again after it has been picked. We find plum leaves, especially young ones, are easily scorched by Bordeaux mixture. They stand a 2% lime sulphur spray better and this may also help to control red spider.

Plum Mildew (*Podosphaera oxyacanthae* (DC.) de Bary var. *tridactyla* (Wallr.) Salm.)

This mildew forms a white, flour-like coating on leaves and tips of shoots. The leaves may wither and fall prematurely. Perithecia of this mildew were noted on plum leaves in England in 1937 (Moore, W. C., 1943). In addition to plum it attacks also sweet and sour cherries, bullace, sloe, and bird cherry, but is seldom of much importance. When it does become prominent it can probably be controlled by a 2:100 lime sulphur spray or by a sulphur dust.

Pocket Plums (*Taphrina pruni* (Fuck.) Tul.)

Pocket plums occur especially on prunes, but may also occur on other plums and on myrobolan, bird cherry, bullace and sloe. According to Jørstad (1940), Czar, Rivers' Early Prolific and Yellow Egg plums are very susceptible to this disease in Norway while Victoria and Reine Claude are practically immune.

Symptoms. The first symptoms of the disease often appear soon after flowering but they are then often overlooked. When the fruit has attained to size of a small hazel nut the affected ones are noticeably paler than the others and have a longer, somewhat pointed shape. The diseased fruits grow much quicker than healthy ones and become flat, somewhat curved or crooked and up to 2–3 inches long by ½–1 inch broad (Fig. 128). The surface is slightly wrinkled and covered with a greyish bloom, formed of a tightly packed layer of asci. The flesh is greenish, tough, dry and tasteless, and in place of the stone there is a long, hollow space. As the season advances the pocket plums become brownish, and are often attacked by other fungi so that they rot and fall, but some may remain on the tree until the following year. Shoots and leaves may also be infected and become distorted.

Cause of the disease and its life history. Pocket plums are caused by the fungus *Taphrina pruni*, but it is possible that different

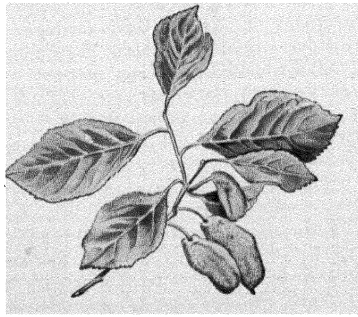


Fig. 128. Pocket plums on myrobolan.

physiological races of this organism exist. In 1936 we had an unusually large number of reports of pocket plums on myrobolans in gardens where the plums were unaffected. This suggests that the strain attacking myrobolan differs from that which attacks plum.

The fungus overwinters in infected branches and grows out thence into the young fruit. It is accordingly usually found that the same branches always bear the diseased fruit. The severity of infection varies greatly from year to year and seems to be affected by growth conditions. Drought checks it, while trees growing on enclosed sites on heavy, cold and sour soil are readily attacked.

The disease has been known since 1583, when it was mentioned by Caesalpin, but the life history of the fungus is still not completely known. Probably spores from the asci on the surface of pocket plums infect young buds during the summer and from them the fungus grows into the branches.

Importance. Pocket plums are not uncommon in Denmark, especially in certain seasons. Sometimes every plum on an affected tree has been attacked. In England the disease is seldom seen outside the western counties, where it was common in 1937 (Devon) and 1941 (Cornwall) (see Moore, W. C., 1943). It is known to occur to a greater or lesser extent in all countries where plums are grown.

Control. Spread of the disease can be checked by cutting out and burning all branches that bear pocket plums, before the bloom of asci has developed on them. Happily such drastic treatment is seldom necessary. In Norway good control has been obtained by spraying with a 10:90 lime sulphur mixture just before the buds burst and with 8:8:100 Bordeaux mixture or 1:40 lime sulphur just before blossoming. A final 1:40 lime sulphur spray may be applied when the fruits are the size of hazel nuts. Hand picking of pocket plums as soon as they are noticed is also advisable. Vigorous growth increases the resistance of plums to this disease.

Witches' Broom (*Taphrina insititiae* (Sadeb.) Johans.)

This fungus, which causes witches' broom on plum and bullace, is very closely related to that causing witches' broom of cherry, but the brooms on plum form thicker clusters. Control is the same as for the cherry fungus (see p. 154). It is seldom seen in England, but has been reported a few times from the western counties, as far North as Westmorland on plum and damson.

Scabby Fruit

Among the minor diseases of plum is scab, caused by the fungus *Fusicladium carpophilum* (Thüm.) Oudem. The scabs appear as circular brownish spots on the half-grown fruit and if they are numerous they may cause cracking of the skin. The disease occurs sporadically in England, and was observed in Denmark in 1929. Quite often, however, corky patches arise on the skin of plums as a result of mechanical injury, as by rubbing against a twig, damage by the weather or by sprays, or as a response to insects damaging the young fruit.

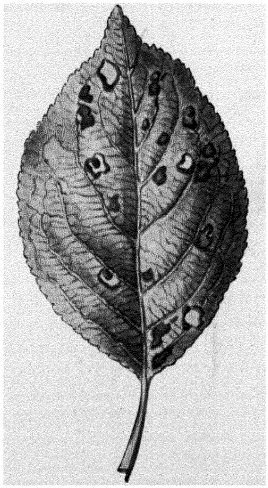


Fig. 129. Shot-hole of plum caused by spraying with lead arsenate.

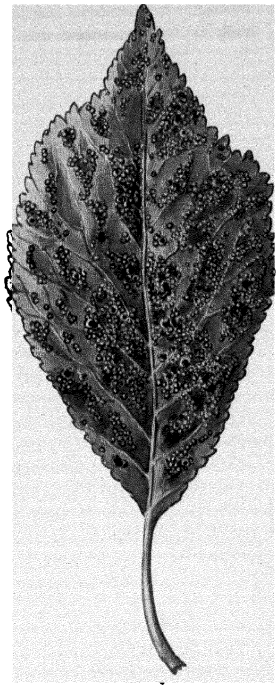


Fig. 130. Shot-hole of plum, small spots whose centres are falling out.

Shot-hole

The type of shot-hole disease caused by the bacterial canker organism is described on page 200. This form of the disease is usually recognizable by the translucent, watersoaked margin to the young spots. Other forms of shot-hole start as a dry brown leaf spot, surrounded by a reddish-brown border. The spot seldom grows to more than ¼ inch across, usually it is not as large, and after it has attained its full size the centre becomes loose and falls out, though this may not happen under very dry conditions. This kind of shot-hole may be due to spray damage, to physiological disturbances, or to fungus infection.

We have seen instances in Denmark where trees, sprayed with lead arsenate, Bordeaux mixture, or lime sulphur, developed shot-hole in their leaves about six weeks later, while neighbouring unsprayed trees showed little or none of the disease. As long ago as 1899 Duggar showed that many different chemicals used as sprays could induce this reaction in leaves and that too heavy a spray with the standard fungicides could also induce it. He also described instances where shot-hole arose as a result of physiological disturbances. On a tree blown down in a heavy summer rainstorm, followed by a warm muggy afternoon, numerous holes appeared in the leaves two days later. There also seems to be a tendency for small islands between veins to fall out of the leaves when late frosts occur after the buds have burst, as in 1935. The fungus usually responsible for shot-hole in leaves of stone fruit trees is *Clasterosporium carpophilum* (Lev.) Aderh., also sometimes referred to as *C. amygdalacearum* Sacc., *Stigmina briosiana* Farn., *Coryneum beijerinckii* Oud. and *C. laurocerasi* Prill. & Del. The pycnidial fungus *Phyllosticta beijerinckii* Vuill. and the ascomycete *Ascospora beijerinckii* Vuill. have been claimed as parts of the same life history, but this requires confirmation as the biology of the fungus is not well known.

Clasterosporium carpophilum may attack any stone fruit. In Norway it occurs on fruit and leaves of sweet and sour cherries, less often on plums (Jørstad, 1940). In Germany and Switzerland, where it has been most investigated and is fairly common, it is most prevalent on cherries. In England it is best known on almond, nectarine and peach leaves and as a cause of scab on peach fruit, but occurs occasionally on cherries. If shot-holes are very numerous they may cause premature leaf fall. The same fungus causes sunken brown or black spots or scabs on fruit and may cause it to be deformed. On young twigs, especially of peach, it causes dry, brown scabs with a red rim, often associated with small drops of gum. It passes the winter in these twig lesions and in bud scales. Under favourable conditions the short conidiosphore of the fungus develop

on the leaf spots, each crowned by a minute brown, septate, spindle-shaped spore. Spores are more often found on the fruit and twig lesions.

Control. In Norway a Bordeaux spray before and after flowering, at 8:8:100 and 4:4:100 strengths respectively, is recommended to control *Clasterosporium carpophilum*. In Switzerland a $1\frac{1}{4}\%$ lime sulphur spray before flowering and twice more with 10 days' interval soon after the fruit has set is recommended against shot-hole on cherries and prunes. As, however, the reaction to spray damage is very similar to that following *Clasterosporium* attack, spraying should only be undertaken when the fungus has been particularly destructive the previous year. When shot-hole has been due to unfavourable cultural conditions, possibly impeded root action, spraying will, of course, be of no avail.

Trees whose growth has been checked by lack of nitrogen are said to be particularly susceptible to *Clasterosporium carpophilum*. Hence, especially in the first year after planting, care should be taken to see that nitrogen is not deficient. In a Zealand plantation shot-hole disease was considerably reduced by ploughing in a cover crop of medick in autumn. Care should also be taken to see that the roots do not suffer either from drought or from waterlogging.

Branch Dieback

By "dieback" is understood the condition when individual branches wither and die. This condition occurs in many trees, but is particularly prevalent in stone fruit, where it is associated with a number of fungi and with unsuitable conditions of growth.

One of the fungi associated with dead branches is *Valsa leucostoma* Pers., which is usually found in its conidial state *Cytospora leucostoma* (Pers.) Sacc. This forms a large number of tiny pimples on the bark within which the fructifications are embedded. There is considerable doubt if this fungus is really a primary parasite. It is usually believed that it attacks tissues damaged by frost, sun scorch, drought or bacterial canker, in which case it may perhaps increase the amount of damage. Gummosis generally takes place below the lesions.

Diseased branches should be cut out and destroyed, though not in autumn for fear of admitting bacterial canker, and the wounds should be sealed with grafting wax or paint. Direct control measures against the fungus are pointless; one should endeavour to find out what primary damage allowed it access to the branch.

Diaporthe perniciosa Marchal, another pyrenomycete with, in this case, a *Phomopsis* as its conidial state, is also often a follower of bacterial canker, but can sometimes attack branches in the absence of bacteria, especially if the trees are lacking in vigour, as when grown on unsuitable soil. It can be found on plum, apricot, peach, myrobolan, pear and apple, especially on young trees. Infection may take place long before definite disease symptoms are seen, sometimes years before. As soon as twigs begin to wilt and die, however, the whole infected branch soon dies and the fungus fructifies in cracks in the dead bark.

Young trees that have been overmanured and pruned too hard are susceptible to *Diaporthe* attack, otherwise it occurs mainly on trees damaged by bad growing conditions, by frost or by bacterial attack. The lines of control indicated are similar to those for *Valsa leucostoma*.

The discomycete *Dermatea prunastri* (Pers. ex Fr.) Fr., which forms clusters of tiny blackish brown gelatinous button-like fructifications on dead branches, is also sometimes associated with dieback, especially in greengage. Its conidial state, known as *Sphaeronema spurium* (Fr.) Sacc., projects from the bark like a stiff black hair up to $\frac{1}{8}$ inch long (Dowson, 1913).

Another fungus often seen on the underside of large dead branches on old neglected plum trees is the bracket fungus *Fomes pomaceus* (Pers.) Lloyd. This is hard, woody and crust-like, or hoof-shaped, with a cinnamon-coloured lower surface covered with minute pores. It causes a white rot of the heart wood,

but is not a very active parasite. It also attacks cherry and is occasionally found on very old sloe bushes (*Prunus spinosa*). Branches bearing the fructifications should be cut out and burnt, care being taken to cut back until only healthy wood is left.

Bacterial Canker (*Pseudomonas mors-prunorum* Worm.)

It has long been known that large plum branches or the whole tree may suddenly die. The cause of this sudden death long remained a mystery and the condition was referred to as " apoplexy " when the whole tree died at once or " dieback " when a few branches died at a time. Observant gardeners often noticed, however, that death occurred of trees or branches on which the leaves had turned yellow and fallen prematurely the previous year.

Thanks mainly to the researches of Wormald at East Malling we now know that most of this trouble is due to the bacteria *Pseudomonas mors-prunorum* and *P. prunicola*. Other causes are sometimes involved, however (see gummosis, p. 159; *Verticillium* wilt, p. 156; and dieback, p. 198).

The death of so many stone fruit trees in Denmark after the hard winters 1940–2 may largely be attributed to this disease. Probably there was interaction between frost and parasites, especially the bacterial canker organisms and *Stereum purpureum* Pers., the cause of silver leaf. Parasites easily gain access to parts affected by frost and spread from them so that in the course of a year or two they are able to kill the entire tree. There have been several instances of trees which appeared only slightly damaged after a hard winter dying some time later. In England bacterial canker seems to have been unusually prevalent in 1936–8 and 1945.

Symptoms. Bacterial canker is most serious on young trees, but older ones may also be attacked.

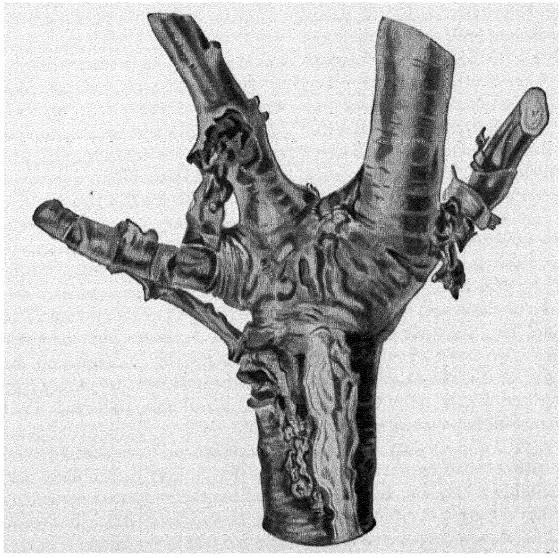


Fig. 131. Plum trunk showing symptoms of bacterial canker. A strip of bark has been removed to show the sharp dividing line between sound and diseased wood.

When the trunk is infected the tree develops sickly-looking leaves in spring or autumn. The first sign of disease is a slight yellowing of the leaves, visible very soon after the leaves appear. In a few weeks the leaves become distinctly yellow, especially in the variety Czar. Usually growth of the young shoots is noticeably retarded, especially in those at the tips of the branches. The leaves tend to be narrow and to roll up; this is shown more distinctly in Victoria than in Czar. The trees now appear to have "thin" foliage. Usually all the branches of such trees die, but the lower part of the trunk remains alive and healthy and so do the roots. Some trees die back when the fruit has attained its full size, on others the leaves stay green till autumn, but if the canker has girdled the trunk their buds do not break in the following spring.

Sometimes only one or a few branches are attacked but not the trunk and sometimes only one side of a tree is affected. In such cases the leaves on the rest of the tree remain perfectly normal. The cankers are very difficult to see on trunk or branches, until some growth has taken place in the healthy bark. Then the diseased patches can be detected because they remain slightly sunken. If then the bark is cut, that of the sunken areas is seen to be brown with a sharp division between healthy and diseased tissue (Fig. 131). Callus may form on the edge of the cankers. There is seldom more than one canker on a trunk. Usually it forms an oblong patch from one to three feet long. The long ones often twist spirally round the trunk but seldom reach down to the base or the roots.

Cankers a yard long may not completely girdle the trunk; only when they meet round it does the whole tree die. The lower part of the trunk below the canker often produces numerous suckers with large green leaves, in striking contrast to the sickly leaves above. Gumming is associated with bacterial canker in cherries, but seldom in plum, except on the variety Bradley's King.

Little, round, dark spots appear on the leaves and eventually drop out, giving a shot-hole effect (Fig. 132). Shot-hole in plum leaves may be due to several causes, including spray damage (p. 196), violent fluctuations in weather, and the attack of various fungi (p. 197). When it is due to the bacterial canker organisms it starts as tiny, translucent, watersoaked spots that dry out and turn brown in the middle but retain a narrow, translucent margin, at most $\frac{1}{16}$ inch wide. In time the dry brown centre becomes loose and falls out leaving the shot-hole which is seldom more than $\frac{1}{4}$ inch across, usually less. Bacteria swarm in the watersoaked tissue, and in severe attacks all the leaves of the tree become perforated in this way. Buds may also be attacked, especially in cherries. A sunken area develops round them and they fail to break in spring.

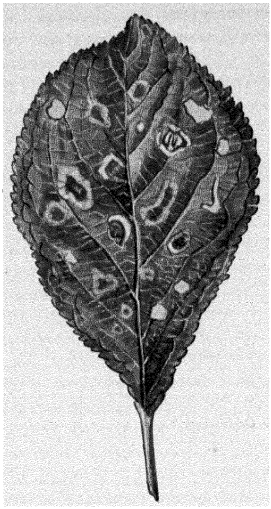


Fig. 132. Plum leaf with shot-holes caused by *Pseudomonas mors-prunorum*.

On the young green shoots bacterial canker takes the form of dark oblong patches. Infected blotches may also occur on the fruit, both in plum and cherry, but seem to be of little practical importance.

Causes and development. In *East Malling Annual Report* for 1924 Wormald drew

attention to dieback of plums and cherries, a well-known phenomenon which he tentatively attributed to bacterial infection. Subsequent investigations confirmed this hypothesis, and the cause of the disease was shown to be the bacterium *Pseudomonas mors-prunorum* Worm. Later he demonstrated that a similar but distinct organism *Pseudomonas prunicola* Worm. could cause a similar disease. Both bacteria isolated from plum are able to attack cherry and vice versa, but the strains are more virulent on the hosts from which they were isolated. Bacteria isolated from leaves, buds, branches or trunks can cause the appropriate symptoms on each of the other organs so that all the diverse symptoms described above are phases of a single disease bacterial canker.

Most of Wormald's work was done with *Pseudomonas mors-prunorum* and he isolated this organism from leaves of myrobolan, cherry and plum sent him from North Zealand, Denmark. With the culture from myrobolan he inoculated branches of Victoria plum and produced typical canker. As the two species of *Pseudomonas* can only be distinguished by prolonged cultural work in the laboratory it is not certain whether *P. prunicola* occurs in Denmark as well. In other countries still other bacteria have been described causing similar diseases of stone fruit, e.g. *Pseudomonas pruni* E. F. Sm. and *Phytomonas cerasi* Griff. in U.S.A.

By numerous inoculation experiments Wormald showed that infection of trunk and branches was easiest from October to December and hardly ever succeeded during the summer, from March or April to September. During the winter half of the year the bacteria live in the bark of branches and trunks, while they pass the summer in lesions on young shoots and in the shot-holes on the leaves. By the time the cankers in the bark have become visible the bacteria have usually died out there and the dead tissue has been invaded by various fungi. Hence in the past these have often been claimed to be the cause of the disease, though it cannot be reproduced when they are introduced into healthy branches in inoculation experiments (see dieback, p. 198). It is not clear how the bacteria pass from the bark cankers to the young leaves in the spring. There seem to be two possibilities, either that they are transported by insects, or that they pass the winter in or on the buds and infect the leaves as the buds break.

Not much is known about the influence of environmental conditions on susceptibility of plums to bacterial canker. Wormald concluded that manurial treatment had little or no effect, but that liming increased susceptibility. In English sand culture experiments over a number of years the greatest resistance was found with a small supply of phosphoric acid. Very heavy phosphoric acid manuring led to highest susceptibility as did also full manuring which included ample potash. With half this potash supply or with no potash, the lesions were much smaller. It seems, therefore, that heavy phosphate or potash manuring is to be avoided. The increased susceptibility with high phosphoric acid supply may be due to its effect in reducing solubility of iron salts. Soil acidity and the supply of iron in a readily soluble form check the disease but pH4, which is most favourable in the sand cultures, is difficult to obtain in the orchard and might be injurious in other ways. One might suppose that supplying iron sulphate to the soil or its use in sprays would have a beneficial effect, but we have no experience of this.

Observations in Danish plum orchards agree with the results of the English sand culture experiments. In a plantation made on heavy soil in 1934, 45 out of 144 trees had died by 1937. The attack was worst where the marl reached the surface. In another plantation the rows with most diseased trees were on soil of pH 7.0, there was less disease where the pH ranged from 4.8 to 7.2, but as different varieties were involved no very reliable inference can be drawn. In a third plantation the attack was worst on heavily manured soil near the chicken run. It is not certain whether bacterial canker is the same disease as was investigated in Germany by Aderhold and Ruhland (1906–7), who ascribed it to a bacterium they called *Bacillus spongiosus* (*Pseudomonas spongiosus* (Aderh. & Ruhl.) Braun.). The symptoms on trunk and branches

described by the German workers agree with those of bacterial canker, but they did not mention the shot-hole phase. Possibly it was overlooked or was not recognized as forming part of the same disease cycle.

We first observed this disease on plum trees in Denmark in August 1930. From information that has accumulated since then we believe it to be the main cause of death of stone fruit in that country, but there are considerable differences in susceptibility between the various species and varieties grown. Of two varieties growing side by side one may be severely attacked, the other not at all. In 1937 we visited eleven Zealand plum orchards and inspected them for bacterial canker. In all 2,347 trees were examined, and a general impression of the relative susceptibility of varieties was secured. On this basis, supplemented by reports received from various sources, we are able to give the following summary of varietal susceptibility in Denmark. We have also included some English data. It must be remembered that varieties listed here as resistant may prove susceptible under some circumstances or in other countries.

VARIETY OF PLUM	SUSCEPTIBILITY TO BACTERIAL CANKER
Apricot plum	Individual cases seen at three places.
Bastard Victoria	Susceptible in England.
Blue Belgian	Diseased branches seen at one place.
Belle de Louvain	No diseased trees at one place, many at another.
Bradley's King	Very susceptible in England.
Czar	Very susceptible in Denmark and in England.
Denniston's Gage	Very resistant in England.
Early cherry	None diseased out of 29 at one place, 3 out of 12 at another.
Early Laxton	Susceptible.
Early Orleans	Susceptible.
Egg Plum (yellow)	1 out of 15 attacked at one place.
Egg Plum (red)	5 diseased or dead out of 14 at one place.
Emma Leppermann	Seems to be resistant.
Evesham Wonder	Susceptible in England.
Farleigh Cluster Damson	Susceptible in England.
First	Susceptible.
Giant	Susceptible, death of 25% of a plantation recorded in England.
Golden Drop	Susceptible in England.
Hackmanns	4 diseased out of 31 at one place.
Jefferson	Apparently susceptible in Denmark but seems able to recover from the attack.
Kirkes	6 diseased out of 187 at one place, none out of 36 at another.
Mirabelle précoce	A single attack seen.
Monarch	Seems to be fairly resistant.
Pershore Egg	Susceptible in England.
President	Susceptible in England.
Prince of Wales	Severely attacked in two localities, much branch dieback.
Prince Engelbert (Kathrine)	50 trees seen, all healthy.
Prune Pêche	9 dead and 7 diseased out of 29.
Reine Claude Althans	Severely attacked at one place, not at another.
Reine Claude d'Oullins	Attacks seen at several places.

Plum	Bacterial Canker
Reine Claude noire	A single diseased tree seen, variety healthy in two other localities.
Reine Claude, transparent early	Severely attacked at one place, not at another.
Rivers Early Prolific	Single diseased trees noted at several places.
Prunes, Experimental garden..	Attacked at several centres, not at others.
Prune, Italian	Seems not to be susceptible.
Prune, Early English.....	Seems not to be susceptible.
Prune, Hungarian	Seems very susceptible, but may sometimes recover from an attack.
Prune, Wangenheimer	10 dead out of 14 at one centre, 2 diseased out of 12 at a second, 25 all healthy at a third.
Tragedy	Seems very susceptible but can sometimes recover.
Utility	Seems quite resistant in England.
Warwickshire Drooper	Susceptible in England.
Victoria	Very susceptible in Denmark and in England.
Washington	4 diseased out of 16 at one centre, 2 out of 30 at another.

As regards cherry varieties we have only been able to make a single estimate of relative susceptibility at a plantation in North Zealand. The varieties seen are arranged below in order of susceptibility, starting with the least affected.

SYMPTOMS ON TRUNK OR BRANCHES	FOLIAGE SYMPTOMS
Black Spanish	Heart Cherries
Heart Cherries	Yellow Spanish
Hedelfinger	Black Spanish
Napoleon	Krogerup
Krogerup	Napoleon
Yellow Spanish	Hedelfinger

Of the 35 Black Spanish trees seen only one had lesions on trunk or branches. This plantation contained 130 trees of the above sweet cherry varieties and in addition there were 12 sour cherry trees interplanted all of which were free from branch or trunk lesions and showed only a trace of shot-hole symptoms.

Hans Larsen (1943) mentioned Napoleon and Black Tatarisk as especially susceptible.

Wormald (1937a) in 1936 examined 350 cherry trees of 9 varieties in a Kentish orchard. His estimate of relative susceptibility is given below, with the least susceptible at the top.

SYMPTOMS ON TRUNK OR BRANCHES	FOLIAGE SYMPTOMS
Frogmore	Frogmore
Governor Wood	Governor Wood
Roundel Heart	Roundel Heart
Early Rivers	Amber Bigarreau
Napoleon	Emperor Francis
Waterloo	Napoleon
Amber Bigarreau	Waterloo
Bradbourne Black	Bradmore Black
Emperor Francis	Early Rivers

Other observations showed Black Tartarian, Cluster Black Heart, Bigarreau de Schrecken, Black Eagle and Bradbourne Black to be the most susceptible varieties, but Waterloo, Turk, Amber Heart and Early Rivers were also severely attacked in several localities.

There is also considerable mortality among myrobolans in Denmark, especially in trees between 5 and 12 years old, and all evidence points to bacterial canker being the cause of death. Wormald found the disease also on peach, almond and *Prunus pissardii*, so that ornamental *Prunus* species must be regarded as a possible source of infection for plum and cherry orchards.

Control. Trees or branches affected by bacterial canker should be removed and burned. We cannot, however, recommend destroying trees showing only the shot-hole

phase of the disease for they may still bear fairly well.

Of late years attacks on the bark of fruit trees, including plums, by a bark beetle *Anisandrus dispar* have been noted rather frequently. It is possible the insect may assist the spread of bacterial canker, partly by the wounds it causes allowing entrance to the bacteria and partly by actually transporting the bacteria on its body and introducing them into the wounds. Unfortunately control measures against it have scarcely been worked out, apart from cutting out and burning the infested branches.

As the bark of the trees is most prone to bacterial infection in October and November special care should be taken to avoid injuring it during these months. Pruning should be done during the summer or later in the winter; the best time is between early April and the end of August. This recommendation agrees with the experience of many growers that stone fruit trees stand summer pruning best.

Experiments in England have shown that copper sulphate and tar-oil sprays kill the bacteria. Unfortunately the leaves of stone fruit trees are easily damaged by Bordeaux mixture, especially after petal fall, but before blossoming cherries stand it well. Tar-oil sprays can only be used late in the autumn when much of the bark infection has already taken place. Danish experiments with a Bordeaux spray at various times during autumn and just after leaf fall gave no control of the disease. On cherries in England, however, some control has been obtained by spraying with 10: 10: 100 Bordeaux mixture in autumn and with 6: 6: 100 or 3: 3: 100 to check the shot-hole phase in spring and summer.

As most of the valuable commercial varieties of plum and cherry are susceptible to bacterial canker one cannot avoid it by planting only resistant varieties. It may, however, be possible to grow those susceptible varieties by working them on resistant stocks. In a large Danish nursery bacterial canker was, on the whole, worse in trees grafted on myrobolans than in those on St. Julien. In England selection of suitable stocks has been undertaken, for apparently myrobolans are not all equally susceptible. A very susceptible variety like Victoria will suffer much less if it is grafted high, so that a resistant stock forms the trunk, than if it is grafted low, so that the Victoria forms the trunk. It will suffer still less if the framework of main branches is formed by the stock as well.

Myrobolans and ornamental *Prunus* species should not be used to form hedges or windbreaks near plum or cherry orchards. Source of the stock may also have an influence on its susceptibility (Gram, N., 1938). Myrobolans of Danish origin were found to be more hardy and resistant than imported ones. Hence where a grower has good sound myrobolans he would do well to take cuttings from them in August or September and use them for stocks instead of raising seedlings.

Virus Diseases

Virus diseases of stone fruit are very destructive in North America, especially to peaches and cherries. They seem to be of minor importance in Europe and are almost unknown in the British Isles. A disease called **plum pox**, well known in Bulgaria, probably occurs as far north as Bohemia and Holland and a single tree apparently affected by it was discovered in Hampshire in 1934 (Moore, W. C., 1943). Leaves of affected plums bear light-green or yellowish blotches and rings. The fruit becomes marbled and pockmarked with patches of dead tissue and there are drops of gum in the flesh. Growth of the branches is checked. The disease has been shown to be transmitted by grafting (Atanasoff, 1933). It attacks several varieties of plum, and a similar disease occurs on sour cherries, bird cherries and peaches (" buckskin," see Rawlins and Thomas, 1941).

Mosaic disease with distinctly mottled leaves occurs on plums, apricot and peach in Bohemia. A similar trouble has been observed several times on plums in Worcestershire (Moore, W. C., 1948).

In Holland three probable virus diseases of plum have been described:

(1) Narrow mis-shapen leaves associated with poor bearing of fruit; healthy shoots

grafted to narrow-leaved trees also become narrow-leaved.

(2) Variegated leaves, especially in plums, sometimes in cherries.

(3) Leaves with rounded marginal teeth, and buckled uneven surfaces with translucent or perforated spots. Eventually the leaves become narrow and pointed. This condition occurs also in cherries and peaches.

Cracked Fruit

If heavy rain occurs when plums and cherries are nearly ripe the fruit often splits, especially if the rain follows a period of drought. Apples, pears and gooseberries may also split, but less frequently. It is generally supposed that the skin splits because it cannot keep pace with the expansion of the flesh of the fruit caused by the sudden increase in water absorption by the roots. In West Virginia, however, Verrer has shown that cracking of the skin is not correlated with water content of the soil. Nor is it rain that is directly responsible for the cracks, but a period of excessive atmospheric humidity when evaporation of water from the surface of leaves and fruit can no longer take place.

Cracks appear first and are most pronounced on fruit that is scabby, wrinkled or highly coloured by sun, wind or spray. On the wrinkled and highly coloured side of a fruit the osmotic pressure in the cells may be appreciably higher than on the other, e.g. a pressure of 22 atmospheres as compared with 17. Fruit growing quickly is more likely to crack than that growing slowly. Fruit of different varieties may also differ somewhat in their tendency to crack. Not much can be done to prevent cracked fruit apart from avoiding scabbiness and care in spraying. Watering in dry weather may be helpful as normal skin does not crack as easily as the hard skin produced during a drought.

Gum Drops

Often drops of gum are found on the surface of half-ripe or ripe plums or in lumps in the flesh of the fruit. Sometimes they are exuded as a result of insect damage, but often there is no sign of such a stimulus and the gum production is apparently due to some growth condition not yet understood. Claims that it is due to boron deficiency have not been substantiated.

Summary of Plum Diseases

		page
I. Roots and Crown		
Tumours	Crown Gall	81
II. Trunk, Branches and Twigs		
A. Twigs of this season's growth:		
(1) Dark oblong patches	Bacterial Canker	199
(2) Small dry brown spots with red margins	Shot-hole	197
(3) Whole twig withering after flowering	Blossom Wilt and Die-back	193
B. Older branches:		
(1) Slightly sunken patches on the bark, bark brown in section	Bacterial Canker	199
(2) Gumming	Gummosis	159
(3) Brown stain in the wood, sometimes leathery fungus growths, greyish above, mauve beneath, on dead branches	Silver Leaf	191
C. Dead twigs and branches	Dieback	198
D. Dense bunches of twigs here and there	Witches' Broom	196

		page
III. Leaves		
A. Malformations:		
(1) Rolled leaves, not associated with aphides	Bacterial Canker	199
(2) Abnormally small or distorted leaves	Virus	204
(3) Arched, thickened leaves	Witches' Broom	196
B. Widespread discoloration:		
(1) Pale leaves with irregular brown patches or scorched margins	Potassium Deficiency	42
(2) Light-green leaves	Chlorosis	36
(3) Silver sheen to the leaves, associated with brown stain in the wood	Silver Leaf	191
(4) Silver sheen to the leaves but normal wood	False Silver Leaf	193
(5) Yellowing and premature leaf fall	Bacterial Canker	199
C. Discoloration in patches:		
(1) Mottled leaves	Mosaic	204
(2) Light green or yellowish rings or spots	Plum Pox	204
(3) Small light spots bearing powdery brown (later black) fungus pustules	Plum Rust	194
(4) Small round brown spots that eventually fall out:		
(a) Margin of the spot translucent	Bacterial Canker	199
(b) Margin of the spot not translucent	Shot-hole	197
D. With a superficial coating of mould:		
(1) White coating	Mildew	195
(2) Black coating	Sooty Mould	126
IV. Flowers		
(1) Flowers and often young leaves wilted	Blossom Wilt	152
(2) Dead flowers (only in North America)	Fire Blight	186
V. Fruits		
A. Small patchy discolorations:		
(1) Black spotted skin	Sooty Blotch	126
(2) Round dark patches on skin	Scab	196
(3) Sunken blackish-brown patches	Shot-hole	197
(4) Skin pock-marked and marbled. Dead rubbery patches in the flesh	Plum Pox	204
B. Cracking	Cracked Fruit	205
C. Brown rot, later drying to shrivelled mummy fruit:		
(1) Conidial pustules yellowish	*Sclerotinia fructigena*	121
(2) Conidial pustules grey	*Sclerotinia laxa*	152
D. Black sticky fungus coating	Sooty Mould	126
E. Long tough pale fruit	Pocket Plums	195
F. Small gum drops on the surface	Gumming	205
G. Scurfy, corky patches on the skin	Scabby Fruit	196
H. Brown patches in the flesh (in cold storage at 32° F.)	Cold Breakdown	141

QUINCE

Leaf Blight (*Entomopeziza mespili* (Sor.) v. Höhn = *Fabraea maculata* Atk. = *Entomosporium maculatum* Lév.)

Symptoms. On the upper surface of young quince leaves there commonly appear spots which are at first a faint carmine by reflected light and distinctly red by transmitted light. They soon become visible also on the lower surface of the leaf (Fig. 133). Later they turn

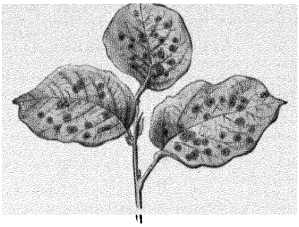


Fig. 133. Quince leaf blight.

brown and finally almost black. Conidial pustules can be detected on them under a hand lens, appearing at first as minute dots which enlarge and may become confluent. Severely infected leaves may be shed. Spots may also appear on the tips of the shoots and occasionally on the fruit.

Cause. The conidial stage of the fungus occurs in the pustules on the spots in summer while the perfect stage develops on the fallen leaves in winter. There has been some disagreement about the systematic position of the perfect stage. Sorauer referred it to *Stigmatea*, Atkinson (1909) to *Fabraea*, while Klebahn (1918) placed it in a new genus, *Entomopeziza*.

Infection occurs in spring from ascospores produced on the overwintered leaves and probably from conidia produced on the spots on the twigs. The latter is probably the more important source of infection as the perfect stage has yet to be found in Britain, though the disease is common there.

Importance. The disease is found especially on quince propagated in nurseries as root stocks for pears, but Rostrup (1902) also observed it occasionally on wild pear. Klebahn (1918) considered physiologic races exist, each restricted to a few hosts. Besides quince, leaf blight is also known on medlar, apple, hawthorn, cotoneaster and amelanchier. Wormald and Harris (1937) found Malling Quince C very susceptible, and Quince A less so.

As the fungus sometimes causes premature defoliation it may have a weakening effect and prevent nursery stocks from attaining graftable size as quickly as is desired. Williams pear may also be attacked, but it is usually only on the quince stock that the disease is of any importance.

Control. The fallen leaves should be collected and buried. Frequent Bordeaux sprays should be given during the summer, especially in wet years and when the disease is prevalent.

Leaf Blotch (*Sclerotinia cydoniae* Schellenb.)

Large dark-brown or blackish patches appear on the leaves especially along the veins soon after they have unfolded. From these the fungus may spread into the shoots and overwinter there. The leaf lesions bear a whitish sweet-smelling mould consisting of chains of conidia. These may infect the flowers which become permeated by mycelium and mummified. Eventually they fall to the ground and from them the sclerotinia fructifications may be produced in the following spring.

Control is obtainable by cutting out the infected leaves in June and July and removing the diseased shoots during the winter, when they are recognizable by their expanded bud scales. The fungus is not known to infect any other host.

Summary of Quince Diseases

I. Branches

		page
Small cankers	*Phacidiella*	118

II. Leaves

A. Small reddish-brown spots	Leaf Blight	207
B. Yellow or orange spots with cylindrical cluster cups	Hawthorn Rust	440
C. Blackish-brown blotches	Leaf Blotch	207

III. Flowers

A. Dead flowers with slimy exudate	Fire Blight	186
B. Dead flowers, becoming mummified	Leaf Blotch	207

IV. Fruit

A. Dark-brown patches of rot bearing yellowish conidial pustules	*Sclerotinia fructigena*	121
B. Black rotten spots with small, spherical, fructifications, exuding cream-coloured spore masses	*Phacidiella*	118
C. Young fruit mummified	Leaf Blotch	207
D. Yellow spots with cluster cups	Hawthorn Rust	440

RASPBERRY

Cane Spot (*Elsinoë veneta* (Burkh.) Jenk.)

Symptoms. Cane spot starts as tiny purple spots on the surface of the young canes at the end of May or beginning of June. These enlarge and turn whitish and slightly sunken at the centre, with a narrow purple border. The individual spots do not exceed ¼ inch in length, but they may be so numerous as to fuse and form large irregular lesions (see Fig. 134). The mycelium slowly spreads into the underlying tissues and destroys first the bark, then the bast and finally reaches the wood. The whitish dead tissue in the centre of the spot then cracks and falls away during the winter so that the cane becomes pitted with tiny brown cup-shaped cankers that may penetrate even to the pith.

The leaves are attacked with very small spots, white at the centre, dark round the edge. Similar spots occur on the leaf stalks and even on the young fruit, which then becomes irregular and one-sided.

Cause. The disease is caused by the fungus *Elsinoë veneta* which has an ascospore stage, seldom seen, and a conidial stage, formerly known as *Gloeosporium venetum*, which consists of minute black dots just visible to the naked eye. On the surface of these black dots microscopic, one-celled, conidia are produced



Fig. 134. Raspberry cane spot, grey spots with purple edges (after C. A. Jørgensen).

during the summer. The ascospore stage appears as tiny brown warts, about $\frac{1}{10}$ mm. across, on the spots in autumn and winter. The fungus passes the winter in the spots on the canes and fallen leaves and spores from these infect the new shoots and spurs in the spring.

Importance. The small deep spots on the stems interfere with transport of sap to the shoots and may lead to withering of the berries in dry summers. In severe attacks the fruiting spurs, the leaves and even the fruit may be directly attacked. The last-named phase of the disease may cause a serious reduction in the crop of marketable berries. Affected fruit may be quite normal on one side, but on the other a number of its constituent drupels remain hard and green and bear the minute *Gloeosporium* pustules. Cane spot is regarded as a serious disease in Australia and North America. It is prevalent in Britain and is known also in Holland, Denmark and Norway. In Denmark it was found by C. A. Jørgensen in 1926 on raspberries but the conidial stage had been found on brambles at Horsens by Lind in 1901. It can spread from brambles and wild raspberries to cultivated ones, so the 1926 outbreak need not have originated from imported diseased canes. C. A. Jørgensen found it at many places and it is still to be found in Denmark but seldom to such an extent as to cause serious loss.

Control. Raspberry varieties differ greatly in their susceptibility to cane spot; Superlative and Fajstrup are seldom much attacked.

Badly spotted canes should be cut out and burned and in a small plantation it may be worth while to cut down and burn all the canes one autumn, even though it means losing the following year's crop. Dutton (1918), in America, recommended spraying with lime sulphur 5:95 when the buds were $\frac{5}{8}$ inch long and with Bordeaux mixture 4:4:100 about one week before flowering. Anderson (1920), also in America, reported good control by a single spray with 24:24:100 Bordeaux mixture and oil applied just as the buds began to show green at the tips. Lime sulphur 8:100 was also effective. The oil he used was one not available in Europe, but probably $\frac{1}{2}$ or 1% spraying oil could be used with the Bordeaux mixture instead. At East Malling Harris (1931) recommended spraying with 12:12:100 Bordeaux mixture or 7:100 lime sulphur when the buds had begun to swell, but were not more than $\frac{1}{2}$ inch long, and again with 6:6:100 Bordeaux mixture or $2\frac{1}{2}$:100 lime sulphur when the first flower buds were showing white at the tips. Bordeaux mixture is preferable to lime sulphur in this instance. If this programme cannot be carried out in full it would be better to do one spraying every year than to do both one year and none the next.

Spur Blight (*Didymella applanata* (Niessl) Sacc.)

Symptoms. Symptoms of spur blight usually do not appear on the young canes before August. Then dark reddish-brown patches with a purple sheen, 2 or 3 inches long, appear on the canes round the leaf bases. They are most abundant on the lower half of the cane. During the autumn months the patches increase in size and two or more may coalesce (Fig. 135). Leaves of infected canes usually bear large irregular, dark-brown blotches from June onwards and purplish streaks occur also on the leaf stalks. The diseased leaves may fall early and leave the lower part of the canes bare before the upper.

As the canes ripen in autumn and turn colour from green to brown the patches become less distinct and by winter they have taken on a light-grey silvery appearance. At the same time little black dots appear on them, which are the fructifications of the fungus (Fig. 136). These are of two kinds, pycnidia containing minute colourless one-celled conidia and perithecia containing asci with two-celled ascospores. The silvery sheen of the patches is due to small air spaces which arise between the outer skin and the inner layers of bark, which have been killed by the fungus and shrunken inwards a little. Most of the buds surrounded by the grey patches are killed outright and do not break in spring. In seasons with sufficient rain evenly distributed through

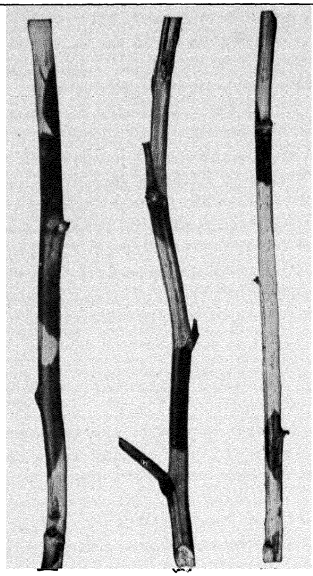


Fig. 135. Raspberry spur blight on young canes in September, dark purplish brown blotches (*S. p. F.*).



Fig. 136. Raspberry spur blight in October, grey patch dotted with black pycnidia, surrounding a bud.

the summer the remaining buds on the infected canes produce normal flowering spurs and set fruit. If there is any tendency to drought, however, these berries are apt to dry up and the shoots to wither. Probably this occurs because of interference with the water supply through the spur blight lesions but the exact nature of the damage has not been investigated. In spring one usually finds healthy inner bark, bast and wood below the grey lesions, and discoloration of the wood and pith generally only occurs just before the shoots wither. *Didymella* mycelium has not been detected in the inner tissues of the canes, so the immediate cause of the withering remains obscure.

Cause. Various fungi can be found on the dead grey patches of bark. In Denmark Rostrup first described the symptoms of spur blight in 1902 and attributed it to *Didymella applanata*. Dalskov in 1912 described the disease in *Gartner Tidende* and ascribed it to the fungus *Diplodina pallor* (Berk.) All. When H. Gram mentioned it in *Gartner Tidende* in 1916 he followed German authorities in attributing it to *Hendersonia rubi* West.

Finally in 1917 Osterwald in Switzerland proved by inoculation experiments that the cause of the disease was *Didymella applanata*. There has still been much uncertainty about the relationship of its conidial stage to the various species of *Phoma* previously described on raspberry canes. Schellenberg (1922) thought the conidial stage was the same as *Phoma idaei* Oud., but Karthaus did not agree with this.

In 1926–8 C. A. Jørgensen and A. Weber made a thorough reinvestigation of spur blight in Denmark, showing that it was principally caused by *Didymella applanata* and that in

single-spore cultures of this fungus a *Phoma* conidial stage was produced. This stage did not agree with *Phoma idaei* Oud., nor with any other *Phoma* species described from raspberries. It was therefore an undescribed imperfect fungus, but as it is only a stage in the life history of *Didymella applanata* it does not require a separate name of its own.

The "spur blight" attributed in America to *Mycosphaerella rubina* Peck is probably identical with the European disease.

In addition to *D. applanata* inoculation experiments were made with various fungi isolated from raspberry canes. One of these, *Leptosphaeria coniothyrium* (Fuck.) Sacc. (imperfect stage *Coniothyrium fuckelii* Sacc.), was able to produce similar lesions to those of *Didymella*. It was, however, much less common in Denmark. In England *L. coniothyrium* causes a disease known as cane blight, in which cankers occur at the base of the canes which then wilt, die, and break off at soil level.

Development. Spores from the fructifications on the old canes infect the new ones as they grow up. The *Phoma* conidia are more important than ascospores in spreading infection. Infection may occur first in the leaf stalks and spread from there into the canes or infection may take place through the buds. Canes may also be infected direct so that patches occur on them between the nodes. Sometimes the disease is not observed until the buds are found to be dead in the spring. Jørgensen and Weber's investigations showed that blotches on the petioles began to appear singly from the end of June onwards and became more numerous during July. Most lesions on the canes appeared during the first three weeks of August. Thereafter the number of new infections decreased but some still occurred even in September.

Importance. As far as is known the *Didymella* mycelium occurs only in the bark of the young canes and does not penetrate deeper. The hyphae grow through the spaces between the cells and probably kill them by producing soluble toxic substances that diffuse through the cell walls. The protective layer of cork and bark which surrounds the canes in autumn is inclined to crack and split open over the diseased patches, exposing the healthy tissue inside.

The diseased buds do not break in spring or do so only feebly, and this may cause considerable loss of fruit. The non-attacked buds produce normal flowering and fruiting spurs but many of these also do not attain their full development. They wither before the fruit is ripe and it is then, of course, completely spoiled. Often these shoots do not wither completely but the development of the berries is retarded and they remain small and dry. An infected raspberry plantation may give a satisfactory crop one year and yield only small, dry fruit in another. Apparently transport of water in the cane is impeded so that only when water is plentiful does sufficient reach the developing berries on infected canes.

Varietal susceptibility. Raspberry varieties differ greatly in their susceptibility to spur blight. Superlative and Marlborough are very susceptible, Fajstrup and the related variety Kejserinde Dagmar are very resistant. In England Pyne's Royal has been found to be resistant, and in Denmark Preussen has often been severely attacked. According to Wormald, Reader's Perfection and Lloyd George are particularly susceptible to cane blight caused by *Leptosphaeria*.

Control. It is important to avoid introducing the disease into a new plantation. Young canes should only be planted on ground that has not carried raspberries for several years. Spawn should be taken from a special nursery plantation which is cut down every year so that there are never any old canes remaining when the new ones come up in spring. In such a nursery bed there is, of course, no yield of fruit, but its maintenance is in any case desirable for the propagation of a virus-free stock (see p. 214) and is a small price to pay for the health of the fruiting plantations.

If spawn cannot be obtained from a nursery bed the old canes can be cut out, either when planting or in early spring before the new

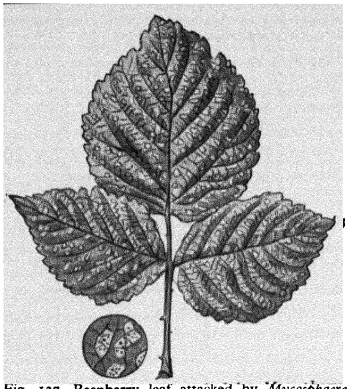


Fig. 137. Raspberry leaf attacked by *Mycosphaerella rubi*. Inset: lens view, showing the pycnidia.

ones appear. The source of infection for the new canes during summer is thus removed.

In old-established raspberry beds one should try to keep the disease in check by cutting out the old canes the day the last of the fruit is picked. Most infection, however, takes place during August, i.e. before the crop is over. At the same time weakly new canes and those severely attacked by spur blight should also be removed. Or it may sometimes be wise to leave thinning out the young canes until some time after the old ones have been removed to give time for all the spur blight lesions to become clearly visible. Thinning also makes conditions less favourable for infection than they are in a dense plantation.

In cutting out the old canes care should be taken to remove them right down to the ground as most spur blight lesions occur towards the base. In the case of canes killed by *Leptosphaeria* all the canker on the cane at the soil level must be cut away with the rest and burned.

Spraying with Bordeaux mixture before the fruit matures has been recommended but no spray programme has given really satisfactory control. The best time to spray would no doubt be when the fruit is ripening, but this is obviously impracticable.

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth. or *V. dahliae* Kleb.)

This disease, often called blue stripe wilt, was studied in England by Harris (1925, 1928) and in Holland by Karthaus (1927). During the winter the buds of infected canes shrivel and in spring the effects of the disease become conspicuous. Some canes are seen to be quite dead, others have a number of dead buds above one another along one side of the cane. When such a cane is cut a discoloured strip of wood is found along the side where the buds are dead. In other canes the spurs may grow out, but are poorly developed and die before the fruit ripens. Sometimes all the canes of a stool are affected, and in that case it usually dies. Often only one or more show the symptoms.

On the new shoots the first symptoms usually appear on the leaves about the end of June. Yellow or reddish-brown patches appear between the lateral veins so that the leaves appear striped. Gradually their edges roll up and they wither, starting at the base of the cane. Often the striping appears first on all the leaves along one side of the cane, and sometimes one side of an individual leaflet is affected before the other.

Later on a bluish or bluish-brown stripe appears along the surface of the young cane, and if this girdles it the whole cane dies.

As is usual with this fungus, infection seems to occur mainly through the roots and underground parts of the stems.

Spawn should not be taken from an infected plantation. All severely affected stools should be dug up and burned, care being taken to dig out all their spawn. Stools in which only one or two canes have been attacked may sometimes recover if their growing conditions are improved. No varieties immune to this trouble are known.

Raspberry Rust (*Phragmidium rubi-idaei* (Pers.) Karst.)

All four stages of this rust occur on raspberry leaves and closely resemble those of the common rust of blackberries, *P. violaceum*.

The spermogonia and æcidia appear on the upper surface of the leaves, the yellow uredo pustules and black teleutospore pustules are found on the lower surface. Usually the attack is not noticeable before the autumn and causes little damage.

Crown Gall (*Pseudomonas tumefaciens* (E. F. Sm. & Towns.) Stevens)

Crown gall tumours on raspberry canes may occur on ground level or higher up. In the latter case there may be a row of small galls. Wild raspberries, blackberries, especially Himalaya berry, youngberry and loganberry may all be attacked.

Galls on raspberry canes were examined as early as 1908–9 by Th. Wulff, but were attributed to growth disturbances ("*heteroplastische Gewebewucherungen*") or to the fungus *Coniothyrium*. Somewhat similar galls may arise as a result of frost damage (p. 215) or as callus growths over a wound on the crown. True crown galls are rather soft and fleshy, but Wormald has described spindle-shaped hard woody swellings which occur on the roots of some raspberry varieties. The cause of these is unknown.

As a rule crown gall does little damage to raspberries, even to infected stools. Infected canes may be cut out and burned, and care should be taken not to take spawn from the stools concerned.

Raspberry Mosaic

This is today by far the most destructive disease of cultivated raspberries in Britain and is common in Denmark in the variety Lloyd George. Similar diseases are prevalent in North America, but it is not certain that the viruses concerned there are the same as those found in Europe. It is still not known how many viruses attack raspberries in the British Isles, but some progress in disentangling them has been made by using the very susceptible variety Baumforth's Seedling B as an indicator. The widely grown variety Lloyd George has been found to carry almost without symptoms a virus known as Mosaic 2 (Fig. 138). Another virus, Mosaic 1, causes a comparatively mild

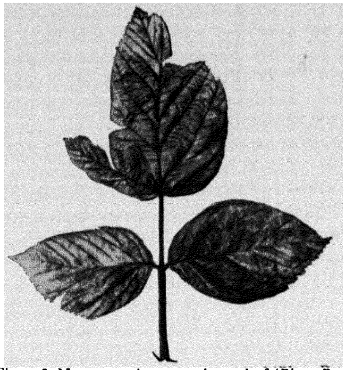


Fig. 138. No. 2 mosaic on raspberry leaf (*Photo Dept. Agric. Scotland*).

mottle in Lloyd George (Fig. 47), but when Mosaic 1 and Mosaic 2 are combined in that variety a severe bright-yellow mosaic disease results, which is that commonly found in commercial plantations of Lloyd George. Later work has shown that both Mosaic 1 and Mosaic 2 are probably mixtures rather than single viruses. Even the best British strains of Lloyd George so far tested are infected with viruses which affect their vitality and apparently bring about slow degeneration, even when they cause no distinctive symptoms of mottling on the leaves. A virus-free strain of this variety has recently been found in New Zealand. The comparatively new variety Norfolk Giant can be obtained in the virus-free condition, but is liable to suffer from a distinct disease, leaf curl, due to a virus which is "carried" by Lloyd George (Harris and Cadman, 1947). The variety Preussen seldom shows mosaic symptoms, but is a carrier of one or more of the raspberry mosaic viruses.

For a long time all attempts to find an insect vector of these diseases were unsuccessful. In 1947, however, Cadman and Hill reported that they had been able to transmit two raspberry viruses by aphides. *Amphorophora rubi* transmitted mainly a virus of the Mosaic 2 type from Lloyd George to Norfolk Giant; *Aphis idaei* transmitted a virus which induced in

Norfolk Giant an apparently new disease called curly dwarf. Both species of aphides require feeding periods of over 12 hours on the infected plant before they can pick up these viruses, and very large numbers are required to ensure transmission to a healthy plant. This probably accounts for the comparatively slow spread of virus diseases in raspberry plantations.

Once a raspberry stool has become infected with any of the mosaic viruses it never recovers; the symptoms gradually get worse, and the canes become dwarfed and unfruitful.

Control. Because Lloyd George may carry a virus which kills Norfolk Giant these two varieties should never be planted in close proximity. Where a plantation has been recently made from a healthy stock it may be possible to check the spread of mosaic into it by lifting and destroying all infected stools as soon as they are seen, together with those on either side which may have become infected as well. In old plantations roguing of this kind is not likely to be very effective. It is advisable to maintain a separate spawn nursery, well away from the fruiting canes, to start this nursery from disease-free stock and to keep it strictly rogued of all plants which show a trace of mosaic. The fruiting plantations can then be maintained by spawn from this nursery. A certification scheme for healthy spawn nurseries has been launched by the Ministry of Agriculture and the Department of Agriculture for Scotland.

For suggestions for maintenance of spawn nurseries, see Grubb and Harris, 1945, and *The Fruit Grower*, xcix, p. 43, 1945.

Raspberry Leaf Curl

This very destructive virus disease is prevalent in Scotland on the variety Norfolk Giant. Symptoms appear on the new canes when they are about a foot high, at the end of May or beginning of June. The uppermost leaves curl tightly downwards towards the cane, followed progressively by the lower leaves (Fig. 139). At the same time they acquire an abnormally dark-green colour, often with a waxy look, and their stems appear glossy. Similar symptoms may develop on the fruiting spurs, combined with a blotchy yellow mottle. Affected leaves and canes are very brittle and snap readily. Stools infected with leaf curl die quickly, at the latest during the season following that in which the symptoms first appeared. This virus is believed to have been responsible for the failure of Baumforth's Seedling B as a commercial variety in Scotland, but is apparently carried with few or no symptoms by Lloyd George.



Fig. 139. Raspberry leaf curl (*Photo Dept. Agric. Scotland*).

The insect vector of leaf curl virus has not so far been discovered, but the disease is transmissible by grafting. Until more is known about it the principal control measures can only consist in strict roguing of Norfolk Giant plantations wherever leaf curl is observed. Care should be taken to keep plantations of this variety as far removed as possible from all plants of Lloyd George and from wild raspberries, some of which may also prove to be carriers of the leaf curl and other viruses.

Raspberry Yellows

Raspberry yellows, which is very general on the variety Marlborough in Denmark, is apparently the same as a condition described on

raspberries in Norway and Bohemia, but is not identical with the yellow mosaic disease described from England and America. In yellows the yellow colouring appears in bands and patches between the lateral veins. These patches eventually dry out. Sometimes the leaves are almost entirely yellow with only a little green left along the midrib and lateral veins. At first the suckers have dark-green leaves. They may turn yellow in early autumn but mainly become green again later. The yellowing is most pronounced in dry, warm weather and on poor soil. If the soil and weather are not very unfavourable the yellow plants may still crop fairly well. Yellowing may spread considerably through a plantation in two or three years. It is probably a physiological disturbance, possibly due to malnutrition, but its cause is still not understood. Some observations in Norway suggest that it may be due to infection by a parasite.

Frost Damage

A frequent cause of complaint amongst raspberry growers is the failure of all or most of the buds on their canes to break in spring. This form of frost damage is especially common when there has been a mild spell, with rising sap, followed by frost as the buds were about to burst.

Frost damage is also particularly severe where unfortunate growing conditions have impeded ripening of the buds in the preceding autumn. One spring we had the opportunity of examining a plantation where the soil reaction at one end was near pH8. The raspberries had survived the winter much worse on this soil than at the other end of the plantation, where the reaction lay between pH6 and 7. In the spring of 1938 many flowers were damaged by frost and the berries were deformed. As a rule, however, malformed berries are due to attack by raspberry beetles and other insects.

In Finland Rainio (1936) concluded that the cracks and irregular corky knots found on raspberry canes were forms of frost canker. In a very mild autumn the canes, supplied with too much nitrogen, developed extra soft wood and bark which was split by the first frost of winter (cf. Fig. 23). Then, when the sap rose in spring, irregular, knotty outgrowths of callus developed on the living edges of the wound. In the past these have been attributed to various causes, including spring frosts (Sorauer, 1909), excess nitrogen and moisture (Wulff, 1908) and attacks of the fungus *Coniothyrium fuckelii*, which, however, does not produce cankers on this host (Stewart and Eustace, 1902; Jørgensen, C. A., and Weber, A., 1929).

Summary of Raspberry Diseases

I. Root and Crown

		page
A. Irregular fleshy tumours	Crown Gall	213
B. Woody, spindle-shaped swellings on roots	Cause Unknown	213

II. Canes

A. Dead buds, mainly near tip of cane	Frost	215
B. Dead buds, surrounded by grey patches of bark usually towards the base of the cane	Spur Blight	209
C. Dead buds associated with brown stains in the wood	*Verticillium* Wilt	212
D. Cankers at base of cane which easily breaks off at ground level	Cane Blight	211

		page
E. Dead shoots:		
(1) Shoot withered at the tip in spring, bark often cracked	Frost	215
(2) Shoots die during winter	*Verticillium* Wilt	212
(3) Fruiting spurs die in summer	Spur Blight	209
F. Superficial markings:		
(1) Blue or bluish-brown stripes on young canes	*Verticillium* Wilt	212
(2) Small purple spots, often with grey centres	Cane Spot	208
(3) Purplish-brown blotches, mainly round leaf bases on the young canes. Grey patches dotted with black round the buds after leaf fall	Spur Blight	209
G. Irregular tumours:		
(1) Small wart-like outgrowths associated with split-bark	Frost	215
(2) Irregular tumours not associated with frost damage	Crown Gall	213
III. Leaves		
A. Large, irregular, dark-brown blotches spreading down the leaf stalk	Spur Blight	209
B. Leaf edges discoloured:		
(1) Light-coloured dried-up margins	Chlorine Poisoning	34
(2) Brown marginal scorching	Potassium Deficiency	42
C. Discoloration between the lateral veins:		
(1) Bright yellow, sometimes recovering later	Yellows	214
(2) Yellow blotches, turning brown and drying out, leaves eventually rolling up	*Verticillium* Wilt	212
D. More or less bright yellow irregular spots, surface sometimes crumpled	Mosaic	213
E. Very tightly curled, brittle leaves on young canes	Leaf Curl	214
F. Well-defined spots of fungus origin:		
(1) Yellow cluster cups on the upper surface in spring, yellow, then black, pustules below later	Rust	212
(2) Small circular spots with whitish centre and dark border	Cane Spot	208
IV. Fruit		
A. Grey mould on the ripe fruit	Botrytis	59
B. Grey, dull, surface on ripening fruit	Sun Scorch	27
C. Misshapen fruit:		
(1) Irregular one-sided berries, the drupels on one side remaining green and bearing tiny blackish fructifications	Cane Spot	208
(2) Irregular berries with many drupels aborted, no fungus fructifications	Frost	215
(3) Dried-up berries on a wilting spur	Spur Blight	209

VINE

Mildew (*Uncinula necator* (Schw.) Burr = *Oidium tuckeri* Berk.)

Symptoms and development. Vine mildew may attack all the green parts of the vine; young shoots, leaves, flowers and fruit, covering them with a floury white layer of mycelium and conidia. The haustoria penetrate the underlying epidermal cells which turn brown and the leaves curl, dry and may be shed.

The attack usually commences at the base of the young shoots and spreads thence to the leaves, where the fungus may grow over both surfaces, and the bunches, where it does most damage. It may appear on the flowers, in which case they set no fruit. More often it appears on the young fruit soon after they have set, causing them to crack and fall before they attain half their normal size (Fig. 140).

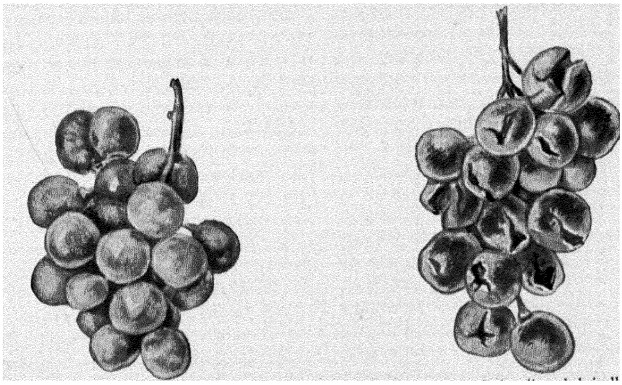


Fig. 140. Vine mildew. (*a*) early stage on the fruit; (*b*) late stage, fruit split and shrivelled.

Ripe grapes are not attacked. The cracking is due to the infected skin being unable to stretch to keep pace with the normal swelling of the flesh as the grape grows. Under dry conditions mildewed grapes may ripen prematurely without cracking and bear only dry patches of infected skin. Under damp conditions the cracks soon appear, giving easy access to other fungi, such as grey mould, which complete the destruction of the grapes.

During the growing period the fungus spreads readily from vine to vine by means of the conidia, but its method of overwintering is less clearly understood. In North America vine mildew is believed to overwinter largely as perithecia which mature on fallen leaves during the winter. Perithecia are rare in Europe, and overwintering there probably occurs mainly as mycelium in the buds or on the young twigs. This has not been demonstrated with certainty, however. In Germany, Seeliger (1939) found that perithecia are formed in plenty in hot summers, especially on certain varieties.

History and importance. Vine mildew was first discovered in an English glasshouse in 1845, when it received the name *Oidium tuckeri* because only the conidial stage was found. In 1848 it was seen at Versailles, but may have been present in France before that. In 1852 it was found in many places in central and southern Europe, in the Levant, North Africa and on Madeira, where it did much damage. In 1854 and 1855 it caused catastrophic losses in many places. For example, in some districts in France vine growers were forced by them to leave their holdings and emigrate. The average yield in the French vine-growing provinces fell to one quarter from 1850–5. Thereafter various control measures

were adopted, especially dusting with sulphur which kept vine mildew under control. But in years particularly favourable to the fungus it can still cause a serious disease.

Vine mildew was found in Denmark in 1857, where it appeared in one of the Rosenborg greenhouses in such intensity as to make all the grapes uneatable. It is now generally distributed in that country, both in greenhouses and out of doors. It is worst where the shoots are overcrowded and in sunless, damp weather, or in excessively shaded houses.

Control. Cultural conditions should be kept as favourable as possible. Vines must not be overcrowded and thinning of shoots and leaves must be thoroughly done. Usually each branch should bear one bunch and be cut back two internodes above the bunch. All shoots produced late are to be pinched back above the first leaf. Dusting with sulphur is generally employed in vine-growing districts. Where mildew is to be feared the dusting is done at least once before flowering and again immediately after flowering, or, if necessary, even during flowering as it does not prevent fertilization. Dusting is repeated later, there being 4–5 applications in all. The most favourable temperatures for dusting are 77–95° F., for below 77° F. the sulphur is not sufficiently active and at temperatures exceeding 95° F. it may cause scorching. After dusting with sulphur one may sometimes notice a number of tiny brown spots on the young grapes. These are not necessarily due to scorching, but may be dead epidermal cells killed by the mildew and only rendered visible after the latter has been destroyed. We have seen corky spots on the skin of grapes which were attributed to scorching by a sulphur dust. It seems that damage by the sulphur may occur not only at high temperatures, but also when the dusting has been very heavy and if the grapes were wet when dusted.

In greenhouses vaporization of sulphur is often more effective than dusting.

In 1911 Lind recommended spraying with strong lime sulphur in winter in addition to the usual summer sulphur dusting or vaporization. In experiments carried out by the State Plant Pathological service in Denmark in 1914–18, excellent results were obtained by scraping and brushing the vine bark, followed by spraying with 1:9 lime sulphur before the buds broke. In subsequent experience these control measures have given somewhat varying results. Sometimes mildew has disappeared completely after the treatment, in others it has reappeared, often late in the season. In the latter instance it may have been reintroduced from outside. As soon as the beginning of an outbreak is noticed either dusting or vaporization of sulphur should be resorted to.

The beneficial results of scraping of the bark and winter spraying are best explained by the overwintering of the fungus on the surface of the shoots. Most of the infectious material is removed by the scraping, the rest is killed by the spray. Mycelium overwintering in the buds would not be affected directly. Wormald (1939) suggests that when vine stems are painted during winter with a mixture of sulphur and soft soap the high spring temperatures vaporize enough sulphur to control the mildew. All fallen leaves, prunings and bark scraped off should be removed and burnt.

American varieties descended from *Vitis labrusca* are less susceptible to mildew than European varieties descended from *Vitis vinifera*, but differences in susceptibility occur even among the latter.

Downy Mildew (*Plasmopara viticola* (Berk. & Curt.) Berl. & de Toni.)

Downy mildew is one of the most serious vine diseases in south Europe, but in the north it is of minor importance. In Denmark it occurs occasionally; for example, at one locality on an outdoor vine in 1909 and again in 1918 and at several places in 1937. In England it was first observed in 1894 and not found again until 1926 (Harrison and Ware, 1926). In 1936 it reappeared on a very old vine 100 yards from the latter outbreak and in 1941 it occurred again ¼ mile away. All these outbreaks have been on outdoor vines in Kent (Moore, W. C., 1943).

Symptoms. Light-green patches appear on the upper surface of the leaves and a little later

a downy whitish mildew appears on the lower surface of these areas. The diseased areas dry up, causing the leaves to curl and fall off. Tips of the shoots may also be attacked, and so are the berries which shrivel and become brown and leathery.

Control. All diseased leaves and tendrils should be removed and burnt in autumn, but this is not sufficient to prevent further outbreaks, for the fungus also overwinters in bud scales and shoots.

Spraying with Bordeaux or Burgundy mixture or some comparable copper spray should be repeated as often as necessary until the grapes are so far advanced that further spraying would cause spotting of the fruit. Spraying during flowering should be avoided.

Downy mildew is encouraged by nitrogen deficiency, and affected vines should therefore receive a dressing of nitrogenous manure at the beginning of the season.

Ripe Rot (*Glomerella cingulata* (Stonem.) Spauld. & v. Schrenk = *Gloeosporium fructigenum* Berk.)

This disease is caused by the same fungus as bitter rot of apple and occurs occasionally on greenhouse grapes in England. Fruit is infected when it is almost full grown and develops a discoloured patch which soon spreads over the whole berry. Fructifications of the fungus appear on the skin as tiny whitish pustules arranged in concentric circles. Eventually the affected grapes shrivel and become more or less mummified.

Control measures recommended include thorough ventilation, removal of all infected berries and badly damaged bunches and avoidance of syringing, which might splash the spores from bunch to bunch.

Anthracnose (*Elsinoë ampelina* Shear = *Gloeosporium ampelophagum* (Passer.) Sacc.)

This is a serious disease in the vineyards of Europe and North America but has only occasionally been reported in England. All the green parts of the vine are attacked, young shoots, tendrils, leaves and berries. The leaves bear irregular greyish spots with a dark margin. Eventually they dry up and may fall out to give the leaves a ragged shot-hole appearance. On the berries the spots are small and circular, at first brown, later grey with spores in the centre, with a dark margin. Similar spots appear on the young green shoots.

Diseased twigs should be cut out and burnt. Continental experience suggests that the disease may be kept in check by a winter spray with 1:9 lime sulphur and a summer spray with 8:8:100 Bordeaux mixture.

Grey Mould (*Botrytis cinerea* Pers.)

The vine is often attacked by grey mould, especially on the grapes. They rot and the bunches become covered with a shaggy grey fungus growth. In virulent attacks the leaves and shoot tips may be damaged and infection may also occur at the base of shoots so that their leaves turn yellow or become spotted with rusty brown. Prevalence of grey mould is an indication of excessive atmospheric humidity. Grapes damaged by other causes such as shanking are especially easily attacked.

There should be thorough ventilation and the temperature of the house should be kept down. Watering should be done as little as possible. When an outbreak has begun, spraying with Pomarsol or Shirlan may possibly check it.

When grey mould is seen the diseased grapes should immediately be cut off and collected in a pail of disinfectant, for example strong lime water, to prevent the conidia from being blown about. The fungus may also occur in propagating frames. To avoid it a mixture of $\frac{1}{3}$ charcoal and $\frac{2}{3}$ peat moss litter can be recommended, or else the use of only disinfected soil.

In vine-growing countries it is well known that when the grapes that are almost ripe are attacked they dry up like raisins. Oddly enough this gives the wine a high quality and is hence called *Edelfäule*. It is due to infection with *Botrytis acinorum* Pers.

Sooty Mould

On vines infested with scale insects, aphides or woolly aphis, the two former are most plentiful. A sooty coating is apt to occur on the leaves and grapes. This is made up of various species of fungi that live on the sugary exudations of the insects. Sooty mould fungi do not penetrate the plant tissue, but shade the leaves and thus hinder carbon assimilation. The affected grapes are also rendered unappetizing in appearance.

The insects responsible may be controlled by removing old loose bark from the stock in winter and by spraying or painting with lime sulphur or some insecticidal oil (see further, p. 126).

Shanking

Symptoms and cause. Shanking is the name applied to a brown spot that appears on the stalk of the unripe grape. This spreads until the stalk is girdled and the berry fails to ripen. It may become covered with brown spots and shrivel or, if shanking occurs shortly before ripening, the grapes remain sour and do not attain the normal colour. Fruit of black varieties turns red and that of white varieties remains green.

Shanking is a non-parasitic condition usually associated with inadequate functioning of the root system. It is most prevalent on late varieties. Sudden pronounced changes in temperature and humidity in the house may cause shrivelling of grapes, as may a sudden spell of strong sunshine after a prolonged period of dull weather.

If the soil is too rich there may be a copious growth of new roots that have not time to mature before winter. They may therefore be destroyed by frost or rot away. Waterlogging of the beds may have a similar effect. Lack of these roots is felt especially after the pips begin to form and shanking then sets in. Vines which are themselves within a warm house but have much of their root system in colder soil outside are also prone to it. Shanking also often happens the year after an unusually heavy crop unless the depleted soil has been carefully manured.

Grapes affected by shanking are easily infected by grey mould.

A young vine that is cropping too heavily may be unable to ripen its grapes normally. They tend to be sweet, but only reddish instead of black, and lack the stalk lesions characteristic of shanking.

Control. Violent fluctuations in temperature and humidity in the house should be avoided. The beds must not be allowed to dry out in hot weather. It may easily happen that soil may be kept damp on the surface by frequent watering, but is actually dry lower down round the absorbing roots. The aim should be to maintain a warm, porous, well-drained soil. Recently it has been suggested that potash deficiency is an important factor in causing shanking and that it can be corrected by applying sulphate of potash to the soil in August and January at the rate of 1½ oz. per square yard (Moore, W. C., 1943).

Vines should not be allowed to carry too many bunches and they should be shaded by the leaves. When bright sunshine follows a period of dull weather it may be necessary to shade the vines in the middle of the day. Shading may also be advisable in the mornings when dew has condensed on the grapes during the night.

Aerial Roots

When vine stocks put out numerous aerial roots it is an indication that the condition of the underground root system is unsatisfactory. Generally the reason is that the soil is too cold for the roots to function adequately and the plant attempts to supplement them by growth of aerial roots. These generally begin to appear a little before flowering, as though the vine had been able to make do until then with the food stored in the stock.

As soon as aerial root formation is observed the soil conditions should be investigated and, if necessary, corrected (see shanking).

Œdema

Œdema is not uncommon on vines under glass. It occurs partly on the bunches, where small warts develop on the grape stalks, and

partly on the under-side of the leaves, which become covered with small, irregular, yellowish pimples.

Though conspicuous, the condition is a non-parasitic one and of little economic importance. It is due to transpiration from the leaves failing to keep pace with water absorption by the roots. Œdema is therefore associated with too humid an atmosphere or with too warm and moist a soil.

May Sickness

A vine disease has been reported from Holland under the name "May sickness." It occurs as dead spots on leaves, stalks, floral parts and young grapes. According to Dutch observations it is due to low night temperatures and especially to dew settling on the vine and remaining there far into the day. The damage may be avoided by shaking the vine the morning after a cold night.

Summary of Vine Diseases

		page
I. Roots		
Root rot, often associated with white mycelium under the bark	*Armillaria mellea*	456
Dead roots (see under shanking)		
II. Stem		
A. Whole plant wilts and dies, brown stain in wood	*Verticillium* Wilt	60
B. Fungus coating on young shoots:		
(1) Floury white coating	Vine Mildew	217
(2) White downy mould	Downy Mildew	218
(3) Shaggy grey mould	Grey Mould	219
(4) Black coating	Sooty Mould	220
C. Dead spots on young shoots	May Sickness	221
D. Irregular tumours	Crown Gall	81
E. Aerial roots		220
III. Leaves		
A. All leaves wilted and dead	*Verticillium* Wilt	60
B. Fungus coating, sometimes causing shrivelling:		
(1) White floury coating	Vine Mildew	217
(2) Downy mould on lower surface	Downy Mildew	218
(3) Shaggy grey mould	Grey Mould	219
(4) Sooty black coating	Sooty Mould	220
C. Leaf spots:		
(1) Rust-coloured spots eventually bearing grey mould	Grey Mould	219
(2) Yellowish-green patches with mould on underside	Downy Mildew	218
(3) Dead spots	May Sickness	221
(4) Irregular grey spots with a dark border	Anthracnose	219
D. Widespread discoloration:		
(1) Pale-green or yellowish leaves	Iron Deficiency	36
(2) Light-green leaves with brown edges	Magnesium Deficiency	38
(3) Irregular brown patches	Sun Scorch	27
(4) Bronzed leaves	Potassium Deficiency	42
(5) Dingy yellow leaves, eventually turning red at the edges	Phosphorus Deficiency	41
E. Pimples forming yellowish coating on the lower surface	Œdema	220

IV. Bunches		page
A. Soft grey berries	Sun Scald	27
B. Fungus coatings:		
(1) Black coating	Sooty Mould	220
(2) Dusty white coating, berries often cracked	Vine Mildew	217
(3) Whitish mould, berries often brown and leathery	Downy Mildew	218
(4) Rotting grapes covered with grey mould	Grey Mould	219
C. Spotted or mummified fruit bearing minute fungus pustules:		
(1) Mummified grapes	Ripe Rot	219
(2) Reddish spots, turning whitish at the centre with a dark rim	Anthracnose	219
D. Small warts on the stalks	Œdema	220
E. Corky brown spots and patches without mould:		
(1) Dead patches on young fruit	May Sickness	221
(2) Brown spot girdling fruit stalks, shrivelled or undeveloped berries	Shanking	220
(3) Spotting of older berries, soon followed by grey mould	Grey Mould	219

WALNUT

Blotch (*Gnomonia leptostyla* (Fr.) Ces. & de Not. = *Marssonina juglandis* (Lib.) Magn.)

Symptoms. The leaves develop irregular, rounded, brown spots with darker edges, somewhat greyish on the under-side (Fig. 141). Several spots often become confluent to form large brown patches. On the surface of the green fruit dark spots appear that soon turn black and may be conspicuously sunken (Fig. 142). Heavily infected leaves and fruit often fall off. The conidial stage (*Marssonina juglandis*) appears as minute pustules on the spots in damp weather and the spores are dispersed especially by rain splashes. The *Gnomonia* perithecia form on the fallen leaves during winter, and ascospores from them reinfect the new leaves in spring.

Control. All fallen walnut leaves should be raked up and destroyed during the winter. If this is insufficient to prevent an outbreak a Bordeaux spray as recommended against bacterial blight may be tried.

Mouldy Walnuts

Stored walnuts are often attacked by various fungi and are then popularly described as "mouldy." The fungi generally appear first on the shell but soon penetrate it and destroy the kernel.

Hamond (1931) found the following fungi on walnut shells: *Penicillium* sp., *Botrytis cinerea*, *Rhizopus nigricans*, *Fusarium* sp., *Coniothecium* sp., *Alternaria* sp., and *Cladosporium* sp. The first four were also found on the kernels. The following procedure in preparing walnuts for storage is recommended at East Malling. The green shell should be taken off, the last remains being removed with a soft scrubbing brush and water, but the nuts should only be in the water for a few seconds. They should be dried in an airy place at about 70° F. They may be bleached and disinfected in a solution of 6 lb. chloride of lime and 3 lb. washing soda in 10 gallons of water. The chloride of lime and soda are dissolved separately in small amounts of water and the mixture is then made up and left to stand for 24 hours. The nuts are then dipped for 3 minutes in the clear solution and dried out at 70° F. Nuts with holes in the shell are rendered inedible by this treatment which is useful more for bleaching the shell than for preventing mould growth.

Walnuts keep well if stored at a temperature of about 38° F. and a relative humidity of 90%.

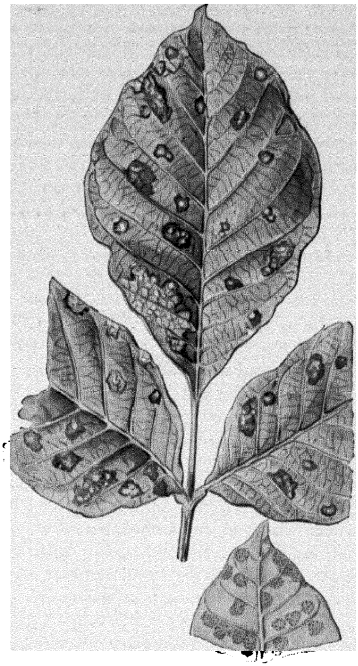


Fig. 141. Walnut blotch. Above: upper side of leaf. Below: lower side.

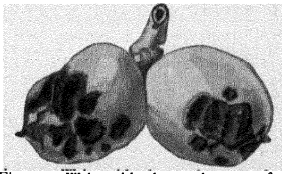


Fig. 142. Walnut blotch, on the green fruit.

At higher temperatures they easily go mouldy, at lower humidities the kernels shrivel. Shrunken nuts can be softened by soaking for 24 hours in milk or salt water.

Storage in a mixture of 90% salt and 10% sodium phosphate also proved successful at East Malling, but in our experiments in a fairly damp cellar the salt became too moist. Storing nuts by packing them in bran is practised to some extent. **Washing** the nuts for 3 minutes in a 2 pM Sterisol solution can arrest a mould attack that has already begun (Lange, 1936). Probably 2pM chloramin solution would have the same effect.

Bacterial Blight (*Pseudomonas juglandis* Pierce = *Xanthomonas juglandis* (Pierce) Dowson)

Symptoms. Very small angular black spots, bounded by the finer veins, appear on the leaves. If they are numerous they join up to form large withered patches. Blackish elongated patches may also appear on the young shoots and may girdle and kill them. On the young green fruits there are black blotches which may cover the whole surface. They may spread inwards and cause the kernel to shrivel and the nut to fall. There may also be infection of the male catkins.

The bacterium overwinters in the lesions on the shoots and in the buds. From these it spreads to the young leaves in spring, probably in rain splashes, but possibly also on the bodies of aphides.

Distribution and importance. The disease has been longest known and is most destructive in western North America, especially in California. In Europe it has been reported from Russia, Italy, Switzerland, France and England, where it was first observed in 1925. In America the disease has been very destructive on old fruiting trees, but not on young ones. In England it has been found mainly on young trees and is not yet very dangerous, though a severe attack was reported in Devon in 1937 (Moore, W. C., 1943). In Oregon the same bacterium has been found to attack both wild and cultivated hazel.

Control. Every attempt should be made to eradicate the disease from nurseries and to prevent the sale of young infected trees. All infected shoots should be cut out and burnt. Towards the end of winter the trees should be sprayed with lime sulphur 1:20. According to experience in California the summer sprays are far more important. A Bordeaux spray is recommended while the buds are breaking (before the female flowers appear) and another

as soon as possible after flowering (Rudolph, 1933).

Resistant varieties of *Juglans nigra* and *J. hindsi* are being propagated. These species may also be used as stocks on which to graft more susceptible varieties.

Graft Disease (*Chalaropsis thielavioides* Peyronel)

This is a disease of young grafted walnuts raised under glass. The fungus grows over the cut surfaces of scion and stock, penetrates the wood, turning it brown, and kills the living cells. Union of scion and stock therefore becomes impossible and the former dies a few weeks after grafting. A sooty powder formed of resting spores of the fungus will be found on the ununited surfaces. This disease has been studied by Hamond (1935).

Control measures include disinfecting the grafting house with 1:39 solution of commercial formalin. Before the stock is cut the place of union should be painted with the same solution and the knife used should be sterilized.

Ring Rot (*Fusarium* sp.)

Ring rot is a disease of green walnuts causing them to fall prematurely. Sometimes they show only a small black sunken area on the husk near the stigma. Others have a roughly circular patch about one inch in diameter on one side. This patch is slightly raised, pale green or yellowish and is surrounded by a wavy black band about $\frac{1}{12}$ inch wide. Gradually the patch turns black and decays, when orange-coloured pustules of *Fusarium* spores break out on its surface. The rot slowly spreads down the immature shell inside the husk, and ultimately the kernel, too, decays to a soft yellow-brown mass.

Moore (1945), who described this disease in England, did not identify the species of *Fusarium* present. In Germany Wollenweber and Reinking (1935) found *F. lateritium* Fr., *F. sambucinum* Fuck. and *F. avenaceum* (Fr.) Sacc. associated with walnut fruit rot.

Soft Shell

This is a non-parasitic disorder in which the fruit has a thin shell, usually with two irregular holes in it, one on each side of the suture near the apex. Such nuts cannot be stored. The trouble is characteristic of certain varieties but its prevalence varies with the season. It is generally most prevalent after dull, wet summers, as in 1931 in England.

No control measures are known apart from avoiding susceptible varieties.

Summary of Walnut Diseases

I. Branches and Twigs

		page
A. Irregular swellings	Crown Gall	81
B. Blackish blotches on the season's growth, and dead shoot tips	Bacterial Blight	223

II. Leaves

A. Brown well-defined rounded spots	Blotch	222
B. Minute angular spots on leaves and petioles	Bacterial Blight	223

III. Fruit

A. Black blotches on green fruit	Bacterial Blight or Leaf Blotch	222–3
(To distinguish these diseases examine the spots on the leaves)		
B. Mouldy stored fruit		222
C. Thin shells, often with holes towards the tip	Soft Shell	224
IV. Failure of scions in grafted trees	Graft Disease	224

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III

DISEASES OF VEGETABLES AND HERBACEOUS FRUIT

3 DISEASES OF VEGETABLES AND HERBACEOUS FRUIT

ARTICHOKE (GLOBE)

Leaf Spot (*Ramularia cynarae* Sacc.)

The leaves may develop greyish spots with brown margins. In severe attacks the leaves wither and the flower heads remain small. Outbreaks that are just beginning may be checked by a fungicidal spray or dust.

Grey Mould (*Botrytis cinerea* Pers.)

Grey mould causes great damage to globe artichokes in U.S.A., especially during transit (Ramsay, G. B., and Wiant, J. S., 1944). The attack begins, however, in the field and even there may be of some importance. It has also been noted in Denmark.

The fungus attacks the flower heads, where the first symptom is the appearance of small brown spots especially near the tips of the surrounding bracts. Gradually it spreads to form a hairy grey coating over the greater part of the flower head. When the heads are cut the fungus may invade the cut surface and from there, during transit, grow into the tissues and cause rotting of the head. It may also infect the stem left in the field through the wound left at cutting and grow downwards, killing part of it.

Control is difficult when conditions favour grey mould, e.g. in damp weather, for the fungus will then be found sporing on many kinds of plant debris or weakly plants in the garden. All such plant debris should be removed, placed on the compost heap and covered with lime, or buried beneath a good layer of soil. Globe artichokes in store should be kept as cool and dry as possible, though cold storage will only slow down rotting that has started. It can only be stopped altogether at a temperature of 28° F., which is too cold for the artichokes to endure (see also p. 59).

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

This disease is easily recognized by the white, woolly mycelium and large black sclerotia (see p. 77). It is more common as the cause of a foot rot and stem rot of Jerusalem artichoke.

Downy Mildew (*Bremia lactucae* Regel)

An extensive outbreak of this disease on globe artichoke occurred in Sussex in 1944–5 (Moore, 1948). For a description of the fungus see p. 288.

ARTICHOKE (JERUSALEM)

Sclerotinia Disease

Jerusalem artichoke may be severely attacked by stem rot due to *Sclerotinia sclerotiorum* (Lib.) de Bary, especially in old plantings. The disease is easily recognized by the cottony white mycelium and large white, later black, sclerotia which appear on the stems, usually at or near soil level. The same fungus may cause a rot of the stored tubers as also may other fungi, especially *Botrytis cinerea* Fr. and *Rhizopus nigricans* Ehrenb. Tuber rots are best prevented by storing the artichokes at a low temperature, a few degrees above freezing point.

ASPARAGUS

Asparagus Rust (*Puccinia asparagi* DC.)

In some seasons asparagus is severely attacked by a species of rust fungus peculiar to that crop. The ornamental species, *Asparagus sprengeri* and *A. plumosus*, are not attacked by it. A species of *Puccinia* has, however, been found on *A. sprengeri* and a third species, *A. falcatus*, is subject to infection by another rust, *Puccinia phyllocladia* Cooke.

Symptoms. Yellow spots appear on the stems and branches in May or June and on these the cluster cup stage of the rust develops (Plate 10, Fig. C). As the crop is being cut at this time from the beds in use the cluster cups should be looked for in young plantings. Spores from the cluster cups infect an asparagus top if they are blown on to it and the infected plant bears the first pustules of cinnamon-coloured summer spores, or uredospores. Every 12–14 days throughout the summer a fresh generation of these spores may be produced until the stems, branches and needles appear brown and powdery all over. As the above-ground parts of the plant begin to fade the black pustules of teleutospores appear, which form the overwintering phase of the fungus (Fig. 143).

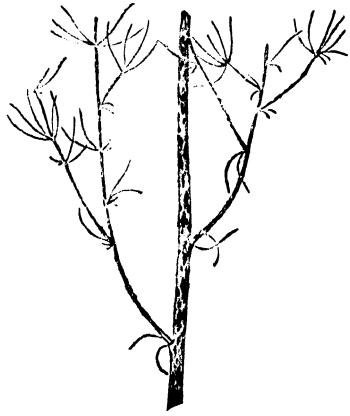


Fig. 143. Asparagus rust, teleutospore pustules on the stems.

Occurrence and importance. In England asparagus rust was first recorded in 1865, at Swanscombe, Kent. Since then there have been severe outbreaks of the disease, notably in 1895, 1904–6 and 1933–5, separated by periods in which the fungus was of little or no importance. A map showing the known distribution of asparagus rust in England was published in *Min. Agric. Bull.* 139, 1948. In Denmark where new plantings are infected early the disease may be destructive; in districts where there are only old plantings it generally appears late in the year and is less important. Severe attacks decrease carbon assimilation by the plant and increase evaporation as the skin is broken over every rust pustule. The results are seen in the shape of feeble shoots the following year. Grey mould and secondary fungi on roots and shoots add to the loss (Hassebrauk, 1938) and the weaker plants may be killed outright.

The teleutospores survive the winter better covered with soil than on top of it, otherwise winter has little effect on them. Some lose their viability in warm, moist air, but if 25% survive the winter that is quite enough infectious material. By the end of June the teleutospores have lost the capacity to germinate, hence epidemics of asparagus rust are only to be feared in young plantings, which are not cut, and in wild asparagus, which may be infected early in the year (Gassner and Hassebrauk, 1934). A warm, dry period from June to August favours the uredo stage.

Control. We have seen excellent results from spraying with Bordeaux or Burgundy mixture, but it is essential that the spraying be done early. A copper lime dust may also be used. The first spraying should be on the " bare " stems, that is, before the needles have unfolded. Where there are new plantings protection is especially important, both for the plants themselves and for the older ones in use. Spraying should be repeated frequently. We have seen beds where the stems were blue with spray on one side and yellow with cluster cup rust on the other. Use of a good spreading agent is strongly recommended (p. 601). Where there are many new plantings and the

rust is prevalent spraying should be done several times during the summer to keep the tops protected. In the drier parts of western North America sulphur dusting has given good control.

The needles attacked by rust fall fairly early. Hammarlund recommends that the plants be cut down altogether before normal withering takes place. They should then be left to dry for some days and then burnt together with some straw, by which means any spores that are lying on the ground are rendered harmless.

It is desirable to make young plantings a long way away from the beds in use. Where rust is to be feared one should not cut small or weakly plants or those whose stems are fasciated (grown together). No wild asparagus should be allowed to grow in the neighbourhood, nor any self-sown seedlings on the beds.

In America there are resistant varieties, Mary Washington and Martha Washington. They have been tried in Germany with varying success and are used in Denmark for forcing. As in all breeding programmes, both the power of disease resistance and the value as a crop in the place where the variety is to be grown have to be considered.

Pythium Rot (*Pythium* sp.)

In the last century Rostrup noted a serious disease of asparagus which was found again on material received at the State Pathological Experiment Station from two localities in 1930 and one in 1931. A similar disease was reported in Herefordshire in 1936 (Moore, W. C.).

Symptoms. The disease appears on the shoots of plants several years old, both when they have just emerged above ground and when they have attained a height of 5–6 inches. At the point of attack the shoot curves over, often so sharply that its tip bends downwards and later it bends right over at the same place. In moist air, a white mould-like coating appears, especially at the bend, which may be an inch or two long and $\frac{1}{10}$ inch thick (Fig. 144). The shoot may then rot completely, but in dry air the mould-like coating quickly vanishes and the shoot dries up.

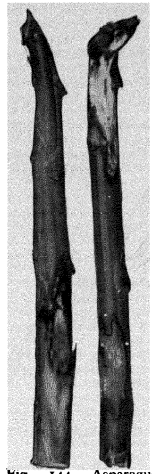


Fig. 144. Asparagus shoots attacked by *Pythium* (*S. p. F.*).

In 1930 healthy asparagus shoots were successfully inoculated with an unidentified species of *Pythium* isolated from diseased shoots (Jørgensen, C. A.).

Control. As species of *Pythium* may attack many plants, especially seedlings, the asparagus beds should be kept as free from weeds as possible. The surface soil should be kept loose.

Watering the soil round the diseased plants with Cheshunt compound (p. 593) may be tried. This is likely to be more effective than corrosive sublimate as *Pythium* is more sensitive to copper than to mercury.

Fusarium Rot (*Fusarium* spp.)

Attacks of *Fusarium* on asparagus assume various forms. In Denmark they occur on young plants which will not grow after replanting so that a very irregular growth is obtained. Observations indicate such plants were of poor quality and that the fungus infection only completed their destruction. *Fusarium* also occurs on the commercial shoots of asparagus which then become bent, spotted and tough (Fig. 145). The disease looks much

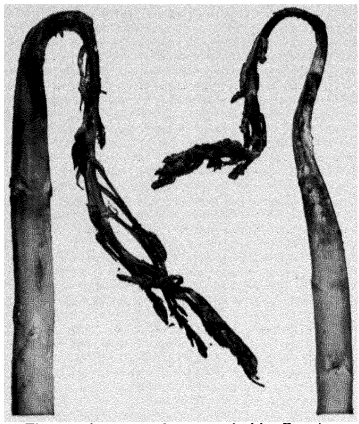


Fig. 145. Asparagus shoots attacked by *Fusarium*.

like that caused by *Pythium* but the two fungi are readily distinguishable under a microscope. If a red colouration appears in the mould when it is kept in a damp chamber one can feel sure it is *Fusarium*.

In Saxony *Fusarium* is associated with a basal rot of asparagus shoots in beds on calcareous, humus-free, sandy soil. In June–August the tops turn yellow on parts of the bed, in damp weather the stalks become dark brownish, with red pith, near the surface of the soil. In dry weather the stalk 5–6 inches down in the soil becomes soft and red. In the following autumn the attack continues on both root stock and shoots and the affected patches of soil increase in size.

Both in Denmark and in Saxony the species involved has been *Fusarium culmorum* (W. G. Sm.) Sacc., common also on cereal crops, but other species of *Fusarium* have also been found on asparagus. In Holland the danger of infection from China aster (*Callistephus*, p. 427) has been pointed out. Species of *Fusarium* may be left in the soil from previous cereal crops or may be brought into the asparagus beds on straw that remained undecayed in stable manure. Many wounds and punctures inevitably occur on the asparagus shoots through which these fungi can gain an entry.

Control. In Saxony control of the root rot has been claimed by levelling the beds as soon as cutting is over; obviously the fungus does not like dry conditions. In autumn the tops are cut out as far down as possible. Well-rotted stable manure is used with the necessary phosphoric acid and potash fertilizers added (Weise, 1939).

Disinfection of the soil has been suggested when the diseased plants are dug out, but does not seem to have been generally practised.

Watering with fungicidal solutions containing mercury salts may be tried (see p. 594), or digging in Brassicol (p. 592). Resistant varieties have not been observed.

Violet Root Rot (*Helicobasidium purpureum* Pat.)

The violet-coloured mycelium of this fungus attacks the roots and underground stem; the roots rot and the tops wither. The disease occurs mainly on old-established plants and is rather widespread in asparagus-growing districts of England, especially Worcestershire and the vale of Evesham (Moore, W. C., 1943). See further under carrot, p. 262.

Zopfia rhizophila Rabenh.

This fungus forms small black perithecia which look like dots scattered over dead brown



Fig. 146. Asparagus top deformed by night frost, photographed 12th June, 1939.

roots. In Italy it is regarded as a parasite, in France as harmless. In England it has been frequently reported on dead asparagus roots, but its ability to attack healthy roots has not been demonstrated.

Frost Damage

Where asparagus is grown in exposed situations, as on the Lammeford in Denmark, night frosts late in the season may cause considerable damage. The young plants not yet to be cut are especially exposed. The day after a hard night's frost the stems may be quite slack or their tips may be soft and bend over like a shepherd's crook. Many shoots are killed, others escape with a malformation in the form of a twisted tip or low bushy growth (Fig. 146).

Protective measures will usually be economically impracticable (see also p. 24).

Summary of Asparagus Diseases

I. Root Stock and Roots

A. Root stock spotted, more or less softened, roots often hollow and soft:		
(1) Violet-coloured mould on root stock and base of stems	Violet Root Rot	page 234
(2) Small black perithecia on dead roots	*Zopfia*	234
(3) In damp weather a coating of mould appears:		
(a) Thin white layer of mould	*Pythium*	233
(b) Whitish or pink mould	*Fusarium*	233
B. Root stock feeble, but showing no sign of decay. After-effect of severe outbreak of	Asparagus Rust	232

II. Young Shoots

A. Shoots flaccid, transparent	Frost	235
B. Dead shoot tips	After-effects of Frost	235
C. Sharp bends on shoots at or near soil level, glassy brownish spots coated with mould in wet weather:		
(1) Shoots brittle, mould white	*Pythium*	233
(2) Shoots tougher, mould whitish to pink	*Fusarium*	233

III. Older, Green Shoots

A. Twisted shoots, often with a little dead tissue on the inner surface of the curve	Frost	235
B. Dead shoot tips	Frost	235
C. Yellow spots on stems followed by cluster cups	Rust	232
D. Cinnamon-coloured pustules all over stems and shoots, followed at end of summer by black pustules	Rust	232
E. Top withers early, stalk brown at soil level, possibly with a little white or pinkish mould	*Fusarium*	233

BEAN—BROAD

Chocolate Spot (*Botrytis fabae* Sardiña, *B. cinerea*, etc.)

Chocolate spot is one of the commonest diseases of broad beans and field beans. Dark brown spots appear on the leaves and stems. They may be small, more or less circular, and scattered, in which case little harm is done, or they may spread rapidly, cover the whole leaf surface and cause decay of the stems and death of the plants. In severe outbreaks very few pods mature. Brown spots occur on the pods as well and may penetrate to the ripening seed. Typical chocolate spot may be caused by the ubiquitous mould *Botrytis cinerea*, but there is also a specialized species, *B. fabae*, peculiar to broad beans, which causes a similar disease. This fungus was found to be the principal cause of chocolate spot in England in 1946 (Moore, 1948).

Control. Chocolate spot is favoured by moisture, ill-drained soil, shade and crowded planting. Hence it is a much more disastrous disease of field beans than of isolated rows of beans in gardens. It is also encouraged by potassium deficiency or by malnutrition of any kind. Control measures therefore include well-balanced manuring and planting in open sites on well-drained soil.

Minor Leaf Spots

Cercospora fabae Fautrey causes small chocolate-coloured spots $\frac{1}{16}$ inch or so across on broad-bean leaves. Under favourable conditions they become larger with a sunken grey centre and a raised dark-brown margin. In wet weather both sides of the spot may bear a silver-grey layer of conidia (Woodward, 1932). Similar spots may be caused by the pycnidial fungus *Ascochyta fabae* Speg. This fungus may also cause light sunken spots with a broad dark-brown margin on the pods and is known to be seed-borne.

Both *Cercospora fabae* and *Ascochyta fabae* are widespread in England, but neither is usually considered important enough to warrant control measures.

Sclerotinia Disease (*Sclerotinia trifoliorum* Erikss. var. *fabae* Keay)

Broad beans, and especially field beans, are susceptible to a stem rot similar to that caused in other plants by *Sclerotinia sclerotiorum* (see p. 77). In the case of these beans, however, the cause of the trouble is usually a distinct fungus, *S. trifoliorum* var. *fabae*, very closely related to the fungus of the well-known red clover rot. Although the race commonly found on broad beans has been distinguished from *S. trifoliorum* by small technical differences it can also cause rotting of red clover, and the typical *S. trifoliorum* can cause rotting of broad beans. Both fungi also attack common vetch, garden pea, sainfoin, lettuce, and possibly other plants (Keay, 1939).

Control measures are similar to those for *S. sclerotiorum*.

BEANS—DWARF AND RUNNER

Anthracnose (*Glomerella lindemuthiana* Shear = *Colletotrichum lindemuthianum* (Sacc. & Magn.) Bri. & Cav.)

Except in the warmest and driest parts of the earth dwarf and runner beans are liable to suffer severely from attack by the anthracnose fungus, the conidial stage of which is only too well known in Denmark. It has become fairly common in England especially in cool wet summers, but the ascospore stage has only been found in America. All kinds of dwarf and runner beans may be attacked.

Symptoms and development. The disease is seed-borne and the seedlings may show brown stripes on their stems. In damp weather new spots readily arise, extending lengthwise into sunken brown stripes. On the leaves the spots generally start on the under-side of the veins, though the surrounding leaf tissue may also be killed (Figs. 147, 148). The most conspicuous symptoms are those on the pods, which are circular or elliptical sunken brown spots, up to $\frac{1}{2}$ inch across, with a darker edge (Fig. 149). From these the fungus grows into the seed, in which it remains alive until germination, and then attacks the seedling. On dark-coloured seeds it is difficult to see the

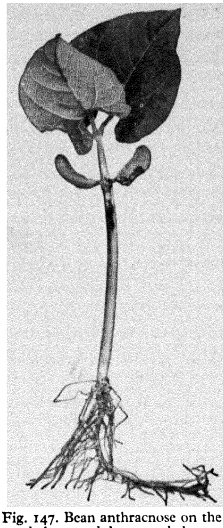


Fig. 147. Bean anthracnose on the cotyledons and hypocotyledonary stem (after C. A. Jørgensen).



Fig. 148. Stem of bean seedling attacked by anthracnose, enlarged to show conidial pustules (*S. p. F.*).

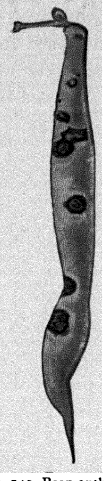


Fig. 149. Bean anthracnose on pod.

spots resulting from infection. On white-seeded varieties the larger spots are pale brown (Fig. 150) but small ones may escape notice. The fungus overwinters in the soil, especially on dead pods and other plant fragments. Plants attacked in the seedling stage or soon after may be killed outright; at later stages the damage is mainly to the pods. The fungus fructifies as minute pinkish pustules of slimy spores on the surface of the spots and these are scattered, especially by splashes of rain and by handling when the pods are being picked. Enclosed localities where the air is apt to be moist and still favour the disease. Manuring with potash and superphosphate may make the plants a little less sensitive; sulphate of ammonia should only be used on a table crop, not in seed production (Schaffnit and Böning, 1925).

Bean varieties differ greatly in susceptibility so that considerable success has been attained in developing resistant types. Resistance may be dependent on one or more inherited factors but so far these have always been dominant in hybrids. It soon became evident, however, that the fungus, too, exists in numerous races different in infective capacity (Barrus, 1918; Rands and Brotherton, 1925). Hence a bean variety may be resistant to several races of the fungus but not to others. Hence new varieties have to be exposed to infection by all known races of the anthracnose fungus before they can be described as resistant, and preferably this property should be combined with resistance to other diseases like halo blight, rust and mosaic. So successfully has this been done that some bean varieties now contain three hereditary factors for resistance and are resistant to 34 races of the anthracnose fungus. Actually they all become infected but in

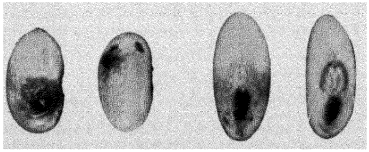


Fig. 150. Beans from a pod attacked by anthracnose (*S. p. F.*).

resistant varieties the fungus only penetrates the skin and then dies (Leach, J. G., 1923). Breeding resistant varieties has mainly been done in North America, where were produced the varieties Nova Scotia Marrow, Perry Marrow, Wells Red Kidney and White Imperial.

According to German researches none of the varieties grown there is resistant to all the 14 races of the fungus found in Germany but some have been found to show considerable resistance in Denmark. Field observations suggest that runner beans are less exposed to infection than dwarf beans. The following varieties seem to be resistant: Holstensk, Hinrichs Champion, Saxa, Imperator, Phenomenon, and of kidney beans Alabaster and Nordstjerne. The resistant property of scarlet runners is transmitted to varieties with " scarlet runner blood " such as Sutton's Prizewinner. The American varieties developed to resist the fungus races present in North America cannot be expected to prove resistant to all the races of the fungus found in Europe, but they will surely form a valuable addition to the stocks from which future European beans can be bred.

Control. The importance of growing resistant varieties and of breeding fresh ones has already been pointed out. All remains of bean plants should be removed from the plots and burnt. As infection is mainly seed-borne it is important to sow healthy seed. In seed production the unspotted pods should be selected. Hand picking the seed to remove spotted ones is also useful but lightly infected ones are likely to be missed. Attention should particularly be directed to beans which bear a spot at the scar (Jørgensen, C. A., 1934). In North America seed is largely grown in parts of Colorado, Idaho and California, with dry, warm climates which do not favour the disease. The fungus remains alive at least two years in infected seed but the percentage of infection may be reduced in old seed.

Disinfection of the seed by soaking for one hour in $\frac{1}{4}\%$ formalin or $\frac{1}{4}$–$\frac{1}{2}\%$ Uspulun solution is effective when the infection is not too deep-seated. Such wet treatment may aid germination or check it, depending on local conditions and the quality of the seed. The effect should be tested on a small sample before a large consignment is treated. Disinfectant dusts may aid germination but have little effect on the disease. Hot-air treatment damages the seed.

Spread of infection in the seedling stage can sometimes be avoided by protecting the plants from rain. This is not often practicable but its efficacy shows how the fungus is largely spread. One should avoid hoeing and picking while the plants are wet with dew. Copper lime dusts have proved injurious to the beans. Bordeaux and Burgundy sprays may cause a temporary check to growth and do little to protect against the fungus. Strong sprays on the pods have been tried, in an endeavour to produce healthy seed, but without success (Jørgensen, C. A., 1934).

Beans that looked healthy when picked may become conspicuously spotted during storage. At summer temperatures the incubation period is about five days. The disease may also spread to unwounded pods. Picking in dry weather, protection from sun and rain, removal of all spotted and wounded pods and storage at temperatures from 38–44° F. will help to keep consignments free from anthracnose and other pod diseases such as *Sclerotinia* rot, grey mould and bacterial spots.

Fusarium Wilt (*Fusarium vasinfectum* Atk. var. *lutulatum* (Sherb.) Wr.)

This disease was first reported in England from Worcestershire by Ogilvie in 1931. The chief symptom is a rolling inwards of the leaf edges, followed by wilting of part or the whole of the plant. The fungus occurs only in the vascular strands which are stained dark brown as in *Verticillium* wilt, especially at the base of the plant. It is apparently not seed-borne and infection presumably comes from the soil, through wounds made by soil insects or by the mechanical splitting of the stem base by secondary roots. The optimum temperature for its growth is 79–82° F.

Control measures include an adequate rotation of crops, destruction of infected plants

when the crop is cleared and cultivation of resistant varities. Giant Painted Lady, Czar and Sutton's Scarlet have shown some resistance to *Fusarium* wilt (Davies, D. L. G., 1939; 1942).

Foot Rot (*Fusarium solani* (Mart.) App. & Wr., var. *martii* (App. & Wr.) Wr.)

Foot rot is caused by a soil-borne fungus prevalent in the Evesham district of Worcestershire. It attacks the roots and stem bases of dwarf beans, turning them reddish brown. The leaves turn yellow and only the first-formed pods swell properly. Foot rot causes most loss in dry seasons, when it may reduce the yield by one half. Strong-growing varieties like Flageolet are somewhat resistant as they put out fresh roots from the lower part of the stem to replace the diseased ones.

Control measures are similar to those for *Fusarium* wilt (Ogilvie, L., 1939).

The same fungus has been found to cause a similar disease in French beans grown under glass (White, H. L., 1941). In this case sowing in sterilized soil or peat is recommended, followed by protecting the stem base with the rim of a bottomless pot when the seedlings are planted out in the house.

Rust (*Uromyces phaseolorum* de Bary)

This is found on dwarf and runner beans all over the world. In the last century it was widespread in Denmark and was prevalent in some places in 1917 but has seldom been seen since because resistant varieties are grown. Scarlet runners are resistant to all but one physiological race (Straib, 1943). The cluster cup stage of this rust is white (Fig. 151). This is usually followed by cinnamon-coloured pustules of uredospores on both sides of the leaf (Fig. 152) and finally by blackish-brown pustules of teleutospores. Bean rust is only occasionally reported in England.

Sprays and dusts give no control of rust and if it breaks out the diseased plants should be burnt or composted and replaced by a resistant variety.

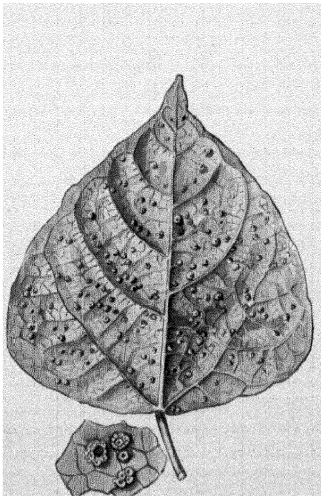


Fig. 151. Bean rust, aecidial state, lens view below.

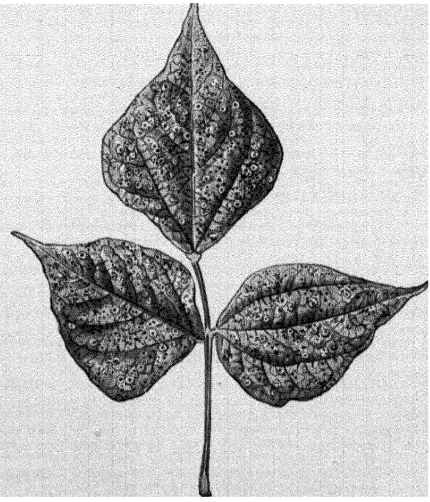


Fig. 152. Bean rust, teleutospore state.

Red Nose (*Pleospora herbarum* (Fr.) Rabenh. and damaged seed)

When beans are germinated in the seed-testing laboratory a slight pink discolouration is sometimes noticed at the micropyle or germ pore. This has been called " red nose " and is due to infection with the common fungus *Pleospora herbarum* (Fr.) Rabenh., which has as its conidial stage the mould *Stemphylium botryosum* Wallr. This fungus cannot cause disease of the growing crop but the infected seeds often decay or give sickly seedlings that die early. If they survive this stage they grow into normal healthy plants (Brinkman, A., 1931). Treatment with one of the proprietary organomercury seed dressings often gives improved germination (Doyer, L. C., 1948).

Beans harvested under unfavourable conditions, or otherwise of poor quality, may be attacked during germination by bacteria and moulds like *Penicillium*, which reduce the percentage germination. Treatment with an organo-mercury seed dressing or with red copper oxide ($\frac{1}{2}$–$\frac{3}{4}$ oz. per pound) may give a definite improvement in germination.

Some beans germinate and develop their cotyledons (seed leaves) normally but the terminal bud or plumule is seen to be lacking. An axillary bud of one of the cotyledons may grow out to replace it but only gives a poor plant. The cause may be bacterial infection or damage at threshing, which has cracked the embryo just above the attachment of the cotyledons. Microscopic examination will decide which cause is involved.

Finally, beans may germinate badly (and cook badly) after a very hot, dry autumn or after storing in a dry warm place which hardens the skin of the seed. Storage in a damp warm place also brings about certain enzyme reactions within the bean and leads to formation of a " hard kernel," which also reduces germination and cooking quality.

Halo Blight (*Pseudomonas phaseolicola* (Burkh.) Dowson = *Bacterium medicaginis* (Sack.) E. F. Sm., var. *phaseolicola* (Burkh.) G. K. K. Link & Hull)

A number of bacterial diseases are known to affect dwarf and runner beans but their symptoms are usually somewhat similar so that they can only be distinguished by bacteriological investigations. In England the disease is usually attributed to *Pseudomonas phaseolicola*. This is seed-borne, and infected seed gives rise to stunted, spotted or yellow deformed plants which usually soon die. From them the bacteria are spread, probably largely by rain splashes, to healthy adjacent plants. On the leaves small, irregular, watersoaked spots appear, that are at first pale, but later turn brown with a transparent halo-like margin. Similar spots may appear on stems and petioles and from them small drops of bacterial slime may ooze out. When the pods appear they too become spotted and sometimes deformed, or even killed (Fig. 153).



Fig. 153. Halo blight on bean pod.

Seeds from infected pods are likely also to be infected with the bacteria. Sometimes they are spotted, wrinkled, or shrivelled, but they may bear no obvious mark of infection and yet give rise to an infected seedling. This disease is common in England and in North America. It was found in Denmark in 1932 and subsequently also in Holland, Switzerland and Germany. Of dwarf beans the varieties Canadian Wonder and Early Prolific are very susceptible to it, whereas Black Windsor, The Prince and Abundant are resistant to stem and leaf infection. Runner beans may also be infected.

Another bacterium, *Phytomonas viridiflava* Burkh., has been isolated from a leaf spot of Scarlet Runner beans in Kent and from bean pods in Switzerland (Burkholder, W. H., 1930). In 1931 E. J. Petersen isolated a race (var. *concentrica*) of this organism from diseased beans in Denmark. These had on their leaves small irregular spots, first pale, then brown with a transparent margin (Fig. 154). These spots became so numerous that the leaves became brown and ragged. Spots also appeared

on the stems, and petioles and plants infected while still young cast their leaves and died. On older plants the pods were also spotted.

A third bacterial disease of beans, caused by *Pseudomonas phaseoli* E. F. Sm., is widespread in North America and has been found in France, Switzerland, Bermuda and the Philippine Islands. It too is seed-borne and infected seedlings may be killed by it as they emerge above ground. On older plants it forms minute watersoaked spots or pale-green

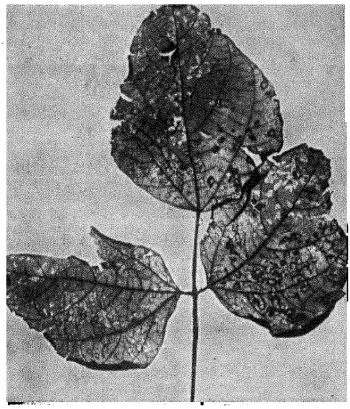


Fig. 154. Bacterial disease of bean leaves, caused by *Phytomonas viridiflava* var. *concentrica* (*S. p. F.*).

wilted areas which enlarge, dry out and turn brown. They usually have a yellow border. Reddish streaks appear on the stems, or reddish lesions may girdle the stem at the level of the cotyledons, or one of the lower nodes. The stem easily breaks at these points. On the pods there are dark-green, watersoaked spots that become dry, brick red and sunken (Burkholder, 1930). *Pseudomonas syringae* van Hall has been found causing a leaf and pod spot of beans in Australia and has been isolated from infected dwarf bean seeds in England (Moore, W. C., 1943). In North America there is also a vascular wilt disease of beans caused by *Bacterium flaccumfaciens* Hedges. Burkholder isolated this organism from seed obtained from France and Germany so it evidently occurs also in Europe.

Control. Where halo blight has appeared the bean straw should be burnt; the ground should be regarded as infected the following year and not used for beans. When selecting seed only that from healthy unspotted pods should be taken. In the United States seed is to some extent supplied from districts in California that are free from infection. Soaking seed for 20 minutes in corrosive sublimate solution 1: 1000 may reduce the number of infected seedlings. It is best not to soak runner or dwarf bean seed in water before sowing as this may spread infection through the sample. Prolonged storage of the seeds or exposing them to dry heat seem of little avail. The attack may be worst in early sowings. In crops grown for seed, spraying with Bordeaux mixture gives more healthy pods (Hähne, 1942).

The best means of preventing heavy losses in commercial bean production seems to be to grow the most resistant varieties.

Mosaic

Ordinary bean mosaic (*Phaseolus* Virus 1) is known all the world over, in Russia as long ago as 1899. Though it is common, losses from it are usually slight but in Denmark severe attacks have been noted on French beans (crinkled leaves and deformed pods).

Symptoms vary greatly as different bean varieties react very differently. The pale-green patches may follow the veins or lie between them. The leaf may be more or less curled and in the worst cases becomes thread-like. Often the green colour remains in large blister-like islands on a pale yellow-green leaf. The plant may be stunted and abnormally bushy.

The pale-coloured parts of the leaves are abnormally thin and very hairy. Some forms of inherited variegation, sun damage and red-spider injury may simulate mosaic but can be distinguished from it microscopically (Burkholder, W. H., and Muller, A. S., 1926).

The virus is transmitted by aphides of various kinds and also in the seed, especially

in that from the first-formed pods. Seed from diseased pods may give 50% mosaic-infected plants. Seed 32 years old has still given mosaic-infected seedlings. The virus can be transmitted to broad beans and lupins but its relationship to the viruses causing mosaic diseases in peas, sweet peas, clover and melilot is uncertain.

Control. Treatment of the seed with wet or dry heat kills the seed before destroying the virus.

Some varieties are very susceptible but both in Japan and U.S.A. resistant varieties of commercial quality have been developed. A small white-seeded bean, Robust, bred at Michigan Experiment Station, has won great popularity on this account.

In another group of bean varieties, descended from Corbett Refugee, a high degree of resistance to bean mosaic in the field is due to hypersensitivity to the virus (cf. top necrosis in potato, p. 338). In these varieties a few plants exposed to virus infection develop a systemic necrotic disease known as black root and are killed, but in most spread of the virus is prevented by death of the tissues round the place of entry (Grogan, R. G., and Walker, J. C., 1948).

Yellow bean mosaic (*Phaseolus* Virus 2) is a conspicuous virulent disease not transmitted by the seed. The variety Robust is not resistant to it. So far this disease is known only in North America, where White Melilot is an important host in which the virus overwinters. The virus is spread by aphides and can infect also broad bean, soya bean, alsike and other clovers (Grogan, R. G., and Walker, J. C., 1948).

Summary of Dwarf and Runner Bean Diseases

I. Roots

		page
A. Roots and stem base brown and stinking, plant dies	*Bacterium carotovorum*	257
B. Reddish-brown roots but no soft smelly rot	*Fusarium* Foot Rot	239

II. Stem

A. In seedlings:

(1) Shoot twisted and deformed	Halo Blight	240
(2) Brown streaks on the stem	Anthracnose	236
(3) Stem covered with grey hairy mould	Grey Mould	59
(4) Growing point dead	Threshing Damage, Manganese Deficiency, Halo Blight or Other Bacteriosis.....	240

B. In larger plants:

(1) Grey hairy mould	Grey Mould	59
(2) Woolly white mycelium with large white or black sclerotia	*Sclerotinia* Rot	77
(3) Brown, dry, sunken streaks	Anthracnose	236
(4) Brown spots, often exuding slimy white drops ..	Halo Blight	240
(5) Reddish-brown canker at base of plant	*Fusarium* Foot Rot	239
(6) Wilting of plant, brown stain in vascular strand	*Fusarium* Wilt	238

		page
III. Leaves		
A. Light spots or markings:		
(1) Pale spots between the veins	Manganese Deficiency	39
(2) Leaves curl back and turn yellowish brown from the margin inwards between the veins	Potassium Deficiency	42
(3) Leaves blotched with yellow or yellow with dark-green blisters	Mosaic	241
(4) Leaves turn yellow and growth stops	*Fusarium* Foot Rot	239
(5) Small slightly swollen spots bearing cluster cups	Bean Rust	239
B. Dark markings or spots:		
(1) Brown lines along the main veins	Anthracnose	236
(2) Tiny powdery brown pustules	Bean Rust	239
(3) Circular brown spots, pinkish spore pustules visible under lens	Anthracnose	236
(4) Circular brown spots, dark dot-like pycnidia seen under lens	*Ascochyta phaseolorum* cf.	236
(5) Small watersoaked spots, becoming larger and brown with a translucent edge, leaves often become torn	Halo Blight	240
C. Leaves wilt suddenly and plant dies	*Fusarium* Wilt	238
IV. Pods		
A. Mouldy decay:		
(1) Mould grey or hairy	Grey Mould	59
(2) Mould woolly and white with black or white sclerotia	*Sclerotinia* Rot	77
B. Dry, brown spots:		
(1) Spots very small	Halo Blight (or Similar Disease)	240
(2) Spots larger:		
(a) Spots more or less round with dark edges, pinkish conidial pustules visible under lens	Anthracnose	236
(b) No dark edges, dark pycnidia seen under lens	*Ascochyta phaseolorum* cf.	236
C. Greasy-looking or translucent spots, often turning dark green or red-brown, often oozing drops of slime	Halo Blight	240
D. Small warts or hairs on the surface	Intumescence	18
V. Seeds		
A. Brownish spots	Anthracnose	236
B. Small pale blisters on skin	Halo Blight	240
C. Pinkish stain round micropyle (germ pore)	Red Nose	240
D. Plumule damaged, germination poor	Retarded Germination	240

BEET

Rust (*Uromyces betae* (Pers.) Lév.)

Beet rust is one of those which has all its stages on the same host plant. The orange-coloured cluster cups (æcidia) appear in May on the under-surface of leaves of young plants, and occasionally on the leaf stalks. This stage is seldom seen on seedlings but more often on the young leaves produced by overwintered roots kept for seed production. Spores from the cluster cups spread to young beetroot plants, on which successive generations of uredospores appear as rust-coloured pustules all through the summer. Towards the close

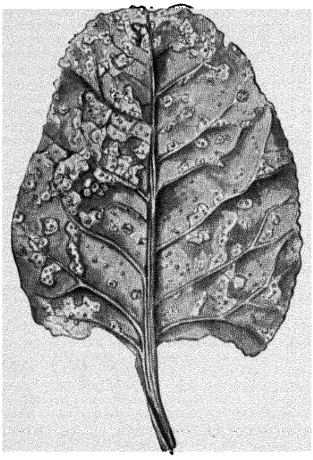


Fig. 155. Rust pustules on beet leaf.

of summer they become replaced by pustules of the dark-brown teleutospores (Fig. 155).

Beet rust is most prevalent on mangolds and sugar-beet, but occurs also on garden beet, spinach beet and the wild sea beet (*Beta maritima*).

The most important control measure is to keep seed crops as far away as possible from fresh sowings. It may also be as well to inspect seed crops at regular and frequent intervals in spring as long as the cluster cups appear and remove the leaves that bear them. They should at once be put in a pail of lime water or other disinfectant fluid to prevent spores from being spread about the crop.

Mother roots for seed crops should, if possible, be chosen from plants free from rust and stripped of leaf stalks which might bear the fungus.

Leaf Spots

Ramularia beticola Fautr. & Lamb. and *Cercospora beticola* Sacc. cause very similar circular leaf spots with a whitish centre and dark-purple margin on beetroot. On spinach beet the spots are pale with brown edges (Fig. 156). Usually they are less than $\frac{1}{4}$ inch across, but may be very numerous.

When necessary, spraying or dusting with a copper fungicide should be practised.

Hughes (1945) has described a leaf spot of mangolds caused by *Stemphylium botryosum* Wallr (see red nose, p. 240).

Black-leg

The young seedlings turn brown at the soil level and shrivel up. Black-leg is worst on wet and sour soils which should, therefore,

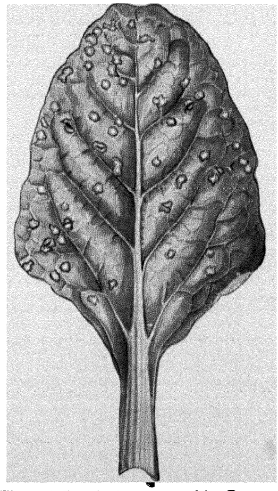


Fig. 156. Beet leaf spot caused by *Ramularia beticola*.

be improved. Various fungi may play a part in the disease, including species of *Pythium* in the soil and *Phoma betae* Frank which is seed-borne. Hence treatment of the seed with the standard seed disinfectants may have a beneficial effect (see also damping off, p. 68).

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

As a rule this only affects beetroots that have been lifted and stored. They become overgrown by a dense white mycelium in which large sclerotia, at first white and then black, are embedded (see *Sclerotinia* rot, p. 77).

Scab (*Actinomyces* spp.)

Beetroots often have round spots or low warts with an uneven, somewhat scaly upper surface. These are caused by infection by a number of *Actinomyces*, including those which cause common scab of potato (see p. 322). The disease is worst on soils rich in lime. In Denmark the varieties Spangsbjerg and Crosby were less attacked than Egyptian flat round and Detroit.

Beet Mosaic (*Beta* Virus 2)

Mosaic is very general in seed crops of mangold and sugar-beet, but seems less prevalent in garden beet, where it is only found in individual plants with conspicuously mottled leaves. In our infection experiments red beet proved not very susceptible and spinach beet even less so. Hence attacks are only to be feared when these crops are grown in the vicinity of sugar-beet or mangold seed crops. Beet mosaic will, however, attack spinach (see p. 357) and hence overwintered spinach may be a source of infection from which the virus may be spread to beet by aphides. In our experiments the common goosefoot was not infected but wild sea beet did develop mosaic. Recently, however, beet mosaic has been shown to infect several common weeds of the beet family, viz. *Chenopodium album* (fat hens, goosefoot), *C. murale*, *Atriplex bracteosa*, *A. rosea* and *A. patula* var. *hastata* (orache) (Severin and Drake, 1947).

Heart Rot, Dry Rot and Canker (Boron Deficiency)

The innermost leaves turn black and die and gradually all the leaves on the plant may wither. Later new clusters of healthy leaves may appear on the crown outside the dead heart. On the root a slow, dry brown decay starts at the shoulder and gradually penetrates the tissues. In garden beet black spots may appear in the flesh.

Heart rot and dry rot are much better known in sugar-beet and mangolds than in red beet. In the former they are known to be associated with limed soils and with dry years. Of late years excellent control has been obtained by manuring with borax at the rate of about 20 lb. per acre.

On soils where previous experience has shown that beet is liable to suffer from trouble of this kind borax should be applied in spring. Care must be taken not to exceed the recommended rate or the plants will be poisoned. Not more than 1 oz. to 12 square yards should be given. Watering with very dilute borax or boric acid solutions may check an outbreak that is just beginning.

Beet Yellows (*Beta* Virus 4)

Yellows is a common disease in sugar-beet and mangold crops where it affects all the plants in large patches or over whole fields. The virus may be spread by aphides to garden red beet, spinach beet and spinach (p. 357) and to goosefoot (Roland, 1939a).

Infected spinach beet develops stiff yellow leaves. Leaves of infected red beet are also stiff, but the yellow colouration tends to be masked by the red pigment in the leaves. Leaves of the diseased plants are gorged with starch, and are often prematurely attacked by the black mould (*Alternaria*) which normally only appears on the dying leaves in autumn.

The disease is not carried by the seed and there is no infection from the soil. The virus overwinters in beet plants, especially in seed crops, and is spread from them by aphides. As far as garden beet and spinach are concerned it seems to be prevalent only in the vicinity of sugar-beet crops.

CABBAGE, CAULIFLOWER, etc.

Club Root, or Finger and Toe (*Plasmodiophora brassicae* Wor.)

Even in the seventeenth century this disease was known as "the devil" on cabbages in Spain; it was found in England in 1736, but not until club root devastated the cabbage fields around Leningrad (St. Petersburg) in 1870 and was fully described by Woronin (1878) was its prevalence in Europe fully realized. It was said to have devastated gardens at Viborg for several years when Rostrup recognized it there and at Randers in 1884. The following year it was found at Hadsund and Copenhagen and was prevalent in turnip and swedes on dry soils deficient in lime.

As late as 1905 Amager and the southern islands of Denmark were thought to be free from it, but by 1909 experiments for its control had been started in Amager! The disease was then widespread in Jylland and North Zeeland and occurred at various places in Fyn. Increased seed production and swede cultivation led to further spread of club root in the succeeding years. The heavy liming and land drainage of more recent years, coupled with earlier sowing and the development of valuable resistant varieties of swede, should make the trouble less prevalent in agricultural crops and so reduce the danger of contamination in manure purchased for gardens and nurseries.

Host plants. The club root fungus attacks only plants of the family *Cruciferae*, the cabbage family. A great many of these have been tested systematically (Cunningham, 1914; Jamalainen, 1936), and only 14 or 15 kinds have been found not to be attacked. Similar-looking diseases on plants belonging to other families have never proved to be club root when investigated critically. Among susceptible plants the following may be mentioned:

ORNAMENTALS

Alyssum	*Hesperis*
Arabis	*Iberis* (candytuft)
Aubretia	*Lunaria* (honesty)
Cheiranthus (wallflower)	*Matthiola* (stock)
Erysimum	

CROPS

Cabbage and all its allies	Rape
Mustard	Seakale
Radish	Swede
	Turnip

WEEDS

Sinapis arvensis (charlock)	*Alliaria officinalis* (garlic mustard)
Raphanus raphanistrum (runch)	*Thlaspi arvense* (pennycress)
Cardamine pratensis (cuckoo flower)	*Lepidium campestre* (field pepperwort)
Capsella bursapastoris (shepherd's purse)	*Rorippa* spp.

Of the *Brassica* crops kale and marrow-stemmed kale are least attacked, and after them Brussels sprouts (Hercules is said to be particularly resistant). All kinds of cabbage are very susceptible and cauliflower most of all. Chinese cabbage, used as a salad, is also attacked; so are radishes, though seldom to any extent. Cress is said to be resistant as also are some strains of kohlrabi.

Various suggestions that there may be different physiological races of the fungus specialized on different hosts are not confirmed by our own observations. The relative resistance of different species and varieties seems to remain fairly constant.

Symptoms and development. In cabbage and its allies the spindle-shaped irregular swellings on the roots are the most obvious symptoms (Fig. 157), and are often accompanied by poor development of the head and, in dry weather, by flagging and a bluish tint to the foliage. In swedes, turnips and radishes, where the stem swells to form a storage of reserve foodstuff, this is also attacked, and becomes covered with tumours and cracks both above and below soil level. In the swelling the cells of the flesh are enormously enlarged and developed at the expense of the woody tissue so that the tumours easily rot, the decay being completed by bacterial activity.

The galls caused on roots by the cabbage gall weevil can easily be distinguished by their more spherical shape and by always having a

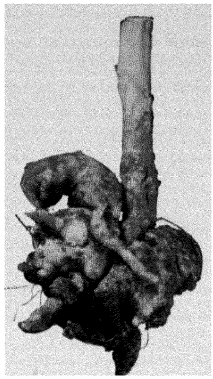


Fig. 157. Club root of cauliflower.

cavity at the centre, containing the larva. The so-called hybridization nodules on swede roots are also more round, have a more wrinkled surface and never become very large. True club root can always be recognized under the microscope by the gigantic malformed cells of the host. These cells contain innumerable minute spores of the fungus which are liberated into the soil when the swellings decay. They may remain alive in soil for at least 5–6 years. Living roots of cruciferous plants stimulate the spores to germinate and from each emerges a swarm spore, a naked speck of protoplasm able to creep and swim through the soil moisture to the root. It may penetrate the latter through wounds or cracks, as where side roots have emerged (Larson, 1934), but often swarm spores perforate the wall of root hairs and grow and divide within the hair cell. From the hair the fungus protoplasm penetrates deeper into the root, following especially the cells of the medullary rays between the vascular strands. These thin-walled cells are stimulated to enormous development and cause distortion and disruption of the whole tissue. Within the giant cells many swarm spores unite to form a large mass of fungus protoplasm (plasmodium) which ultimately breaks up into hundreds of tiny resting spores, ready to be set free in the soil by decay of the root.

Conditions for infection. Infection can take place at temperatures between 43° and 81° F., but from 64–77° F. is the most favourable temperature (Wellmann, 1930). Ledingham (1944) obtained no infection at 53–55° F., slight at 56–58° F. and moderate infection even at 92° F. in moist soil. This is why late sowings generally suffer much more than early sowings made while the soil is cold.

Soil reaction plays a very important part; pH 5.8 is most favourable for spore germination. Swarm spores can emerge from the resting spores at pH 7.5, but are then sluggish. When the soil is packed with spores, however, infection may occur even at pH 8.3 (Chupp, 1928; Lindfors, 1924). Similarly, in liming experiments in Denmark it has been found that club root infection is distinctly diminished by increasing dressings of lime or marl, but that severe infestations cannot be stopped by liming alone. Sometimes even slight differences in soil reaction have an effect and nitrochalk gives a healthy crop where sulphate of ammonia gives a diseased one. Sodium, magnesium and barium salts that raise the pH value in the same way as lime have a corresponding effect on the fungus (Naumoff, 1927; Osterwalder, 1929).

In an experiment at Amager cabbage was grown in 1909, then crops not susceptible to club root, and cabbage again in 1913. Amongst other things the results showed that lime has some effect in the year it is applied, but much more in succeeding years.

APPLIED 12 . V . 1909	% DISEASE IN CABBAGE PLANTS	
	1909	1913
No lime	21.1	17.0
Agricultural lime ..	13.6	2.0
Quicklime.....	13.0	0.3

Soil moisture is essential for club root infection; if the soil contains only 50% of the water it is able to hold, cabbage can still grow in it, but the swarm spores are unable to move; as the water content rises above this level so does the percentage infection rise in proportion (Monteith, 1924). High soil moisture

during winter and heavy spring rains lead to virulent outbreaks. Summer droughts make the plants flag conspicuously so that they are weakened, but infection by the fungus does not increase. Plants from seed sown in August may escape infection in the autumn, but in the following spring, when new roots are produced, these are liable to be attacked. Heavy summer rains may speed up rotting of roots that are already attacked. Club root does not spread in stored produce but if conditions are moist the diseased tissues quickly decay. The sites of turnip or swede pits should never be used for cultivation of a susceptible crop.

The type of soil is not very important, except that well-drained soils rich in lime are least prone to club root.

Spread of infection. Soil on which a crop infected with club root has been grown may be regarded as infected for at least 5–6 years. If a cruciferous crop is taken before this time has elapsed, or if many cruciferous groundkeepers and weeds occur, the period of soil infection is correspondingly extended. The sale of soil to gardens, where the purchaser did not know it to be contaminated with club root, has led to a lawsuit in Denmark, judgment being given for the purchaser, so that the seller was obliged to make good the damaged crop.

Soil is easily spilt in transport and infection may thus be spread to other areas. Infected material may also be carried by tools, on horses' hoofs, tractor treads and the boots of workers. It seems that it may be also spread to hitherto uninfected areas by particles of soil blown by the wind. Infection may also be water-borne, as when diseased turnips have been thrown into a pit which overflowed as a result of heavy rains.

Infection in farmyard manure is a very serious problem. Observations indicate that the spores may pass through the intestines of ruminants in a living condition, but this is probably not of practical importance. The damage is done by contamination of stable manure with waste infected fodder such as turnips. The sediment of liquid manure may also be infectious. Hence bought stable manure and kitchen refuse are to be mistrusted, but on the whole consistent use of heavy dressings of non-infected stable manure seems to lead to less club root (Reinh. Kristensen, 1941).

Cabbage plants frequently become infected in the seed bed, and are sold when bearing only a slight swelling easily overlooked. These are a frequent source of infection in private gardens. The seed itself cannot be infected, but infection may be present in soil particles in a seed sample.

Control. In gardens and nurseries the most important thing is to avoid introducing infection. Stable manure should only be bought from farms where the animals are not fed on diseased turnips—summer stable manure is least likely to be contaminated. Bought cabbage plants should be very carefully inspected for suspicious swellings on the roots and should be planted in non-infected soil. Soil disinfection may be recommended for benches, seed beds and other places where cabbages and their allies are sown, as a precaution against club root and damping off and to destroy weed seeds. Baking or steaming may be used or the soil may be well soaked with a suitable disinfectant solution (see p. 595). Heavy liming may be resorted to, to kill club root spores, but it may lead to manganese deficiency or other nutritional disturbances. Neither copper salts, sulphate of iron, potassium permanganate, nor carbon disulphide are effective against club root. But dipping plants in clay gruel to which $\frac{1}{4}$ to $\frac{1}{2}$ oz. Danish tillantin has been added per gallon may be useful. Similarly, watering with corrosive sublimate to control cabbage root fly may give considerable control of club root as well. Preston (1944) has shown that calomel paste may be equally effective. Soil disinfection with proprietary preparations like Brassicol and Brassisan has given promising results in England (Smieton, 1939) and also with us. Use of nitrochalk has sometimes been helpful.

Draining very wet land is very important in fighting club root; next, on contaminated soil, as much agricultural lime or marl should be applied as the soil can stand. Cabbages and all other cruciferous crops should not be grown for 5–6 years or more, and the land

should be kept clear of charlock, shepherd's purse and other cruciferous weeds for that period or longer. Early sowings are likely to be least infected.

Cabbage stumps, unused turnips and similar rubbish should be collected into heaps and burnt as soon as dry, so as to destroy the spores remaining in them.

Canker (*Phoma lingam* (Tode) Desm.)

Symptoms. *Phoma lingam* may attack seedlings, half-grown plants, cabbage heads in storage, seed-bearing plants and the seed itself.

It is one of the causes of damping off in cabbage, broccoli, and turnip seedlings. The plants rot at soil level, and when the disease was caused by *P. lingam* small dark pycnidia appear on them, out of which, under moist conditions, ooze amethyst-coloured drops containing the spores (Fig. 158). *P. lingam* infection at this stage comes from infected seed.



Fig. 158. Cabbage canker, caused by *Phoma lingam* (after Olaf Nielsen).

These seedlings attacked by the primary seed-borne phase of the disease almost always die, but the disease spreads from them to other plants in the seed-bed. These when planted out develop the canker phase. In this the stem is usually invaded just below soil level. It turns black and decays so that the plant becomes loose in the soil and may eventually fall over (Fig. 159).

Secondary roots may be produced from the

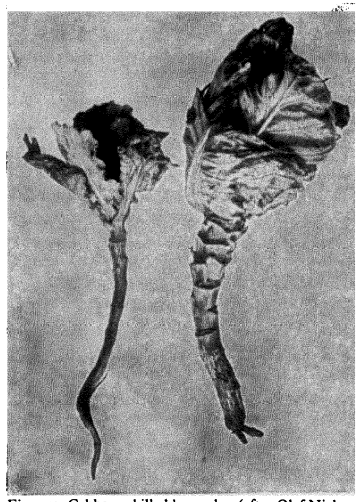


Fig. 159. Cabbages killed by canker (after Olaf Nielsen).

stem above the canker and keep the plant alive for some time. The leaves often take on a blue or purplish hue, but similar colouration may result from other injuries to the root system as by larvae of the cabbage fly. Pycnidia form on the rotting tissue or at the edge of the canker next to the healthy stem (Fig. 160).

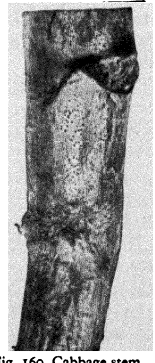


Fig. 160. Cabbage stem showing pycnidia of *Phoma lingam* (after Olaf Nielsen).

In mild or late attacks the disease may progress further in cabbage heads stored for the winter. The fungus grows up through the stump into the leaves, which turn black from the base upwards and outwards. They become loosened from the stem so as to produce a curious crater-like hollow round it. Ultimately pycnidia appear in the diseased tissue.

If there are a few infected plants in a seed crop the disease may easily spread considerably on the ripening pods. *Phoma lingam* infection is often confused with " dark spot " caused by *Alternaria brassicae* and *A. oleracea*, but it is of great importance because it leads to production of infected seed. In a seeding crop numerous dark spots appear on leaves, stems and pods and on these the pycnidia are gradually produced. From the spots on the pods the fungus penetrates the seeds where it occurs as resting mycelium between the embryo and the seed coat. Thus infection of the seed takes place between flowering and seed ripening, but the seedling becomes infected during germination.

It is not possible to tell whether a seed sample is infected or not merely by inspecting it, though some infected seeds may be lighter, greyish and somewhat shrivelled. These would float if the sample were shaken up in water. If a germination test is made, however, the diseased seeds can be detected by the pycnidia which appear on them and on the seedlings grown from them. Such a test takes from 3 to 4 weeks.

Cause. The same fungus causes a disease of swedes called dry rot. On that host it has been named *Phoma napobrassicae* Rostr. Numerous other names have been given to it, such as *P. oleracea* Sacc., *P. brassicae* Thüm, *P. siliquastrum* Desm. and *Aposphaeria brassicae* Thüm., but they are all superseded by the oldest, viz. *Phoma lingam* (Tode) Desm.

Importance. *Phoma lingam* can attack all kinds of cabbage, savoy, brussels sprouts, cauliflower, broccoli, kale, turnip, swede, wallflower, stock and a number of cruciferous weeds. In the summer of 1927 it was found causing canker on cabbage from Amager and in 1928 causing damping off of cauliflower seedlings. There have been several subsequent outbreaks in Denmark but the disease is not considered plentiful there and is of little importance in cabbage cultivation. In North Holland, however, it has become very serious and it causes much loss to broccoli growers in England especially in Yorkshire and the West Midlands. It also causes enormous losses in some seasons by attacking the swede crop.

In Denmark it is most important as a disease of the seed crop because of the important export trade in cabbage seed, for example, to the United States. Of 80 seed samples of cabbage, red cabbage, cauliflower and brussels sprouts tested, about a quarter were found to be infected, the amount of infection being very low, from 1 to 3.1 per thousand seeds (Olaf Nielsen, 1932). If the seed is sown under conditions favourable to the disease even this minute quantity of infection may lead to a serious outbreak. In Holland samples have been found where as much as 60% of the seed was infected.

Control. Seed suspected of being infected should be steeped in water at 122° F. for 30 minutes (see p. 576). Soaking for the same period in 1: 1000 corrosive sublimate solution does not give complete control as it does not affect mycelium already inside the seed coat. A good rotation of crops is important; one should not grow any cruciferous crop, especially a crop for seed production, on land that has borne diseased cabbage, broccoli, swedes or the like within the past 3 or 4 years. It is also important to avoid using for such crops any farmyard manure containing remains of swedes, turnips or kale.

Ring Spot (*Mycosphaerella brassicicola* (Duby) Oudem = *Phyllosticta brassicicola* McAlp.)

This disease was very serious in Denmark in 1917–20, but in subsequent years declined until it became almost unknown. Of late years there have been a few outbreaks but none of great importance. It is always present in the West and North of England, Wales and Scotland, but is seldom seen in the East and

South-east. It is well known in North America and Australia. Ring spot occurs mainly on cabbage, cauliflower, broccoli and brussels sprouts.

Symptoms. Almost all above-ground parts of the plant may be affected. On the leaves the spots are usually numerous, round, and light brown to grey-brown with a narrow olive-green border. They bear innumerable small pycnidia or perithecia, usually arranged in concentric rings, whence the name ring spot (Fig. 161). The fructifications may be so

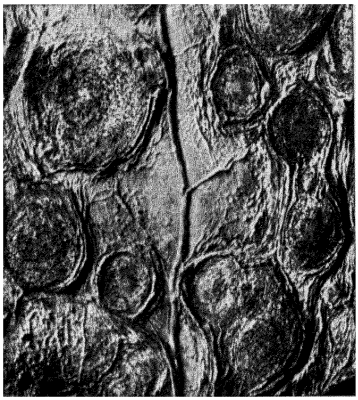


Fig. 161. Cabbage ring spot (slightly enlarged) (*S. p. F.*).

numerous that the whole spot looks black. In severe attacks the outer leaves of cabbage turn yellow and fall. Spots on the outer leaves of a cabbage head may be so numerous that from a short distance it resembles a red cabbage. Similar spots occur on stems and pods of plants grown for seed. Whereas pods spotted by *Alternaria* tend to open prematurely, those attacked by ring spot are apt to remain closed even when the seed is ripe.

Cause. *Phoma lingam*, the cause of cabbage canker, may be responsible for somewhat similar spots, but there is no connection between it and the true ring spot fungus. *P. lingam* spots bear far fewer pycnidia. The ring spot perithecia form during autumn and ripen about December. Recent investigations in America have shown that the spores produced by the pycnidia are not conidia and are unable to infect leaves or to grow in artificial culture. Presumably they are male cells or spermatia, analogous to those produced by rust fungi, but their precise function remains unknown (Snyder, 1946). Ascospore germination is slow at 36° F., but rapid at temperatures between 60° and 72° F. At the latter temperature infection may take place in about a day, but there is an incubation period of 2 to 3 weeks before the spots are clearly developed. The fungus is believed to survive from one crop to the next on old leaves, stems and seed pods. Infection is mainly by ascospores blown by the wind.

Control. As the fungus attacks the seed crop it is as well to disinfect the seed as recommended against canker. It is more important, however, to clear away the debris of seed crops as quickly as possible and burn it. Seed beds should be placed as far away as possible from seeding crops and cabbage fields, as the amount of wind-blown infection from an old crop is greatly reduced by distance. The sprays recommended against dark leaf spot may also be helpful. Seed crops may be sprayed in August and possibly in early September, but newly sprayed cabbages are rather susceptible to frost and should not be sprayed in late autumn.

Downy Mildew (*Peronospora brassicae* Gäumann)

Symptoms. The upper surface of the leaf bears small, pale-yellow, angular spots, often speckled with brown, which may grow together to form irregular patches (Fig. 162). On the under-surface the corresponding areas are covered with a light-grey mould formed by the much-branched conidiophores and conidia of the fungus. The disease is commonest on seedlings in the seed bed and on the lower leaves of young plants, but it may occur on older plants and even on stored cabbage heads. In the latter circumstances the infection came through the stalk from infected lower leaves of the mature cabbage. The pith turns dark grey, and grey lesions with a yellow-

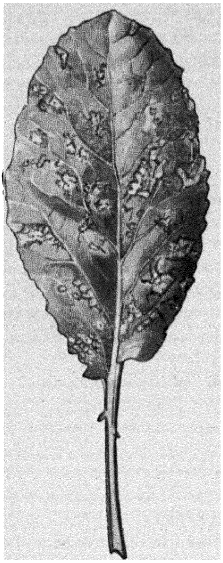


Fig. 162. Cauliflower leaf with downy mildew.

brown margin occur on the leaves throughout the head (Ramsay, 1938). Downy mildew may also spoil the sprouts in brussels sprouts.

Cause. The downy mildew affecting most cruciferous plants, cabbage and its allies, turnip, swede, seakale, mustard, radish, stock, wallflower and weeds like shepherd's purse and bitter cress, has traditionally been regarded as a single species, *Peronospora parasitica* (Pers. ex Fr.) Tul. This name is still used for it in British literature. In Switzerland, however, Gäumann has shown that *P. parasitica* in this wide sense includes a considerable number of races specialized on particular host plants and these he prefers to call different species. In his nomenclature the downy mildew of cabbage and its allies becomes *P. brassicae* Gäumann. Because of this degree of specialization there is no risk of cabbage being infected from, for example, the downy mildew so common on shepherd's purse.

Within the tissue of the diseased leaves the fungus forms its sexual reproductive bodies, oospores, which can remain alive in the decaying leaves in soil for several years. They are, however, not very abundantly produced and the fungus probably overwinters mostly in the form of mycelium in living plants. Laubert mentions the possibility of the mycelium growing into the seed pods and seeds, but seed-borne infection is not usually regarded as important in this disease. The fungus can grow and form conidia at a temperature as low as 40° F. and the spores germinate readily in water between 46° and 54° F., less freely at 66° F. and scarcely at all at 73–77° F.

Importance. Downy mildew does most damage to plants indoors and in frames. Only occasionally under moist conditions does it become important out of doors. Plants infected in the seedling stage usually grow away from the disease. In Holland, however, it has damaged heads of savoys particularly by persisting through the summer on the lower leaves, attacking the outer leaves of the head and growing in from there to cause brown spotting of the inner leaves in stored heads. Kale seems less susceptible than cabbage.

Control. Soil in which cabbages or their allies have been grown during the last few years should not be used for another cabbage crop. Development of the fungus is checked by dry air, hence the plants should be kept dry and the atmospheric humidity low by giving plenty of ventilation to seed beds in frames and glasshouses.

Spraying with Bordeaux mixture and other copper sprays also definitely checks downy mildew, especially when the under-side of the leaves is wetted, but spraying should be done early in the day and in weather when the moisture will dry quickly. Copper lime dusts may also be helpful, sulphur dusts are less effective.

Mildew (*Erysiphe polygoni* DC. = *E. communis* (Wallr.) Link)

Powdery mildew is often very common on swede leaves in autumn, but is seldom seen on cabbage, cauliflower or brussels sprouts.

It is sometimes quite severe on Hungry Gap and Marrow Stem Kale.

The same physiologic race of mildew attacks cabbage and swede, but mildew on weeds like pennycress and shepherd's purse seems to belong to other races. For control see p. 63.

White Blister (*Cystopus candidus* (Pers. ex Chev.) Lév. = *Albugo candida* (Pers. ex Chev.) Kuntze)

Symptoms. This disease is also sometimes called white rust, but that is a misleading name as the fungus which causes it is allied to the downy mildews and not to the true rust fungi. It is found on almost all members of the family *Cruciferae*: turnip, swede, cabbage, and its allies, mustard, cress, horseradish, arabis, honesty, wallflower, and numerous weeds, especially shepherd's purse. Any above-ground part of the plant may be attacked and in flowering plants the stems are often twisted, contorted and much swollen. The spores are formed in large white blisters under the epidermis, which burst open to expose powdery white pustules. For control see horse-radish, p. 285.

Dark Leaf Spot (*Alternaria brassicae* (Berk.) Sacc. and *A. oleracea* Milbrath)

Symptoms, cause and importance. The dark leaf spot fungi are commonly seed-borne and may attack seedlings, causing brown spots and streaks on stems and leaves. This phase of the attack is of minor importance in western Europe, but spotting of mature leaves and especially of the flower stalks, flowers and pods of seed crops may be of some significance. Two related fungi are concerned in the disease, which differ mainly by the size of their spores and other microscopic characters. Both cause rounded spots up to an inch across on leaves (Fig. 163). The spots are pale yellow with a yellow margin on green leaves and green on red cabbage leaves, best seen on the upper side of the leaf. In damp weather the upper surface, and to some extent the lower also, becomes covered by conidiophores and conidia. In *A. brassicae* they are pear-shaped with long

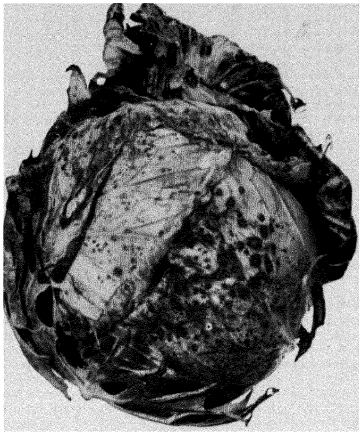


Fig. 163. Cabbage head damaged by dark leaf spot.

beaks and form a pale-greyish felt-like coating; in *A. oleracea* there is a dark olive-green, closely velvety growth, bearing the conidia in concentric rings. The spots can be found on leaves all the year round.

In seed crops, when the weather is damp and warm, these fungi spread rapidly over the stems after flowering and form pale oblong spots, soon covered with a dark layer of conidia, on the ripening pods (Fig. 164). In a few days of favourable weather the appearance of a whole seed bed may be changed from green to brown or almost black.

On the pods the mycelium grows through the wall into the seeds. Severely attacked pods shrivel and die. In others the seeds ripen but are weak and germinate with only a weak shoot soon killed by the fungus. Slightly infected seeds can be protected by seed disinfection and will in any case give a vigorous seedling if conditions favour rapid germination.

Presence of these moulds causes the pods to split open prematurely so that much seed is lost by shedding and the yield obtained from the crop is reduced. The quality of the seed is also adversely affected. Spots on the leaves are seldom of much importance in themselves,

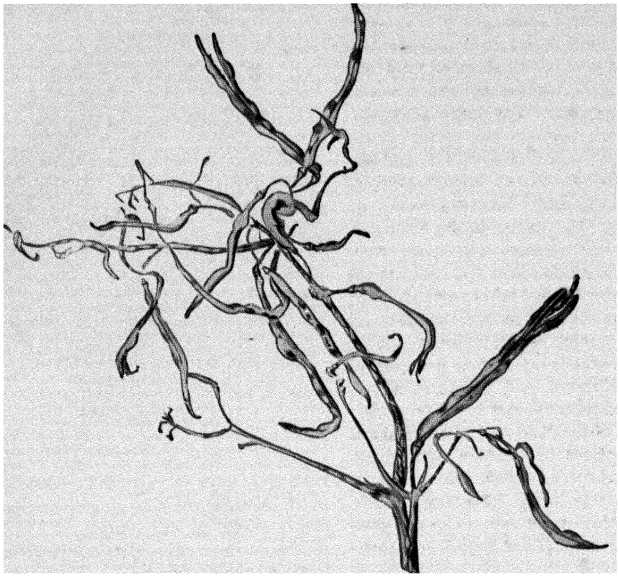


Fig. 164. Cabbage seed pods attacked by *Alternaria*.

but help the fungi to persist in the crop all the year round. The lowest leaves of a young seed crop are usually infected. They wither in the spring and on them are produced enormous numbers of conidia by which the flowering and fruiting stems become infected later on.

Control. In practice loss from these fungi has long been prevented by harvesting the seed early and ripening it by drying afterwards, either in the field or in a barn. This checks the attack but at the cost of reduction in yield of seed.

Spraying with Bordeaux mixture (8: 8: 50) retards ripening for about 10 days, but gives healthier seed with a higher germination. The first spraying is done just before flowering and the second and third after flowering, with a fortnight between them. If only two sprays are given they should both be after flowering, but at least 14 days before the crop ripens or their effect will be lost. The spray should be applied at a good pressure.

A good distance between the rows in the seed bed reduces the risk of infection, and there will be fewer yellow fading leaves to be attacked early and serve as foci of infection. Close planting also encourages dampness and favours growth of the fungi.

Replanting in autumn also checks the disease, perhaps because the infected leaves are covered. All withered leaves should be removed from overwintered cabbage crops in spring and destroyed.

Harvesting should not be delayed after ripening, as this encourages a severe attack and loss of seed by shedding.

Remains of the seed crop left in the field and after threshing should not be put with compost heaps or ploughed in, but should be burnt to destroy the fungi present on it.

Seed disinfection may improve germination and prevent infection of the seedlings, but it is not likely to prevent infection of the mature

plants by windblown spores from other sources.

Light Leaf Spot (*Cylindrosporium concentricum* Grev. = *Gloeosporium concentricum* (Grev.) B. & Br.)

This fungus was described by Greville in Edinburgh over a century ago and is still not uncommon on cabbage, cauliflower, broccoli and brussels sprouts in Great Britain. In 1902 Rostrup recorded it as at times destructive to cabbage leaves in Denmark, but of late years it has only once been observed there, on leaves from Amager. It causes roundish patches of tiny white pustules arranged in more or less distinct concentric circles, somewhat like those of white blister, on both surfaces of the leaves, but mainly on the underside. Usually only the outer, older leaves are affected and little damage is done but it may cause cabbage heads to become loose-leaved and spoiled (Thomson, 1936).

Control measures are unlikely to become necessary. If the disease becomes troublesome spraying with Bordeaux mixture may be tried.

Grey Mould (*Botrytis cinerea* Pers.)

Cabbage heads, especially when stored, may be attacked by the common grey mould, which causes watersoaked grey-green to brownish patches on the leaves. These become covered with the familiar grey coating of conidiophores and conidia and eventually irregular black sclerotia form in the rotting tissue. Grey mould thrives under damp conditions, especially at a moderate temperature, and may cause great loss to cabbages in storage. It is important that the cabbages be dry and well aired for some days before being stored, and that care be taken to avoid bruising them, as the fungus easily gains entry to the tissues through wounds. Hearts of spring cabbage may also be attacked in the field, especially after damage by frost.

Botrytis cinerea may also damage brussels sprouts even out of doors during the winter months. Early Dwarf and Long Island are particularly susceptible to it. Early varieties generally suffer most (see also p. 59).

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

In cabbage this is principally a disease of the stored heads. Larger or smaller parts of them turn black and slimy and on these the white, cottony mycelium characteristic of the fungus develops. Associated with it are large sclerotia, at first white, then with a black rind. Cabbages should be dry when stored and should be handled carefully so as not to be bruised, for damaged tissue forms an easy means of access for this fungus and for *Botrytis cinerea*, etc.

The disease is worst when the straw in the pit becomes damp. Cabbage varieties differ in susceptibility to *Sclerotinia* rot; of red cabbages Amager and spring red cabbage are resistant, and of ordinary cabbages high and half-high Amager. For further information see p. 77.

Bacterial Leaf Spot (*Pseudomonas maculicola* (McCull.) Stev.)

This disease, first described in North America in 1911, has been found on cabbage, cauliflower, broccoli, savoy and brussels sprouts in England (Moore, 1943) and on several cruciferous crops in Denmark (Ferdinandsen, C., 1923). At Lyngby we first noticed it in 1924 on radish, turnip (Snowball), swede (Wilhelmsburger) and kohlrabi, and to a lesser extent on savoy, cauliflower, cabbage, and red cabbage.

Numerous small watersoaked spots appear on the leaves and soon turn brownish or grey with translucent edges. At first they are round but become angular, limited in size to $\frac{1}{8}$ inch or so by the fine veins of the leaf (Fig. 165). If they are very numerous several may coalesce and the leaves may become ragged and torn. In severe infections the leaves may turn yellow and fall. The causal bacterium enters leaves through the stomata and can also attack flowers and the curd of cauliflower heads. It grows best at 77° F., not at all at temperatures over 86° F. and is favoured by high humidity. In California it has been shown to be spread on the bodies of insects.



Fig. 165. Bacterial leaf spot of cauliflower (after C. Ferdinandsen).

Seed disinfection with corrosive sublimate may help to prevent infection, and spraying with Bordeaux mixture plus a spreading agent may check its spread.

Black Rot (*Xanthomonas campestris* (Pamm.) Dowson)

Black rot may affect almost any cultivated cruciferous crop and some wild species as well, especially cabbage, red cabbage, savoy, brussels sprouts, broccoli, cauliflower, kohl-rabi, swede, turnip, mustard, charlock, radish, wallflower and stock.

Symptoms. Plants may be attacked at any age but especially when nearing maturity. In cabbage a larger or smaller section of a leaf typically turns yellow and bears a network of blackened veins. This blackening spreads down the vascular bundles (Fig. 166), to the upper part of the stem. Especially in turnip, infection may also start in the roots, which have been damaged by larvæ of the cabbage fly and other insects. In severe attacks so many of the outer leaves are destroyed that the cabbage plant appears " leggy " and its head becomes lopsided and loose-leaved. The blackened vascular bundles may appear in the head leaves too.

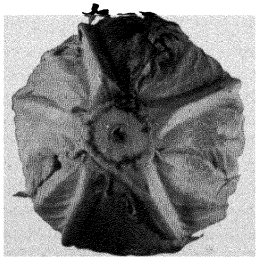


Fig. 166. Black rot of cabbage showing the brown vascular ring (*S. p. F.*).

When infection comes from the seed the cotyledons become infected 12–20 days after sowing. Many plants may be killed in the seed-bed or, if they survive, spread the disease when they are planted out. The rot caused by *X. campestris* alone is rather dry and limited in extent but it may open the way to secondary organisms which set up a rapid evil-smelling soft rot.

Development. Infection may come from the seed or the soil, especially if diseased cabbage or turnip heads, roots or leaves have come in with manure. From diseased plants and from infected soil the bacteria are spread by rain splashes, also by snails, by the larvæ of insects like the cabbage fly and by workers among the plants. Most infections occur through the water pores on the edge of the leaves. These exude drops of water during the night and if bacteria get into these drops they multiply and enter the terminal strand of the vascular system below the water pore. The bacteria can grow at temperatures between 40° and 100° F. and do best at about 86° F. They thrive on cabbage which have heated in a silo. Freezing and thawing does not kill the bacteria, which are able to remain alive in the soil from one year to the next. Nor are they killed by drying and survive for one year, possibly for three years, in stored seed. Extensive seed-borne infection has not been proved in Denmark but in North America this is considered one of the chief sources of infection and seed disinfection is strongly recommended.

Importance and distribution. Black rot seems to occur throughout the world wherever members of the cabbage family are grown. In North America it is a major disease of cabbage; in Denmark it occurs on cabbage in some

seasons, more often on swedes. Heavy manuring is said to encourage it. In England the disease occurs sporadically on cabbage, cauliflower, broccoli, seakale, turnip and stock, but is usually of little importance.

Control. Seed-borne infection can be destroyed by steeping the seed for 30 minutes in 1:1000 corrosive sublimate solution and then rinsing in water. Considerable control can also be obtained by hot water treatment, soaking the seed in water at 122° F. for 25–30 minutes, then cooling and drying. Seed beds may be disinfected by watering three times with corrosive sublimate solution. When the disease breaks out in a seed bed the whole stand should be discarded, or apparently healthy plants will carry the bacteria to the planted-out crop. Among older plants all those diseased should be destroyed and special care taken to prevent diseased plants or debris getting into the manure heaps or compost heap. Only heads free from black rot should be used in seed production. The disease may have become less prevalent than formerly with the increased use of artificial manures instead of infected dung in agricultural practice. Soil that has born an infected crop should be regarded as infected for 2–3 years.

Soft Rot (*Bacterium carotovorum* (L. R. Jones) Lehm)

Plant organs that are used for the storage of foodstuffs are often affected by a soft stinking rot caused by bacteria. Probably the commonest cause of the trouble is *Bacterium carotovorum* which has received several other names in the past according to the host plant in which it was found, e.g. *Bacillus oleraceae* Harr., *B. omnivorus* Van Hall, *B. apiovorus* Worm. and *B. hyacinthi septicus* Heinz. Closely allied species of soft-rotting bacteria are *Bacterium aroideae* (Towns.) Stapp, which can attack arum corms, iris leaves, potato tubers, etc., and *Bacterium phytophthorum* (Appel) Burgwitz, the cause of black-leg of potato.

Host plants. Soft rot is most frequently found in mature turnip roots, next in carrots and quite often in radish, swede, cabbage and its allies, potato tubers, especially in wet, warm soil, celery, seakale, parsnip, onion, leek, cucumber, melon, horseradish, tomato, capsicum, egg plant, arum, iris, hyacinth, cyclamen, and probably parsley, wallflower and violet.

Symptoms and life history. It is always soft and fleshy organs that are attacked, usually storage organs, like carrot and turnip roots, melon fruits, iris rhizomes, but sometimes other organs like the soft stems of cucumber and melon and the fleshy leaf bases of iris. It is characteristic of soft rot that the tissues dissolve into a sloshy mess unable to support their own weight. The rotting organ therefore collapses while watery drops and slime containing swarms of bacteria ooze out. There is often a peculiar smell such as one associates with rotten cabbage.

Bacterium carotovorum is able to move freely in water by means of its flagellæ. It is emphatically a wound parasite but once it has obtained entrance to plant tissues it secretes a poison which diffuses into neighbouring healthy cells and kills them. The middle lamella, the substance between plant cells which binds them together, becomes dissolved, so that the tissue becomes a structureless mass. Usually there is no change in colour but sometimes there is a slight brown discoloration. The bacteria multiply most rapidly at 80–86° F., hence their activity is restricted by cold storage.

In Denmark parsley has been affected by a serious rotting of the root, both under glass and out of doors, which may have been due to *B. carotovorum*, perhaps liberated into the soil from rotting turnips.

Kale, cabbages and the like are sometimes attacked by a wet blackish-brown rot that has been attributed to other bacteria (*Pseudomonas destructans* Potter, *Bacillus oleraceae* Harr.), but is probably only soft rot aggravated by secondary bacteria. The larvæ of the cabbage fly carry infection and the bacterium has been found still alive in the chrysalis of the fly (Johnson, D. E., 1930). Rotting at the neck in swedes and turnips similarly often follows injury by the swede midge. The heart leaves of celery are often destroyed by this organism.

In England and elsewhere *B. carotovorum* has been found to cause a soft rot of tomato fruit, as does its near ally *B. aroideae* in North America (see p. 563).

History. Soft rot of celery was described as long ago as 1892 by Halsted, an American, and in 1901 his compatriot L. R. Jones gave a more detailed account of *Bacterium carotovorum* and its peculiar capacity for dissolving plant tissues. In 1902–3 the Dutchman Van Hall described similar bacterial attacks on iris, wallflower, and other plants.

Control. Rotation of crops is important in preventing soft rot but one must remember what a large number of plants are liable to be attacked and hence to leave bacteria behind them in the soil. Infection is not spread by stable manure unless it contains infected debris.

Storage places may be disinfected by spraying or scrubbing with formalin solution 1: 80. Surface drying of carrots and other stored products before they are stowed away is to be recommended, but it is far more important that the silos or stores be cool and well ventilated. Pits should not be covered too soon or too heavily and provision should be made for ventilation. Carrots poorest in invert sugar were found by Kristoffersen (1921) to keep best. He concluded that carrot roots would probably rot during the winter if they had more than half their cane sugar converted to invert sugar at the beginning of storage.

Broccoli Mosaic (*Brassica* Virus 3)

The first symptom of infection in broccoli is a clearing of the finer leaf veins which appears first towards the base of young leaves. It may become more obvious as the leaf develops so that the veins become quite yellow. This condition is slowly replaced by vein banding, in which a dark-green band of tissue follows the larger veins and the intervening portions of the leaf blade become pale or yellowish. Small dead spots, at first white, then pale brown, may appear in the yellow areas. The leaves may be twisted and deformed and there is a tendency for the outer leaves to bend backwards and downwards instead of arching over the curd. In severe infections the whole plant is stunted and no marketable curd is produced. The virus found in England is known to infect brussels sprouts, cauliflower, cabbage, red cabbage, colewort, kale, savoy, sprouting broccoli, turnip, kohlrabi, rape, swede, radish, and charlock, and what is probably the same virus has been shown in America to infect shepherd's purse, ten week stock, and candytuft. Under English conditions the virus is transmitted mainly by the mealy cabbage aphis *Brevicoryne brassicæ* but several other aphides including *Myzus persicæ* can act as vectors. Symptoms of broccoli mosaic are completely masked when the plants are grown at temperatures above 77° F.

Diseases caused by this virus are common throughout Great Britain and mosaic is particularly destructive to the broccoli crop in South West England (Caldwell and Prentice, 1942). These authors recommend placing the broccoli seed bed in the middle of a field, far from hedges, and strict roguing of early infections afrer the broccoli have been planted out. The practicability of controlling the aphis vectors by spraying is extremely doubtful.

Cabbage Black Ring Spot (*Brassica* Virus 1)

This disease is common on cabbage in Europe and North America and is easily recognized by the small black ring-like markings which occur scattered over the leaves of infected plants. On cauliflower the virus causes a fine light-green mottle. The virus also attacks a number of ornamental plants such as arabis, wallflower, hesperis, and stock, and causes " breaking " in the colour of self-coloured flowers. It is transmitted by aphides.

Control measures have not been fully worked out. The virus passes the winter in ornamental plants and overwintering green crops and it is obviously desirable to separate new plantings as far as possible from such old plants. Infected crops should be destroyed as soon as all marketable produce has been cut. As far as possible multiplication and spread of aphides should be prevented.

Browning of Cauliflower (Boron Deficiency)

Boron deficiency on cauliflower is widespread in some districts of North America. Small transparent areas appear in the pith of the stem and on the surface of the maturing head. These turn brown or even almost black (Dearborn, C. H., 1942). Affected heads do not decay but are very bitter and unpalatable. In severe boron deficiency the leaves may be narrow and the formation of a head may be delayed or even prevented (Brandenburg, E., 1942). The disease can be controlled by manuring with borax at the rate of 5 to 25 lb. per acre according to the severity of the trouble.

Boron deficiency in cabbage has been fully described by Jolivette and Walker, 1943. The usual symptoms are watersoaked spots in the pith of the main stem. These turn brown and may break down into cavities. In extreme cases the oldest leaves may be mottled at the edges, be curled and irregular in shape and develop cross-cracking on the upper surface of the leaf stalk. Early maturing varieties are most prone to the trouble.

Potassium Deficiency

It seems that when potassium is deficient cabbage and all its allies develop pale leaf edges which later shrivel, also, sometimes dead spots on the leaf blade and brown streaks on the leaf stalks. The heads may be loose and defective, brussels sprouts develop few sprouts, there are green leaves in cauliflower heads and red cabbage leaves turn greenish. Potassium-deficient plants seem more susceptible to aphis and downy mildew than normal plants.

Whiptail (probably Molybdenum Deficiency)

Whiptail is a condition in which the leaves, usually of cauliflowers, are narrow, irregular and wavy and in extreme cases consist merely of a bare midrib or of a midrib with irregular lodes of leaf blade on each side of it. The condition has been reproduced artificially by growing cauliflower and broccoli plants in sand cultures lacking molybdenum. Whiptail usually occurs on acid soils very deficient in lime, but liming alone does not always cure it.

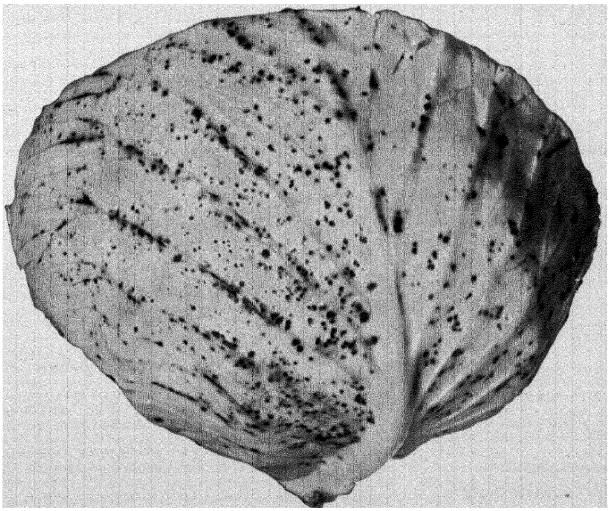


Fig. 167. Black spots on cabbage head.

Molybdenum in the soil is believed to be more available to plants in alkaline or limed soils than in acid ones. The disease has been cured by manuring with very small quantities of sodium or ammonium molybdate (Davies, E. B., 1945; Hewitt, E. J., and Jones, E. W., 1947).

Black Spot of Cabbages (Non-parasitic)

In 1934 there were complaints from several places in Denmark that some of the leaves in mature cabbage heads were covered with small black spots (Fig. 167). The appearance of the diseased cells resembled that of cells in turnips affected with brown heart, and boron deficiency may have been involved in the cabbage trouble. Similar black specks in cabbage and cauliflower leaves may arise in transit and during storage. They have been produced experimentally by keeping cabbages at room temperature in an atmosphere with little or no oxygen and by keeping them in a well-ventilated room at temperatures near 32° F. for 2–3 weeks. Black spot has also been found in the field after a sharp fall in temperature (Ramsey, G. B., *et al.*, 1938). Some cabbage virus diseases also cause black spot on the leaves (see p. 258).

Blindness

The cause of blindness in cauliflowers and broccoli is somewhat uncertain. The term applies to the condition in which the growing point is lacking so that no head is produced.

Often it is due to attack by the larvæ of the swede midge (*Contarinia nasturtii*) but it may arise with no sign of injury by any pest.

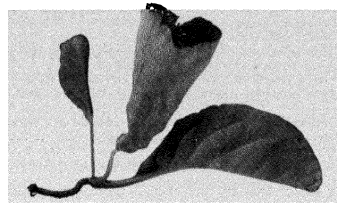


Fig. 168. Blind cauliflower seedling (*S. p. F.*).

Growth merely ceases with the production of a cup-shaped or funnel-shaped leaf, often the third leaf after the cotyledons (Fig. 168).

In many cases blindness is inherited and characteristic of the strain of seed used, or due to hybridisation with kale or cabbage. In 1936 some strains of cauliflower seed gave progenies of which 60–70% were blind.

Probably the tendency to blindness may also be intensified by cultural conditions, but the influence of these is not understood.

Splitting of the Head in Cabbage

In very wet weather the roots absorb so much water that growth of the inner juicy tissues of the cabbage head becomes too rapid and the outer tissues are burst open (see Fig. 2). This is especially likely to happen when heavy rain follows a drought. By driving a spade into the soil on both sides of the plant or by twisting it half round in the soil a considerable number of roots will be severed and excess absorption of water will be prevented.

Summary of Diseases of Cabbages and Their Allies

I. Roots

		page
A. Seedlings and bedding-out plants:		
(1) Plants fall over, neck pale and soft.....	Damping Off (*Pythium*)	68
(2) Plants wilt, neck dark, dry and shrivelled	Wire Stem (*Corticium solani*)	68
B. Larger plants:		
(1) Tumours on roots.....	Club Root.....	246
(2) Vascular bundles blackened, plants wilt	Black Rot	256

II. Stems *page*

A. Seedlings:
(1) Shrivelled neck, see 1 (A) above
(2) Blackish-brown lines on stem ..... Dark Leaf Spot 253

B. Larger plants:
(1) Stalk rotting:
(a) Dense white mycelium with white or black sclerotia ..... *Sclerotinia* Rot 255
(b) Brown or black vascular bundles ..... Black Rot 256
(c) Soft stinking wet rot ..... Soft Rot 257
(2) Stalk with dead leaves:
(a) Brown-purple or black spots ..... Ring Spot 250
(b) Blackish spots, sometimes sunken ..... Canker 249
(3) Stems with white pustules of spores, twisted and deformed ..... White Blister 253

III. Leaves—stem leaves and outermost leaves of the head

A. Superficial fungus coatings:
(1) White mealy coating..... Mildew 252
(2) White pustules, leaves twisted ..... White Blister 253
(3) Fine white mould in patches on under surface . Downy Mildew 251

B. Leaf spots:
(1) Minute white or cream-coloured pustules on pale spots ..... *Cylindrosporium* 255
(2) Pale round spots soon turning brown or blackish, rather large and covered with minute black specks ..... Ring Spot 250
(3) Small, black spots with translucent margins ... Bacterial Leaf Spot ... 255
(4) Round, greyish spots with dark-olive mould ... Dark Leaf Spot 253
(5) Small, often angular, pale spots, usually a fine white mould on lower surface ..... Downy Mildew 251
(6) Small black rings on leaves ..... Black Ring Spot Virus . 258

C. Leaf edges pale and shrivelled, sometimes dead spots and lines on leaves, leaves of red cabbage turn greenish ..... Potassium Deficiency . 259

D. Yellow patches spreading in from leaf edge (often triangular) and covered with black network of veins. Lower leaves may fall ..... Black Rot 256

E. Yellowish fading leaves, growth poor (on over-limed soil) ..... Manganese Deficiency . 39

F. Pale yellowish leaves, green along the veins only. On soil poor in lime ..... Magnesium Deficiency 38

G. Leaves variegated, mottled or deformed ..... Mosaic 258

H. Leaves funnel-shaped ..... Blindness 260

J. Leaves narrowed or reduced to midrib only ..... Whiptail 259

		page
IV. Heads		
A. Head lacking, often a funnel-shaped leaf present ..	Blindness	260
B. Head cracked	Cracked Head	260
C. Head lopsided or loose-leaved:		
(1) Leaf edges pale and shrivelled (green in red cabbage)	Potassium Deficiency ..	259
(2) Vascular bundles, black or brown	Black Rot	256
(3) Leaf with pale spots, minute white pustules visible under a lens.....	*Cylindrosporium*	255
D. Head spotted or decaying from the outside:		
(1) Yellowish or brown spots, sometimes with fine, white mould	Downy Mildew	251
(2) Pale spots with grey or olive mould	Dark Leaf Spot	253
(3) Leaves brown, vascular bundles black	Black Rot	256
(4) Leaves pale, wet, soft and evil smelling	Soft Rot	257
(5) Leaves black and slimy with white or black sclerotia	*Sclerotinia* Rot	255
(6) Large brown patches, often covered with grey mould	*Botrytis*	255
(7) Brown markings on surface of head in cauliflower	Boron Deficiency	259
E. Spots inside the head:		
(1) Black vascular bundles	Black Rot	256
(2) Translucent brown spots in pith of cauliflower, sometimes breaking down into small cavities ..	Boron Deficiency	259
(3) Small black spots on normal looking leaves	Bad Ventilation or Over-heating	260
F. Cauliflower heads with small green leaves in the curd	Potassium Deficiency ..	259
V. Inflorescence and Pods		
A. White pustules, twisted stems and pods	White Blister	253
B. Pale spots with grey or dark-olive mould on pods which may shrivel, but if not, easily spring open	Dark Leaf Spot	253
C. Brown to purplish-black spots, pods spring open with difficulty	Ring Spot	250

CARAWAY

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

The stem and leaf stalk become soft, rotten and overgrown with white woolly mycelium containing large hard sclerotia, at first white, then with a black rind (see p. 77).

CARROT

Violet Root Rot (*Helicobasidium purpureum* Pat. = *Rhizoctonia crocorum* (Pers.) DC)

History. This fungus was described by Persoon in 1815 under the name *Rhizoctonia crocorum* and re-described by Tulasne in 1851 as *Rhizoctonia violacea*, under which name it was long spoken of in plant pathological literature. Buddin and Wakefield (1927) showed

it to have as its perfect stage *Helicobasidium purpureum* Pat.

Symptoms. The fungus has a reddish-purple mycelium which grows in a network of fine threads over the underground parts of diseased plants, which gradually collapse as the tissue beneath the mycelium decays. It may grow through the soil to the next plant and attack it also. In dry weedy soil the fungus may persist in the same place for many years. Many different kinds of plants may be attacked. The earliest name, *Rh. crocorum*, was given to it because it was found on *Crocus* corms. Moore, 1943, records it in England as attacking potato, turnip, swede, sugar-beet, mangold, broad bean, red, white and Alsike clover, asparagus, carrot, celery, chicory, parsley, salsify, seakale, carnation, holly, and the weeds wild carrot, plantain and creeping buttercup. It is the most injurious root disease of sugar-beet and becomes a serious problem where carrot growing is practised intensively.

Baudys and others consider that different races of the fungus exist and call that on carrot *f. dauci*. This view does not find general acceptance and it is wiser to consider the possibility of infection spreading from any of the above-mentioned crops to any other. The fungus persists in the soil for many years and little can be done to get rid of it once it has appeared in a field. Long rotations, avoiding root crops for a considerable number of years, offer the best hope of control. On a small scale it may possibly be killed by soaking the soil with corrosive sublimate solution at the rate of 2 oz. sublimate plus 18 oz. saltpetre to 22 gallons of water.

Carrot Fungus (*Phoma rostrupii* Sacc.)

This disease seems to be virtually unknown outside Denmark. It was first found by Rostrup in 1888, who called the fungus *Phoma sanguinolenta* Rostr., not knowing that name had already been used for another species. Saccardo, therefore, re-named Rostrup's fungus *P. rostrupii* Sacc. C. A. Jørgensen (1934) has succeeded in reproducing the disease artificially by inoculating carrot roots and seed-bearing stems with a pure culture of the fungus.

Symptoms. The disease appears in late summer on one-year-old carrots as sunken grey spots on the upper part of the root, often forming a sunken ring round the rosette of leaves. Many blackish pimple-like pycnidia appear on the surface of the spots, from which there ooze out in damp weather red globules or tendrils, composed of innumerable conidia embedded in mucilage (Fig. 169).

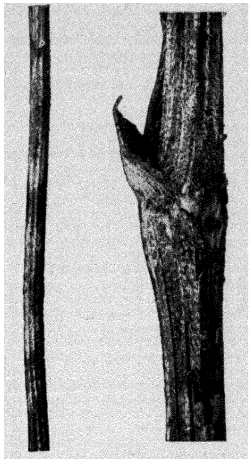


Fig. 169. *Phoma rostrupii* on carrot stems, stem on right enlarged to show the pycnidia (after C. A. Jørgensen).

In C. A. Jørgensen's artificial infections the tissue did not sink in as much as in Rostrup's description (1902). The spots became blackish-brown, covered with a short loose grey mycelium mingled with white mycelial tufts. Pycnidia were gradually formed in the grey patches.

The fungus may spread freely during storage and may grow from infected roots into healthy ones and destroy a whole consignment. Numerous other fungi (*Stemphylium radicinum*, *Sclerotinia sclerotiorum*, *Botrytis cinerea*, *Fusarium* spp.), however, often co-operate in this decay.

The fungus does most damage to the seed crop. When infected roots are planted out

to seed, spots gradually appear on the stem and leaf stalks, especially at the axils of the leaves. Often the spots are situated one above another and run together to form a streak down the stem because the mycelium tends to spread along the furrows. Infection becomes widespread by means of the conidia during the summer. When flowering commences the disease has usually advanced so far that the root decays, the stem withers, the leaves hang down and the plant dies without setting seed. Seeds that do mature may also be infected and bear small black pycnidia, but according to C. A. Jørgensen commercial seed stocks are not usually infected.

Importance. The disease was formerly much more important than at present. Commercial seed production is now usually from carrots that overwinter where they were sown and are less exposed to the disease than carrots taken up in autumn, stored over winter and replanted in spring.

Control. When young carrots for seed purposes are lifted in autumn they should be sorted very carefully and all spotted ones should be destroyed. It is a good plan to store the roots in sand to check spread of the fungus from any remaining infected roots to adjacent healthy ones. Any found to be spotted when the carrots are replanted in spring should also be destroyed.

Spraying or dusting may check spread in the seeding crop. Diseased seed heads should be burnt. For seed disinfection see black rot.

Black Rot (*Stemphylium radicinum* (Meier, Drechsl. & Eddy) Neergaard = *Alternaria radicina* Meier, Drechsl. & Eddy)

Symptoms. Dry blackish spots appear on the leaves, leaf stalk and stems of growing plants but the phase of the disease most often seen is a slow rather hard blackish rot on the stored root. It usually occurs on the neck but may also occur farther down the side of the carrot. Under dry conditions the rot is slow and dry but under damp conditions it becomes a wet rot covered with a blackish-grey mould



Fig. 170. Black rot of carrot (after C. A. Jørgensen).

(Fig. 170). *Fusarium* sp. and other secondary fungi often appear on the decaying spots. In carrots planted for seed the tops may wither. The disease is seed-borne and kills seedlings by rotting the roots and hypocotyledonary stem.

Cause. Black rot is caused by the fungus *Stemphylium radicinum*. According to Neergaard's examination of old preparations preserved at the Agricultural School and Botanical Museum this is the same fungus as Rostrup formerly called *Macrosporium dauci* Kühn.

Development. Infection may come from the seed or, according to American investigations, from the soil or from contaminated plant debris introduced into a pit with the stored roots. Stored carrots may be infected when lifted or may become infected by spread of the disease through the consignment in the pit, especially if the roots have been bruised or wounded.

Importance. The disease was particularly noticeable in carrot seed crops in Denmark during 1923, 1927 and 1932. Neergaard (1936) found it in the carrots in four out of six greengrocers' shops examined in Copenhagen. He also found various seed samples to be infected and to have their germination adversely affected by the fungus.

Control. Soaking the seed for from ½ to 1½ hours in a ¼% solution of corrosive sublimate or shaking up with a dry seed disinfectant greatly reduces the amount of seed-borne infection and increases the percentage germination. When carrots are being stored

or selected for a seed crop all roots marked with spots should be rejected.

Downy Mildew (*Plasmopara nivea* (Unger) Schroet.)

The upper surface of the leaf bears yellowish spots which later turn black and may cause the leaves to shrivel and fall. On the under-side of the spots is a loose white mould. This fungus is much more common on parsnip and races of it exist on many wild umbelliferous plants such as goutweed, hedge parsley and angelica. These should not be tolerated in and around gardens as, even if the race of mildew attacking them does not infect carrot, they may also harbour the carrot fly. An attack of downy mildew may be checked at the outset by spraying or dusting with a copper fungicide. Remains of infected plants should be burnt or otherwise destroyed in autumn.

Cracked Roots (Boron Deficiency or Fluctuating Water Supply)

Boron deficiency has not been reported in English carrot crops. In pot experiments with carrots on moorland peat, however, Ødelien in Norway found the roots very much cracked. Manuring with small quantities of boron compounds prevented the cracking. On slightly acid peat soils in Canada longitudinal cracking of carrots has similarly been controlled by manuring with 20 lb. borax per acre. Carrots grown by Hill (1943) in sand cultures without boron also developed wide cracks, and leaves which turned yellow round the edges. Longitudinal cracking may also be due, however, to a sudden acceleration of growth when rain follows a dry spell.

Other Root Diseases

Stored carrots are liable to be rotted by bacterial soft rot (*Bacterium carotovorum*), p. 257; *Sclerotinia sclerotiorum*, p. 77; and *Botrytis cinerea*, p. 59. A non-parasitic trouble called Clayburn has been described by Moore (1943). Affected roots have irregular, superficial, scurfy patches that turn deep blue-black when washed. The condition is said to occur on soils too heavy for carrots.

Summary of Carrot Diseases

I. Root		page
A. Seedlings die, root blackish-brown	Damping Off	68
	Black Rot	264
B. Larger roots:		
(1) Cracking	Too Rapid Growth	265
	Boron Deficiency	265
(2) Covered by reddish-purple network or crust, sometimes decaying	Violet Root Rot	262
(3) Obviously rotting, under moist conditions they bear:		
(a) White mycelium with black sclerotia	*Sclerotinia* Rot	77
(b) Hairy grey mould, sometimes with black sclerotia	Grey Mould	59
(c) Loose grey-black mycelium	Black Rot	264
(d) Blackish pycnidia oozing red globules of spores	Carrot Fungus	263
(e) No mycelium but a soft stinking rot	Bacterial Soft Rot	257

		page
II. Stems		
A. Death of seedlings, roots decayed	Damping Off	68
	Black Rot	264
B. Older plants:		
(1) Dark spots	Black Rot	264
(2) Light-brown spots and streaks dotted with blackish pycnidia	Carrot Fungus	263
(3) Pale spots with:		
(a) Hairy grey mould	Grey Mould	59
(b) White mycelium and black sclerotia	*Sclerotinia* Rot	77
III. Leaves		
A. Yellowish and shrivelling	Manganese Deficiency	39
B. Yellow spots with white mould underneath	Downy Mildew	265
C. Reddish-yellow leaf stalk, sometimes twisted (only in North America)	Aster Yellows	428
D. Dry spots and streaks	Black Rot	264
IV. Umbels		
Withered umbels, diseased spots on the stem or neck of the root:		
A. Blackish pycnidia on the spots	Carrot Fungus	263
B. Grey-black mycelium present under moist conditions	Black Rot	264

CELERY AND CELERIAC

Celery Leaf Spot (*Septoria apii* Chester and *S. apii-graveolentis* Dorogin)

For many years celery leaf spot was attributed to one fungus, *Septoria apii*, but more recently it has been shown that two related species are involved. *S. apii* causes large brown spots on the leaves, bearing a few pycnidia; *S. apii-graveolentis* causes more numerous, smaller, greenish spots crowded with pycnidia. The latter is much the more common in Britain, but the life history of the two species is similar and they will not be further distinguished in this account.

Symptoms. These fungi are carried by the celery seed and only a slight magnification is needed to see their small blackish pycnidia on the infected seeds. They are about 1/10 mm. across and are often arranged in groups. There may be as many as 20 on a single seed and others may occur on fragments of seed stalks mixed with the seed sample.

During germination the spores ooze out of the pycnidia and are carried on to the cotyledons of the seedling in drops of water or by rain splashes, etc. As the incubation period lasts about three weeks the attack is first seen on somewhat larger seedlings and the infected cotyledons are nearly always overlooked in practice. On the first small true leaves and on those that follow there appear somewhat angular spots, varying from greyish to grey-brown or dark-brown, in part according to the species of fungus concerned (Fig. 171). Individual spots even of *S. apii* seldom attain ½ inch diameter but several may unite to cover

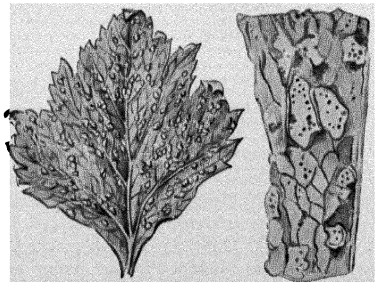


Fig. 171. Celery leaf spot. On right, part of a leaf enlarged × 5, showing the pycnidia.

a large part of the leaf and give it a withered appearance. Numerous spots appear also on the leaf stalks. Small black pycnidia, like those on the seed, are embedded in the tissue of the spots with only their mouths protruding from the surface (Fig. 163).

Occurrence and importance. C. A. Jørgensen (1934) examined 89 different samples of celery seed in Denmark. They were of different varieties, from various countries, harvested over the period 1921–6, but only one was free from pycnidia of the leaf spot fungi. In the others from 1 to 67% of the seeds were infected. Moore (1943) recorded a similar situation in England, where of 870 samples examined 637 were found to be infected and in 45 of them infection ranged from 60 to 80%.

It must, therefore, be recognized that most seed samples are infected, or at least were so up to 1942, and it is not surprising that the disease is very widespread. Lin (1939) found 142 out of 280 seed samples examined in New York state contained seeds bearing pycnidia. By counting the number of conidia in a pycnidium and the number of the latter on the leaf spots he concluded that if there were only 10 spots derived from seed-borne infection in a seed bed these would produce 1,500,000 conidia to spread the disease further. Where no control measures are undertaken leaf spot is often so severe that the leaves are weakened. In celeriac the crowns are very poorly developed and in celery the stalks are spotted and growth of the whole plant is checked. The commercial quality of the crop is thus greatly impaired.

Control. Control measures include preventing infection of the seed and checking further development of the fungus after the first spots have begun to appear on the leaves.

By spraying the seed crop 2 or 3 times with 2% Bordeaux mixture, or a similar copper spray, it is possible to obtain a seed sample virtually free from infection.

When the seed is infected the fungus in it may be killed by suitable treatment before sowing. The following were the best methods of seed disinfection among those tested by C. A. Jørgensen: soaking for 24 hours in 2% copper sulphate solution, 6 hours in ¼% solution of commercial formalin, or 6 hours in ¼% Germisan solution. All these treatments gave plants free from the disease. If the treated seed was kept the germinative capacity of that treated with formalin rapidly declined; so, gradually, did that of seed treated with copper sulphate; but seed treated with Germisan kept well for 2 years and was quite good after 3 years. Germisan treatment is the one most practised in Denmark but it has been shown to check the energy of germination so that the seed germinates irregularly. Hence several growers have used a weaker solution but the effect of this on leaf spot disease is unknown.

Krout (1921) stated that steeping the seed for half an hour in water at 113–117° F. killed the fungus without affecting germination.

The fungus can remain alive for 2 years in compost and can easily overwinter in the remains of diseased celery in the soil, so that rotation of crops is advisable (Klebahn, 1910). If celery has to be planted in the same place, or in an adjoining plot in the following year all diseased tops should be burnt in autumn.

Several investigators (Krout, 1921; Coons and Levin, 1916; Campinile, G., 1926) contend that the fungus on the seed dies before the seed loses its germinative capacity, so that it may be avoided by using seed 2–3 years old. British growers are reminded that a report on the health of a sample of celery seed can be obtained for a small fee from the National Institute of Agricultural Botany, Huntingdon Road, Cambridge.

As spraying with 1 or 2% Bordeaux mixture (8: 8: 100 or 16: 16: 100), or comparable copper spray, gives excellent control it is probably best in most cases to rely mainly on a rational spray programme. It is advisable to spray the seedlings once while still on the bench but the solution used for this should not be stronger than 1% Bordeaux mixture. Spraying should be repeated as soon as the first leaf spots are seen in the transplanted crop and in wet years it should be repeated

several times at intervals of 2 or 3 weeks. Copper lime dusts may also be used.

As the spores of the fungus may be spread on clothes and tools of the workers when the tops are wet, one should avoid walking amongst a celery crop in damp weather.

Differences in varietal susceptibility have not been demonstrated and observations on this point are complicated by the fact that celery seed samples are not always true to type. Non plus ultra is regarded as very susceptible in Denmark but all other varieties seem to become infected to a considerable extent.

Celery that has received a heavy dressing of nitrogenous fertilizer is more susceptible than that moderately manured. Hence large dressings of nitrogen should be avoided or, if they are required, a close watch should be kept on the crop and sprays applied at the first sign of leaf spot.

Root Rot of Celery and Scab of Celeriac (*Phoma apiicola* Kleb.)

These diseases are of general occurrence but seldom attain any real importance.

In celeriac, brown smooth spots appear on the skin of the crowns and gradually develop a cracked, warted skin. In early and severe attacks development of the crowns is checked and their upper surface becomes covered with a brown or yellowish-brown, scabby skin which may crack, and the crowns do not keep well. This condition, too, is sometimes called "rust" but it has no connection with true rust caused by *Puccinia apii*.

In the scabby patches tiny dark pycnidia are formed and these may also be found on seedlings at the base of the leaf stalk and occasionally on the seeds and leaves. According to Goossens, in Holland infection of the seed is very rare; according to Moore, a small amount of *P. apiicola* infection is found in about half the celery seed samples examined at Cambridge.

In severe attacks seedlings still in the seed box may develop damping off symptoms, i.e. they rot at the soil level and die. Celery is attacked by *P. apiicola* mainly at the base of the leaf stalks and the rot spreads thence into the crown and the roots. The decayed leaf stalks are bluish-green or black and the rotting roots are brown.

Celery roots may also sometimes be attacked by soil fungi such as species of *Pythium*, causing a reddish root rot, *Thielaviopsis basicola*, causing a black root rot, and the ubiquitous *Corticium solani*.

Control. Soil which has carried a diseased celery crop may be regarded as infectious for several years and an interval of 3–4 years at least should elapse before another celery crop is planted in it. Mild attacks may easily be overlooked but leave sufficient infection in the soil for a worse outbreak in the following year.

All remains of a celery crop should be burnt, deeply buried or, if composted, the compost should not be used for 3 or 4 years.

Downy Mildew (*Plasmopara nivea* (Unger) Schroet.)

Yellow spots, which later turn black, appear on the upper surface of the leaves and a close white mould on the corresponding parts of the lower surface. Ultimately the leaves wither. The fungus may penetrate the upper part of celeriac crowns which become brown and mouldy in the centre (see parsley, p. 305).

Black Rot (*Stemphylium radicinum* (M. D. & E.) Neerg.)

The name black rot was applied to the disease caused by this fungus on carrots. In 1934 Neergaard discovered it causing a dry, brown, cracked, superficial, scab-like rot of celeriac crowns, rather like that of *Phoma apiicola*. How widespread it may be and how far it has been confused with *P. apiicola* scab we cannot say.

By inoculation experiments Neergaard has shown that the fungus can kill seedlings by rotting the roots and stem, which turn brown or black and shrivel so that the plants fall over. Seedlings may also be killed before they emerge above ground. Inoculation of the crown of a year-old celeriac plant led to production of a brown or blackish-brown scab

about 1/10 inch thick on the upper surface round the point of inoculation. All the above-ground parts, leaf stalk, leaves and flower stalk, may be attacked; 3 or 4 days after inoculation a brown sunken area with a yellow margin appeared. Gradually the leaf stalks cracked at the points of infection and later both leaves and leaf stalks became blackish-brown and translucent, looking as though frosted. Flower stalks were similarly affected.

Cercospora apii Fres.

This fungus is known in most countries where celery is grown and is sometimes troublesome. In Denmark it is far less important than *Septoria* leaf spot and it seems to be as yet almost unknown in Britain; the only British record is from the west of Ireland (*Trans. Brit. Mycol. Soc.* xxii. p. 11, 1938).

As a rule the spots do not appear before the plants are 6 weeks old. The first symptoms are small yellow spots on both sides of the leaves. These quickly enlarge and become ash-grey and papery. They may be irregular, angular or semi-circular and have a slightly darker margin. Under very damp conditions the conidia of the fungus appear like a grey coating to the spots on both leaf surfaces. Similar spots may also appear on the leaf stalks and in severe outbreaks these may crack at the point of attack.

The disease is less destructive to vigorous plants than to weak ones. As the spores are easily spread by tools, boots, etc., when the plants are wet, all work and movement among wet celery crops should be avoided if possible. The disease may be controlled by spraying with a copper fungicide such as Bordeaux mixture. The fungus may survive for a long time in the debris of a diseased crop which should therefore be destroyed in autumn.

Rust (*Puccinia apii* Desm.)

This disease was evidently more frequent in Denmark at the end of the last century than at present. In 1902 Rostrup wrote of it as not infrequent but during the last 20 years there have been only two authenticated records. The varieties now grown are not very susceptible to the disease. Celery rust seems to be unknown in Britain where growers commonly apply the term " rust " to brown markings of diverse origin, not usually caused by fungi.

The patches of cluster cups (*aecidia*) are found in spring in yellow spots on the leaf stalks and leaves. Later on appear numerous pustules of cinnamon-brown uredospores and later still the dark brown teleutospores which remain for a long time covered by the skin of the host.

Two sprayings with Bordeaux or Burgundy mixture have given good control in Bavaria (Flachs, 1926). One is made as soon as the disease is observed and the second twelve days later (see also rust, p. 74).

Heart Rot and Other Bacterial Diseases

Bacterial soft rot involving the heart leaves and spreading up the leaf stalks and down into the stock is not uncommon in celery. The cause is probably the common soft-rotting bacterium (see p. 257). Damage by the fly larvæ may begin the trouble and it is likely to become prevalent when there is no proper rotation of crops. Danish experiments indicate that excess lime and boron deficiency are another important cause of heart rot (see p. 270). A non-parasitic form of black heart is widespread in celery fields in North America. It seems to follow periods of either drought or excessive soil moisture, especially if the crop is growing rapidly (Ramsey, G. B., and Wiant, G. S., 1941).

A purely bacterial disease is that caused by *Pseudomonas jaggeri* (Jagger) Stapp, so far known only in the United States. This organism causes small rusty-brown spots on the leaves. It can be controlled by dusting the seed beds weekly with copper lime dust and in the field crop by spraying with Bordeaux mixture.

Mosaic and other Virus Diseases

Several kinds of virus disease are known in celery in North America. In England celery mosaic has been shown to be due to infection with cucumber mosaic virus, which causes

whitish vein clearing, followed by a conspicuous whitish and green mottling of the leaves. The plants may appear stunted owing to the leaf stalks curving outwards instead of remaining stiff and upright. There may also be brown spots on the leaf stalks.

Tomato spotted wilt has been found to cause small yellow leaf spots and brown spots on the leaf stalks of celery in California (Severin and Freitag, 1938).

Two other diseases known only from North America are those caused by celery yellows virus (*Callistephus* Virus 1A) and western celery yellows virus (*Apium* Virus 1).

Bitterness

Celery and celeriac belong to the group of plants which profit by chlorine. Hence plentiful manuring with a chlorine-containing potassium fertilizer should give a good flavour and good keeping quality (high sugar percentage, low nitrogen content); the bitter taste comes from too high a nitrogen content.

Boron Deficiency

In celeriac boron deficiency results in hollow crowns with dark tissue lining the cavities. The crowns tend to turn black when cooked. In severe outbreaks there is only a thin layer of normal tissue surrounding a large hollow.

In some cases there is also a yellowing of the leaves, particularly of the heart leaves, which may die completely and the tops have a tendency to lie along the ground. In such plants there is usually a cavity in the heart extending down into the root stock, into which one can stick one's finger. In other cases there is no outward sign of the damage, which is only discovered when the crowns are cut.

This disease was particularly noticeable in 1932 at Hornum, Lyngby and in S. Jutland, and in 1934 at the first two centres and at Copenhagen.

The disease appears to be purely physiological in nature and to be connected with particular soil and weather conditions. It seems most serious where a good growing period has been followed by drought and then by a further good growing period. Different strains of celeriac differ in susceptibility to heart rot. A high soil reaction clearly favours the trouble and it can be prevented by manuring with borax. At the experimental station at Hornum about 1 oz. of borax to 36 square yards entirely prevented the appearance of these hollow crowns on ground where untreated celeriac was severely affected.

This kind of hollowness seems to have no connection with the small hollow so often found in celeriac as to be regarded as more or less normal. This latter is often no larger than a hazel nut or a walnut and the surrounding tissue is quite white, though it may be a little soft around the cavity. The size of the cavity may vary somewhat with variety and conditions of growth but is much less than that due to boron deficiency with its unsightly dark walls.

With ordinary celery in Florida, boron deficiency has been found to cause browning of the heart leaves and the appearing of brown cracks and lines all over the ribs of the outer leaf stalks (Purvis and Ruprecht, 1937). This "Cracked stem" condition has once been reported in England (Moore, 1943). Borax manuring at the rate of 15–20 lb. per acre prevents the disease, or it may be checked by spraying the crop with a borax spray, 2–3 lb. borax per 100 gallons of water. In celery on peat soil in Quebec, cracked stem has been accompanied by heart rot so severe that at maturity of the crop there remained only a ring of outer leaf stalks with brown corky inner faces surrounding a cavity.

Rusty Markings in Celeriac

The swollen crowns of celeriac are sometimes stained by reddish markings either in winding lines like worm tracks or in flame-like shapes. This red colouration is often, mistakenly, called "rust," but it has nothing to do with the rust fungus. It is therefore best to restrict the name celery rust to the disease caused by *Puccinia apii*.

Unsatisfactory manuring and sour soil may co-operate to produce these golden-reddish patches in the crowns. Very close planting

has the same effect. Apparently also other factors, as yet undetermined, have some influence on it.

Blackening of Celeriac

At times celeriac tends to become black when cooked. Unfortunately the reason for this is unknown. Varieties grown on the same soil may differ considerably in their tendency to darken after cooking. Soil and weather conditions may influence it to some extent, but in recent experiments no clear differences have been demonstrated in the behaviour of celeriac grown with different manurial treatments.

Frode Hansen has shown that the presence of iron during cooking may cause blackening of celeriac. Thus in comparative experiments celeriac turned dark when cooked in iron saucepans, or in those with the enamel chipped, whereas when cooked in saucepans with undamaged enamel it was not discoloured.

Summary of Celery and Celeriac Diseases

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(7) Sunken brown spots	*Stemphylium*	268
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(2) Greenish or blackish decay at the base	*Phoma apiicola*	268
(3) Light-brown or grey spots	Leaf Spot	266
(4) Light red-brown soft stalks, white dense mycelium with large sclerotia	*Sclerotinia* Rot	77
C. Whole top discoloured or deformed:		
(1) Yellow, prostrate leaves, heart leaves often dead	Boron Deficiency	270
(2) Poorly developed red-brown tops	Potassium Deficiency	42
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III. Celeriac Crowns

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(2) Reddish or golden irregular markings	Rusty Markings	270
(3) Flesh darkens on cooking	Blackening	271
(4) Bitter taste	Bitterness	270

CHERVIL

Foot Rot (*Corticium solani*, *Pythium* spp.)

Under unfavourable circumstances young chervil plants may develop a constriction at the soil level due to infection by one of the damping off fungi, fall over and die (see p. 68).

Downy Mildew (*Plasmopara nivea* (Unger) Schroet.)

The lower surface of chervil leaves may become quite white with a layer of conidiophores of this fungus. The corresponding parts of the upper surface show as yellow spots which later turn black and may cause the leaves to wither. Races of this fungus attack many cultivated and wild umbelliferous plants. Weeds belonging to this family should, therefore, be destroyed in and around the garden. In the early stages of an outbreak an attempt may be made to stop it by spraying with a copper fungicide. The remains of an infected crop should be burnt or otherwise destroyed in autumn.

SPANISH CHERVIL

Rust (*Puccinia chaerophylli* Purt.)

Numerous little yellow pustules of spores appear on the leaves. Control measures have not been tried; fungicidal sprays or dusts would only be effective if applied at very frequent intervals, i.e. at an uneconomic expense.

CRESS

Damping Off (*Pythium de Baryanum* Hesse)

Under damp conditions, especially when too closely sown, cress seedlings develop a pale shrivelled lesion at soil level and fall over (see damping off, p. 68). The usual cause is the water mould *Pythium de Baryanum* but *Corticium solani* may also cause a similar trouble. Cress grown for seed may suffer from white blister (p. 285).

CUCUMBER

Anthracnose (*Colletotrichum lagenarium* (Passer.) Ell. & Hals. = *C. oligochaetum* Cav.)

Rostrup described this disease in 1902 under the name *Gloesporium orbiculae* Berk. It has been very destructive in the past in England and in Sweden. In Denmark it was first definitely identified in a greenhouse at Odense in 1943 where it appeared in a virulent form and recurred in 1944.

The seedlings may be attacked and fall over but this seldom happens. Leaf spots are the usual symptom at all stages of growth of the crop. They are pale green and transparent, later becoming dry, reddish brown, with a pale margin and tend to fall out. They enlarge more quickly than those of blotch and are more apt to fuse together into large

withered areas, which may kill the whole leaf. Sunken dry patches, later becoming dry and floury, appear on leaf stalks and stems. Sunken pale spots also appear on the fruit. The diseased tissue commonly bears pinkish conidial pustules of the fungus, easily distinguishable under a magnifying glass.

In greenhouses that have contained an infected crop the fungus occurs in the soil, on the glass, especially at joins, on decaying wood, old paper, etc. (Bewley, 1923).

When the disease has occurred the houses should be thoroughly cleaned after the crop has been removed, sulphur should be burnt in them, all crop debris destroyed, the woodwork and glass should be sprayed with formalin 2:100 and the soil should either be renewed or thoroughly disinfected. In Denmark it was found in 1944 that cleaning and sulphuring alone were not sufficient to prevent recurrence of the disease in the same house.

Gummosis (*Cladosporium cucumerinum* Ell. & Arth.)

Symptoms. When the seed has been sown in infected soil the seedlings may be attacked. As a rule the first symptom is a water-soaked spot on the hypocotyl below the seed leaves and often a dark stripe extending as far as the latter, on which dark spots may also develop. If the attack is severe the stem cracks at the attacked places. Under favourable conditions the infected seedlings may survive but the fungus is easily spread further from these lesions.

In older plants oblong spots with yellowish-brown margins may develop on the stems and leaf stalks.

On the leaves the fungus causes yellow or greyish-yellow spots which burst open. They are larger and more irregular than the small regular spots characteristic of blotch.

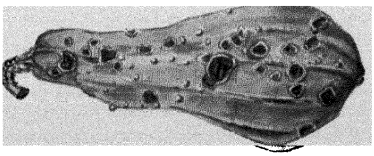


Fig 172. Gummosis of cucumber.

The disease on the fruit is the most general, most characteristic and best-known phase. Depressions occur in the skin, each individually seldom exceeding $\frac{1}{2}$ inch in diameter and $\frac{1}{5}$ inch deep, but several such spots often fuse to form large irregular patches (Fig. 172). The spots often exude a gummy liquid, which hardens into a yellow or brown bead, entirely or partially filling the depression. This characteristic gummy discharge gives the disease its common name "gummosis". The conidia of the fungus form a grey or greenish-black layer over the depressions and on the edge of the spots.

Cause, development and importance. The disease is caused by the mould fungus *Cladosporium cucumerinum.*

Gilbert has shown that races of this fungus isolated from many different places varied greatly in their capacity for infection. It can over-winter in the soil, on woodwork, glass, etc.

Pfältzer (1927) examined cucumber seed extensively but was unable to demonstrate seedborne infection; its possibility is not, however, to be excluded.

The fungus can grow at temperatures between 32° and 85° F. but thrives best about 70° F., whereas cucumber plants grow best about 80° F. If the atmospheric humidity is kept below 85% development of the fungus is greatly checked.

The disease is general in Denmark and in England, where it became prominent with the expansion in acreage of outdoor cucumbers during the last war (Moore, 1948). It does most damage in inadequately heated houses and on outdoor crops, where in wet years it may destroy the crop. In Denmark too it became more important during the war because of lack of heating.

Control. One should take advantage of the fact that the optimum temperature for growth of the cucumbers is higher than that for the fungus. If germination takes places at 86–90° F. the fungus has little prospect of development. A temperature of 77–82° F. would, however, suit the plants better than 90° F. In greenhouses the temperature is kept well up during the day, i.e. from 77–86° F., and by

firing at night it is prevented from falling appreciably. During the dark days early in the year it may be necessary to maintain a lower temperature to prevent the plants from becoming too long and spindly. The house should be kept no damper than is necessary for the health of the crop.

Diseased fruits should be removed as soon as possible and should preferably be placed in a pail, e.g. containing lime water. If they are collected in a box or basket there is a risk of a cloud of conidia rising from them every time another affected fruit is thrown in.

As soon as the first attack is seen the plants should be dusted with sulphur and this should be repeated at frequent intervals. When the last fruit has been picked from a house sulphur should be burnt in it (see p. 600).

For the outdoor crop it is important to make the new cucumber beds as far as possible from those of the previous year and always to remove and burn all the remains of infected cucumber plants. Where there has previously been trouble with gummosis and it is not possible to obtain fresh soil the soil of the beds should be disinfected, e.g. by heating, or by watering with 1: 100 formalin solution at the rate of 18–20 pints per square yard. It is especially important to disinfect soil intended for seed boxes and potting.

The following varieties are said to be resistant to gummosis in Denmark: Danish pickling cucumbers Tagenshus, Lyngby, and Guldagergaard and Langelands Kaempe Lyngby. The variety Delikatess is said to be resistant in Germany (Schultz and Röder, 1939).

Blotch (*Corynespora melonis* (Cooke) Lindau — *Cercospora melonis* Cooke)

Symptoms. This is a fairly widespread disease which may become serious. The first signs are small, light-green, water-soaked spots on the upper surface of the leaf. This stage only lasts for a short time and often escapes observation by the grower. The spots increase very rapidly in size and in numbers so that the leaves become closely covered by them (Fig. 173). They become grey, then brown and quite dry in the centre, where they crack

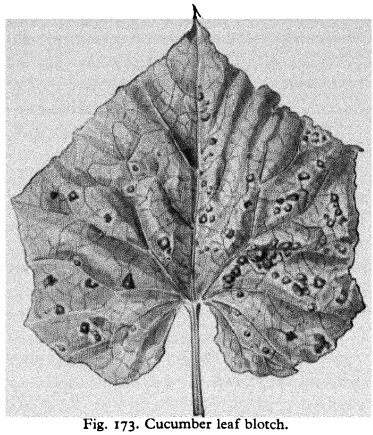


Fig. 173. Cucumber leaf blotch.

open. Gradually they fall out leaving numerous holes in the leaves. The spots often cross the smallest veins but their spread is restricted by veins that are somewhat larger.

The fruits may also be attacked. There the spots are at first small, dark and somewhat sunken, not marked off from the surrounding healthy tissue by a sharply defined border. They quickly increase in size and may sometimes cover half the upper side of the fruit or more. Often the diseased tissues sink in to form a constriction in the fruit. This usually happens at one end of the cucumber; if it is at the stalk end naturally the entire development of the fruit stops.

Cause. The spots are produced by the fungus *Corynespora melonis*, first described by Cooke in 1896 under the name *Cercospora melonis*, which is still commonly used.

On the spots many conidia are produced which are easily spread by workers and their tools, by watering and by insects and other pests, such as aphides and mites.

The conidia can germinate quickly in warm, moist conditions so that the disease may spread very rapidly.

Control. The fungus overwinters in the soil, on woodwork, glass, etc. Infection with the seed seldom occurs, but the possibility of

it has to be reckoned with. (As a precaution the seed should be disinfected, see p. 576.) By growing a good resistant variety like Butcher's Disease Resister it is possible to avoid the disease altogether. Weigelts Beste von Allen is also said to be resistant.

Excessive moisture during the night should be avoided by firing and keeping good ventilation and at all times the air should be kept no moister than is absolutely necessary. If the attack is discovered while only a few leaves are involved, these should be carefully collected and burnt (cf. gummosis, p. 273). In England spraying in the evening with liver of sulphur at a temperature of about 70° F., followed by ventilating next morning, has been recommended. Other suggestions include evaporation of sulphur, dusting with sulphur and spraying with copper fungicides.

Sulphur should be burnt in the house before the plants are removed. In houses where attacks have occurred it is best to grow Butcher's Disease Resister for the next three years to avoid the disease. If that is undesirable it is advisable to resoil throughout the house, not only in the borders, or to disinfect the soil thoroughly (see p. 577).

Mildew (*Erysiphe cichoracearum* DC.)

Mildew may be very serious on outdoor cucumbers in Denmark; it is of general occurrence but usually less serious on the hothouse crop. The disease begins as small white spots which may gradually coalesce to form a continuous powdery white coating over the shoot tips and leaves, particularly on the upper surfaces (Fig. 174).

When mildew is found in a greenhouse the ventilation should be improved and the old leaves removed as soon as possible. The usual remedies for mildew may also be tried (see p. 63).

In two experiments on outdoor cucumbers at Lyngby, mildew was checked by two sprays with Bordeaux mixture 16: 8: 100. At Cheshunt, Orchard (1933) obtained good results by spraying greenhouse cucumbers with Shirlan plus the spreader Agral N. The spraying was at frequent intervals, every 3–4 days for the first month after the disease appeared, thereafter once a week. In Denmark the disease is not common enough to warrant much spraying in the houses. The variety Delikatesse has proved resistant to mildew in Germany.

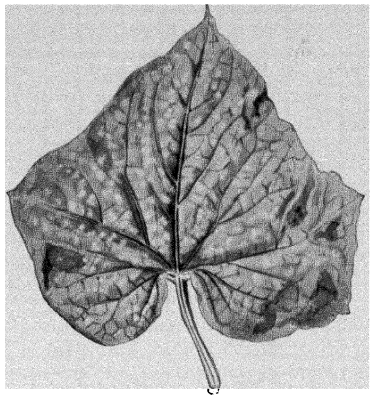


Fig. 174. Cucumber mildew.

In warm climates cucumbers suffer also from a downy mildew (*Peronoplasmopara cubensis* (Berk. & Curt.) Clint.).

Leaf Spot (*Phyllosticta cucurbitacearum* Sacc.)

This is a minor disease due to a pycnidial fungus which causes dirty whitish spots on cucumber leaves. Dotted over the spots are the minute fructifications of the fungus, each containing innumerable one-celled spores which ooze out in a sticky mass. The disease is known in South and Central Europe and has occasionally been reported in England (Moore, 1943).

Leaf Spot (*Sporidesmium pluriseptatum* (Karst. & Har.) Peck)

In Denmark outdoor cucumbers sometimes develop a disease which, with its brown-edged leaf spots that soon fall out, is very reminiscent of blotch. The attack is due, however, to the fungus *Sporidesmium pluriseptatum*, the conidia of which occur in great numbers on the under side of the spots (Fig. 175).

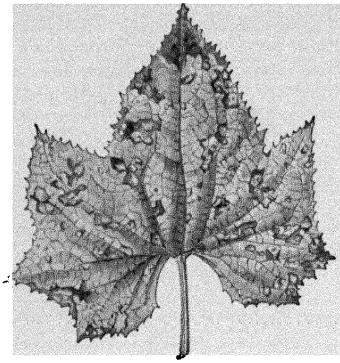


Fig 175. Cucumber leaf spot.

The usual fungicidal sprays and dusts give very irregular results against this fungus. Cucumbers should be planted as far as possible from the site of the previous year's crop and after the last fruit has been cut the plants should be collected and destroyed.

Pythium Fruit Rot (*Pythium ultimum* Trow and other species)

Rotting of greenhouse cucumber fruits at various stages of maturity due to infection by *Pythium ultimum* has been described by F. T. Brooks (1945). The fruits become soft and covered with a cottony white mould. Infection can take place through the uninjured skin, often at the tip of the fruit. The disease is encouraged by a moist atmosphere and by the cucumbers coming into contact with the soil or staging.

Other species of *Pythium*, notably *P. aphanidermatum* (Edson) Fitzpatrick, may cause a similar rot. So may *Sclerotinia sclerotiorum*, but this is easily distinguished by its large sclerotia (Fig. 176).

Control measures have not been worked out but it is obviously important to prevent the fruit from coming into contact with the soil or being splashed during watering.

Species of *Pythium* may also cause damping off but this is probably more often due to *Phytophthora* spp. (see tomato, p. 375).

Pythium aphanidermatum has also been found to cause a stem wilt and rot of mature glasshouse cucumber plants (Hickman, C. J., 1944). The stems were girdled by a dingy yellow lesion, extending upwards for 2–3 inches from the level of the soil, separated from the healthy tissue by a dark-green, watersoaked band. Watering with a proprietary fungicide containing a copper-ammonium compound checked the disease.

Canker (*Bacterium carotovorum* (L. R. Jones) Lehm.)

Symptoms. This disease is widespread, especially on hothouse cucumbers. The plants are attacked by a soft, wet rot just at the soil level. It gradually spreads downwards into the roots and a little way up the stem, killing the plant if it is not checked.

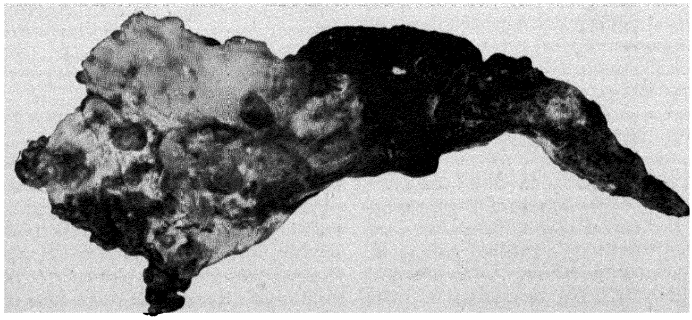


Fig. 176. Cucumber rotted by *Sclerotinia sclerotiorum*.

Cause. The damaged tissue is full of various kinds of bacteria and fungi whose rôle in the disease has not been fully investigated. The common soft-rot organism *Bacterium carotovorum* (L. R. Jones) Lehm. (see p. 257) is frequently found as well as species of *Fusarium*.

The disease appears especially where fresh manure has been placed close to the stem and where conditions are very moist.

Control. The tissue of the plants should be quite firm, not too soft. A couple of handfuls of gravel, sand or ashes placed close round the stems help greatly in warding off an attack. As far as possible one should avoid watering close to the stem. To ensure this it is suggested that a metal collar, a pot with the bottom knocked out or some similar device might be put round the base of the stem so that the soil inside remains dry while watering ensures adequate moisture outside and underneath.

Metal rings or similar devices will probably only be used where the grower has experienced much trouble from canker or where valuable seedlings are concerned. But it is not very costly or troublesome to cut pieces of thin tin to a suitable size and bend them round the stems, though they should not be pressed further into the soil than necessary, or they may impede the growth of new roots.

One should avoid using compost, or field soil containing remains of diseased turnip or carrots, as these are often full of *Bacterium carotovorum*.

In the early stages of an attack one can try to save the plant, for example by washing away the diseased tissue with a strong jet of water and packing gravel, mixed with a little flowers of sulphur and lime, round the base of the stem, or by dusting the stems with a mixture of 10 parts slaked lime, 2–3 parts powdered copper-sulphate crystals and 2–3 parts of sulphur dust (Bewley, 1923). The dusting should be repeated, especially wherever new soil is added to the beds.

Sometimes one can best save the plants by placing fresh field soil or very coarse sphagnum close round the stem. The cucumbers then root out again more easily, but the danger of a recurrence of the disease is greater than when chemicals have been applied round the stems.

Excellent results have been obtained by watering attacked plants with a solution of 1¼ oz. corrosive sublimate plus 3½ oz. saltpetre in 10 gallons of water followed by watering with 1:1000 potassium-permanganate solution. In some nurseries they water several times at intervals of a few weeks with about 1 oz. sublimate in 10 gallons weak saltpetre solution. Good results have also been obtained by watering every 14 days with 2–3 oz. Danish Tillantin per 10 gallons water.

Bacterial Leaf Spot (*Bacterium lachrymans* E. F. Sm. & Bryan)

This disease first appears as round or irregular transparent spots on the shoots and later on as well-defined, watersoaked, brown leaf spots, which dry up and fall out, just as in blotch. In the morning one often sees on the under surface of the spots small, clear, bacteria-filled droplets which dry to leave a chalky deposit. Watersoaked spots yielding a slimy white discharge may also arise on stems and leaf stalks. On the fruit the spots are at first very small, round and watersoaked, but later turn chalky white at the centre as the tissues die and dry out.

The disease is spread at pruning, by water splashes, and perhaps by insects. It can be transmitted on the seed, which may be disinfected by soaking for 5–10 minutes in 1:1000 corrosive sublimate solution, following by rinsing in clean water and drying.

Bacterial leaf spot is common in America and of late years has been reported in Germany. It has not yet been indentified in Denmark. There are several reports of its occurrence in England, notably in 1943, but Moore (1948) considers the identity of the disease requires confirmation by a competent bacteriologist.

Bacterial Wilt (*Bacillus tracheiphilus* E. F. Sm.)

This disease is due to a bacterial infection of the vascular bundles and effects cucumbers, melons and gourds. The leaves develop pale-

green spots that quickly spread and are followed by a general withering of the leaves and shrivelling of the stems. When the latter are cut a sticky, whitish, bacterial slime exudes from the bundles.

This is a serious disease of outdoor cucumbers in North America, where it is spread by the knife at cutting and by insects, especially by a leaf beetle. It is unknown in Denmark and in England, where, however, *Verticillium* wilt of cucumbers occurs sporadically every year (see p. 60).

Cucumber Mosaic (*Cucumis* Virus I)

Diseases of the mosaic type may be caused in cucumber by more than one virus. Ainsworth (1935) distinguished three forms of the disease, green mottle mosaic, yellow mosaic and mosaic, of which the first-named is the most prevalent and least destructive.

Cucumber mosaic was found in Copenhagen in 1918, and in 1923 it was widespread in cucumber houses. In 1924 mosaic was found in melons in houses in Odense (Danvig) but the disease is not general on melon. In North America cucumber mosaic is a virulent disease of glasshouse and outdoor cucumbers and has been very fully studied. In England it is widespread on glasshouse cucumbers, outdoor ridge cucumbers and vegetable marrows. The following account refers to the green mottle mosaic caused by *Cucumis* Virus 1.

The first indication of infection is the appearance of small, light-green spots on the young leaves. The leaf tips may turn yellow and the leaves become slightly distorted and curled backwards. In more advanced stages of the attack the leaves are conspicuously mottled and curled and the whole growth of the plant is stunted. The flowers are often few, small, and a little paler than normal. If the temperature exceeds 80° F. the fruit may be mottled with a lumpy skin and be unsaleable. In 1945 mosaic in English ridge cucumbers assumed such a severe form as to kill the plants. The leaves showed a greenish-yellow mottle with rusty marks along the veins and the terminal internodes were noticeably shortened. Then one or more runners wilted at the tip and within 48 hours the plant died (Ware, W. M., and Glasscock, H. H., 1943).

Host plants. *Cucumis* Virus 1 has a very wide host range including many common garden plants in addition to most members of the cucumber family (*Cucurbitaceae*), though not the water melon. In musk melon and the wild North American species *Micrampelis lobata* infection has been shown to be carried by the seed. The last-named plant, as well as species of *Physalis*, *Phytolacca*, and *Asclepias* may all serve as sources of infection there. So may the white bryony (*Bryonia dioica*) common in European hedges. The virus is transmitted by the common green aphis *Myzus persicae* and may also be spread by contact. To transmit the disease it is sufficient to break a few hairs on an infected plant by touching it with the fingers and then handle a leaf of a healthy plant. In hothouses the incubation period is 6–7 days, in the open air 12–14 days.

Infection from the soil has never been demonstrated, probably because *Cucumis* Virus 1 is unable to survive long outside the plant tissues. In leaves dried slowly, however, it remains viable for 2–3 weeks.

Seed from mosaic-diseased cucumber plants may give quite healthy plants. Of 10,000 plants grown in insect-free houses Doolittle found only one that had been infected from the seed. But in practice there is often circumstantial evidence pointing to infection from the seed, and Bewley considered this had been proved to occur at Cheshunt. Seed-borne infection is, however, in any case low, at most 1–2% of the seedlings.

Effect of environment. Cucumber mosaic is most virulent at 86° F., infection seldom occurring below 68° F. Soil conditions and manuring have an indirect effect, for strongly growing plants are the most easily infected. Plants whose growth is at a standstill are not very receptive to the virus.

Cucumis Virus 1 produces pale leaf colouration and perhaps curling of spinach. In North America it causes a mosaic disease of celery and capsicum. In tomato it causes a

mild form of mosaic, and under certain conditions leads to the production of strap-like or thread-like leaflets. In aquilegia and delphinium it causes stunted growth and pale veins. In lupins it causes " browning" which may be fatal. Other known host plants include arum, catmint, china aster, chrysanthemum, dahlia, honesty, lettuce, onion, periwinkle, petunia, primula, swede, tulip and watercress.

Cucumis Virus 2 causes mosaic disease of cucumber and melon but does not infect marrow or plants of the nightshade family (*Solanaceæ*). In North America there seems to be a separate virus that causes variegation and buckling of the leaves and fruit in vegetable marrows.

Cucumis Virus 1 is much less resistant than *Nicotiana* Virus 1 (see p 383); it dies in expressed sap in 3–4 days; it cannot withstand drying, heating to 140–158° F., nor an hour's treatment with 50% alcohol.

Control. Use seed from healthy plants. Keep cucumber houses and their surroundings free from any of the above-mentioned weeds and ornamentals which may harbour the virus. The young cucumbers should be looked over at frequent intervals and suspected individuals should at once be removed and burnt. The houses should be fumigated regularly to control aphides which would spread infection. Knives and hands should be cleaned by washing in soapy water after touching the plants. In cucumber houses individual infected plants should be cut off at soil level. When they have completely withered they should be removed and burnt. No varieties completely resistant to mosaic are known.

Ammonia Scorching

Ammonia fumes from fresh manure may scorch the leaves, causing white to brown withered spots to appear, especially along the edges of the lower leaves. These become dome-shaped, for the leaf surface continues to grow while the scorched margins cannot do so.

Scorching of this kind may somewhat check growth but is not particularly harmful when confined to the lowest leaves.

Chlorine Poisoning

Cucumbers have often been injured by being given stable manure, or latrine contents, to which chloride of lime had been added as a disinfectant.

The leaves suddenly develop yellow areas at the tips, along the edges, or between the main veins. The newly set fruits suddenly stop growing and are spoilt; often they shrivel from the tip. Generally the injury appears at irregular intervals, i.e. it appears suddenly, then an improvement occurs, followed by another bad period, and so on. When one realizes that the damage is due to injury to the roots by chlorine from the chloride of lime one can understand that the first outbreak follows application of the manure. Then when the chloride has been partly washed out the plants recover until damage to the roots again takes place.

One should make sure that chloride of lime is not added to the manure or latrine contents that are to be used. If one discovered that it has been added one should water the plants very heavily to try to wash it out as quickly as possible.

Bitterness

A tendency to bitterness in cucumbers is unfortunately quite common, especially amongst those grown in hothouses, but it has also occurred in cucumbers grown out of doors for sweet pickles. The bitterest fruits are dark outside and yellow inside but in most cases one cannot see from the outside whether a cucumber is bitter or not. The bitterness begins at the stem end and spreads through the flesh from there. There may be a noticeable difference in taste between two slices cut close to one another. A cucumber which is not bitter when pickled may become so later. Bitter cucumbers are most frequently met with from June to August. Hunderup Hothouse Cucumber and Tottenham Prolific may have a tendency to become bitter while Butcher's Disease Resister and H. V. Larsen's Improved are definitely less liable to it. On the whole the smooth types are far more liable to bitterness than the spiny-skinned kinds.

This is well known to many housewives and it is, therefore, easier to sell a spiny variety than a smooth one.

Various factors encourage bitterness in cucumbers. There is some difference of opinion as to which are most important but it appears that sudden changes in growing conditions are of great importance. The phenomenon is often encountered when a period of sunshine follows a long period of cloudy weather, when the plants are too dry, or when they are very severely pruned.

Other factors predisposing to bitterness are: damage to the roots by parasites, overwatering, use of too strong manure or of fertilizers containing chlorine, wide variation in atmospheric or soil temperatures and, finally, setting of too many fruits. Individual plants may vary greatly in their tendency to bear bitter fruits.

One should endeavour to maintain steady growth by supplying heat during cold nights, ventilating and shading in strong sunlight, watering and cutting at the appropriate times, and avoiding too strong manuring.

Bitter cucumbers are stained deeply by potassium iodide solution which may be useful in detecting them (T. Bacher).



Fig. 177. Shrivelled cucumbers.

Shrivelled Cucumbers

Frequently cucumbers on hothouse plants cease growing and shrivel when about 2–4 inches long, without any obvious cause (Fig. 177).

The phenomenon is most frequent at high temperatures, with a moist atmosphere and wet soil. It also seems to be worst on plants carrying many large fruit.

Several other conditions may stop growth of the young fruits: damage to the roots by chlorine (p. 279), too strong farmyard manure, too cold water, drought, draughts, big ranges in temperature, or heavy pruning, leaving too few leaves so that transpiration is reduced. A similar " non-parasitic damping off " of fruits in England is ascribed by Moore (1943) to faulty root action when the soil is too wet and compact.

Stoppage in Root Growth

The roots may be damaged, so that they turn brown and no new roots are produced for a time, if too fresh manure or manure containing chlorine is used (see p. 279), or if the plants are watered with very cold water.

This check to root development may cause injury to the fruits (see shrivelled cucumbers, above) or of the leaves (see drooping leaves, p. 281).

A root rot of cucumbers, associated with species of *Fusarium*, is common in England, where the primary cause is believed to be bad cultural conditions, such as lack of drainage and poor aeration of the soil.

Blind Shoots

Cucumbers in houses sometimes develop " blind shoots", i.e. the shoots suddenly stop growing and the tip ultimately withers, when side shoots develop to replace it. Even if these succeed, there has been a check to growth and, moreover, some side shoots may be destroyed also, so that it is some time before the plant begins to grow again.

Shoots may be killed by bugs and aphides

but usually these are not the cause of blind shoots.

The trouble is not due to parasites but to unsatisfactory conditions of growth which cannot always be precisely identified. For example, it may occur where there is a draught from a hole in the glass, or from the ventilator. Good results have been obtained by putting cones over shoot tips exposed to draughts. The most frequent cause, however, seems to be that the plants were growing too vigorously, i.e. they had been given too strong manure, especially an excess of nitrogen and also too much warmth. Blind shoot tips are therefore most frequent on plants with large dark-green leaves.

The larger the plants are when planted out the better. Large plants have a shorter time in which " the top can go " than small ones that take longer to attain their full height. The plants should not receive extra nitrogen until they have begun to set fruit. Temperatures during growth should not exceed 86° F.

Drooping Leaves

Cucumber leaves may develop transparent withered edges and in the course of a few hours the condition may spread over the whole leaf, which collapses as though scalded. The stalk remains normal. In some cases the leaves may have been noted beforehand to be pale yellowish-green between the veins; in other cases there was nothing peculiar about them.

This phenomenon is almost confined to the spring months. No parasite has been found associated with it, and after a time it ceases to occur, though affected plants suffer some check in growth.

In one instance the damage seemed to be associated with the use of too fresh cow dung in the beds.

Circumstantial evidence indicates that the trouble is due to some cultural fault. Cucumbers are a forced crop, grown at a high temperature and richly supplied with both food and moisture. If, therefore, they are checked even for only a short period, by a sudden rise or fall in temperature, or if the roots are damaged by too warm or too cold water or by too fresh manure, they receive a shock which finds expression in some kind of malformation of growth.

Sudden Wilting

In Denmark it frequently happens, generally in August, that outdoor cucumbers suddenly lose their turgidity and wilt. The trouble was particularly prevalent in 1939 and especially in 1934, when rain came after a long dry period. There was no sign of disease on the root, stalk or leaves except that the plants drooped and eventually withered altogether (Fig. 178).



Fig. 178. Sudden wilting of cucumber. Healthy plant on left.

In a locality where the matter was examined more closely the phenomenon was equally prevalent in soil where cucumbers had been often grown and where there had been none for 10–15 years.

Attempts to correlate appearance of the disease with some cultural condition in the various districts were unsuccessful except that everywhere the plants were said to have withered " after rain". In some places they

had been watered during dry periods and still many plants withered. Those that died were about to set fruit; they seem to be particularly susceptible to the trouble at that stage of growth.

Sudden wilting and death of ridge cucumbers at about the same time of year, due to infection by *Cucumis* Virus 1, has been described on p. 278.

Floral Abnormalities

In 1930 it happened at various places in Jylland that the outdoor cucumbers bore only male flowers or only an abnormally small number of female flowers. At one place where only Danish pickling cucumbers are grown, those raised in the greenhouse and planted out gave at first both male and female flowers and set normal fruit, but after a definite period the internodes became shorter, the leaves darker and only male flowers were produced. Some plants sown out of doors, which therefore developed later, produced only male flowers, while those sown in the hothouses and kept there remained normal throughout.

In another district the plants were of different varieties but the ovaries were not normal except on one or two and these set only a few fruits. Seed of the same varieties and consignments gave only normal plants in a third district.

From a place where there were only a few female flowers, seeds were saved from the few fully developed fruits produced, and these were sown in the following year alongside seeds from normal plants. Seed from the abnormal plants gave almost exclusively abnormal plants while the others were normal. Thus the abnormality was inherited but its first appearance was due to some external condition. It may perhaps have been, for example, a sudden spell of sunshine after a dull period with low soil temperature that caused the appearance of male flowers.

Summary of Cucumber Diseases

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A. Root and stem of seedlings soft and shrivelled	Damping Off	68
B. Roots of older plants turned brown	Stoppage in Root Growth	280
C. Soft wet rot at base of stem	Canker	276
II. Stems		
A. (1) Soft rot and shrivelling of stem in seedlings	Damping Off	68
(2) Spotting and sometimes cracking	Gummosis	273
B. Shoot tips diseased:		
(1) Cotyledons brown-edged, growing point dead ..	Blind Shoots	280
(2) White, mealy coating	Mildew	275
C. Older parts of the stem diseased:		
(1) Transparent spots with white surface	Bacterial Leaf Spot	277
(2) Oblong yellow-edged spots	Gummosis	273
(3) Sunken pale spots, turning dry and mealy, pink conidial pustules visible under lens	Anthracnose	272
(4) Stems wilt:		
(a) Slime exudes from the vessels	Bacterial Wilt	277
(b) Vascular bundles brown and ultimately black	*Verticillium* Wilt	60
(5) Stems rotting:		
(a) White woolly mycelium with large sclerotia..	*Sclerotinia* Rot	77
(b) Hairy grey mould	*Botrytis* Rot	59
(6) Soft wet rot at soil level	Canker	276

III. Leaves

		page
A. Leaves wilt suddenly:		
(1) Leaves flaccid, stem normal	Sudden Wilting	281
(2) Leaf edge transparent, then shrivelled; leaf looks as though boiled	Drooping Leaves	281
B. Leaf edges turn white-yellow-brown:		
(1) Cotyledons affected	Blind Shoots	280
(2) Leaf edges yellow, leaves sometimes yellow between veins at the tip	Chlorine Poisoning	279
(3) Leaf edges white, leaves possibly becoming dome-shaped, mainly affecting lower leaves	Ammonia Scorch	279
(4) Dark-greyish leaf edges, leaves somewhat crinkled	Potassium Deficiency	42
C. Pale yellow-brown leaf spots:		
(1) Angular brown spots that fall out	Leaf Spot	275
(2) Angular pale-green or transparent spots, becoming grey-brown and falling out	Blotch	274
(3) Rounded, brown spots that fall out, sometimes with white drops of mucus on the stems	Bacterial Leaf Spot	277
(4) Large round or irregular pale-green transparent spots, turning reddish-brown with pale margin and falling out (pink conidial pustules visible under lens)	Anthracnose	272
(5) Large yellowish or greenish-yellow spots that burst open	Gummosis	273
(6) Faint spots, wilting leaves, mucus in the vascular bundles	Bacterial Wilt	277
(7) Spots with grey hairy mould	*Botrytis cinerea*	59
(8) Indistinct pale-green or yellowish discolouration between veins; leaf edges transparent and shrivelling	Drooping Leaves	281
(9) Yellowing between veins, leaves gradually fading	Manganese Deficiency	39
(10) Small light-green spots or spotted crinkled leaves	Mosaic	278
D. White mealy covering	Mildew	275

IV. Fruits

A. No fruit sets	Male Flowers	282
B. Bitter taste to ripe fruit	Bitterness	279
C. Arrested growth:		
(1) Small shrivelled cucumbers	Shrivelled Cucumbers	280
(2) Larger fruit, constricted at one end	Blotch	274
(3) Fruit thin at stem end	Potassium Deficiency	42
(4) Fruit shrivelled from tip	Chlorine Poisoning	279
(5) Fruit yellow at tip	Nitrogen Deficiency	40
D. Fruit lumpy or variegated yellow and green	Mosaic	278

		page
E. Fruit spotted:		
(1) Transparent spots with chalky centre	Bacterial Leaf Spot	277
(2) Sunken spots with drops of gum	Gummosis	273
(3) Pale sunken spots with pink conidial pustules visible under lens	Anthracnose	272
(4) Sunken darker spots	Blotch	274
F. Rotten fruit:		
(1) Hairy grey mould	*Botrytis cinerea*	59
(2) Cottony, white mould:		
(a) with large sclerotia, first white then black	*Sclerotinia* Rot	276
(b) No sclerotia	*Pythium* Rot	276
V. Whole Plant Limp or Withered		
A. Stem and root normal	Sudden Wilting	281
	Drooping Leaves	281
B. Roots brown and shrivelled	Stoppage in Root Growth	280
C. Soft rot at soil level or below	Canker	276
D. Vascular bundles brown or blackish	*Verticillium* Wilt	60
E. Vascular bundles yield slimy mucus when the stem is cut across	Bacterial Wilt	277

DILL

Rust (*Puccinia petroselini* (DC.) Liro)

Rust occurs on dill in Sweden though not yet in Denmark. The plants may become studded by small rust-brown pustules of uredospores, followed by darker or almost black pustules of teleutospores. In very severe attacks the plants may wither. The rust of dill is probably a physiologic race of that found on parsley. Control measures are unknown.

Black Spot (*Phoma anethi* (Pers.) Sacc.)

This fungus, which is known in Denmark but not destructive there, was very destructive in a large seed nursery in Skane (Sweden). Small dark spots appear all over the green parts of the plant and many fuse together to produce large, irregular, dead patches. In these spots groups of minute dark pycnidia can be detected. Affected plants may die prematurely.

Control has not been attempted, but spraying with Bordeaux mixture at the beginning of an outbreak may probably check it.

EGG PLANT

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

Growth of infected plants is stunted, those most severely affected turn yellow and their lower leaves fade and fall off. These symptoms become progressively worse until the plant is killed. The wood of root and stem is stained dark brown and this discolouration may extend throughout the vascular system even into the fruit. In severe attacks the roots and base of the stem decay (see *Verticillium* wilt, p. 60).

Phomopsis vexans (Sacc. & Syd.) Harter

The disease caused by this fungus occurs in most places where egg plants are extensively grown but is as yet unknown in Denmark. In U.S.A. cases have been reported in which the average yield of the egg-plant crop in a district has been reduced to a fifth by this disease.

Many different parts of the plant may be attacked. On seedlings the fungus causes damping off and foot rot. Leaves may be attacked at all ages but the spots usually appear first on the lower leaves. They are well

defined, circular, grey or brown with a paler centre. Gradually as the spots age they become irregular and numerous small black pycnidia appear dotted over them. When the spots are large or numerous the infected leaves turn yellow and fall off.

Infection of the stems may either lead to a constriction at the point of attack, or to a grey, dry rot of the tissues. Sometimes the cortex falls away in a canker, leaving the woody cylinder exposed. The pith is generally discoloured. Adventitious roots appear on the stem above the lesion or the stem may break at that point.

Spots on the fruit are pale and sunken and may spread over the whole surface. The numerous small black pycnidia give them a characteristic appearance. Infection of the fruit seems to occur less readily than attack on the stems, but the fungus often grows from the stem through the fruit stalk into the fruit and sets up a black dry rot there.

Control. The crop should be grown in soil free from any egg-plant debris. Seed disinfection by soaking for 10 minutes in 1: 2000 corrosive sublimate solution may kill the conidia on the surface of the seed, but cannot reach any mycelium already inside the seed. Frequent spraying with Bordeaux mixture or lime sulphur sprays has given good control (Chupp, C., 1925).

HORSERADISH

White Blister (*Cystopus candidus* (Pers. ex Chev.) Lév.)

This fungus causes white conidial pustules on all above-ground parts of the plant but especially on the leaves. The infected parts are often thickened, contorted or twisted.

Shepherd's purse, cabbage and other cruciferous plants may also be attacked, but Togashi and Shibasaki (1934) have indicated that various physiologic races of the fungus occur. Klebahn's experiments (1937), however, indicated that shepherd's purse may become infected from horseradish. It is therefore necessary to reckon with a possible transference of infection from shepherd's purse to cruciferous crops and to try to get rid of that ubiquitous weed. Resting spores (oospores) are formed in the dead tissues and can survive for several years in the soil, hence rotation of crops is desirable. Debris from a diseased crop should be burnt.

Spraying with Bordeaux mixture, 16: 8: 100, has a good effect; it should be done as soon as an attack is seen and perhaps again twice later.

As the fungus can overwinter in the form of resting mycelium in the roots, planting material should only be taken from healthy plants.

Pale Spot (*Ramularia armoraciae* Fuckel)

This fungus causes a large number of small pale spots, with narrow red-brown edges, on horseradish leaves (Fig. 179). They may be so numerous as to destroy half the leaf surface.

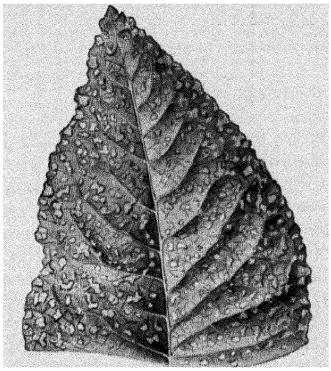


Fig. 179. *Ramularia armoraciae* causing spotting of horseradish leaf.

No experiments have been made to control this disease but it could probably be kept in check by a fungicidal spray or dust, such as Bordeaux mixture or sulphur powder.

Bacterial Leaf Spot

On horseradish the bacterium *Pseudomonas campestris* var. *armoraciae* McCullock may cause translucent spots which finally become yellow or brown. Similar spots have been produced by it on cabbage and bean but only in inoculation experiments. This disease

occurs in North America but has yet to be found in Britain.

Rot

In eastern North America stored horseradish roots have for years suffered losses from a soft rot which partly arises in the cortex and spreads throughout the thickened root stock and partly starts in the core where the bacteria follow the vascular bundles. The trouble has been attributed to *Bacillus alliariae* Amori but this has not been proved. Some control has been attained by rolling the planting material in sulphur dust so that it is completely coated with the powder, or by dipping it for 15 minutes in 1: 1000 corrosive sublimate solution; soil lacking lime is used for protection in storage.

Stored horseradish may also be attacked by *Sclerotinia* (p. 77), by a species of *Fusarium* and by putrefactive bacteria (Böning, K., 1936).

Dark Rings

In a cross-section of a horseradish root a dark ring may sometimes be seen, a little way in from the surface. In longitudinal section the discolouration forms two dark parallel streaks. Often the dark colour occurs in spots more or less close to one another, rather than in a continuous band. The attack may be so severe, however, that the whole root becomes grey or black. The discolouration may also start as yellow spots which slowly turn brown.

Gum is often produced in the diseased tissues and both *Verticillium* (Blattný, 1927) and bacteria (Klebahn, 1937) have been isolated from them. Workers are not in agreement regarding the species of *Verticillium* present. Klebahn, who worked with the bacteria, did not identify them, but in his experience bacterial infection was much more general than that by *Verticillium*. Soft rot (p. 257) may attack damaged roots and a form of black rot (p. 256) may occur on the leaves.

Control. Propagating material should, if possible, be taken only from plants which have been carefully examined and found to be quite free from discolouration in all parts of their roots. If such careful selection is impracticable all planting material should be examined on both cut surfaces and if the slightest discolouration is observed it should be discarded.

It is best to disinfect the knife between each cut, either by dipping it in boiling water or into a liquid disinfectant such as 2% formalin and then in clean water. The shoots should be started in clean sand or in soil which has not been used for horseradish before.

As long a period as possible should elapse before horseradish is replanted in soil where an infected crop has grown, and potash and stable manure should not be spared. Damp soil rich in humus, but not too rich in lime, is best suited to the crop.

Mosaic

In North America Pound (1948) has described a mosaic disease of horseradish caused by a virus closely allied to that causing black ring spot of cabbage (see p. 258).

LEEK

Smut (*Tubercinia cepulae* (Frost) Liro)

Light-grey blisters appear on the leaves of leek seedlings and soon burst, exposing a black spore powder (see onion smut, p. 298).

Heterosporium allii Ell. & Mart. and *Stemphylium botryosum* Wallr.

These two fungi cause spots on leek leaves, covered respectively with a grey and black powder of spores. Both are fairly common in Denmark and cause withering, especially of the outer leaves (see onion, p. 302).

White Tip (*Phytophthora porri* Foister)

This disease, described by Foister in Scotland in 1929 probably has long been known to leek growers. He found considerable spread of white tip during the three years of his investigation, 1928–30 (Foister, 1931).

Symptoms. The leaf tips turn yellow, then die and become white; these white tips may be as much as six inches long but become overgrown by dark secondary moulds in the process of decay. They may be twisted or curled but more often hang limply. Whitened areas may be found at the edge anywhere in the upper

part of the leaf. Halfway down or at the leaf base there is often a translucent zone.

Large plants rot if severely attacked and disintegrate at about soil level if one attempts to pull them.

If all the leaves are infected the value of the crop is greatly reduced. Plants severely attacked while still young remain small. Even those only lightly attacked have a reduced market value because they wither more quickly than normal plants when prepared for sale.

Cause. The disease is caused by a particular species of *Phytophthora* which has been fully studied by Foister. The fungus can easily be found in the yellow translucent tissues, less easily in the dead white parts.

Control. In dealing with *Phytophthora* diseases it is only natural to try controlling them by copper fungicides. Sprays and dusts of this description have given promising results in English and Scottish experiments. When the leek leaves are small it is doubtless desirable to add a spreading agent to the Bordeaux or Burgundy mixture.

Fusarium Wilt (*Fusarium* spp.)

The roots rot, especially while the plants are standing on the bench (see onion, p. 303).

Rust (*Puccinia porri* (Sow.) Wint.)

Rust is not uncommon on the leaves of leeks, especially in seed crops, but is seldom responsible for much damage.

LETTUCE

Ring Spot (*Marssonina panattoniana* (Berl.) Magn.)

This fungus causes oblong sunken spots, at first watersoaked, then yellowish and finally reddish or light brown, like small patches of slug damage, on the stem and the under-side of the midrib of lettuce leaves. Similar spots appear on the leaf blade, where they may be round or angular, defined by the larger veins. In later stages of the disease the tissue in the centre of these spots dies and falls out, so that the leaves look as though perforated by hail. The holes are usually about $\frac{1}{10}$ inch in diameter with brown edges (Fig. 180). The attack starts on the outer leaves and spreads inwards. Endive is also attacked by this fungus, so is the wild lettuce *Lactuca scariola* and probably the weed *Crepis*.



Fig. 180. Ring spot of lettuce (*Photo Dept. Agric. Scotland*).

Ring spot is known in North America and in Europe, especially in Great Britain, Holland and Germany. In England it is seldom seen in glasshouses or frames but is particularly prevalent in spring on plants that have overwintered out of doors, especially if there has been much rain. Diseased seed plants give diseased seed, though there is no visible sign of infection in the seed. Seedlings become infected from the seed coat during or after germination.

Control. Stevenson (1939) recommends disinfecting lettuce seed to control ring spot. Bleaching powder is well mixed with ten times its weight of water and left to settle for half an hour. The clear liquid is then strained through muslin and the seed steeped in it for 4–6 hours, dried and sown at once. A small quantity of

a wetting compound may be added to the solution.

Other control measures include rotation of crops and destruction of the remains of all infected crops after the marketable heads have been cut. In greenhouses care should be taken to keep watering at a minimum and to avoid splashing soil on the plants. Seedlings may be sprayed with a copper fungicide, but this is impracticable with maturing lettuce as the copper deposit on the leaves would render it unfit for human consumption.

Other Leaf Spots

The fungus *Pleospora herbarum* (Fr.) Rabenh., which has the conidial stage *Stemphylium botryosum* Wallr., was found to cause leaf spot of lettuce in West of England greenhouses in 1930. A similar disease was afterwards found on outdoor lettuce.

The first symptoms are small light-brown spots on the leaves, usually round but sometimes angular or oblong. The spots enlarge, join up and may cause large parts of the leaf to become brown and wrinkled. On the outer leaves the centre of the spots may fall out so that they look like hail-damaged plants. Often the spots have a dark-brown edge or bear a number of darker concentric lines.

Septoria lactucae Pass. causes light-yellow irregular blotches up to $\frac{1}{2}$ inch across, which turn brown and become covered with minute dot-like pycnidia. In later stages of the disease the spots may join together, or fall out as described for *Pleospora* above. The fungus is seed-borne and its pycnidia can be detected on infected seed by examination under a binocular microscope (Moore, W. C., 1940). The disease is widespread in Europe and North America.

Control measures are similar to those for ring spot.

Rust (*Puccinia opizii* Bubak)

This is rather a rare rust which has its cluster cup (æcidial) stage on lettuce as well as on wild species of *Lactuca*, *Crepis*, *Sonchus* and *Lapsana* and its alternate uredo and teleuto stages on sedge, especially *Carex muricata*. It has occurred in East Anglia, also in Poland, Germany, Rumania and Holland and has occasionally been detected on lettuce imported from the last-named country (Moore, W. C., 1946b). Other rusts produce æcidia on cultivated lettuce in North America and Australia.

Downy Mildew (*Bremia lactucae* Regel)

Lettuce out of doors in frames and in greenhouses is often attacked by downy mildew. The upper surface of diseased leaves is marked by rather vague pale or brownish spots, the under-side of which is covered by a thin whitish mould (Fig. 181). When conditions are favourable the fungus may spread very rapidly.

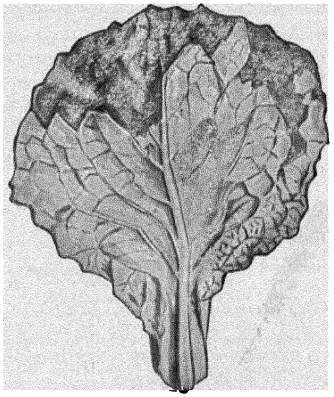


Fig. 181. Downy mildew on underside of a lettuce leaf.

It has a low optimum temperature, 43–50° F., and is hence favoured by cool weather and by low temperatures in houses and frames. Young plants are more susceptible than older ones. The fungus may overwinter in the soil but seems unable to survive severe frost.

Morphologically similar downy mildews occur on many weeds of the lettuce family (*Compositae*) but it is unlikely that most of these can attack lettuce. Ogilvie (1945) has found two races of downy mildew on lettuce, one of which infects the wild lettuce *Lactuca virosa* while the other does not. Both occur on

L. serriola (= *L. scariola*). It is often impossible to establish where the infection of cultivated lettuce comes from, but one must remember that the conidia can be spread from a diseased crop in many ways, through the air, by adhering to boxes and baskets used for lettuce and on clothes or boots of workers. Seed-borne infection has not been demonstrated.

Control. Soil disinfection has been tried once or twice in America without satisfactory results, perhaps because of reinfection of the house by some other means.

It has been shown to be very useful to spray seedlings once or twice with Bordeaux mixture 8: 8: 100, but not for 3–4 days prior to transplanting, and to spray again about 4 days after transplanting. Just before or after transplanting spraying is likely to be injurious.

It is very important to water carefully so as to avoid wetting the leaves. Watering should only be done when absolutely necessary and the houses should be kept well ventilated. If necessary the temperature may be kept 5 or 6° higher than usual at night. When lettuces are grown in pots the leaves can be kept drier than when they are planted out in a bed.

In all parts of the world lettuce varieties have been found to differ greatly in susceptibility to downy mildew. In Denmark the varieties King of Hearts and May King are considered resistant while Leppermann is susceptible, as is Tempo if grown during the short days. Schultz and Röder (1938) found Buse's brown, Maikönig forcing, Böttner's forcing and Riesenkristellkopf fairly resistant in Germany. At Long Ashton, Ogilvie (1943) found Gotte à forcer, Golden Ball, Loos Tennisball and Early French Frame resistant to downy mildew while Cheshunt Early Ball, Trocadero, Sutton's Imperial and May King were very susceptible. Owing to the existence of physiological races of the fungus, varieties found resistant in one experiment may not always prove to be so.

Grey Mould and Red Leg (*Botrytis cinerea* Pers.)

Grey mould may cover a greater or smaller portion of a lettuce head and cause it to rot. This is especially likely to occur if the head has been damaged in some way and if there is a little dead tissue about, as from marginal spot or tip burn (see also p. 290). In forced lettuce and outdoor white lettuce much more serious losses may arise by infection of the stem of young plants. This causes a reddish sunken wound, "Red-leg" which rapidly kills the plant, especially in overwintered seedlings transplanted in spring (Abdel Salam, 1934). Later in the season the stem wounds may heal and the plant mature more or less normally. Dusting with Folosan is said to afford some protection (see further, p. 595).

Sclerotinia Disease (*Sclerotinia* spp.)

These fungi gain access to withered leaves at the base of the plant and spread rapidly from them into the living head, which after a couple of days becomes limp and appears as though boiled. The heads may also appear to have fallen over, or to have been cut at the base. These fungi are easily recognized by their profuse development of white mycelium and their large, black sclerotia.

Sclerotinia sclerotiorum is one cause of this rot, but so also are other allied species such as *S. minor* Jagger, found in North America, Bavaria and Alsace (Flachs, K., 1931; Labrousse, F., 1930). The former species will attack almost any herbaceous crop, the latter has already been found on beans, asparagus, radish, etc. The clover rot fungus (*S. trifoliorum* Erikss.), common in red clover fields, also occasionally attacks lettuce.

The control measures described for *S. sclerotiorum* on p. 79 are applicable to all these species.

Rhizoctonia Disease (*Corticium solani* (Prill. & Delacr.) Bourd. & Galz.)

This disease is particularly feared in North America. The fungus is very common in European soils also and may therefore probably from time to time cause the death of lettuce plants. Abdel Salam (1933) has described its attack on frame and glasshouse lettuce in England.

Symptoms. When the heads are formed,

small rusty or chocolate-coloured spots appear on the under-side of the midrib of those leaves in contact with the soil. The midrib and leaf blade soon wither but the stem of the plant is less susceptible. Wilting of the outer leaves on sunny days is one of the first signs of the disease. Gradually the heads decay completely; at first they are brown and slimy, then they dry up and turn almost black. The brown mycelium of the fungus with its small dark irregular crust-like sclerotia permeates the dead tissues.

The disease is favoured by damp and even more by high temperatures. See the detailed description of the fungus on p. 68.

Mosaic (*Lactuca* Virus 1)

The first symptom of lettuce mosaic is a clearing of the leaf veins, making them appear more prominent. Later a rather vague irregular mottling with shades of green may develop or the whole leaf may become yellow and hard-looking, as though damaged by wind or frost. The leaf surface may be abnormally crumpled and its shape modified. There may also be tiny brown spots between the veins, or brown lines along the veins, and scorching of the leaf edges. The whole head is dwarfed and does not heart properly. When the crop " bolts " plants infected with mosaic remain stunted and may have brown spots on the stem and flower head. In cos lettuce browning of the veins is rare, but scorching of the leaf edges often occurs. Cabbage lettuces differ considerably in reaction, according to Ainsworth and Ogilvie Trocadero, Premium, Lee's Immense, Harrison's Green Winter and Stanstead Park seem to suffer least.

The virus is transmitted by lettuce seed and is spread in the field and glasshouses by aphides, including *Myzus persicae* and *Macrosiphum gei*. It can infect groundsel and sow thistle (*Sonchus asper*) and also sweet pea and culinary pea, causing a " streak " disease in the former and mottling of the leaves in the latter. It appears unable to infect dandelion.

Control measures include the use of seed from virus-free plants and eradication of weeds, especially groundsel, which may carry the virus over from one lettuce crop to the next. In glasshouses regular nicotine fumigation should be done to control aphides. With winter lettuces sown out of doors, fairly late sowings or sowing in frames tend to reduce aphis infection and so help the crop to escape infection. For the same reason lettuces planted in open or hilly situations are liable to suffer less than those surrounded by trees or buildings. Consideration may also be given to growing the varieties which suffer least from the disease.

Dandelion Yellow Mosaic on Lettuce

This virus causes conspicuous bright yellow spots and rings on dandelion leaves. When transmitted to lettuce it causes the young leaves to be bronzed, narrowed and stunted. Minute light-brown dots appear between the leaf veins and join up to form patterns of dark-brown or black lines bordering the veins. Leaf tips and margins may be scorched. In glasshouse lettuce this necrotic phase is followed by yellowing of the leaves which remain small and malformed. The disease has been well described and illustrated by Moore (1946), who believes it to have been present in the south of England at least since 1933.

Dandelion yellow mosaic is transmitted by the aphides *Myzus ornatus*, *M. pseudosolani*, and *M. ascalonicus*, but not by *M. persicae*. Control measures obviously include eradication of mosaic-infected dandelions and discouragement of aphides.

A less severe brown spotting and distortion of lettuce leaves may be caused by infection with the virus of tomato spotted wilt (Tompkins, C. M., and Gardner, M. W., 1934).

Tip Burn

Shrivelling of lettuce leaf edges may be due to infection by *Pseudomonas marginalis* (Brown) Stapp and scorching to potassium or phosphoric acid deficiency. Probably more common than either is the physiological disorder known in North America as tip burn. Similar symptoms occur in Denmark but it is not clear if the cause is the same; indeed, the cause is somewhat obscure even in America.

The disease begins as small brown spots near the edges of the leaves. As a rule they occur where the finer veins come together ¼–½ inch in from the leaf edge. These small spots seem to hinder water from reaching that portion of the leaf which lies beyond them, hence it withers, turns yellow and dies so that a brown band appears along the edges of the leaves. The dead leaf edges may be found inside the head so that they are not seen until the outer leaves are stripped off.

Factors conducive to the production of dead leaf edges are: excessive moisture, great variations in the amount of moisture, lack of light, high temperatures and overmanured soil, especially an excess of nitrogen. Vigorously growing plants are more likely to develop the condition than those growing more slowly. According to Dutch experiments, lettuce is sensitive to an excess of salt in the soil. Good results have been obtained by using fertilizers free from chlorine, viz. monoammonium phosphate and potassium nitrate. Experiments have also shown that varieties rich in sugars are less susceptible than those with a low sugar content.

In Denmark Leppermann and May King have proved fairly resistant to tip burn and Tempo very susceptible.

In English glasshouses Western finds that steam sterilization of the soil predisposes to tip burn and that it is worst in heated houses and after prolonged dull weather. These factors are all conducive to soft growth, and tip burn sets in when the plants are exposed to strong sunlight.

Control measures include maintaining steady moderate growth and avoiding excessive moisture.

Stoppage in Growth

Naumann described the result of watering lettuce seedlings with very cold water. The cotyledons become curved, cease growth and development of the plant is checked.

Summary of Lettuce Diseases

		page
I. Young Seedlings		
Brown roots	Damping Off	68
II. Larger Plants		
A. Shrivelled and dead leaf margins:		
(1) Leaves arched, margins dry, heads loose and worthless	Potassium Deficiency	42
(2) Leaves die from the edge inwards, leaf blades may be blue-green and stiff	Phosphoric Acid Deficiency	41
B. Leaf spots and blotches:		
(1) Yellowish or brownish blotches with a fine whitish mildew on the lower surface	Downy Mildew	288
(2) Yellow or reddish spots, their centres often dropping out	Ring Spot	287
(3) Small brown spots with darker edges or dark concentric rings, centre sometimes falling out	*Pleospora* Leaf Spot	288
(4) Yellow or brown spots dotted with tiny pycnidia	*Septoria* Leaf Spot	288

		page
(5) Large dead areas with a hairy grey mould	Grey Mould	289
(6) Brown blotches on outer leaves, brown mycelium visible under a lens	*Corticium solani*	289
(7) Brown blotches on outer leaves and reddish colour of heart leaves in packed lettuce heads..	Suffocation	41
(8) Small groups of orange-yellow cluster cups	Rust	288
C. Generalized decay of the head:		
(1) Outer leaves destroyed, heads spotted or decayed, or red-brown lesions on stem	Grey Mould	289
(2) Dense white mycelium with large sclerotia	*Sclerotinia* Rot	289
(3) Fine brown mycelium, sometimes with small scurfy brown sclerotia	*Corticium solani*	289
(4) No fungus present, heart leaves watersoaked and rotting	Bacterial Rot	257
D. Leaves deformed or mottled all over:		
(1) Vague mottling of leaves and stunting of the plant	Mosaic	290
(2) Dead brown or black spots and lines along the veins, young leaves much stunted and distorted..	Dandelion Yellow Mosaic	290
(3) Leaves with pale spots, bearing fine white mould on under surface	Downy Mildew	288
(4) Leaves stiff and erect	Nitrogen Deficiency ...	40
(5) Seed leaves curved, growth stopped	Check to Growth	291
E. No head, plants small (seldom occurs)	Phosphoric Acid Deficiency	41

MELON

Fruit Rot

A melon may rot very suddenly. The whole of the inside may be converted into an evil-smelling liquid mass without any obvious mark of disease becoming visible on the outside. Finally the fruit collapses or the liquid leaks out through holes.

This trouble had occurred sporadically in Denmark before 1935 but in that year there were very extensive outbreaks of melon fruit rot. Innumerable bacteria were present in the liquid but they were not identified precisely.

Such a disease has been attributed to *Bacillus melonis* Giddings, but Erwin F. Smith considered that identical with *Bacterium aroideae* (Towns.) Stapp, one of the common soft-rot organisms which can attack many different plants.

One should try to ensure that the fruits remain fairly dry, and avoid damaging them. As soon as one is discovered to be rotten it should be removed and destroyed.



Fig. 182. Wilting of melon.

Sudden Wilting

It frequently happens that when the first fruits on a melon plant are about to ripen the plant suddenly wilts and then shrivels up completely (Fig. 182), without any obvious decay at the foot or other indication of attack by a parasite. Varieties differ somewhat in their susceptibility to this trouble.

As renewal of soil or its disinfection seems to help prevent wilting it seems that it is probably due to soil fungi such as *Verticillium* (see p. 60). In England melon wilt has been attributed to *V. albo-atrum* and to rotting of the roots by species of *Fusarium*.

Foot Rot

Melon plants may rot just above soil level and if the decay spreads the plant is killed. Both bacteria and species of *Fusarium* may be found in the diseased tissue but apparently no experiments have been made to decide which is the primary cause of decay and which are secondary invaders. Stem rot of melon in England has been attributed to *Bacterium carotovorum* (Jones, L. R.) Lehm (Moore, W. C., 1943).

One should endeavour to avoid the disease by using clean soil, perhaps spreading gravel or ashes round the base of the stem, planting the plants high and avoiding splashing the stem during watering. One should also remove enough leaves to ensure free ventilation.

Several melon growers regularly give a couple of waterings with a solution of corrosive sublimate and saltpetre to avoid attack (see p. 73).

We have seen a single case where the leaf edges turned yellow after this treatment.

If the disease appears the soil should be removed round the base of the stem, the diseased tissue should be scraped out and the stem base surrounded with charcoal, or with a mixture of gravel, flowers of sulphur and lime, or of two parts finely ground copper sulphate, two parts flowers of sulphur and ten of dry slaked lime (Bewley, W. F., 1923).

Cracked Fruit

Melons may develop numerous cracks in the skin so that it looks as though the usual network of veins is too deeply sunk. Gummy drops appear in the cracks.

In practice cracking has been successfully treated by smearing the affected fruit with thick milk of lime.

The cause of the trouble is uncertain but potassium deficiency and irregular watering have been suggested as responsible.

Gummosis (*Cladosporium cucumerinum* Ell. & Arth.) may also attack melon fruit (see p. 273).

Summary of Melon Diseases

		page
I. Root		
A. Base of the stem soft and slimy	Foot Rot	293
B. Roots brown, dry, perhaps cracked	Brown Roots	376
II. Stems		
A. Plant wilts and dies, either as a whole or individual branches:		
(1) No visible rot, whole plant wilts, usually as fruit begins to ripen	Sudden Wilting	293
(2) White mycelium with large sclerotia	*Sclerotinia*	77
(3) Brown vascular bundles	*Verticillium* Wilt	60
(4) Whitish slime, stems shrivelled	Bacterial Wilt	277
B. Sunken dry, pale, mealy spots (light red conidial pustules visible under a lens)	Anthracnose	272
III. Leaves		
A. Surface mottled with light and dark green	Mosaic	278
B. White, powdery covering	Mildew	275
C. Leaf spots:		
(1) Small, brown, angular	Blotch	274
(2) Large, pale-yellow, translucent spots, turning brown and becoming confluent (pale-red conidial pustules under lens)	Anthracnose	272
D. Wilted leaves	see II. Stems, above	
IV. Fruit		
A. Surface cracked (network appears too coarse) sometimes with exudation of gum	Cracked Fruit	293
B. Pale sunken spots, pink conidial pustules visible under lens	Anthracnose	272
C. Fruit rotten:		
(1) White mycelium with large sclerotia	*Sclerotinia*	77
(2) Inside liquefied and leaking	Fruit Rot	292

MINT

Rust (*Puccinia menthae* Pers.)

Puccinia menthae is common on wild and cultivated mint and sometimes occurs in forced crops. All four stages of the rust occur on the same plant; there is no alternate host. In the spring the young infected shoots are distorted and twisted with conspicuous swollen stems and leaves covered with the orange cluster cups of the æcidial stage. Later in the season small brown pustules of uredospores break out on the under-side of the leaves of shoots that were hitherto normal, and are followed by the darker brown teleutospore pustules. When these pustules are numerous they render the shoots unsaleable.

Control. The rust overwinters as spores on the buds of the plants which were rusted in the previous summer. These shoots are reinfected as soon as they begin to grow. Steeping the plants in water at 112° F. for 10 minutes destroys the fungus without damaging the mint and should be a regular practice with roots to be forced. Plants freed from rust in this way and planted out of doors are likely, however, to contract the disease again from wind-blown spores sooner or later,

Ogilvie and Brian (1935). Mint also sometimes suffers from *Verticillium* wilt (see p. 60).

MUSHROOM

Brown Blotch or Bacterial Spot (*Pseudomonas tolaasi* S. G. Paine)

Mushrooms grown in too warm and moist an atmosphere may be destroyed by purplish-brown spots on the cap (Fig. 183), in which

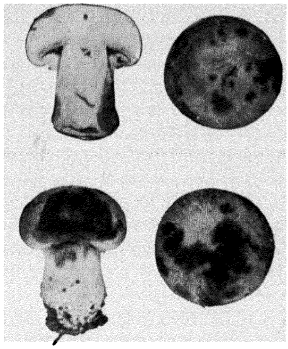


Fig. 183. Bacterial spot of mushroom (*S. p. F.*).

slimy drops may be found by closer examination. These drops teem with bacteria, which may be spread about the crop by various insects. Mushrooms may be destroyed by this disease before they attain the size of a pea.

The houses must be well ventilated after watering and the temperature should not rise above 55° F. Diseased fruit bodies should be removed and the attacked parts of the bed watered with chloride of lime (1 lb. in 20 gallons water) (see p. 298).

Brown Plaster Mould (*Papulaspora byssina* Hotson)

Occasionally large crusts of felted mycelium appear on the surface of the manure and of the casing soil and slowly turn brown, owing to the production of minute sterile resting bodies by the fungus. These look like tiny yellow or brown sand grains.

Brown plaster mould is introduced with the manure. In North America it is prevalent; in Denmark, where it has been known since 1931, and in England it is much less troublesome. Ware (1934) found that if the manure is good and in good condition mushrooms will be produced in infected beds and even in the brown mould patches. On the whole, however, presence of the mould seems to reduce productivity of the bed.

Green Plaster Mould (*Myceliophthora lutea* Cost.)

On examining unfruitful mushroom beds one sometimes finds that the white mycelium of the mushroom has become surrounded by that of another fungus, which is at first also white, but soon turns yellow and ultimately grey-green. The fungus comes in with the manure and is said to be able to spread in sour wet manure. It may also be found on the surface of the beds. Direct control measures seem hopeless (see further on p. 297).

White Plaster Mould (*Scopulariopsis fimicola* (Cost. & Matr.) Arn. & Barthelet = *Monilia fimicola* Cost. & Matr.)

This fungus has a world-wide distribution and is of great importance in mushroom cultivation, especially where wet or too old manure is used. Then the fine white mycelium spreads in great quantity over the beds until their surface appears to have been powdered with chalk. Old patches of the fungus may take on a faint pinkish or greyish hue. Whether or not cropping of the mushrooms is affected by the plaster mould apparently depends on whether the latter succeeds in invading much of the bed before the mushroom spawn gets a start.

Control is first and mainly by preventing infection of the beds. When the attack breaks out before the beds have been covered with casing soil the infected manure and some of that surrounding it should be removed. Bewley and others (1937) suggest the following control measures for white plaster mould. Remove the infected casing soil, placing it in a paper-lined box close at hand. Then remove the surrounding casing soil until the compost

below is uncovered over an area slightly greater than the infected patch. Remove the white infected compost and fill up the hole gradually with acid peat, moistening it thoroughly with dilute acetic acid from a small syringe (1 part of 33⅓% acetic acid in 7 parts water) and re-cover with fresh casing soil.

White Mould (*Mycogone perniciosa* Magn.)

This is a parasite of the fruit body in mushrooms and other gill fungi. Diseased fruit bodies are often transformed into unrecognizable lumps or, if the infection occurs comparatively late, they have a short thick stalk and hardly any cap. Their whole surface is covered by a close white mould and they soon break down into a stinking rotten mass. The term " bubbles " has also been used for this disease in England.

The fungus is said to be introduced with the casing soil, and use of fresh field soil from arable land is recommended. Local outbreaks in a bed can be treated by watering with formalin 1:50. The brown race of cultivated mushroom is resistant to white mould. The white mould layer of the fungus contains two-celled resting spores and sometimes also a conidial stage something like that of *Verticillium*. A similar fungus, *Mycogone rosea*, has been found attacking mushrooms in England by P. H. Williams (1939). The surface of the diseased caps was dark brown and the underlying flesh was stained yellow.

Dactylium dendroides Fr. is a fungus attacking mushrooms and wild toadstools, which covers them with a close downy mycelium often with a pinkish tinge, and causes them to rot. It, too, probably comes from the casing soil.

Verticillium and Cephalosporium Diseases

Several species belonging to these allied genera of moulds have been described as causing disease in cultivated mushrooms.

Verticillium malthousii Ware was first discovered in Kent in 1929 and is now known to be widespread in England (Ware, W. M., 1933). Sometimes it causes shapeless, deformed mushrooms, covered with a fine white short mould, sometimes greyish-white spots on mushrooms of a normal shape, sometimes only white patches on the gills or stalk. This difference in symptoms depends on the stage of development at which the mushroom becomes infected. Even the most deformed fruit bodies do not rot but become firm and somewhat leathery in texture. Conidia of the *Verticillium* are produced in vast numbers in minute slimy heads on the mould and are spread about the crop by *Sciara* flies and other insects. Control measures suggested by Ware include keeping down insect infestations, removing and destroying all diseased fruit bodies, sprinkling the beds only after this has been done, and maintaining the temperature at 50–55°F. F. C. Atkins (1945) recommends spraying diseased patches of the bed with Bordeaux mixture, 1:1:50 or 2:2:50, or dusting with copper lime dust.

A closely related fungus, *Verticillium psalliotae* Tresch., has been described on the brown variety of cultivated mushrooms in Denmark (Treschow, C., 1941). This forms a fairly thick layer of mycelium over the caps, which become deformed and spotted with various shades of brown. Infected fruit bodies often have a very small cap with a greatly swollen stalk and shrivel up, becoming tough and leathery. The fungus does not attack mushrooms at temperatures over 70° F. and seems unable to infect the white-capped strain.

Cephalosporium costantinii F. E. V. Smith was described in England in 1924. It causes mushrooms to become deformed, spherical, or pear-shaped bodies like those resulting from mycogone attack, but dry, brown and elastic in texture. Treschow found that *C. costantinii*, unlike *V. malthousii* and *V. psalliotae*, could attack the white strain of mushroom as readily as the brown one.

Gill Mildew Disease (*Cephalosporium lamellaecola* F. E. V. Smith)

All the above described species of *Verticillium* and *Cephalosporium* cause spots on the

surface of mushrooms and, in favourable circumstances, reduce them to dry, misshapen lumps. The present fungus only attacks the gills, which tend to fuse together and have a mildewed appearance. Affected mushrooms are still edible but do not keep well, as their gills tend to rot.

Competitive Fungi

Stable manure inevitably contains the spores of many kinds of fungi. Many of these are killed by the high temperature during composting, and if the mushroom spawn is introduced to the bed at the correct moment it usually grows away ahead of the remaining fungi. But under unfavourable conditions and if there are other sources of contamination, as from boots or tools, infection of the bed by competing fungi may forestall growth of the mushroom mycelium. Competition of this kind has already been mentioned under the heading of brown and white plaster moulds. Between the mycelium of species of *Fusarium*, especially *F. oxysporum* Schlecht. and *F. solani* (Mart.) Sacc. var. *martii* (Appel and Wollenw.) Wollenw., and the mushroom mycelium there is, however, a reciprocal hostility or "antagonism." Growth of one mycelium excludes that of the other even at some distance. Presence of these *Fusaria* may lead to individual mushrooms becoming brown inside and tough, or to many small button mushrooms remaining undeveloped. Sometimes the mushroom growth may be restricted to a single crop and sometimes there is no crop at all. Sterilization of the casing soil by heat kills these moulds and hence prevents trouble from that source.

Mushroom Bed Invaders

The false truffle *Pseudobalsamia microspora* Diehl and Lambert produces nodular, cream-coloured or pinkish hard fruit bodies, from $\frac{1}{8}$ inch to 1 inch across, buried in the casing soil (Williams, P. H., 1936).

Xylaria vaporaria Berk., called "seaweed" by the growers, forms tough, branched, black resting bodies in the casing soil. Its white mycelium permeates the underlying compost. According to Wood (1935) the sclerotia send branches up to the surface of the casing soil, where they form pinkish bodies flush with the surface. The sclerotia are said to have a strong smell like cucumber, detectable by crawling over the surface of the beds and smelling the casing soil.

Other invaders of mushroom beds include various toadstools such as *Clitocybe dealbata* (Fr.) Gillet (Buddin and Ware, 1933), *Clitopilus cretatus* (B. & Br.) Sacc. (Ware, 1935), *Panaeolus subbalteatus* B. & Br., species of *Coprinus* and various discomycetes (cup fungi). These are mainly harmless weeds or may even help the mushrooms in breaking down the manure.

Rose Comb (Non-parasitic)

After oil sprays have been used to destroy insect pests, or where oil stoves are in use, or woodwork has been coated with creosote, mushrooms sometimes bear a malformation on the cap somewhat like the rose-comb found in some breeds of poultry. The irregularly lobed excrescence is due to abnormal gill formation on the upper side of the cap as well as on the lower.

Prevention of Mushroom Diseases

Production of a healthy crop of mushrooms depends largely on the care taken in preparing and tending the beds.

Cleaning empty mushroom houses. The usual disinfectant is burning sulphur (see p. 600), but there are other methods whose relative effectiveness has not been fully ascertained. Empty houses may be sprayed throughout with formalin 1: 50, preferably with a power sprayer, so that soil, walls and ceiling are thoroughly soaked. The house is then locked and kept warm for two days and nights, then thoroughly ventilated. The man who sprays should wear rubber gloves and his eyes and mouth should be protected. (For formalin fumigation see p. 595.) Boards and other woodwork may be disinfected by spraying or washing with copper sulphate (1 lb. in 10 gallons water) or one can go over it with a blow lamp so as to burn off all loose

splinters without scorching the wood. The copper sulphate treatment has an advantage over the blow lamp and formalin in that it leaves a small deposit of copper in the wood which preserves it without harming the mushrooms. Milk of lime is also sometimes used on woodwork.

If the compost beds are laid directly on the ground the latter may be covered with a layer of peat dust ½ to 1 inch thick which may be watered with acetic acid at the rate of 2 gallons per 40 square yards. This acidified peat will prevent infection of the bed from the ground.

Shelves used for mushroom growing may be washed in formalin 1: 50 and flamed with a blow lamp before the compost is placed on them.

The quality of the manure is of the utmost importance even in disease prevention. The right consistency of the horse manure, not too fresh and not too old, fermented long enough but not too long, can only be learned by experience. The manure should be moist enough to hold together when squeezed into a ball but not so wet as to drip liquid when this is done. The moisture can be increased by watering, or reduced by mixing with ground gypsum. Addition of gypsum should be done according to one or two definite rules, e.g. first time 28 lb. of gypsum per ton of manure, second time 21 lb. gypsum mixed with 7 lb. of superphosphate (Pizer, N. H., 1938). The compost should have a reaction about neutral, pH7.

The final fermentation (heating) should take place inside the mushroom house, and when it is at its height sulphur should be burnt to destroy fungi and insects on the surface. It is important to keep down mites and insects because, in addition to doing damage themselves, they spread fungus spores.

The casing soil should be obtained from the subsoil. Surface soil contains weed seeds and often *Mycogone* and if it must be used it should be disinfected by formalin 1: 50, or by steam sterilization. A sandy loam is the best type of soil and a neutral reaction is desirable. Steam sterilization sometimes reduces the yield of mushrooms, while after using formalin the house must be left to ventilate for some time.

The spawn is added to the compost as soon as the temperature 3 inches below the surface has fallen to 80° F., so that the mushroom mycelium may spread ahead of competitors.

Diseased mushrooms should be removed with their mycelial strands and with the surrounding soil as well. Before removing them the spot should be watered with formalin 1: 50, chloride of lime or raw carbolic acid 1: 50. It is better to remove diseased fruit bodies wholesale than to see the whole crop destroyed by disease. Treatment with peat dust and acetic acid has been described above. The diseased material should be burnt.

Gypsum dusted over the beds helps to prevent plaster mould. If watering becomes necessary it should be done through holes in the casing soil instead of by sprinkling the surface. The house should be kept damp by watering round about the beds. It is better to water between picking periods rather than during them.

If the soil has been very mouldy one may spray it with very dilute Bordeaux mixture (1 lb. copper sulphate and 1 lb. slaked lime to 40 gallons water) or chloride of lime 1 lb. in 20 gallons.

When greenhouses are cleared after a crop the earth or manure removed should not be put near mushroom houses or on the site where mushroom compost is to be made.

ONION

Smut (*Urocystis cepulae* Frost = *Tubercinia cepulae* (Frost) Liro)

There is considerable uncertainty about the correct scientific name of this smut fungus. *Tubercinia* is the older generic name and is that favoured by modern Continental authors. It has, however, been extensively used in a different sense, and in 1939 the Nomenclature Committee of the British Mycological Society recommended that the name *Urocystis* be conserved. This can only be done at an

International Botanical Congress of which the next is due to meet at Stockholm in 1950. Until then the name of this smut remains a matter of personal opinion.

Onion smut is regarded as the most destructive disease of that crop in U.S.A. (Chupp, 1925). It was known there as early as 1860 and by 1925 it had been found in nineteen of the most northerly states. Onion smut also occurs in many places in central Europe, and in Rostrup's day it was often seen on onion, shallot and leek in Denmark. It has not been seen there for many years on onion but occasionally of late years on leek. In England it is widely distributed, especially in the main areas of commercial onion cultivation (see map in Moore, W. C., 1948). Outbreaks have also occurred in market gardens in Scotland.

Symptoms. By careful examination smut can be detected on quite young seedlings. It appears as opaque dark spots or streaks under the skin of the first and subsequent leaves and sometimes in the bulb scales of young onions. The leaves first attacked are often abnormally thickened and twisted. Sooner or later the skin over the streaks splits irregularly, exposing the black spore powder. These spores contaminate the soil and many remain alive there for twenty years or more.

Many of the infected seedlings are killed outright by the disease; others survive and grow, but are malformed, while still others grow almost normally but develop fresh smut lesions on their leaves and bulbs. Individual plants may completely recover from the attack; diseased leaves and bulb scales are thrown off, and the bulbs ripen more or less normally, though they are always small.

Cause. The smut fungus which causes this disease can grow and infect seedlings at temperatures between 50° and 85° F. High temperatures after sowing therefore favour the fungus.

Infection takes place from the soil and can occur only in the seedling stage, from about the second day after germination until the first leaf is developed. Older plants cannot be infected. The fungus enters that part of the seedling which is beneath the soil or just above its surface. It penetrates the epidermis and permeates the tissues as far as the growing point, after which the new leaves become infected as they develop. The incubation period is 5 days.

Control. Wherever possible onions should be sown in soil free from smut spores. When they are big enough to be transplanted they can safely be transferred to infected land.

In North America a popular method of treatment is to allow formalin solution to trickle into the seed drill immediately after the seed has been sown. The formalin drips from a can fixed on the back of the drilling machine. Care must be taken, however, to avoid damaging the seed by the solution. Anderson (1923) has given a detailed description of suitable apparatus. The procedure recommended in England is to add 1 pint of formalin to 16 gallons of water, enough to treat 800 yards of drill, but on wet soil a formalin solution of double this strength may be needed.

A few smut spores may be found in some onion seed samples, but it appears that the disease is seldom spread in that way.

It can attack many species of *Allium* and no resistant varieties of onion or leek are known.

Various methods of dry seed treatment have been tried in Germany (Bremer, H., 1936) to avoid the troublesome formalin treatment. Good results were obtained with Brassicol but the method should be tested in larger-scale experiments before being generally adopted. The onion seed was slightly damped and then mixed with half its weight of Brassicol.

Importation of onion sets into Great Britain is subject to strict control under the Importation of Plants Orders of 1939 and 1940. Sale for planting of any onion or leek plants substantially affected by smut is prohibited under the Sale of Diseased Plants Orders 1937–43.

Neck Rot (*Botrytis allii* Munn)

At least four species of *Botrytis* may attack onion foliage but the worst disease due to these fungi is the neck rot of stored bulbs, caused chiefly by *B. allii*. In 1935 this was found to

be the most important species on onions in Holland, and in 1943 E. Hellmers showed it was also the most common in Denmark. In an investigation at Long Ashton, Croxall found 72% of rotted onion bulbs contained *B. allii* and 26% contained a related species, *B. byssoidea* J. C. Walker.

Infection usually occurs at the neck of the bulb and the disease becomes apparent after the fungus enters through a wound it easily spreads even in coloured varieties.

Withering of onion leaf tips and white spotting of the foliage caused by a third species, *B. squamosa* Walker, have been described in England by Hickman and Ashworth (1943). *B. cinerea* can also cause similar damage to leaves of the growing crop, particularly in autumn (Moore, W. C., 1948).

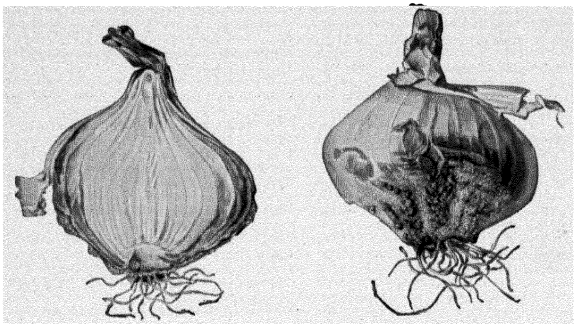


Fig. 184. Grey mould on onion.

lifting. It may spread in storage. The diseased scales become somewhat brown and transparent (Fig. 184), looking as though cooked, and in a moist atmosphere they develop a short hairy coating of grey mould. Bacteria often follow as secondary invaders and set up a rapid soft rot. In the later stages of decay small black sclerotia form on the diseased part or even over the whole bulb. They may sometimes form a continuous scab, especially at the neck of the bulb.

The disease is usually controlled when the bulbs are dried thoroughly and quickly after lifting and are then stored in a cool, dry, well-ventilated place. If they remain damp for long, neckrot may be very destructive. It is advisable to bend over the tops before growth has ceased. Onions are best stored at 35° F.

Onions that have made very luxuriant growth seem more susceptible than those grown at a normal rate.

White-scaled varieties are more susceptible than those with coloured scales. If, however,

Downy Mildew (*Peronospora destructor* (Berk.) Casp.)

Symptoms. This disease does not usually break out until the bulb is about to form. Then a pale coating of the fungus appears on leaves and stalks (Fig. 185). As the leaves wither the fungus turns brownish purple. In damp weather this coating may appear before there is any discolouration of the infected leaves, but they soon become pale and translucent and shrivel from the tip downwards. In mild attacks one may mistake this yellowing for normal ripening. Gradually the infected leaves wither to the base, often being replaced by new ones which are also usually attacked. The dead parts are often covered later by a nearly black layer of the mould *Stemphylium botryosum* (see p. 302). In severe attacks the bulbs remain small, shrivelled or unripe. The disease also attacks shallots and in them it appears first on those grown from infected bulbs. The leaves are small and wither quickly, becoming pale, flaccid and covered by



Fig. 185. Downy mildew of onion (*Photo Dept. Agric. Scotland*).

the greyish-purple layer of mildew. From these plants the disease spreads to the rest of the crop and causes symptoms like those described on onion. Both on onion and shallot the fungus grows down from the leaves into the bulb scales and persists there until the following year. Thus shallots and onion sets are a common source of infection. In severely infected bulbs the outer scales become brown, translucent and shrivelled. Some may die during the winter; others survive and sprout prematurely.

Within the tissue of the infected leaves resting spores may be found, which can survive the winter and even remain alive for 5–6 years in the soil where onion tops have decayed.

Cause and importance. Onion downy mildew is caused by the fungus *Peronospora destructor*, formerly often known as *P. schleideni* Unger. It can attack all varieties of onion and shallot, but is seldom seen on leek. It was first described in England in 1841 but even then was well known to onion growers. It is now widespread and does great damage in Denmark in wet summers. In England it is particularly destructive to seed crops (Moore, W. C., 1943).

Cooke (1932) found infection may be seed-borne, but that is not usually the case, even when the seed is taken from diseased plants. A few diseased seeds in a sample may give rise to diseased plants scattered throughout the crop, centres from which downy mildew can spread to the others.

Control. When onion sets are used it is important to obtain them from a completely healthy crop. If that cannot be done the sets should be graded and only the large sound ones planted and these should be carefully polished. The fungus in the sets may be killed by keeping them for more than 8 hours, or up to 24 hours, at a temperature of 104° F. Jørstad recommended soaking for half an hour in water at 104° F.

In some experiments good results have been obtained by dipping sets in copper sulphate solution, or lime water, but there were no untreated controls for comparison. There is much to indicate that the fungus penetrates the bulb scales too deeply to be completely killed by such treatment.

Seed should be harvested from sound plants.

The soil should not have carried a diseased onion or shallot crop in the previous four years. Remains of a diseased onion crop should be burnt. When nearly all the onion tops in one onion-growing district in America were burnt for several years in succession, to control onion fly, the incidence of downy mildew was reduced at the same time. Onion beds should be well drained and exposed to sun and wind.

Spraying and dusting are less effective than one might desire. It is difficult to get the liquid or powder to adhere to the onion leaves. If it is tried a spreading agent should be added. For example, one may use 16: 8: 100 Bordeaux mixture with 2% spraying oil or, better, 2% white oil (see p. 601). This is also effective against onion fly. Spraying may be begun as early as the middle of May and repeated several times at fortnightly intervals.

Other Minor Leaf and Bulb Spots

Heterosporium allii Ell. & Mart. var. *cepivorum* Nicholas and Aggéry is a mould which causes leaf blotch of onions. Elliptical, slightly sunken, pale-brown blotches appear on the leaves and become covered with a fine brown powdery mould. The disease is of little economic importance. Typical *H. allii* has slightly different spores and occurs on leek, shallot, chives and garlic (Moore, W. C., 1946a).

Stemphylium botryosum Wallr. is generally regarded as a saprophyte but it may become parasitic and often follows onion mildew. It is found especially on stalks of seeding onions, where it causes black spots, often marked with rings, at which the stems finally break. It may also attack the bulbs, penetrating through wounds to cause a soft rot with a yellowish or reddish discolouration.

Smudge is the name given to blackish blotches, composed of numerous closely crowded, minute, black, or very dark-green specks, sometimes found on the outer scales of white onion varieties. It is caused by the fungus *Colletotrichum circinans* (Berk.) Vogl. On the fleshy scales it causes sunken yellowish spots that slowly enlarge in storage. It thrives best at 78° F. but can grow over a range from 36–89° F. In the field smudge develops most rapidly with soil temperatures between 68° and 86° F. and with a high rainfall. It is seldom seen in Britain but is prevalent in North America. Varieties with coloured outer bulb scales are highly resistant.

Rust (*Puccinia porri* (Sow.) Wint.)

This is common on chives but only occasionally occurs on onion. The uredospores occur in orange-coloured powdery pustules on stalks and leaves while the teleutospores occur later in leaden-coloured spots that remain covered by the epidermis.

On chives there may be considerable differences in the intensity of the attack so that probably clones resistant to rust could be selected (see rust, p. 74).

White Rot (*Sclerotium cepivorum* Berk.)

This disease is not known to be very prevalent in Denmark but it has a widespread distribution in Europe and North America and in England about one-sixth of the area devoted to commercial onion production is believed to be contaminated with it (Moore, W. C., 1948).

Symptoms. The disease usually appears about the end of May or beginning of June, especially in autumn-sown crops. The older leaves turn yellow and fall over and the plants can be easily pulled up because their roots are rotten. Round the base of the bulb there is a soft, fluffy, white mycelium, hence the name " mouldy nose " sometimes applied to the disease. In later stages this mould becomes matted and innumerable tiny, round, black sclerotia, none bigger than a pin's head, develop in it. They are readily detached and left in the soil where they may remain alive for many years. All varieties of onion may be attacked, though some are more resistant than others. The fungus also attacks shallot, leek, and garlic and may occur on the wild crow garlic (*Allium vineale*), so that land may be infected with it that has never carried an onion crop. The sclerotia may also be found in seed samples and spread in that way. Probably the most important means of spread, however, is by infected sets and transplants.

After an outbreak of white rot, onions, shallots and leeks should not be grown on the same ground for at least 4 years, preferably not for 8 or 10 years. Infected plants should be burnt.

Shanking (*Phytophthora* spp.)

This disease of onion and shallot closely resembles shanking of tulip (see p. 541). The outward symptoms are yellowing and shrivelling of the leaves and softening of the base of the plant. Some or all of the roots are soft and watersoaked. When an affected bulb is cut longitudinally the bases of the youngest leaves and the tissues of the growing point are found to be discoloured, soft and often contracted. The light-green or white healthy

tissue contrasts sharply with the dull buff infected area (Hickman, C. J., 1943).

In some cases infection has been shown to occur from the soil in which the seedlings were raised prior to transplanting. This is not always the case, however, as the disease may occur in onions drilled out of doors. Onion seed should not be sown in soil that has carried a tomato crop, as the *Phytophthora* spp. which cause foot rot and damping off of tomatoes are the same as cause shanking. Onions for transplanting should be raised in sterilized soil.

Root Rot and Foot Rot (*Fusarium* spp.)

Symptoms. Various species of *Fusarium* may attack onion, both seedlings and older plants.

Roots of seedlings often become attacked by *Fusarium* from infected seeds, and they turn yellowish-pink and decay. The plants put out new roots which are often also attacked. The decay spreads upwards from the roots, and if the bulbs have formed the scales may rot at the base and the leaves turn yellow at the tip. The decayed parts of the bulb become reddish-brown or grey-brown, dry, mealy and overgrown by a pink or white mould; finally they become dry, shrivelled mummies. Alternatively there may be secondary infection by bacteria, causing a soft, wet, stinking rot. Slightly infected bulbs may be overlooked at lifting and decay in storage.

In North America pink root rot of onion, formerly ascribed to *Fusarium malli* Taub., has been shown to be actually caused by the fungus *Pyrenochaeta terrestris* (Hansen) Gorenz *et al.* (Gorenz *et al*, 1948).

Mould Rot in Storage

During storage onions may be seen with soft or dry areas on which there is a sparse blue-green covering of mould, usually beneath the outer scales. This is a species of *Penicillium*. A similar trouble occurs on garlic. The powdery black mould *Aspergillus niger* van Tiegh. may occur in a similar situation and in Southern Europe a mould with yellowish-brown spore clusters (*Aspergillus alliaceus* Thom & Church) causes rotting of garlic and onion bulbs.

These moulds attack bruised bulbs or those damaged by frost or sunscald and stored in ill-ventilated places. Control measures consist in avoiding wounding, drying the bulbs well, and storing in cool, dry places.

Virus Diseases

These are common in shallot, much less so in onion, and are commonly referred to under the names yellow dwarf and mosaic, but the viruses causing them have not been extensively studied. Possibly some forms of the disease, however, may be caused by the common Cucumber Mosaic Virus, which has been shown to be transmitted by the shallot aphis, *Myzus ascalonicus* Doncaster (Doncaster, J. P., and Kassanis, B., 1945).

Infected shallots are small with twisted, compressed, limp leaves, usually streaked with yellow (Plate 9, Fig. A). They make only small bulbs, which may be soft and not keep well. This condition is general in Denmark, where half a crop may be affected. It is at least as common in English shallots and occurs to some extent in onions, especially in seed crops or in the vicinity of diseased shallots. Similar symptoms have been seen in the perennial Welsh onion, *Allium cepa* L. clon *perutile* Stearn.

The shallot aphis occurs on stored bulbs as well as in the field, so possibly some transmission of the disease occurs in storage. Cucumber Mosaic Virus is also transmissible mechanically, as by bruising sets or transplants. Soil infection and seed-borne infection have not been proved. Infections may not give rise to visible symptoms in the first year but the bulbs will yield diseased plants if replanted.

Control. Onion and shallot sets should only be taken from healthy crops. If the health of a consignment is suspected a sample may be planted in a greenhouse during the winter, if infected bulbs are present they will give obviously diseased foliage. It was apparently this disease that Buchanan cured by keeping the bulbs at 84° F. for 112 hours.

Summary of Onion Diseases

		page
I. Roots		
A. Rotten, white mycelium, very small sclerotia	White Rot	302
B. Rotten, pinkish mycelium	*Fusarium* Rot	303
II. Bulb		
A. Localized black streaks and blotches:		
(1) Black streaks in the scales, containing black powder	Smut	298
(2) Black superficial blotches on outer scales	Smudge	302
B. Decaying, brownish translucent scales:		
(1) Light-brown watersoaked decay of the innermost scale and leaf buds in growing crop	Shanking	302
(2) Grey mycelium and spores, ultimately small black sclerotia on stored bulbs	Neck Rot	299
(3) White mycelium, large rounded sclerotia, first white, then black	*Sclerotinia* Rot	77
(4) Dry patches on the scales, starting at the base, finally decaying, white mycelium with very small sclerotia	White Rot	302
(5) Slow decay spreading up the scales from the basal plate, pinkish-white mycelium	*Fusarium* Rot	303
(6) Soft rot with reddish-yellow stain	*Stemphylium* Rot	302
C. Soft or loose-scaled bulbs, often small:		
(1) Loose scaled (after late manuring with nitrogen)	Excess Nitrogen	40
	Calcium Deficiency	33
(2) Soft, small bulbs, sometimes slimy and rotting	Virus Disease	303
	Deficiency of Phosphoric Acid	41
(3) Small transparent shrivelled bulbs	Downy Mildew	300
D. Soft or dried-up patches with a blue-green mould between the scales	*Penicillium* Rot	303
III. Leaves and Stems		
A. Leaden-coloured streaks with a black powder under the skin, leaves sometimes twisted and deformed	Smut	298
B. Reddish-yellow pustules, or leaden-grey spots in autumn	Rust	302
C. Fungus coating covering the plant:		
(1) Delicate pale or mauve mould	Downy Mildew	300
(2) Yellow or brown blotches with fine olive-brown mould	Leaf Blotch	302
(3) Blackish mould	*Stemphylium*	302
D. No superficial fungus coating:		
(1) Leaves turn yellow and fall over	White Rot	302
(2) Leaves shrivel at tip, core of the bulb decayed	Shanking	302
(3) Leaves compressed, twisted, limp, often with vague yellow lines (especially in shallot)	Virus Disease	303
(4) Withered tips (root decayed)	Lack of Potassium	42
(5) Yellowish fading leaves on soil rich in lime	Manganese Deficiency	39
(6) White tips and spots on leaves	*Botrytis* spp.	300

PARSLEY

Leaf Spot (*Septoria petroselini* Desm.)

This fungus causes small angular brown spots to appear on the parsley leaves. Dotted over them are minute black pycnidia which may occur also on the seed.

The fungus is closely allied to celery leaf spot (*S. apii*) but the two fungi seem to be distinct and that on parsley is the less common (Laibach, F., 1921). In practice there is no indication of cross-infections between parsley and celery. Control measures are the same for both diseases (see celery leaf spot, p. 266).

Downy Mildew (*Plasmopara nivea* (Unger) Schroet.)

On the under-surface of the leaves there appear patches of short snow-white mould. The corresponding parts of the upper surface show as yellow spots, which later turn black and the leaves wither. The fungus occurs on many cultivated and wild plants of the family *Umbelliferae*. Hence umbelliferous weeds should be kept down in and around gardens. Control by copper-containing sprays or dusts may also be tried. The remains of a diseased crop may be infectious to the following season's growth and should be removed and burnt.

Sclerotinia Disease (*Sclerotinia sclerotiorum* (Lib.) de Bary)

The roots and leaf stalks rot, a white woolly mycelium appears round them and in the later stages black sclerotia occur (see p. 77).

Soft Rot

In 1931 there appeared in some Danish glasshouses, where parsley was grown without renewal of the deeper layers of soil, a rotting of the root stocks which was strongly reminiscent of soft rot in carrot (see p. 257).

PARSNIP

Leaf Spot (*Septoria pastinacae* West.)

Infection by this fungus causes many small pale spots with brown edges to appear on the leaves (Fig. 186). Definite differences in susceptibility of parsnip varieties have been noticed in Denmark.

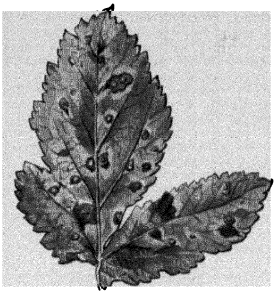


Fig. 186. Parsnip leaf spot.

A somewhat similar leaf spot, caused by the mould *Ramularia pastinacae* Bubak, has been observed in England.

As a control measure fungicidal sprays or dusts may be tried, especially those containing copper.

Mildew (*Erysiphe polygoni* DC = *E. umbelliferarum* de Bary)

A dusty white coating appears on the above-ground parts of the plant (see mildew, p. 63).

Downy Mildew (*Plasmopara nivea* (Unger) Schroet.)

Patches of a short white mould appear on the under-side of the leaves. The corresponding parts of the upper surface become first yellow, then black, and the leaves wither. For further information see parsley.

Rust (*Uromyces scirpi* Burr = *U. lineolatus* (Desm.) Schroet.)

On the under-surface of parsnip leaves and leaf stalks this rust may cause yellow or brownish patches of cluster cups (æcidia). They may also be produced on several other plants such as sea milkwort, mare's tail and weeds belonging to the parsnip family. The uredospore and teleutospore stages occur in summer and winter on a kind of tall sedge (*Scirpus maritimus* L.) which grows in salt marshes. As the fungus alternates each year between the sedge and the parsnip or other æcidial host outbreaks of this disease are

unlikely except near the coast. The æcidia have been found in Britain on sea milkwort (*Glaux maritima*) and hemlock water dropwort (*Oenanthe crocata*) (Fort, M., 1940).

Canker

Canker manifests itself by a larger or smaller portion of the root turning brown, while the skin cracks and decays so that the root looks scabby (Fig. 187). As a rule the

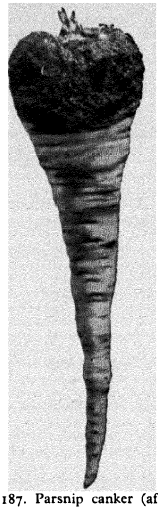


Fig. 187. Parsnip canker (after Cotton).

affected patches spread horizontally round the root; they may be from $\frac{1}{2}$–$2\frac{1}{2}$ inches long and penetrate into the underlying soft tissue. The trouble starts at the crown of the root, which in bad cases may become completely decayed. The initial cracking seems to be a non-parasitic condition caused by soil or climatic factors. The rotting is due to secondary invaders.

Parsnip canker is well known in Denmark but has never been closely investigated there, though it has been observed that varieties are not all equally susceptible.

The disease has long been known in England, where on occasions it has rendered up to 50% of a crop unsaleable. Canker has often, however, been confused with eelworm and carrot fly injury. Cotton (1918), who studied it closely, suggested the following control measures:

(1) Soils in an extremely high state of fertility should not be used for parsnips.

(2) It is better to sow at the end of April or early in May than in February.

Early-sown parsnips tend to grow very big and are then more liable than smaller ones to crack at the crown if a wet period in autumn follows a drought.

(3) Liming should not be neglected. In Denmark, too, parsnips have been found to yield much better at about pH 7 than at pH 6.

(4) Parsnips should not be grown two years in succession on the same ground.

PEA

Leaf Spot, Pod Spot, and Foot Rot (*Ascochyta pisi* Lib. and its allies)

The common leaf and pod spot fungus *Ascochyta pisi* causes small yellow brown



Fig. 188. Leaf and pod spot of pea, *Ascochyta pisi* (*S. p. F.*).

spots with brown edges, dotted with small brown pycnidia, on leaves, stalks and pods. The attack is general and may be destructive (Fig. 188). Very similar fungi occur on sweet pea and on broad bean (*A. fabae* Speg.) but it is not known whether these can infect peas. If the fungus attacks the fruit stalk the pod falls. From the infected pod the fungus often grows into the seeds. Hence the disease is commonly seed-borne. Infected seed may rot or may give rise to a seedling bearing

Control. Pea straw bearing leaf and pod spots should be burnt or ensiled and the sticks used for the crop should be burnt. It is desirable to use new sticks each year and to plant peas on fresh ground for each crop. Potassium manuring is said to make the plants more resistant (Wehlburg, C., 1932). Seed infection can be avoided by selecting seed from healthy pods or by importing seed from a dry climate where these diseases do not flourish. Some control can be obtained by



Fig. 189. Foot rot of pea seedlings due to seed-borne infection with *Ascochyta pisi* (*Photo Dept. Agric. Scotland*).

diseased spots on the stem and the tip of the young shoot. These may die before emerging above ground or may survive and suffer from root rot, depending largely on the soil temperature and speed of germination (Fig. 189).

Two closely related fungi, *Mycosphaerella pinodes* (Berk. & Blox.) Vestergr., which has an imperfect stage *Ascochyta pinodes* Jones, and *A. pinodella* L. K. Jones, also cause spotting of pea leaves and pods and are important causes of foot rot. The leaf spots of *M. pinodes* are small, dark purplish brown, often with concentric rings. On the stems it causes purplish streaks and on the pods small purplish spots which may combine to form large blotches (Fig. 190). *A. pinodella* causes small spots like *M. pinodes* but less numerous.

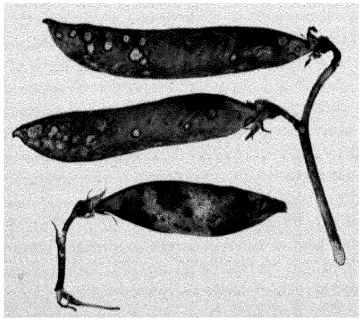


Fig. 190. Spots caused by *Ascochyta pisi* (two upper pods) and *Mycosphaerella pinodes* (below) on pea pods (*Photo Dept. Agric. Scotland*).

seed disinfection with an organo-mercury preparation but this is seldom complete as in some seeds the fungus is already ensconced within the seed coat. In Danish experiments two sprayings with Bordeaux mixture have been beneficial in preventing infection of the pods.

Root Rot and Fusarium Wilt

Very many fungi may attack the roots and kill pea seedlings on wet heavy soils. The water mould *Aphanomyces euteiches* Drechsl. does so especially in late sowings in warm moist soil. In Holland peas are infected mainly by *Fusarium solani* var. *martii* (Appel & Wollenw.) Wollenw. and var. *striatum* (Sherb.) Wollenw. and by *F. oxysporum* Schlecht., while *F. culmorum* Sacc., a common cause of foot rot in cereals, causes some damage to small plants. In Germany *F. redolens* Wollenw. was the species usually found in peas suffering from wilt (St. John's disease, Snyder, W. C., 1935), whilst in North America *F. coeruleum* (Lib.) Sacc. is commonly found associated with the disease (Harter, L. L., 1938). To these may be added root infection by *Ascochyta* spp. (see p. 306) *Thielaviopsis basicola* (Berk. & Br.) Ferr., the cause of black root rot known in Norway and England, *Pythium ultimum* Buism., cause of pea root rot in Holland, and foot rot due to *Corticium solani*. Probably all of these may cause root rot and often several of them may be found together. Finally, Burkholder and Müller (1926) have described a non-parasitic wilt of pea seedlings due to an inherited defect.

Root rot may sometimes be due to seed-borne fungi, but usually a poor seed bed and too frequent cropping with peas are important contributory factors. Repeated cropping with peas may succeed for a few years, but the only safe method of cropping is a long rotation to prevent the build-up of soil infection. Once soil has become heavily contaminated with root-rotting fungi it is difficult to get rid of them and several of the fungi concerned are not particular about the plants they attack.

The best plan is to retain the name root rot for a disease in which either the roots or foot of the plant are attacked by a decay starting from the outside and turn brown or black. In England *Fusarium solani* var. *Martii* is the fungus usually involved (Davis, D. L. G., 1943). If root rot sets in late, or is slow in developing, the above-ground symptoms may be confused with those of wilt. Prolonged drought intensifies the symptoms because the diseased roots and stem bases cannot supply the quantity of water required by the plant.

Fusarium wilt (*Fusarium oxysporum* Schlecht.) is a distinct disease, in which there is no discolouration of the cortical tissues in roots or stem base but the vascular strand turns brown, the plants become yellow, either individually or in patches, and in severe infections they wilt and die. Affected plants yield very little. In Germany the disease is apt to become apparent about St. John's Day (12th June), hence the name St. John's disease. It is encouraged by heat and is therefore worst in warm climates. In the pea-canning districts of North America a very similar pea sickness is caused by *Fusarium orthoceras* var. *pisi* Linf., so far unknown in Europe. In this case infection may be both seed-borne and soil-borne and long rotations become imperative on infected land, a practice little favoured in America. Several resistant pea varieties are now grown, e.g. Green Admiral (Linford, M. B., 1931).

Root rot and *Fusarium* wilt not only cause loss of crop but lead to difficulties in canning peas, because the peas ripen irregularly and also tend to be abnormal in composition and hard when canned.

Control. Seed disinfection may be helpful in certain cases (see poor germination, p. 310), but the chief emphasis is to be laid on maintaining healthy, fertile soil, including the practice of a long rotation of crops. In Wisconsin addition of a complete artificial fertilizer to the seed drill has led to quicker germination and less root rot (Walker and Musbach, 1939). Excess nitrogen and phosphoric acid makes peas more susceptible, while excess potassium makes them fairly resistant to *Ascochyta* and *Mycosphaerella* (Wehlburg, C., 1932). Ensiling pea straw is

insufficient to destroy root-rotting fungi on it; the straw from a diseased crop should be burnt.

Downy Mildew (*Peronospora viciae* (Berk.) Casp.)

On the leaves of field and garden peas this fungus causes yellowish blotches covered on their lower surface by a whitish or pale-mauve layer of conidiophores and conidia. The infected areas may eventually turn brown and in some cases the whole plant may be permeated by the fungus. The pods may be attacked and bear pale-green or light-brown spots. These seldom produce conidia but the resting bodies, oospores, of the fungus are formed within the tissues of the pod. Infected pods often become spotted and deformed but it is not possible to tell whether infection of the seed has taken place. The fungus overwinters in the soil and can infect pea seedlings as long as the soil remains cool, with a temperature below about 65° F.

Races of *P. viciae* also occur on wild and cultivated vetches but cross-inoculation experiments have shown that the race found on pea is specialized and infects only that crop. Hence it is sometimes distinguished as *P. pisi* (de Bary) Syd. (Gäumann, E., 1923).

Spraying with Bordeaux mixture plus a spreader has been suggested as a means of control but must be carried out in the early stages of the disease if it is to be effective. Rotation of crops will help to keep down infection from the soil.

Mildew (*Erysiphe polygoni* DC.)

The race of this fungus found on peas does not attack other plants and hence Continental authors often treat it as a distinct species *E. Pisi* DC. It causes the usual white powdery mildew on both sides of pea leaves, formed by the superficial mycelium of the fungus and its chains of conidia. The tiny brown perithecia are much less often seen. In early and severe infections the mildew may occur also on the pods.

As a rule pea mildew, though conspicuous, causes little damage. In early infections dusting with sulphur or spraying with lime-sulphur may be tried. Severely infected straw should be burnt or ensiled. According to Hammerlund (1925) resistance to mildew exists, based on several inherited factors, so that resistant varieties could no doubt be bred.

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

Under damp conditions peas may be attacked by this fungus but its woolly white mycelium is usually found, if at all, on harvested peas that have not been quickly used. We have found severe attacks on salted green peas. For further particulars of the fungus see p. 77.

Broad Bean Rust (*Uromyces fabae* (Pers.) de Bary)

The cluster cup (æcidia) stage is seldom seen on broad beans but occurs occasionally in late autumn or spring. The powdery cinnamon-brown pustules of uredospores, however, tend to be very common on broad beans and also on vetches during the summer months. They are followed by the dark-brown, firmer, pustules of teleutospores. Races of *V. fabae* occur on wild species of *Vicia* and of *Lathyrus* and, though rust on each of these genera will not infect the other, races found on either of them are able to infect the garden pea. The disease is usually considered of little importance in Britain.

Pea Rust (*Uromyces pisi* (Pers.) Wint.)

Rust of peas is seldom seen in Britain, and in Denmark when it does occur it is usually caused by the vetch rust, *U. fabae* (see above). There is, however, a distinct rust of garden peas, common in south Europe but seldom seen in the north, because it requires for its life cycle alternate hosts rare in Denmark and North Europe. These are species of spurge (*Euphorbia cyparissias, E. esula, E. lucida* and *E. virgata*) of which only the first and second occur naturalized in a few places in Great Britain. On these spurges the cluster cups (æcidia) are formed, causing the infected shoots to be abnormally long and

broad-leaved. If any of these spurges are found infected with cluster cups they should be destroyed as the rust can pass the winter in the spurge plants. Rust on them may, however, be a strain that infects other legumes and not peas. The common rust on the ordinary spurge weeds of cultivated ground (*E. peplus*, *E. helioscopia*, *E. exigua*) is a distinct species, *Melampsora euphorbiae*, and is harmless to peas. Pea rust forms cinnamon-coloured uredospore pustules on leaves of field and garden peas and blackish-brown teleutospore pustules mainly on the stems. It can infect also yellow vetchling. According to a Danish law of 27th March, 1903, every owner of agricultural land has the right to demand that cypress spurge (*Euphorbia cyparissias*) shall not be grown within 200 yards of his property and to demand removal of any found within that distance.

Pea Mosaic (*Pisum* Virus 2)

Pea mosaic is widespread in North America but not particularly harmful. It has been known in Denmark since 1926 in Witham Wonder and other varieties; in England it is widespread but has been little studied (Ainsworth, 1940). The leaves often become yellow, between the veins rather than along them, or are covered with irregular yellowish and dark-green spots. Eventually they become small and curled. Similar symptoms are caused on sweet pea, and in varieties with coloured flowers the petal colour is " broken " by pale lines or spots. The pods may be uneven and mottled. Infection is spread by aphides, notably *Macrosphum pisi*, *Myzus persicae* and *Aphis rumicis*. In addition to sweet pea the virus can infect also common vetch, broad bean, red clover, sweet clover and lupin. On the last-named it causes the fatal disease " sore shin." Dwarf and french beans are not infected. The incubation period in peas is 1–2 weeks. Occurrence of seed transmission has been both reported and denied, in any case it seems to be of little significance (Zaumeyer and Wade, 1936). According to Murphy and Pierce (1937) pea varieties differ greatly in susceptibility to mosaic. If the disease became important the first step in controlling it would be to weed out susceptible varieties.

Another pea virus, *Pisum* Virus 1, causes a disease called " enation " pea mosaic in North America. This has been reported in England once by Ainsworth (1940) and is possibly the same as a virus found on broad beans in Germany. It causes severe mottling and crinkling of pea leaves with distortion and wrinkling of the pods. The symptoms in broad bean are a conspicuous spotty mottle, especially along the veins. It must be remembered that in Europe distortion of pea pods is mainly caused by thrips, not by virus infection. *Pisum* Virus 1 is spread by the aphides *Macrosiphum gei* and *M. pisi*.

Marsh Spot (Manganese Deficiency)

In samples of pea seed, especially in that grown on wet soils, one often finds a more or less circular brown depression in the centre of the inner face of each cotyledon. This occurs especially in varieties with large wrinkled seed but there is no sign of disease on the seedcoat, or until the pea is cut across. Often, too, the tip of the tiny shoot, the plumule, is damaged, or when an affected pea germinates the tip of the shoot may die when only a few inches high. Usually secondary lateral shoots develop to replace the dead tip, and if the seed is sown in fertile soil the plant ultimately suffers little from marsh spot. The trouble has been shown to be due to a deficiency of manganese and may be worst when ripening is slow. It may be prevented by applying manganese sulphate to the seed crop at the rate of about $\frac{1}{2}$ to $\frac{3}{4}$ cwt. per acre (Pethybridge, G. H., 1936; Heintz, 1938). Spraying the foliage with manganese sulphate solution during flowering may be even more effective (Lewis, A. H., 1939). In North America a related phenomenon has been described in which the green peas develop dark spots in the skin but germinate normally.

Poor Germination

Poor germination of peas may be due to pre-emergence blight caused by *Ascochyta*, or to rotting of the seed by various soil fungi, such

as species of *Fusarium*, *Botrytis*, *Pythium* and various bacteria, especially if the seed was of poor quality. Protection against these rotting organisms may be obtained by coating the seed with red copper oxide (1 oz. to 8–12 lb.) or with one of the proprietary organo-mercury seed disinfectants. Marrow-fat peas often germinate badly when sown too early (Hull, 1937; Padwick, 1938; Wain and Wilkinson, 1945).

Summary of Pea Diseases

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(2) Small pale warts	Oedema	18
(3) Light-green or pale-brown spots	Downy Mildew	309

		page
(4) Yellow-brown sunken spots with brown edges ..	*Ascochyta pisi*	306
(5) Dark purplish-brown spots or irregular blotches	*Mycosphaerella pinodes*	306
(6) Twisted pods, blotched with darker green	Mosaic	310
B. Inside of the pods:		
(1) Whitish or pale-mauve mould	Downy Mildew	309
(2) Patches of fine white hairs	Oedema	18
C. The seeds:		
(1) Sunken brown or blackish spot in the centre of the flat side of each cotyledon	Marsh Spot	310
(2) White mycelium with large white, then black, sclerotia	*Sclerotinia* Rot	77

POTATO

Potato Blight (*Phytophthora infestans* (Mont.) de Bary)

History and importance. Potato cultivation had become more or less widespread in Europe by 1750, rather later in Denmark, but it was only about 1830 that people began to notice the "potato disease" which was first seen in Norway in 1841 and in Sweden and Denmark in 1842. The first widespread and devastating attacks were reported from North America in 1843 but it is the outbreak in Ireland with the consequent famine, wholesale starvation and emigration of the peasantry, that is best remembered. The Irish potato crop suffered heavy losses from an attack in September 1845; in the following June it appeared again and Ireland's potato acreage yielded only 10% of the expected crop.

Severe outbreaks occurred in Denmark in 1878, 1879, and 1883, causing J. L. Jensen to experiment in preventive measures. A Bordeaux mixture was tested on potatoes in Denmark in 1885 but there were no practical sprayers available and not until 1904 did spraying slowly begin to be adopted. More than 1400 Danish spraying experiments over a period of 24 years have shown that in 2 of those years the difference in yield of sprayed and unsprayed crops was only 1.5%, in 11 years it was about 11% and in the other 11 years about 20%. The annual loss from potato blight in Denmark may be assessed at an average of about 15% in unsprayed crops (see p. 95).

The potato blight fungus itself was first described in 1845 by the Belgian mycologist Mlle. Libert as *Botrytis devastatrix* and by Montagne as *Botrytis infestans*. After de Bary's extensive studies its true character became understood and the name was changed to *Phytophthora infestans*.

Various attempts have been made to explain why the fungus did not attract attention in Europe until so long after the introduction of the potato. Some have thought that in the days of sailing ships infected tubers rotted during the long voyage from America but that when steamships were introduced the shorter passage permitted infected tubers to reach Europe with the fungus still alive in them.

Others seek an explanation in the change in variety of potato grown between 1750 and 1830, a change from varieties with small, red-skinned, deep-eyed tubers to more attractive types. Even in the present century statements have been made that certain potato varieties have become more susceptible to blight after many years' cultivation.

A third suggestion, put forward by Leonian (1925), is that the potato blight fungus arose as a mutant from another species, *Phytophthora cactorum*, which would explain its sudden appearance and perhaps also its curious aversion to producing any spores other than conidia. Sexual spores (oospores), freely produced by other species of *Phytophthora*, have only rarely been obtained and only under artificial conditions with *P. infestans*.

Such a view might also explain the striking uniformity of the fungus throughout the world. Only after experiments began in crossing the cultivated potato with wild Mexican species immune to blight, did physiological races of the fungus become recognized, differentiated by their capacity to infect individual crossbred seedlings (Müller, K. O., 1933; Schick, R., 1932, 1936; Small, T., 1938).

Host plants. Several of the plants mentioned below are only seldom and feebly attacked by *P. infestans*. A single statement that it can infect cucumber, egg plant and black nightshade has not been confirmed. Natural infections of *Solanum crispum*, *S. dulcamara* and *Lycium halimifolium* have been observed in England (Moore, W. C., 1943, 1948).

CROP PLANTS	WEEDS
Potato	*Solanum dulcamara* (Bittersweet)
Tomato	*Hyoscyamus niger*
Solanum muricatum	(Henbane)

ORNAMENTALS

Anthocercis	*Schizanthus*
Datura	*Solanum atropurpureum*
Lycium (Boxthorn)	*Solanum aviculare*
Petunia	*Solanum crispum*
Physalis	*Solanum marginatum*

Symptoms. The disease on tomato will be described on p. 381. The attack on potato leaves starts as a rule at the edge or tip of a leaflet as a light-green spot, which quickly becomes brown and limp, and later blackish-brown and dry (Fig. 191). Under damp conditions the brown spots have a marginal zone of fine white mould, composed of conidiophores and conidia of the fungus, which burst through the skin of the leaf 2–3 days after infection.

There are at first a few scattered spots, usually on the lower leaves; but others quickly follow and the whole potato haulm may be destroyed in about a week. The diseased leaves have a characteristic sickly sweet smell which can often be detected at some distance from a diseased crop. Brown streaks develop too on the stems and even the flowers and berries may be attacked.

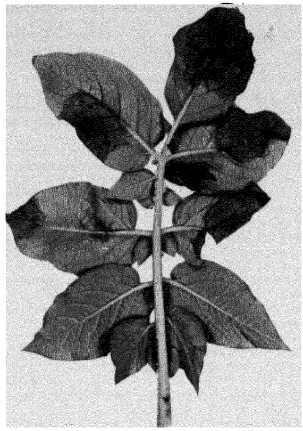


Fig. 191. Potato blight on under-side of the leaf.

Tubers attacked by potato blight show leaden or dull purplish-grey, slightly sunken spots or irregular blotches in the skin. When they are cut through, rusty-brown streaks are seen growing from the skin into the flesh (Fig. 192). In time the whole tissue becomes destroyed by a rather dry brown rot. Commonly, however, *P. infestans* is quickly followed by secondary fungi, especially species of *Fusarium* and bacteria, which obscure the symptoms of blight and may cause an evil-smelling soft wet rot to supervene.

In all its stages the blight fungus needs oxygen and it cannot penetrate rapidly into the flesh by itself. The secondary invaders

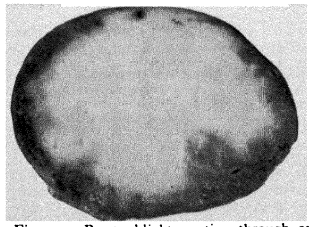


Fig. 192. Potato blight, section through an infected tuber.

feed on the starch grains that the blight fungus has only superficially attacked (Lepik, E., 1929). *P. infestans* conidia may be formed on diseased tubers.

Life history of the fungus. The white mould consists of conidiophores, each of which may produce 8–12 conidia. When these fall into water, such as a drop of dew on a leaf, they function as sporangia, after and about ½ to 3 hours, 10 or so swarm spores emerge from each which swim about, settle down on the leaf and initiate a new infection. At higher temperatures the conidium does not form swarm spores but germinates directly by putting out a hypha.

The fungus enters leaves on the lower surface through the numerous stomata. Rain washes conidia down to tubers exposed on the soil surface, or covered only by a thin layer of soil, and there the fungus enters through the lenticels. Mature tubers are infected especially through wounds, such as the damages unavoidable during lifting, and through cracks in the skin and eyes. Potatoes lifted while unripe, with a thin, easily loosened skin, are highly susceptible to infection for some days after lifting.

Blight seldom spreads from tuber to tuber in the pit or clamp. When tubers that appeared healthy at lifting are afterwards found to have become blighted that is due to infection which occurred during lifting. The incubation period depends on temperature and may vary from 5 to 50 days—at 40° F. blight scarcely penetrates the tubers. At 66–68° F. it may grow nearly ¼ inch into the tissue in a day. Dry storage does not stop growth of the blight fungus into tubers but is useful in checking damage by other fungi and bacteria. The importance of a low temperature in the pit is shown by the following experiment on storage:

Storage temperature in ° F.	40	54	70
Percentage obviously diseased tubers after 2 months	17	53	79

Sources of infection. The most important source of infection every summer is certainly slightly infected seed tubers. These may give rise to (1) quite a normal sprout, (2) infected sprouts which soon die or (3) infected sprouts which come above ground, bear conidia and thus infect the lower leaves of the crop.

The potato blight fungus can also grow out from diseased tubers and small dead shoots into the soil where it lives for a time, and may produce conidia which are splashed on to the leaves by rain. The appearance of the fungus in a crop from diseased seed tubers has been doubted but was seen by de Bary, J. N. Jensen and later by several other research workers (Alcock, N. L., and McIntosh, A. E. S., 1927). In Denmark we have seen small stems attacked in June, in Sweden blight has been noticed on forced potatoes in April. Severely attacked tubers are readily destroyed by bacteria and millipedes, and sprout feebly or not at all. It is only those tubers which are slightly infected which are able, when planted, to produce shoots that come above ground. In June and July spread of infection from these diseased sprouts to lower leaves of the crop proceeds unnoticed. These centres of infection form starting points for the incredibly rapid blazing up of the disease which occurs when conditions become favourable to it in July and August.

It may be supposed that it will break out at many places at the same time if the conidia are carried a short distance by the wind. It may be of importance whether the early potatoes lie on the windward or leeward side of the late varieties. Sometimes one may notice that a hedge has afforded a certain amount of shelter and that blight has started opposite a gateway or other gap in the hedge. Small potato patches in town gardens become attacked at the same time as fields in the country. In damp soil conidia may remain alive for 20–30 days (Murphy, P. A., 1922). In experiments with cultures in sterilized soil or manure, where the blight fungus encounters no competition from other organisms, it has survived the winter out of doors (de Bruyn, H. L. G., 1922). The possibility of it overwintering in ordinary field or garden soil is very small but it must be remembered that there are always some groundkeeper tubers not

killed by frost and the fungus may naturally survive the winter in some of those. Heaps of chats discarded when the crop was graded or left on the site of an old pit are another possible source of infection.

In Denmark we have noticed that blight has broken out in places where the potato haulm was collected the year before. In Germany the fungus has survived in compost for over a year. In North America the overwintering remains of haulm in big tomato fields are regarded as a source of infection and the fungus has been found in seed from diseased tomato fruit (Boyd, O. C., 1935).

If the potato tops are killed early by blight and the tubers are left to ripen in the soil a very small crop is obtained but the tubers do not become infected, provided they were covered by a good thick layer of soil. If blight attack comes late, retarded by weather conditions or by spraying, so that the tops are still green and bear fresh blight spots when the crop is lifted, there is great danger of the tubers becoming infected, especially in wet weather. Liability of the tubers to become infected depends partly on the variety, partly on the soil (see below). Haulm and soil remain infectious for 10, possibly 20, days after the haulm has died. Haulm cut down by frost is at once freed from infection.

Conditions for infection. The weather is the decisive factor in determining the time of an outbreak and its extent. As indicated above, in two years out of twenty-four there was no appreciable potato blight in Denmark and this was due to summer drought. On leaves the fungus grows rapidly at 68–77° F., in tubers at 66–72° F. (In greenhouses with controlled temperature and humidity the optimum temperature for the disease was 77–90° F., and the maximum at which it occurred was 96–98° F., figures which do not agree with outdoor experience.) Conidia form best at 66–72° F. but send out swarm spores most readily at 52–55° F. and at these temperatures the latter's vitality is also greatest. At higher temperatures the conidia generally germinate direct. Temporary drying encourages germination; great heat destroys the fungus, hence in the tropics it is confined to high altitudes. It needs plenty of moisture; conidia are not formed in atmospheres containing less than 75% relative humidity. 90–100% relative humidity favours it most and the conidia can only germinate, by either method, when actually immersed in water, hence the importance of dew and misty rain. Growth of the fungus in the leaves is most favoured by moderate temperatures and plenty of rain.

Attempts have been made in Holland to predict the date at which potato blight will break out, based on the known temperature and humidity requirements of the fungus (Everdingen, E. van, 1926, 1935). Similar attempts have been made in the South West of England where the criterion of a " critical day " are:

Minimum temperature on the day 50° F. or over.

Relative humidity for the two following days 75% or over.

After such a " critical day " the disease may be expected to break out in 5 or 6 days if the temperature is 50° F., or after only 3–4 days if the temperature reaches 70–85° F. Windy weather or bright sunshine, however, may check the outbreak. In Denmark in 1931 potato blight outbreaks followed such " critical days " on 22 out of 26 occasions.

The predictions are based on weather observations at some recording station which cannot be expected invariably to be applicable to conditions within the potato fields themselves. The value of such predictions will be understood when it is remembered that sprays must be applied before blight appears in the crops if they are to be really effective. In Denmark the practice is to publish the news as soon as the first outbreak of blight is seen. In the British Isles the date of the outbreak varies greatly from year to year. It usually occurs first in Cornwall or Devon and at points on the West coast, such as parts of Wales and the Isle of Arran. In the Scilly Isles blight has been seen in January but as a rule the first reports come from West Cornwall and Pembrokeshire in May or the first week in June. As one proceeds north and east outbreaks

tend to occur later, until in most parts of Scotland the disease is seldom noticed before the end of July or some time in August.

Varietal susceptibility. During the devastating outbreaks in the middle of the last century the only remedy thought of was the development of a resistant variety. Magnum Bonum and Up-to-Date originated in this period. There are greater differences in resistance to infection of the tubers than in that of the haulm. Most early varieties are quite susceptible but they generally escape attack in many parts of the country, by ripening before the disease breaks out. Of the commonly grown main-crop varieties King Edward is one of the most susceptible in the haulm, and the first outbreak in a district is often to be found in a low-lying field of King Edward. Varieties with susceptible haulm but rather less susceptible tubers include Great Scot and Majestic. Kerr's Pink haulm is classed as fairly resistant in Denmark.

Haulm of the Scandinavian variety Gustav Adolf is said to be very resistant to blight.

As regards tuber infection a survey of 1600 Lincolnshire potato clamps in 1945–6 led to the most popular varieties being rated as follows in increasing order of severity of attack: Majestic, Doon Star, Arran Pilot, Up-to-Date, Gladstone and King Edward (Moore, W. C., 1948).

Attempts to obtain resistant varieties by selecting plants whose haulm remained green among an infected crop have not given positive results; the green haulms were merely due to accidental factors of development or nutrition in the individual plant, not to an inherited capacity for resistance. Susceptibility of the haulm depends in part on its stage of development (Beaumont, 1934). Hence the first planted crops tend to be those first attacked.

The greater susceptibility of mature haulm may be due in part to the damper microclimate within the crop after the haulms have met in the drills. The swarm spores of the fungus actually penetrate a young leaf more easily than a fully developed one (Crosier and Reddick, 1935; Limasset, P., 1939). Of late years considerable interest has been aroused by the discovery that some wild Mexican *Solanum* species, notably *S. demissum*, are extremely resistant to potato blight, and attempts have been made to produce hybrids between these and the domestic potato. Unfortunately this work has brought to light the existence of physiological races of *P. infestans*, and hybrid seedlings resistant to the common race of the blight fungus have proved to be susceptible to other races (Black, W., 1943, 1945).

There are numerous but somewhat contradictory observations on the influence of manures on susceptibility of potato haulm to blight. Amongst other things the previous crop seems to play an important part, for example in the same potato field the part of the crop that followed barley was attacked much earlier and more severely than that which followed medick. As a rule nitrogen deficiency (or less often an excess of nitrogen) encourages a very strong attack of blight. Deficiency of phosphates checks the disease, excess of phosphates encourages it. Deficiency of potassium may enhance the resistance of the tubers. Susceptibility of the haulm appears to be unaffected by the physical nature of the soil but tubers are most susceptible in clay and fen soils, less so in loams and least in sandy soils. Infection through the eyes is very seldom seen and seems to occur to about the same extent on clay and sandy soils (Löhnis, M. P., 1923, 1929).

Control. In the absence of a resistant variety of commercial value, control of potato blight in the haulm is only possible by spraying with a copper fungicide. The spray must be applied before the disease breaks out and may have to be repeated once, twice, or even three times to maintain a protective covering on new growth and to replace spray washed off by rain. The time to spray will naturally vary from year to year and in different parts of the country. In Denmark the general recommendation is to spray second or early main crop varieties about 20th July and between 5th and 10th August. Earlies may be sprayed about 1st July and 15th July if the top is well developed by that time. The increase in

yield is less in early than in late varieties, but the former should be sprayed to prevent them from acting as a reservoir of infection for the latter. Potatoes forced under glass should be inspected daily and sprayed or dusted at the first sign of an attack.

The standard sprays are Bordeaux and Burgundy mixtures, but there are numerous proprietary copper sprays easier to prepare than these. Bordeaux dusts may also be used, particularly where difficulties of water supply or in carting water are liable to arise.

In North America it is the practice to spray potatoes 6–7 times at the rate of 67–71 lb. copper sulphate per acre. Controlled experiments have shown that early spraying encourages leaf development and gives larger tubers, more uniform in size, and that it may be well to start with Bordeaux mixture 16: 16: 100 or even stronger, and finish with 4: 2: 100 (Blodgett *et al.*, 1933; Mader and Blodgett, 1935). Danish experiments have shown an average yield increase from spraying of about 15% in most varieties and that the tubers of sprayed crops are healthier, keep better and are richer in starch.

In the exceptional years when there is no blight, spraying potatoes in Denmark gives very little increase in yield or may even cause a slight loss. Large horse or tractor-drawn sprayers especially are liable to damage the crop. On a field scale it is best to work with power sprayers that reach a considerable distance. Bordeaux mixture may be beneficial to the potato plant in other ways, as by counteracting a possible deficiency of copper (see p. 36) or by protecting the leaves from strong sun and from night frosts.

In years when the potato haulm remains green late in the season it is advisable to cut and remove it before the crop is lifted. This is impracticable on a field scale and there it is better to burn down the haulm by spraying with sulphuric acid, 1% sodium chlorate, a concentrated copper sulphate solution, a mixture of 2% copper sulphate and 2% common salt or with tar acid compounds (Samuel, 1943, 1944, 1945). Instead of removing or killing the haulm it may be sufficient to wait until a fortnight after it has died naturally, but this may mean postponing lifting until late in the season when bad weather is to be feared.

It is desirable to remove obviously diseased tubers at lifting, but later regrading may be necessary to remove those that become infected during lifting. The bad practice still persists of temporarily covering newly lifted potatoes with the uprooted haulm; that is just asking for trouble. Potato clamps should first be covered with straw, held in place with a few spadefuls of soil, and some ventilation should be left at the ridge when they are finally prepared for the winter.

It is desirable to prevent "seed" tubers from being attacked by blight or they will carry infection into next year's crop and the infected ones by rotting in the drill may cause the crop to be irregular. Disinfection of the tubers in spring has little value but "seed" may be disinfected immediately after lifting, by dipping it in a solution of a proprietary organo-mercury preparation. Greening seed tubers by exposing them to the sun for some days after lifting has met with some favour in the past but seems to have little effect in preventing tuber rots; if the weather turns wet it may expose them to further risks of blight infection.

Alternaria Blight (*Alternaria solani* (Ell. & Martin) Sor. sensu L. R. Jones & Grout)

This leaf spot disease is known and feared in North America under the name "early blight" because it tends to appear earlier than true potato blight, called there "late blight." It is due to the mould *Alternaria solani*, formerly called *Macrosporium solani* Ell. & Mart., which causes leaf spotting also on tomato and egg plant.

Symptoms and development. More or less circular brown or blackish spots appear on the potato leaves from July onwards. They seldom attain ½ inch in diameter and are limited by the leaf veins though in severe outbreaks several spots may become united. Under a lens the spots are seen to be marked with darker, irregularly concentric lines, whence

the name " target spot " sometimes applied to them. In damp weather a fine olive-brown mould may appear on the surface. Some varieties show considerable resistance to the disease and only very small brown spots develop on their leaves (Reiling, H., 1930).

This is the form of *Alternaria* blight usually seen in Britain, where, indeed, the disease is regarded as of no economic importance and where conidia are seldom to be found on the spots. It seems clear, in fact, that not all the " target spot " on potato leaves in this country is attributable to *Alternaria* infection.

In North America and elsewhere decay of the tubers by *A. solani* has been reported. They become infected during lifting, especially in damp weather or when spores fall on open wounds. The fungus only grows slowly in the tubers but it may sometimes cause spots 1–1¼ inches across, at first superficial, then sunken and penetrating ⅛ to ¼ inch. The decayed tissue dries up and becomes hard and is separated from the healthy flesh by a layer of wound cork. When the fungus enters at the heel it may penetrate deeply and ultimately break out to form several small spots (Goossens, J., 1937).

A. solani overwinters in the soil, on potato debris and probably on seed potatoes. Virulent outbreaks in North America occur at day temperatures of 77–86° F. with heavy dews at night. The fungus has been known in Denmark for a generation but was not definitely identified in England until 1933, though probably present there long before.

Control. Very early spraying with Bordeaux mixture prevents an outbreak of *Alternaria* blight; according to one investigator Burgundy mixture is even more effective. Where the disease has been prevalent care should be taken that the tubers do not become infected by spores from the haulm at lifting, and to store them in a dry cool place. The optimum temperature for *Alternaria* rot of the tubers is 60° F. and its growth ceases at 41° F. Alpha, Bravo, King Edward, Majestic, Kerr's Pink, Edzell Blue and Up-to-Date are all susceptible; Juli is fairly resistant and Golden Wonder more so.

P. C. Bolle (1924) and K. O. Müller (1923) have described infection of the flowers by a species of *Alternaria* which penetrates the stigma and prevents the berries from setting.

Cercospora concors (*Casp.*) Sacc.

This fungus causes a minor leaf spot disease which in some parts of Europe causes premature withering of potato haulm in severe seasons. The leaf spots appear from July onwards; they are at first yellow, then brown on the upper surface, and are tinged greyish-purple with the conidia on the under-side of the leaf. The routine sprays against potato blight control this disease as well. *Cercospora concors* has not yet been found in the British Isles.

Powdery Mildew (*Erysiphe cichoracearum* DC.)

A powdery mildew has not yet been found on potato leaves in Denmark, but it has occurred at Berlin (Müller, K. O., 1928). Outbreaks have also been reported in Switzerland and France, where Ducomet (1923) found perithecia when the crop was mature. The conidial stage was noticed on potato crops round Cambridge in 1946 (Thomas, D. G., 1946). It forms a loose, powdery, white covering in patches on the upper surface of the leaves but apparently causes no appreciable damage.

Potato Wart Disease (*Synchytrium endobioticum* (Schilb.) Perc.)

History and importance. This disease was first described from Trencsen, Slovakia, then in Hungary, in 1896. It seems to have been present in Scotland and the North of England since about 1870; it was found in Wales in 1901 and in Ireland in 1908. Since its recognition in Western Germany in 1908 and in Silesia in 1909 it has been discovered in North Germany and in Thuringia, Bavaria and in the neighbourhood of Poznan in Poland. In Holland it appeared in 1915 (or earlier?) and more recently in Austria, Belgium, Bohemia, France, Portugal, Rumania and Switzerland. Italy is said to be free from it but the disease has occurred in Malta. Wart

disease was found in Sweden in 1912 but was not seen there again until 1928. It was then found particularly in Hallande, Blekinge and Central Sweden. Wart disease was found in Russia in 1911, Norway in 1914 and Finland in 1924.

The first cases in Denmark were identified at Hammel, Varde and Padborg in 1923, but the disease had been there for several years at that time; subsequently outbreaks have occurred in various gardens round cities and country towns, especially in South Jutland, but seldom in field crops.

Wart disease increases rapidly on land where potatoes are frequently grown, as in gardens and allotments, and in a space of 2–3 years it may attain such proportions as to ruin the crop. Direct losses of crop from the disease on an agricultural scale have been small but stringent quarantine regulations against it hamper export of produce from infected districts and discourage the cultivation of susceptible varieties. Hence the regulations which aim at excluding wart disease from countries in which it does not yet occur cannot be held to be unreasonable.

On several occasions the disease has been brought in with small consignments of potatoes among immigrants' luggage or by small-scale commercial traffic across a frontier. Thus it entered Newfoundland in 1909 and Canada in 1922. Outbreaks were found in Pennsylvania in 1918, but it had been introduced there before 1912. The disease appeared in South Africa in 1921–2 and in Japan in 1931. It also occurs in Andean provinces of Peru and Bolivia.

Host plants. Only plants of the nightshade family, *Solanaceae*, can be attacked by this fungus. Tomatoes develop swellings on roots and stem, and under experimental conditions small warts on the leaves also (Martin, M. S., 1929). Henbane and a number of species of *Solanum* develop small swellings on the roots and eventually small galls on the stems. It has not proved possible to infect thorn apple (*Datura stramonium*), deadly nightshade (*Atropa belladonna*) or any kind of tobacco (Esmarch, F., 1925).

The following list of susceptible plants is based chiefly on inoculation experiments. Only bittersweet, black nightshade and henbane are attacked to any appreciable extent.

Hyoscyamus niger (henbane)
Lycium spp. (boxthorn, Duke of Argyll's tea tree)
Lycopersicum esculentum (Tomato)
Nicandra physalodes
Physalis Franchetii (chinese lanterns)
Solanum alatum
S. chacoense
S. commersonii
S. dulcamara (bittersweet, woody nightshade)
S. jamesii
S. nigrum (black nightshade)
S. nodiflorum
S. villosum

Symptoms and development. In a wet season warts may be found on the stems and lowest leaves of potatoes, and even on the inflorescence (Gough, G. C., 1920), but as a rule the attack is confined to the tubers and is first noticed at digging. The stolons may be affected but the roots are never attacked. As a rule growth of the haulm is normal and unchecked. The warts on the tubers start as tiny white granular swellings growing out from the eyes. They quickly enlarge into cauliflower-like structures which may be larger than the tuber itself (Fig. 193). Above ground they are light green, below ground at first white, then brown.

Old warts decay and turn black and even at the normal lifting time many will be partially rotten, or break off and decay in the soil, leaving only a small scar on the tuber. Occasionally new small outgrowths may develop in the clamp around the scars, giving a disease picture like that of powdery scab.

Powdery scab infection of the sprouts occasionally produces swellings simulating wart disease, as may also irregular second-growth, covered with scab (Fig. 196). Sometimes microscopic examination is necessary to distinguish between these diseases. The swellings of crown gall also resemble those of

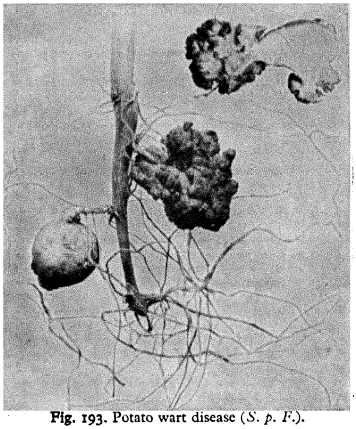


Fig. 193. Potato wart disease (*S. p. F.*).

wart disease but the former is known on potato only from artificial inoculations.

The wart disease swellings contain a vast number of minute, brown, thick-walled resting sporangia which become liberated into the soil as the tissues decay. When they germinate a number of extremely minute, mobile swarm spores emerge and swim about until they meet a young potato tuber. They then penetrate the skin of an eye and settle down inside the potato cells. Here in a fortnight clusters of from 1 to 7 summer sporangia are formed which liberate hundreds more swarm spores. These are partly responsible for multiplying the number of infected cells on and around the developing wart, partly for infecting other tubers. Second generations of summer sporangia form during the summer, and their development requires 10–20 days. They do not overwinter but are replaced in the mature wart by the thick-walled winter sporangia, formed after a kind of sexual process. These not only survive the winter but remain alive in the soil for very many years; they are not destroyed even when an infected potato is eaten and digested by an animal.

The formation of warts is due to the potato cells being stimulated to abnormal growth and multiplication by the fungus cells which invade them. The wart disease organism is one of the most primitive of fungi and never develops any kind of mycelium.

Methods of spread and requirements for infection. The most important and most dangerous means by which infection spreads is the transport of seed potatoes, not only diseased tubers but healthy potatoes from infected land, which may carry resting sporangia in the soil adhering to them. Potato peelings in kitchen waste added to compost heaps or manure and dung of animals fed on raw potatoes may also carry infection.

The fungus may be carried from infected land in soil on carts, tools, boots, and the feet of domestic animals and poultry. Soil particles transported by storms, or floods, or by crows and other wild birds may also spread the disease.

Resting sporangia can germinate over a wide temperature range (40 to 86° F.) but best at 66–68° F. Presence of oxygen and nitrates encourages germination. Invasion of the host cells by the swarm spores also occurs over a wide temperature range (45–86° F.), best at 60–68° F., and oxygen is needed for this process (Weiss *et al.*, 1923, 1925; Esmarch, F., 1924, 1926).

Soil reaction seems to have little effect on infection. It occurs over the whole range between pH 3.9 and 7.5, even pH 5 is said to be quite favourable. Soils more acid than these are seldom cultivated and one can feel no assurance that infection will not occur on soils more alkaline than 7.5 (Weiss. *et al.*, 1923; Crowther, E. M., *et al.*, 1927). No essential difference has been found between infection on clay and sandy soils.

Extreme drought may completely prevent outbreak of potato wart disease (Salaman, R. N., and Lesley, J. W., 1921; Botjes, J. O., 1925). On the other hand the warts become particularly large with a heavy fall of rain. Plants that mature early or form no tubers owing to virus disease, etc., may escape attack, as may very early varieties.

Resting sporangia in the soil survive longest when the ground is under permanent grass or

is left unbroken for other reasons. After a severe outbreak infection has been proved to persist in the soil for 12–16 years (Jørstad, I., 1930).

Immune potato varieties. Even in the last century English and Scottish farmers noticed that the variety Snowdrop remained healthy when others were severely attacked. The implication of this was first pointed out by Gough (1920) and the selection and breeding of wart-immune varieties was begun by English and German workers. In Germany now only immune varieties may be placed on the market, nor does the Swedish plant-breeding institute at Svälof send out susceptible varieties. In Great Britain only wart-immune new potato varieties receive registration.

VARIETIES IMMUNE FROM WART DISEASE

Early:
Arran Pilot
Ballydoon
Di Vernon
Doon Early
Snowdrop
(Witch Hill)
Ulster Chieftain

Second early:
Arran Comrade
Arran Luxury
Arran Signet
Catriona
Dunbar Rover
Edzell Blue
Great Scot
Ulster Monarch

Main crop:
Abundance
Arran Banner
Arran Consul
Arran Peak
Arran Victory
Champion
Doon Star
Dunbar Archer
Dunbar Standard
Gladstone
Golden Wonder
Kerr's Pink
Majestic
Redskin
Sharpes' Express
Up-to-Date
and many others

VARIETIES SUSCEPTIBLE TO WART DISEASE

Arran Chief
British Queen
Duke of York
(Midlothian early)
Eclipse
Epicure
King Edward VII
Ninetyfold
Red King Edward
Royal Kidney

The above lists include all the potato varieties commonly grown in Great Britain. One may assume that no new variety is likely to appear on the market there unless it is immune to wart disease. It will be noted that, apart from King Edward and Up-to-Date, the non-immune varieties still commonly grown are mainly earlies or second earlies. At present immunity to wart disease in the field is complete and has never broken down under British conditions. Early reports of wart disease in crops of immune varieties always proved to be due to the inclusion of a small percentage of non-immune rogues in the crop. Hence the importance attached in "seed" certification schemes to purity of the stock (see p. 341). In Thuringia and Bohemia, however, local physiological races of the fungus have been reported to be capable of attacking some varieties generally regarded as immune (Braun, H., 1942).

New potato varieties are tested for their reaction to wart disease by a laboratory technique which exposes them to infection under conditions very favourable to the fungus. In immune varieties the swarm spores penetrate the cells of the eyes but the infected cells and the fungus within them soon die. All that develops is a minute dead spot visible only under a magnifying lens or dissecting microscope. There are slight differences in reaction of individual varieties but there is no difficulty in deciding which are immune in the field and which susceptible. A few susceptible varieties, such as Bliss Triumph, only suffer slight attack on the tubers but produce large outgrowths from the stolons (Weiss *et al.*, 1923, 1925).

Control. Wart disease is easily and completely controlled by growing immune varieties. In England and Wales the disease is dealt with under the provisions of the Wart Disease of Potatoes Order 1941. Similar regulations are in force in Scotland. The English order requires immediate notification

of any outbreak of wart disease to the Ministry of Agriculture. Diseased tubers must be boiled or burned. No potatoes from an infected crop may be sold for planting and no tubers visibly affected with wart disease may be sold for any purpose. Only approved immune varieties of potato may be grown on land where wart disease has occurred at any time. In a scheduled Protected Area the only potatoes allowed to be planted are tubers from a crop certified to conform to official standards of purity and health and to have been grown on land free from wart disease.

By the Wart Disease of Potatoes (Scotland) Order of 1941 no private gardener or allotment holder in any part of Scotland is permitted to plant non-immune second early or main crop varieties, but a general licence is usually issued annually to permit the growing of non-immune first earlies.

Similar regulations are in force in Denmark with the additional prohibition of removal of any plant with soil attached from an infected garden. It is also forbidden to remove rubbish or compost from such places or to dispose of manure. Export of tubers from boroughs where infected areas occur is also prohibited.

There is no practicable method of destroying wart disease resting sporangia in a field soil. On a very small scale soil can be disinfected by steaming; baking the soil is not effective.

Treatment of soil with 5% formalin for 24 hours also kills the sporangia. Incorporation of sulphur in the soil also has some effect but only in large quantities which make the soil sour and are unreasonably expensive. Implements used on infected land may be disinfected by dipping in 2% formalin solution, but disinfection of potato " seed " has not proved effective. Cooking of potatoes used as fodder helps to prevent contamination of manure and is, in any case, required by the Wart Disease Order as far as infected tubers are concerned.

Common Scab (*Actinomyces scabies* (Thaxt.) Güssow)

The disease called common scab of potato is caused by the ray fungus *Actinomyces scabies*, which also attacks beet, carrot, radish, swede and turnip. Care is needed to distinguish between it and powdery scab (p. 324).

The ray fungi are generally regarded as being somewhat intermediate in structure between bacteria and fungi. Several hundred species occur in soils, where they play a large part in the breakdown of decaying organic matter; others live in water and a few cause disease in animals.

Cause. As early as 1890 *Actinomycetes* were thought to be the cause of potato scab in North America (Thaxter and Bolley) but their presence is difficult to demonstrate and this conclusion was not at first generally accepted. It is now, however, agreed that *Actinomyces scabies* is the cause of common scab. Leach has differentiated two races of the fungus capable of causing somewhat different kinds of scab, and others (Millard, W. A., 1922, 1923, 1926; Wollenweber, H. W., 1920) have sought to divide the scab-producing actinomycetes into thirteen or fourteen species. The usual practice, however, is to group them all in the collective species *A. scabies*. There is considerable competition between species of actinomycetes in the soil and the efficacy of green manures, grass, green rye and the like, in preventing scab depends partly on the capacity of the actinomycetes and other fungi living on the decaying grass, etc., to crowd out the scab-producing species (Millard, W. A., and Taylor, C. B., 1927; Afanasiev, M. M., 1937).

Symptoms. Potato scab appears as rough, scabby spots on the otherwise smooth skin of the tubers (Fig. 194). The spots are from $\frac{1}{16}$ to $\frac{1}{2}$ inch across but several may grow together and the whole surface of the tuber may be covered by them. If careful examination is made when the layer of cork begins to form in the skin the first signs of disease will be detected as small grey-brown spots under the lenticels (or small wounds), where the fungus is stimulating the living cells to production of additional layers of cork. The outer cork is burst open and the new cork follows suit, producing the scabby outer surface of the lesion. The more prone the variety is to cork formation the scabbier it will be.

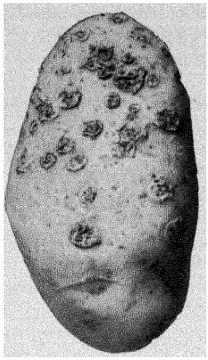


Fig. 194. Common scab of potato.

Roots and stolons of potato may be slightly attacked. Tubers that protrude above ground are not affected, nor do scabs increase in the clamp. Tubers still less than 1 inch long are the most readily infected; if they have a long growing period, the period during which they become scabby is increased, hence, perhaps, the various reports that "spraying induces scab." A strong earthy smell due to the presence of the actinomycete is peculiar to common scab; powdery scabs have no such smell.

Development. The fungus may overwinter either in the soil or in the scabs on tubers. It can survive on cellulose and similar plant products in the soil for years at a time. Several instances have been reported in which disinfected tubers planted in freshly broken forest soil produced scabby crops. On the other hand an interval of several years between potato crops has sometimes led to a striking reduction in the amount of scab (Lutman, B. F., 1923).

The fungus reproduces simply by its fine hyphæ breaking up to form spores, easily disseminated in soil and water.

Manure may be regarded as infected if it comes from farms where scabby potatoes are used raw as fodder. Infection may also be spread by compost and, of course, by planting infected tubers.

Conditions of growth. It has been claimed that where a long rotation is practised with 6–7 years between potato crops, soil infection will be so small that disinfection of seed tubers becomes important in controlling scab (Cairns, H., *et al.*, 1936). In Denmark weather conditions and the kind of soil seem to be the decisive factors. At Lyngby, potatoes have been grown ten years in succession on a neutral clay containing humus without an appreciable amount of scab appearing. The American, Halsted, noticed as long ago as 1895 that lime makes common scab worse and this has subsequently been repeatedly confirmed, by Danish liming experiments amongst others. Ashes and building refuse have a similar effect; so, in the long run, has marl. As a rule *A. scabies* is powerless at pH values below 5.1; it grows between pH 5.8 and 7.7. In very alkaline soils, for example in cultivated chalk pits with pH 8.4, potatoes may be quite free from scab; it has also been observed that if lime is dug into the potato soil in spring the tubers of that summer's crop may be smooth, but scab may appear if potatoes are grown there again the following year.

The scab fungus has a high oxygen requirement which is certainly one reason why light open soils and newly ploughed ground tend to give more scab than heavy, wet ones (Afanasiev, M. M., 1937). Dry soil is generally well supplied with oxygen and generally warm, dry weather encourages the disease while rain discourages it. If, however, late rains lead to second growth, the outgrowths may be severely attacked by scab. The scab fungus can grow at temperatures between 46° and 100° F. but most rapidly at 68–86° F., so obviously it likes warmth. 40° of frost do not kill it, nor does exposure to 195° F. for 10 minutes (Sanford, G. B., 1923, 1926; Jones, L. R., *et al.*, 1922).

Manures that lower the pH of a soil discourage scab. This was first demonstrated in Denmark in 1914 by experiments at Skals. Thus in comparable plots one can get 40–50% of the tubers scabbed when using chili saltpetre and less than 20% with sulphate of ammonia. In North America ploughing in of

sulphur has been much recommended, but this is an expensive treatment and the results may be uncertain (on light or heavily infected soils or in drought) and the low soil reaction produced is not suitable for many other crops. In Denmark the following results have been obtained by Th. Frederiksen:

Pounds of sulphur per acre	0	446	893
Percentage of tubers scabby	26	11	2

Importance. Common scab infection has not been shown to reduce the yield of a potato crop, though the dry matter content of severely scabbed tubers may be less than normal. The real importance of common scab lies in the unsightly appearance of the tubers which makes them less readily saleable. There is also more waste in peeling a scabby potato. There are regulations against exporting scabby tubers. In very severe attacks the eyes of the tuber may be so damaged that sprouting is delayed, otherwise experience has shown that quite scabby tubers may give a normal scab-free crop when soil and climatic conditions do not favour the fungus. Owing to the risk of building-up the *Actinomyces scabies* population of the soil, however, it is generally considered that scabby tubers should not be sold for " seed."

Varietal susceptibility. No potato variety completely resistant to common scab is known and on soil favourable to the fungus all varieties are attacked to about the same extent. Early varieties tend to escape infection, probably because the cork layer in their skin is formed before the soil becomes warm enough to suit the fungus. Main crop varieties do show some differences in susceptibility, probably associated in part with the size and texture of their lenticels. Breeding for scab resistance is in progress in Minnesota where the possibility of physiological races of the fungus is also under investigation.

Control. Disinfection of the tubers is effective in destroying the scab fungus on them but is only of value when they are to be planted on scab-free soil, or in soil treated with sulphur or otherwise disinfected.

Digging in grass cuttings may be effective in gardens or allotments provided they are available in sufficient quantity. Up to a stone per square yard has been recommended. On a field scale ploughing in rye or other green manure in the spring may be helpful (Millard, 1922).

Consistent use of sulphate of ammonia instead of chili saltpetre will gradually reduce the amount of scab, but one must be careful that the soil is not made too acid for other crops that need lime. Combined use of dung and sulphate of ammonia has proved helpful.

The proprietary preparation Bayer P. at the rate of about $\frac{3}{4}$ oz. per square yard or 54 lb. per acre, dug well in before the potatoes are planted, may greatly reduce both common scab and stem canker. It works very well when used with sulphate of ammonia.

Liming may be necessary because of other crops in the rotation, but it is well to have the soil analysed and to apply only the lime requirement. Where it is important to obtain a crop of smooth tubers a piece of ground may be kept unlimed for potato production. Continuous cropping with potatoes is liable, however, to lead to trouble from other diseases and especially from root eelworm.

Powdery Scab (*Spongospora subterranea* (Wallr.) Lagerh.)

Symptoms and development. Powdery scab, sometimes known also as corky scab, first appears on the skin of potato tubers as pale little pimples, $\frac{1}{8}$ to $\frac{1}{4}$ inch across. Either before or after lifting, the skin over the pimple

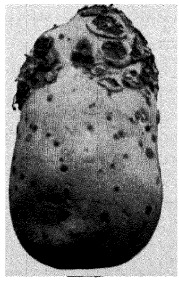


Fig. 195. Powdery scab of potato (*S. p. F.*).

splits and falls off, exposing a snuff-brown powder composed of the spore balls of the fungus (Fig. 195). For a considerable time the individual powdery scab remains as a shallow crater-like hollow, filled with brown powder and fringed by the loose edges of burst skin. Under good cultural conditions the scab seldom exceeds $\frac{5}{8}$ inch, but in potatoes grown in wet land or which have made considerable second growth, large open scabs and cankers may be formed, especially on the ends and protuberances of the tuber. In such extreme forms of the disease microscopic examination may be needed for confident diagnosis (Fig. 196). *Corticium solani* may be

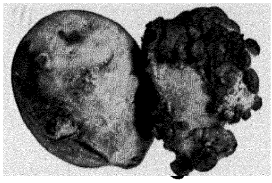


Fig. 196. Powdery scab attacking the sprouts and stimulating wart disease (*S. p. F.*).

found in the wounds and various other secondary organisms may enter and set up a rot. In exceptional circumstances the fungus may continue its activity on tubers in the clamp and stimulate them to production of irregular tumour-like outgrowths closely resembling those of wart disease.

Spongospora subterranea also attacks the roots of potatoes, and less often of tomato, causing irregular white nodules up to about $\frac{1}{8}$ inch across. It is doubtful if these cause any damage to the plant but they contain spores which are liberated into the soil when the roots decay and thus add to the reservoir of infection in the ground.

S. subterranea is one of the most primitive fungi, a relative of the fungus which causes club root in cabbages. Its spores, which are stuck together in peculiar sponge-like balls, can remain alive in the soil for 4–5 years at least and are easily spread to new localities in soil on tubers or by adhering to roots of transplants. When they germinate they liberate swarm spores which penetrate either the roots through root hairs or the tubers through lenticels or small wounds.

Cultural conditions. Powdery scab is found on both sides of the Atlantic and in the potato-growing districts of South America, but is only of importance where there is a heavy rainfall and on ill-drained soils. In dry soils a reasonably clean crop may be lifted even though grown from infected tubers. No varieties are immune from the disease but there is some difference in susceptibility, said to be correlated with the thickness of the cork layer in the skin of the tuber (Wild, N., 1929). Liming is said to make the trouble worse, and application of sulphur at the rate of 3–6 cwt. per acre checks it (Pethybridge, G. H., 1913).

Powdery scab was first described in England in 1846 but for long afterwards it was confused with common scab. It was first found in Denmark in 1889. It is prevalent in parts of the West and North West of England, in Wales, much of Scotland and in the Faroe Isles and has spread as far as Iceland.

Control. Where the disease is prevalent, soil drainage should be made as efficient as possible and the rotation should be arranged to leave 4–5 years between potato or tomato crops. Soil disinfection is impracticable in the field. Diseased tubers should not be returned to the land but may be cooked and used for fodder. Manure from animals fed on raw infected potatoes will contain undamaged spores and should not be used for potato fields, nor should potato peelings and similar kitchen refuse be put in the compost heap.

Disinfection may be practised where infected tubers have to be used on land free from the disease. They may be dipped for 3 hours in formalin solution, 1 pint of formalin in 30 gallons water, or 1: 2,000 corrosive sublimate solution. Moistening the tubers and rolling them in powdered sulphur may also be effective. Spores of closed scabs still in the pimple stage may not be killed by any of these methods.

Under the Sale of Diseased Plants Orders of 1927 and 1936 potatoes substantially affected with powdery scab may not be sold for planting in Britain.

Secondary scab injury. Both in common and powdery scab, especially when associated with second growth, the damage is often increased by the activity of a number of insects and other pests, such as the larvæ of *Sciara* flies, wood lice, and millipedes which penetrate the outer layers of the tuber flesh by way of the scabs and cracks and convert it into a dry crumbling crust. The stem eelworm *Anguillulina dipsaci* produces extreme crust-like rotting of this kind. Secondary fungi may also play a part; even dry rot often starts at a powdery scab lesion. When storage conditions are too damp, or too warm, soft-rotting bacteria obtain easy access to the damaged tissue and the whole tuber decays.

Black Scurf and Stem Canker (*Corticium solani* (Prill. & Delacr.) Bourd. & Galz. = *Rhizoctonia solani* Kühn)

This, one of the commonest and most cosmopolitan of fungi, was first recognized in connection with potatoes, with which it is almost invariably found. But it also attacks many other plants, cabbage, tomato, wheat, lettuce, asters, though sometimes in the form of a specialized strain more or less adapted to a particular host, and has been found on many weeds such as buttercup, shepherd's purse, field thistle, plantain, etc. (Dana, B. F., 1925). It can also live freely in the soil.

Kühn described the infection of potatoes in 1858 and called the fungus *Rhizoctonia solani* as he saw only the sclerotial stage. Subsequently the perfect basidial stage was found and called *Hypochnus solani* Prill. & Del., afterwards transferred to the genus *Corticium*. More recent studies have made it possible to sub-divide this very large genus into a number of smaller more clearly defined genera. When this is done *C. solani* falls in the genus *Pellicularia* and some authors think it identical with a fungus described earlier under the name *Hypochnus filamentosa* Pat., hence it has been re-christened *Pellicularia filamentosa* (Pat.) D. P. Rogers. This name has not yet, however, been generally accepted.

Symptoms. On potato tubers one often finds hard scurfy patches of any size up to about ½ inch across, to which the soil adheres. When the tubers are washed these patches show up as dark-brown or black spots and lumps (Fig. 197) They are the sclerotia of *C. solani*, sometimes described as "The dirt that won't wash off." They are attached to the skin by very fine

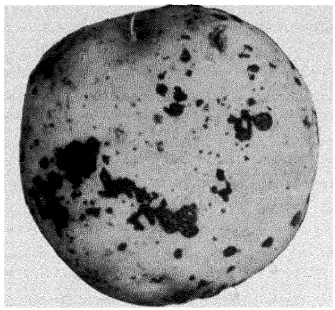


Fig. 197. Sclerotia of *Corticium solani* on potato tuber (*S. p. F.*).

hyphæ and can be scratched off with the finger nail, leaving the uninjured skin of the tuber beneath. Apart from making ware potatoes a little unsightly this phase of the fungus's life history does no immediate damage. Occasionally it has been found to attack tubers through the lenticels before they are lifted, causing small shallow brown pits rather like those of pit rot (Moore, W. C., 1948; Ramsey, 1917). When "seed" tubers carrying *C. solani* sclerotia are planted, however, the fungus becomes active again and many cause more or less serious injury to the sprouts. It may be found on the young sprouts even in the chitting boxes but most damage is done after planting. Then sunken brown spots appear on the sprouts below soil level. Or the tip of the shoot may be killed before it emerges above ground; it then puts out a side shoot which may escape or may also be killed. In the worst cases groups of such dead shoots are found with the result that blanks occur in the potato drill (Fig. 198).

Later in the summer the fungus causes dry, brown, sunken wounds on the underground parts of the stems. If these are deep and girdle the stems the haulm may be weakened and the leaves roll, somewhat as in primary leaf roll

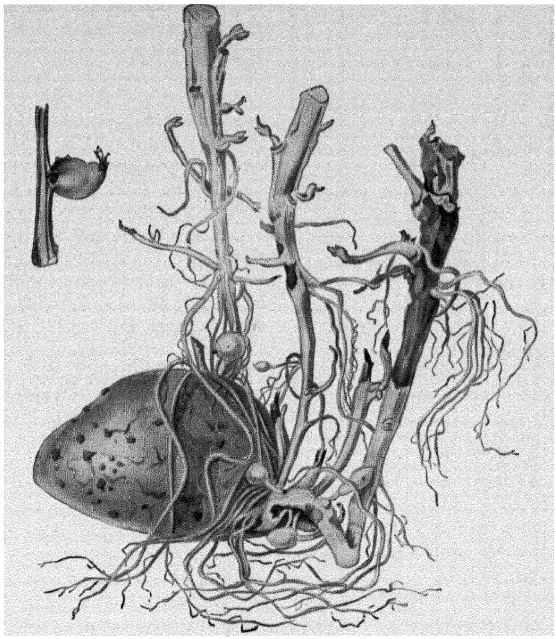


Fig. 198. *Corticium solani* attacking potato shoots; aerial tuber on top left.

though less stiffly. When the starch cannot reach the tubers, green aerial tubers are produced in the axils of the leaves (Fig. 198). Brown lesions also appear on the stolons, which may be killed. The plant then forms merely a cluster of small angular tubers round the base of the haulm. The roots too may be attacked, especially the fine absorbing rootlets (Güssow, H. T., 1917).

In all these brown sunken lesions only the mycelium of the fungus, or occasionally *Rhizoctonia* sclerotia, can be found. But at the base of vigorous haulms where the air remains moist the fungus covers the stems just above soil level with a white powdery film, sometimes extending up them to a height of 6 inches (Fig. 199). This film consists of a layer of basidia and basidiospores. No harm seems to be done to the plants by this sporing stage of the fungus but it offers special possibility for its spread by means of wind-blown spores. The effect of an attack on yield of tubers varies greatly from practically nothing to severe losses where the fungus has caused extensive gaps in the drills.

Conditions of growth. Potato sprouts may be infected partly from the seed tubers, partly from the soil. The fungus seems to thrive in all cultivated soils, is not much affected by soil reaction and can grow over a wide range of temperatures (optimum about 65–75° F.). The rate at which the tubers sprout determines the severity of the disease in spring (Richards, B. L., 1923) because the damage is greater when potatoes are planted early in cold soil; nevertheless early planting generally pays! As a rule lavish use of dung and artificial manures helps the crop.

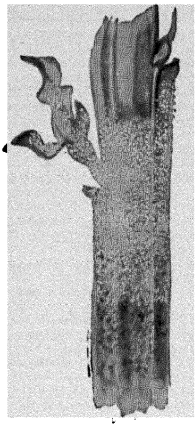


Fig. 199. Basidial layer of *Corticium solani* at base of potato stem.

In autumn the fungus seems first to produce its crust-like sclerotia on the tubers when their skin matures, thus one may find 2–3% of the tubers infected in August, but 20–30% by the end of September (Bisby *et al.*, 1923).

Races. If isolates of *C. solani* from numerous sources are compared they are found to be classifiable into a number of races obviously different in growth habit and nutrient requirement. Cross-inoculation experiments have shown that the races also differ physiologically in their capacity for parasitism, though they are not very strictly confined to a particular host. Races isolated from potato can attack tomato, while potato can also be attacked by isolates from cabbage, from various kinds of cereal and by the strains called *Moniliopsis aderholdi* (see p. 68).

As the fungus can grow in soil, for example, from one potato plant to another, and can survive the winter in the soil, planting of healthy " seed " potatoes affords no guarantee that the crop will not be attacked by stem canker, as had already been observed by E. Rostrup.

Control. In 1938 Th. Frederiksen, C. A. Jørgensen and O. Nielsen published the results of a series of investigations and experiments on the behaviour of *C. solani* in Denmark, with recommendations for its control, and on this report we mainly rely. Early lifting of tubers, before they are ripe, gives the cleanest potatoes. Rotation of crops with at least five years free from potatoes may be recommended, even though the intermediate crops cannot be guaranteed free from infection. Similarly use of stable manure is to be recommended even though there is some possibility that it may be infected (Dorst, J. C., 1923). One should also not be sparing with artificial manures. In experiments in the State of New York, green manure (rye ploughed in), stable manure and artificial manures, both alone and in combination, all definitely reduced damage by *C. solani* (Blodgett, F. M., 1939). The potatoes should be planted as near the surface as experience shows to be safe in relation to drought and blight, for example, at a depth of about $2\frac{1}{2}$ inches. Sprouting the tubers before planting seems slightly to increase stem canker on the young shoots but has so many other advantages that it should not be abandoned on that account.

Potato varieties show little difference in susceptibility to stem canker.

Dipping the " seed " tubers in Aretan, Hortosan or similar preparations may kill the fungus on them but offers no protection against soil infection. Disinfection with solutions of corrosive sublimate and formalin has been recommended in some countries but is liable to injure the tubers. Copper preparations seem to have little effect. Tuber disinfection should take place before the eyes show signs of breaking—to be effective against other diseases it should be done immediately after lifting (see dry rot, p. 330, and skin spot, p. 331). It has the disadvantage that treated tubers cannot be used for food or given to stock.

Soil disinfection with a Bayer preparation (see potato scab, p. 324) has given promising results in Danish experiments.

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

Symptoms and development. In Britain and Denmark the symptoms of *Verticillium* wilt

in potato usually develop only after the haulm has attained a normal size, though in extreme cases it may be stunted, with all the leaves rolled, and yields a negligible crop. More often, from July onwards, the leaves become limp and turn yellow at the tips and edges. Often one side of the leaf turns yellow and dies. Finally the leaves die and hang down brown and shrivelled against the stem, so that the whole plant gradually dries from below upwards. Characteristic of the disease is the brown stain in the woody strands, often extending throughout the whole haulm. In autumn a delicate velvety white mould of conidiophores may appear on the base of the dying plants, and finally the stem may be blackened by the minute resting bodies of the fungus.

Tubers on infected plants are usually also infected from the stolons, and if one slices them at the heel end the vascular bundles will be found to be brown, often far into the flesh. Brown vascular bundles occur also in *Fusarium* wilt, black-leg and other diseases, including leaf roll, and some forms of slight frost damage. There is no natural separating layer between the tuber and the stolon so that when the crop is lifted a small wound is inevitably made at the heel end which takes a little time to heal (Edson, H. A., and Shapovalov, M., 1920).

Before the cause of potato leaf roll was understood it was frequently confused with *Verticillium* wilt and there was consequently a tendency to exaggerate the prevalence and importance of the latter disease.

Importance. In severe attacks and at high temperatures infected plants are very stunted, quickly die and yield only a few tubers like marbles. Generally, however, they give a fair yield of apparently normal tubers. These are, however, infected with *Verticillium* and are not suitable for "seed," as they will give diseased plants.

The disease was first described in Germany in 1879 but was not observed in Denmark until 1917. Observations at Lyngby show that tuber infection plays a considerable part but there are usually only a few diseased plants scattered throughout the crop and losses of the order of 10–20% from this cause are rare. In England the disease is reported every year but very seldom causes appreciable loss.

Effect of environment. *Verticillium* wilt is widespread on both sides of the Atlantic but in warm climates it tends to be superseded by *Fusarium* wilt. High temperatures check the growth of the fungus but the drought and excessive transpiration accompanying them usually kill all the infected potato plants. Experiments have indicated that heavy nitrogenous manuring increases the susceptibility of the crop; soil reaction has little effect (Van der Meer, J. H. H., 1924).

Varietal reaction. In Denmark *Verticillium* wilt is mostly seen in the variety Juli, but Up-to-Date, Magnum Bonum and King Edward are all susceptible. According to Wollenweber, the German varieties Wohltmann, Jubel and Goldpearle are fairly resistant.

Control. Provided a normal rotation of crops is practised the disease is unlikely to become prevalent. Crops containing many infected plants should be used for ware only. If a few plants show *Verticillium* wilt in a crop grown for "seed" they should be dug out and burnt, preferably with those on each side in the drill as otherwise a few infected tubers may be left and lifted with the crop. Also neighbouring plants may have become infected by root contact and if this occurs late in the season they may not show distinctive symptoms before the haulm begins to ripen off.

Pethybridge (1916) showed that the fungus in infected tubers can be killed by keeping them for 20 hours at a temperature of 114° F. but such treatment has never come into commercial practice. Further information about *Verticillium* wilt will be found on page 60.

Fusarium Wilt (*Fusarium oxysporum* Schlecht.)

In warm climates, especially in North America, *Fusarium oxysporum* is an important cause of potato wilt. It has only once been definitely identified in England, in Kent in the hot summer of 1928.

Its presence in Denmark has occasionally been suspected but never confirmed.

In this disease the top turns yellow and wilts during the summer. It is associated with a brown discolouration of the vascular bundles throughout the plant, as in *Verticillium* wilt; indeed, the two diseases can only be distinguished with certainty by isolation of the fungus in pure culture. The *Fusarium* enters the tubers along the vascular bundles from the stolons and in the worst cases it grows through them all round the tuber so that a brown ring is visible when it is cut in half. During the winter the fungus slowly grows into the flesh and may cause rotting of the whole tuber. If those only slightly infected are planted they give rise to infected shoots which grow only into small stunted plants. *Fusarium oxysporum* requires a fairly high temperature; it makes scarcely any growth at 48° F. and its optimum lies between 77° and 86° F. In cool climates it is only likely to be troublesome in potatoes forced under glass. Warm, damp soils favour infection but drought hastens the death of the infected plant.

Trouble is not likely to arise even in forced potatoes if they are raised from " seed " from a normal field crop. If the disease does occur potatoes should not be grown in the same soil for several years.

Black Dot (*Colletotrichum atramentarium* (Berk. & Br.) Taubenh.)

This very common fungus may damage potato haulm in dry seasons. The stems are attacked just below soil level, and become marked or even girdled by dark-brown or blackish lesions from which the skin breaks away. Girdling of the stems leads to yellowing and rolling of the leaves and premature dying of the haulm. The fungus is common on the tubers where it causes silvery grey patches covered with tiny black dots like those of silver scurf (p. 332). It may also girdle the stolons and penetrate some distance up the stems along the vascular bundles. The tiny black sclerotia, sometimes bearing conidia on their surface, may be found on any of the diseased tissues. King Edward, Red King and Doon Star are considered to be susceptible varieties.

The fungus is cosmopolitan and has been described under several other names, of which *Vermicularia varians* Ducom. is that most used on the Continent. It is regarded as a serious parasite in France but is of little consequence in cooler climates. It occurs from time to time on potatoes in Denmark, notably in 1937 on crops weakened by drought and malnutrition. In the English Midlands it attracted some attention in 1928, 1934, 1940, 1942 and 1944–5. The fungus is better known as a parasite of tomato (p. 376) and egg plant.

Control measures have not been worked out but if the fungus becomes troublesome one should practise a long rotation keeping the land free from potatoes or tomatoes for several years, and dip the " seed " tubers in an organo-mercury disinfectant before planting.

Dry Rot (*Fusarium coeruleum* (Lib.) Sacc. and other species)

During storage of potatoes numerous species of *Fusarium* may gain access to the flesh of damaged tubers, sometimes through wounds but mainly through tissues already killed by blight. Dead patches of tissue caused by sun scald or damaged by disinfectant or other chemicals may also afford entry to weak parasites. Susceptibility of the tubers to these rots increases during the winter.

True dry rot, however, is caused mainly by one easily recognized species of *Fusarium*, *F. coeruleum*, so-called because its conidial pustules instead of being salmon pink have more or less of a blue tint. They may be covered with a white or pinkish spore powder but if this is rubbed off the blue colour will be found in the base of the pustules. It also sometimes appears in the mycelium, lining cavities in the rotting flesh. *F. coeruleum* is an active parasite though it may often gain access to the tuber through a scab wound or a bruise.

The first sign of dry rot is a brown patch on the skin, which rapidly increases in size, becomes slightly sunken and surrounded by concentric wrinkles. The underlying flesh is deeply penetrated by a soft brown rot, full of

cavities lined with white or blue mycelium, and giving off a peculiar sweetish smell. In time the rot destroys the whole tuber which becomes hard, dry, shrivelled and very light in weight. The characteristic conidial pustules burst through the dead skin.

Many other species of *Fusarium* have been found on rotting potato tubers but of these only *F. avenaceum* (Fr.) Sacc. seems to be of much importance in Britain (Moore, F. J., 1945).

Control. Infection of tubers with *F. coeruleum* occurs from the soil (Foister, C. E., *et al.*, 1945). Dry rot in tubers intended for "seed" can be prevented by dipping them in a solution of organo-mercury disinfectant immediately after lifting. The treatment is ineffective if applied later in the winter and has not been generally adopted, largely on account of the labour involved. It is also a disadvantage that it cannot safely be applied to the ware intended for human consumption, so that riddling the crop has to be done immediately after lifting, at a time when labour can ill be spared. Attempts to find a dry disinfectant which would be effective against dry rot without damaging the tubers have not yet been successful (Foister, C. E., and Wilson, 1943).

It is important to keep potato clamps cool and well ventilated. They should not occupy the same site two years in succession. Sprouting boxes and chitting houses should be disinfected with 5% formalin solution or 2% copper sulphate solution in good time and then well ventilated before the tubers are placed in them. Slightly infected tubers may be boiled and fed to pigs or poultry. They should not be planted as they will probably decay and leave a blank in the drill. Dry rot causes most loss in early varieties, especially Arran Pilot, May Queen and Ninetyfold. Epicure is fairly resistant. Of main-crop varieties Doon Star is particularly susceptible.

Gangrene (*Phoma foveata* Foister)

This is the name given to a minor rot of potato tubers which starts somewhat like dry rot, as a brown sunken patch rather like a thumb-print, but in most varieties does not become very deep or involve the whole tuber. Another species, *Phoma solanicola* Prill. & Delacr., may cause even smaller, shallow, more or less circular blackish spots on the skin but is of no economic importance (Dennis, R. W. G., 1946). *P. tuberosa* Melh. may be the same species. *P. eupyrena* Sacc. sensu Wollenweber, sometimes mentioned in Continental literature, is not parasitic to potato tubers.

Skin Spot (*Oospora pustulans* Owen & Wakef.)

On certain potato varieties one often finds small hard dark pimples in the skin of overwintered tubers. The individual pimples are above $\frac{1}{16}$ to $\frac{1}{8}$ inch across, and often surrounded by a slightly sunken ring, but they may be crowded together to cover considerable patches of skin. The underlying tissue, seen when the potato is peeled, is blackish brown to a depth of about $\frac{1}{16}$ inch. Not only are affected tubers unsightly but their eyes are

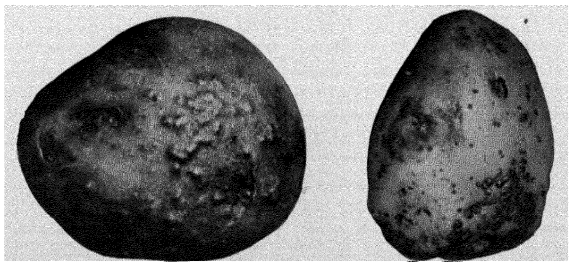


Fig. 200. Skin spot pustules on potato tuber (*Photo Dept. Agric. Scotland*).

often killed. Hence when planted they fail to sprout. Heavy losses due to this cause are often reported in King Edward, Majestic and other varieties, particularly when grown from Scotch or Irish " seed." Blind " seed " in which the eyes have been killed by skin spot does not usually decay in the drill. It may be recovered when the crop is lifted and is often found to have formed a small irregular tumour of callous tissue at the heel end. In bad years losses up to 60% of the consignment may occur from failure of the " seed " to sprout.

Skin spot is due to the wound fungus *Oospora pustulans* which infects the tuber from the soil. Most of the damage is done during storage, and Foister (1943) has shown that dipping " seed " tubers in an organo-mercury disinfectant solution immediately after lifting gives considerable control of skin spot, as well as of dry rot.

Death of the eyes and sprouts is favoured by cold, damp conditions. Hence sprouting the " seed " in a dry, light place tends to reduce the loss from this disease. Whenever possible the " seed " should be boxed when lifted or soon afterwards, not after prolonged storage in a clamp, by which time most of the damage may have been done.

Silver Scurf (*Spondylocladium atrovirens* Harz)

Sometimes silvery patches of skin are noticeable on potato tubers, especially when they are washed. The underlying tissue may be quite normal or a little darker than usual (Fig. 201). Silver scurf is seldom seen at lifting time but develops in the clamp and may be regarded as a slight blemish rather than a disease. In wet clamps the condition may be widespread and extensively affected tubers may tend to shrivel if stored afterwards in a dry place. Otherwise no real harm is done. Under warm, damp conditions a black velvety layer of short conidiophores and conidia appears on the silvery patches, springing from very tiny black specks in the skin, rather like the tiny sclerotia of *Colletotrichum atramentarium* (see p. 330). The fungus is cosmopolitan, known in Denmark since 1904, and

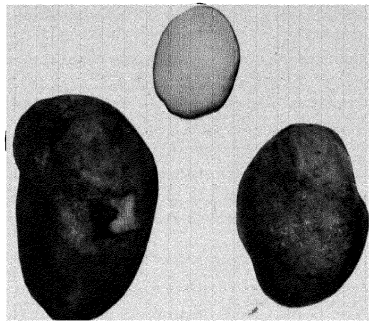


Fig. 201. Silver scurf on King Edward tubers (*Photo Dept. Agric. Scotland*).

likes moderate warmth, its optimum temperature is from 70–80° F. No control measures are normally called for, but if the fungus becomes undesirably prevalent a longer interval between potato crops should be tried, as infection of the tubers is derived from the soil.

Pink Rot (*Phytophthora erythroseptica* Pethybr. and allied species)

This disease occurs sporadically throughout the British Isles but is seldom very destructive except in the West of Ireland.

The fungus, which occurs in the soil, may attack the underground parts of the haulm and the stolons, sometimes causing the plants to wilt, but this usually happens only at the end of the season. Losses from the disease are due to infection of the tubers. This occurs normally at the heel end while they are still attached to the parent plant, but the fungus may also sometimes gain access through an eye or a wound. Inside the tuber it causes rather a rapid rot but the diseased potato remains fairly firm until it is completely killed. When squeezed, however, sap generally oozes from it. If an infected tuber is cut and exposed to the air the flesh turns pink in a few minutes and within half an hour the whole diseased portion becomes salmon-coloured. Later it slowly turns purplish-brown or blackish-brown. Similar colouration may occasionally

occur in healthy tubers or in those attacked by black-leg.

The infected tubers decay fairly quickly and are not likely to survive until planting time, hence there is little risk of spreading the disease with the "seed." As the causal organism is a soil-borne water-mould, thorough drainage of potato fields is desirable and a long rotation should help to prevent infection of the soil being built up to a dangerous level. All infected tubers should be destroyed, not dug or ploughed back into the land.

Watery Wound Rot (*Pythium ultimum* Trow)

This disease is also due to infection of the tuber by a water mould derived from the soil. It is a much more rapid decay than pink rot, and leads to a complete, soft, pulpy rot of the whole heart of a tuber, somewhat like the tuber symptoms of black-leg. As in that disease, cavities commonly occur in the rotten flesh, which may have a fishy odour. The fungus likes warmth and the rot may be checked by storage under cool conditions. Infection occurs through wounds and therefore care should be taken during lifting not to damage the tubers more than is unavoidable. This is especially important with crops lifted early and in warm weather. Diseased tubers should be collected and burnt or deeply buried, not ploughed back into the land to increase the amount of infection another year.

Jelly-end Rot

This is a physiological disorder associated with a form of second growth, especially in Arran Pilot. When second growth occurs evenly over the rose end of the tuber, so that it becomes abnormally elongated, the tissue at the heel end loses its starch and becomes soft, flabby and shrunken. Ultimately it usually shrivels up and falls off leaving an irregular scar.

Black-leg (*Bacterium phytophthorum* (Appel) Burgwitz)

Since 1888, when Sorauer referred to the bacterial rot of potato stems as "Schwarzbeinigkeit" the common name for the disease in all countries has had some reference to the "black-leg." There has, however, been some difference of opinion about the precise identity of the bacterium concerned. It is now generally agreed that *Bacillus atrosepticus* van Hall and *B. melanogenes* Pethybr. and Murphy are synonyms of *Bacterium phytophthorum*, but *B. solanisaprus* Harr., which causes a black-leg disease of potatoes in Canada and rotting of other plants, seems to be a distinct, though related, species. Several writers have contended that *B. phytophthorum* is the same as the ubiquitous soft-rot organism *B. carotovorus* (L. R. Jones) Lehm. The two are certainly closely related, but Dowson (1940) has shown that *B. carotovorum*, though able to rot potato tubers, does not cause the typical black-leg symptoms on the stems.*

Symptoms and development. Bacterial rot of potato tubers may be due to several bacteria, including *B. carotovorum*, *B. phytophthorum*, *B. aroideae* (Towns.) Stapp, *Pseudomonas marginalis* (N. A. Brown) Stapp (Dowson, W. J., 1941), *Bacillus polymyxa* (Prazonowski) Migula (Dowson, 1943), and *Bacillus subtilis* (Allen, 1944). Some of these soft rots are probably secondary to blight, but *B. phytophthorum*, at least, causes an independent rot in tubers harvested from a plant with black-leg. Slightly infected tubers show only faint brown discolouration in the flesh at the heel end round the vascular bundles, or small spots below the lenticels, and cannot be detected when the crop is graded. In more advanced stages of the disease a soft wet rot starts at the heel end. The rotten tissue may have no special colour or smell, but when the tuber is cut it quickly turns pink and then brown and black with exposure to the air. Often there is a reddish or black line between the decayed and healthy flesh and a similar line may appear in the skin outlining the rotting tissue. In advanced stages of decay the potato may be quite hollow. If it is slimy and has a fishy smell other bacteria are present (see wet rot). Slightly infected tubers may decay in the

* More recently Dowson (1949) has agreed that it is impracticable to distinguish *B. phytophthorum* from *B. carotovorum* and the latter name is likely to be that adopted in future literature on Black-leg of Potato.

clamp, especially if frosted or allowed to become moist. The bacteria may then spread to healthy potatoes round about, partly, perhaps, by being transported on the bodies of mites feeding on the dead tissue in scabs and wounds.

There has been much discussion of the relative importance of black-leg infection introduced into the crop in slightly infected seed tubers and that induced by bacteria already present in the soil (McIntosh, T. P., 1941). In the former case the infected tubers may rot in the drill without sprouting. More often they give apparently normal plants, though the haulm may be rather small and pale, until in June or July their leaves suddenly turn yellow and roll up. If the haulm is given a slight tug it comes up easily and the lower part of the stem is found to be soft and black, or perhaps already shrivelled. The seed tuber will have decayed completely. Closer inspection shows that the cortex and pith of the stalk are black and the bacteria may have spread some way up the vascular strands, staining them brown. The plants usually die in 4–5 days, but under favourable cultural conditions the disease may be less acute, the top leaves are somewhat curled, and the plants more or less stunted, possibly with aerial tubers in the axils of the leaves. The underground part of the stem is brown and rather soggy, but not so black and slimy as in the severe phase of the disease. It is probably from these chronic cases that infected tubers are harvested with the " seed " crop.

Sources of infection. *B. phytophthorum* is usually recorded only on potatoes, but of late years it has been isolated from lettuce plants affected by a decay of the leaf edges and from decaying swedes. If it is identical with *B. carotovorum* a much wider host range is indicated (see p. 257). In Denmark the authors are familiar with gardens where the soil has carried potatoes for 30 years in succession without a serious outbreak of black-leg. This does not indicate an appreciable degree of soil infection, and in four experiments designed to test the possibility the black-leg organism was not shown to overwinter in the soil from one crop to the next, whereas the disease was obtained by planting infected tubers. It has been claimed that the bacteria may survive the winter in ground-keeper tubers and in decaying haulm, partly also in the intestinal tract of insects which have fed on diseased plants, but these observations have not been confirmed in Denmark. Infection from dung seems possible, judging by observations in Denmark and Norway (Jørstad, I., 1932), but heavy dressings of farmyard manure tend to delay onset of the disease. Spread of black-leg from one plant to another along the drill seems to be negligible (Botjes, J. G. O., 1928). The bacteria may be transported in soil water, and in North America larvæ of flies and other insects have been credited with carrying the bacteria from diseased plants, or infected soil, and infecting healthy plants on which they feed (Leach, J. G., 1925).

Under Danish conditions, however, the conclusion is that infection comes mainly with the seed tubers. When seed is cut before planting the disease may easily be spread on the knife from a diseased tuber to many healthy ones. If the latter get plenty of air, wound cork will form beneath their cut surfaces, but if they are planted in wet soil immediately after cutting the bacteria will have full play. Infection may also be spread by planting machines; soft black-leg-infected tubers will be crushed and the infected juice rubbed into the small cuts which the machine is apt to inflict on sound tubers passing through it. In Great Britain the tendency of late years has been to emphasize the importance of soil-borne infection (Moore, W. C., 1943).

Importance. In Denmark, as in Britain, black-leg plants are often to be seen among both early and main crop potatoes in gardens and fields. In the fields the disease is worst on light soils deficient in nutrients, especially phosphoric acid. Ill-drained, wet, heavy soils seem to encourage black-leg, which is certainly worst in wet years. A general loss up to about 2% of the crop is to be expected, and occasional severe outbreaks occur. Black-leg appears to have been particularly widespread in Denmark in 1908 and 1918. In England

and Wales the disease was unusually prevalent in 1936, 1942 and 1945 (Moore, W. C., 1943, 1947).

Control. If seed potatoes cannot be obtained from a completely healthy crop all plants showing symptoms of black-leg should be dug out of the seed crop in early summer. Provision exists in the British potato seed certification schemes for rejection of crops showing an unusually large amount of black-leg. Potato clamps should be kept dry and cool but, of course, protected from frost. Sprouting the tubers in the light before planting may have a favourable effect. Some growers dry their potatoes in a barn for a few weeks before putting them into clamps.

Disinfection of the tubers has not given appreciable control and may cause injury (Botjes, 1928).

Whole " seed " is preferable to cut tubers. If the sets must be cut they may be dusted afterwards with a mixture of about ½ lb. powdered sulphur and ½ lb. ground lime per hundredweight.

There is always a risk of spreading infection when uncooked potatoes and peelings are thrown into the manure heap.

On low-lying wet land potatoes should be planted shallow and late.

All potato varieties seem to be susceptible to black-leg.

Wet Rot

Even at lifting tubers may be found that are completely soft and rotten; as a rule they are from a black-leg plant or have been suffocated by flooding or puddling of the soil. Potatoes that have been attacked by blight and black-leg or have been damaged during lifting and bruised by rough handling in transit, packed tightly in railway vans or in a ship's hold, or stored under ill-ventilated conditions, may be transformed in a few days into a rotten mess. Numerous species of bacteria may play a part in this decay (see p. 333).

If heating in the clamp is discovered in time and ventilation is supplied many of the tubers will merely develop black-heart. If not, and the temperature continues to rise, the potatoes will be suffocated and afterwards reduced to a soft, stinking, slimy mass by the common putrefactive bacteria that cannot attack living tissues.

Little Potato (Premature Tuber Formation)

In consignments of potato " seed " individual tubers may be found in which the eyes have grown out into short stolons with new small tubers at the end. Normally this premature tuber formation is of no economic importance. In some years, however, as in Denmark in 1935, it may be widespread after the " seed " has been planted and may then lead to considerable loss. It is more common in early than in late varieties, especially when the " seed " was lifted before being fully ripe. Sometimes the stolon grows inwards and one or more new tubers are formed inside the old one which then splits and shrivels. Wellensiek, in Holland (1924, 1929), has shown that the following factors are conducive to little potato:

(1) Drought with premature ripening of the " seed " crop.

(2) Storage at temperatures from 48–55° F., especially when this leads to rapid growth of sprouts which have to be removed once or twice during the winter or are easily broken off during planting.

(3) Planting in a soil with a temperature of only 40–45° F.

The " seed " tubers should, therefore, be stored dry and at a low temperature, should be sprouted in boxes in the light at 40–45° F. and should not be planted until the soil temperature rises to 45 or 50° F. Potatoes stored dry certainly lose moisture and shrivel but that is of no consequence.

Green aerial tubers in the axils of potato leaves are formed whenever translocation of starch downwards to the stolons is hindered. This may be the result of stem canker, leaf roll, insect damage, injury during hoeing, etc. In 1911 at Lyngby we planted tubers from plants which had borne these aerial tubers; none were formed in the resulting

crop, so the condition is not an hereditary disorder.

Spindling sprout is the name given to very fine thread-like sprouts sometimes produced by " seed " tubers. The condition seems usually to be due to immaturity of the " seed " and is common only after abnormally hot, dry summers, in which the eyes are imperfectly developed and the tubers not fully grown when the haulm dies of drought.

Virus Diseases

Virus diseases occur wherever potatoes are grown, though they only spread rapidly in climates favourable to their insect vectors, hence potatoes grown in some mountainous countries and in exposed wet maritime districts can be kept free from viruses without great difficulty. In commercial potato fields the virus-infected plants occur scattered throughout the crop because they arose from tubers which were already infected when they were planted. Secondary spread of the diseases within the crop is usually evident only late in the season or only in plants grown from the harvested tubers the following year. In lowland districts the decline in productivity resulting from virus infection would render potato production uneconomic if fresh " seed " tubers were not secured every few years from a suitable " seed " producing region.

Tubers from a diseased plant mostly contain the virus or viruses that have attacked it but a few may sometimes escape infection. Once a tuber has become infected, it, and the plant grown from it, always remain diseased. Hence the primary source of potato virus disease is the planting of diseased tubers. It is impossible to tell from its appearance or by any chemical test whether a tuber is virus-infected or not.

Secondary spread within the crop takes place in two ways, by mechanical inoculation as leaves of a diseased plant rub against those of a healthy one in the wind, and by aphides, especially *Myzus persicae* and *Aphis rhamni*. Some viruses are spread only by one means, others by the other, and some can spread by both as shown in the following table:

Solanum Virus No.	1	2	3	4	7	8	9	14
Spread by mechanical inoculation	+	+	(+)	+	—	+	+	—
Spread by aphides (greenfly)	—	+	+	—	—	(+)	—	+

+ = Virus spread by method indicated. (+) = spread only when some contributing factor is satisfied as well. — = Not spread by this means.

Aphides can also spread aphis-borne viruses from sprout to sprout of potatoes in store or in the chitting house. In commercial potato-growing districts ground-keepers, which often persist for years after a potato crop, are a fertile source of aphis-borne viruses.

The **symptoms** caused by infection by a potato virus vary greatly, partly according to the variety of potato infected, partly according to the time elapsed since infection (symptoms in the second years are often very different and more severe than those in the season of infection) and partly according to environmental conditions, for heavy manuring, especially with nitrogen, and warmth may " mask " the symptoms in the haulm. Carriers, varieties which can be or always are infected with a particular virus without showing any symptoms, are common among potato varieties. In practice potato virus diseases are usually referred to one of four categories, irrespective of the particular virus present. These are leaf roll, mild mosaic, severe mosaic and aucuba mosaic.

True leaf roll (Fig. 202) is caused only by *Solanum* Virus 14 (see p. 340). This is the



Fig. 202. **Leaf roll of potato,** healthy plant on right.

most destructive of all potato virus diseases and the most difficult to control. Rolling of the leaves may also be caused by drought, waterlogging, fume damage, *Corticium solani*, black-leg, insect damage to the stem, eelworm and the like, but in all these cases the rolled leaves are limp or at least pliable, and usually it is the upper young leaves that are affected. In true leaf roll, derived from a diseased tuber, the lower leaves are rolled, point more or less upwards and are stiff and hard.

Mild mosaic is defined as a mild light and dark green mottle, in which the leaves are neither distorted nor reduced in size. It may be so faint as to be visible only on a cloudy day or when the leaf is held to the light. Often it is clearest in the young haulm, and less apparent in late summer and autumn. Mottling may also result from manganese or magnesium deficiency, but then the whole haulm is usually affected throughout a field or in considerable parts of it.

Aucuba mosaic, so called from a fancied resemblance to the variegated leaf of *Aucuba japonica*, is recognized by clearly defined, bright yellow spots scattered over the lowest leaves. The leaves are not distorted and are of normal size, and the plant seems to suffer little from the disease. Somewhat similar variegation may result from spring frosts.

Severe mosaic or rugose mosaic. This includes all diseases of the mosaic type in which the surface of the leaves is roughened, crinkled, streaked with dark lines, distorted or reduced in size and in which the haulm is more or less stunted, especially in the year after infection. Different forms of severe mosaic which have been distinguished are:

Crinkle. In this the leaves are greatly reduced in size, much crinkled and distorted and have a conspicuous lighter and darker green mottle. Typical crinkle is always due to simultaneous infection of the plant with at least two viruses of the mosaic group, e.g. Viruses 1 and 2 or 3. Thus, Golden Wonder is always infected with *Solanum* Virus 3 which it carries without symptoms. If it also becomes infected with *Solanum* Virus 1, which causes mild mosaic in most varieties, Golden Wonder develops crinkle, the severity of the disease depending in part on the strain of *Solanum* Virus 1 involved.

Leaf drop streak (acropetal necrosis) is the reaction shown by many potato varieties to infection with *Solanum* Virus 2 in the current season. Brown streaks appear on the underside of the veins and petioles, especially of the lower leaves (Fig. 203). They become very brittle and drop at a touch so that the plant



Fig. 203. Streaks caused by *Solanum* Virus 2 on leaves of Sharpe's Express in the season of infection.

may be left with only a tuft of living green leaves at the top, like a palm tree (Fig. 204); all the others hang dead by the stem. Dark-brown dead spots may also spread over the leaf blade between the veins. These must not be confused with those of *Alternaria* blight or of manganese deficiency. Chlorine poisoning may cause symptoms strikingly resembling leaf drop streak but usually occurs in definite patches where, for example, sodium chlorate has been used. Plants grown the following year from tubers of a plant showing leaf drop streak are stunted, with leaves more or less curled, crinkled and mottled according to the variety and may or may not show streaking and leaf dropping (Fig. 205).

Other Virus Diseases of the Haulm

Paracrinkle is a peculiarly severe form of crinkle caused in the stock when a scion of King Edward potato is grafted on certain other varieties.

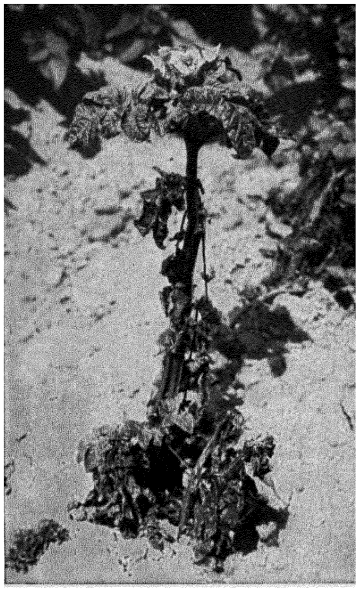


Fig. 204. Severe mosaic of potato, the leaf drop streak phase.

All King Edward plants contain the paracrinkle virus (*Solanum* Virus 7) without suffering any ill effect from it. Paracrinkle is purely a laboratory disease; it has never been seen in the field and no means of transmitting it other than grafting has yet been discovered.

Top necrosis (acronecrosis) begins by the appearance of numerous small brown dead spots on the youngest leaves. These rapidly spread and the shoot dies back from the tip. If any tubers were formed before the plant died, corky spots develop in them during storage and they either die before planting or, at best, give a weak shoot which soon dies. Top necrosis is very seldom seen in a field crop but it is of practical importance in two ways. In the first place different potato varieties react by top necrosis to different viruses. Hence grafting scions from a plant under examination to a selected number of indicator varieties is a standard method of identifying the viruses it contains. This method is particularly valuable in detecting "carried" viruses. Secondly, because the progeny of a plant that reacts by top necrosis are all killed, this is a self-eliminating disease. A virus cannot be perpetuated in a potato variety in which it causes top necrosis. Hence much attention has been paid to breeding new potato varieties which react by top necrosis to as many viruses as possible. It has been possible to produce in this way varieties such as Craig's Defiance which are immune to infection by *Solanum* Viruses 1, 3, 4 and 5 in the field. Unfortunately Viruses 2 and 14 cannot yet be eliminated in this way, as they do not cause top necrosis in any variety.

Virus Disease of the Tubers

Net necrosis is the name given to a fine brown network in the flesh of the tuber. This is a reaction of certain varieties, notably Golden Wonder, in the year they become infected with leaf roll. That is, tubers with net necrosis produce plants with primary leaf roll but these do not, as a rule, yield more tubers showing net necrosis. A somewhat similar brown network may be the result of slight frosting of the tuber.

Tuber blotch refers to small brown spots or blotches in the flesh of the tuber, rather like those known as internal rust spot. The condition may be a result of infection with *Solanum* Viruses 8 or 9 (see p. 340).

Potato varieties all react in much the same way to *Solanum* Virus 14, the cause of leaf roll, though the symptoms are more pronounced in some, e.g. President, than in others like Great Scot. Their reaction to *Solanum* Virus 2 also differs in intensity rather than in kind. To *Solanum* Viruses 1, 3, 4 and 5, however, they react very differently, some by top necrosis, others by a mild mosaic or by showing no symptoms of disease at all. The following table indicates to which of these four viruses the commonly grown British varieties react by top necrosis (N). A much more complete list of reactions will be found in Cockerham, G. (1943).

Variety	*Solanum* Virus 1	3	4	5
Arran Banner	—	—	—	—
Arran Pilot	—	—	N	—
British Queen	—	N	N	N
Catriona	—	—	N	—
Di Vernon	—	—	—	—
Doon Star	—	N	—	N
Duke of York	—	N	—	N
Eclipse	—	N	—	N
Epicure	N	N	—	N
Gladstone	—	N	—	—
Golden Wonder	—	—	N	N
Great Scot	—	N	—	—
Kerr's Pink	—	N	N	—
King Edward	N	N	—	N
Majestic	—	—	—	N
Redskin	—	N	—	—
Sharpe's Express	—	N	N	N
Up-to-Date	—	N	—	N

The viruses of the mosaic group found in Britain are the following:

Solanum Virus 1 (Potato Virus X). This is very widespread, so much so that in America its presence was long regarded as normal and it was called "healthy potato virus"! In most varieties it causes mild mosaic or is carried without symptoms. In varieties such as Golden Wonder and Juli, which always carry *Solanum* Virus 3, it causes crinkle. In King Edward, Epicure and others it causes top necrosis, i.e. these varieties are virtually immune from infection by *Solanum* Virus 1 in the field. No insect vector has ever been discovered, but infection readily takes place by leaf or root contact (Loughnane, J. B., and Murphy, P. A., 1938) and may occur by the knife when seed potatoes are cut.

Solanum Virus 2 (Potato Virus Y) is very prevalent in English potato crops, but is seldom destructive in Scotland outside the south-western counties (Cockerham, G., 1939). A very few varieties, notably Di Vernon, are tolerant of infection by this virus, but most others react to it by developing leaf drop streak and or a form of crinkle. The latter reaction commonly occurs when *Solanum* Virus 1 is present as well. Arran Crest, Arran Victory and Catriona, though stunted when infected with *Solanum* Virus 2, do not develop the characteristic leaf drop streak (Clinch, 1944).



Fig. 205. Leaf of King Edward potato infected with *Solanum* Virus 2.

Yield of tubers from infected plants is greatly reduced but, though small and sometimes misshapen, the tubers show no visible signs of disease. They do, of course, give rise to plants with symptoms of severe mosaic in the following year. *Solanum* Virus 2 can also infect tomato, black nightshade, tobacco, henbane and petunia.

Solanum Virus 3 (Potato Virus A) resembles *Solanum* Virus 2 in being transmitted by aphides, but does not cause leaf drop streak. Some varieties react to it by developing top necrosis (see table) and are hence immune from infection by this virus in the field. In most other varieties it causes only a very mild mosaic *unless* Solanum *Virus 1 is present as well.* When that happens the plant develops crinkle. All stocks of the varieties Golden Wonder and Irish Chieftain are infected throughout with *Solanum* Virus 3 and so are most stocks of Arran Chief and Catriona.

Solanum Virus 4 (Potato Virus B) is almost identical in properties with *Solanum* Virus 1 except that it does not cause top necrosis in Epicure or King Edward and does cause it

in a number of other diagnostic varieties tabulated on p. 339. *Solanum* Viruses 1 and 4 almost always occur together in the variety Up-to-Date without causing visible disease.

Solanum Virus 5 (Potato Virus C) is widespread in Di Vernon, Edgecote Purple, International Kidney and a number of other old varieties seldom grown today. In some varieties it causes a disease resembling severe mosaic, in others it causes top necrosis (see table, p. 339). A few records exist of its transmission in the field but the insect vector, if any, is unknown (Cockerham, G., 1943).

Solanum Virus 7 (Potato Virus E) is carried by all stocks of King Edward but has not been found occurring naturally in any other variety. In Arran Victory and a few other varieties it causes a very severe crinkle (paracrinkle). No insect vector or natural means of transmission is known and the virus appears to be of no economic importance at present.

Solanum Virus 8 (Potato Virus F) can be spread by leaf contact and by aphides if *Solanum* Virus 3 is present as well. In the haulm of most potato varieties it causes only a mild yellow spotting of the basal leaves, but in May Queen, Ninety-fold and Arran Crest it causes distortion of the shoots and foliage for the first few weeks' growth. Tubers of certain varieties, when infected with this virus, develop dead, brown, dry patches in the flesh during storage. The eyes are not affected and sprout normally. This condition, tuber blotch, is shown by tubers of Arran Consul, Arran Signet, Arran Victory, British Queen, Champion, Duke of York, Dunbar Cavalier, Dunbar Standard, Dunbar Yeoman, Gladstone, Great Scot, Majestic, President, Redskin, Ulster Monarch and Up-to-Date (Clinch *et al.*, 1944). Virus 8 is found commonly in the early varieties, May Queen, Forty-fold and Ninety-fold.

Solanum Virus 9 (Potato Virus G) is not known to be spread by insects but is spread by leaf contact, hence very little spread takes place under natural conditions. It causes similar symptoms on the haulm of all varieties, viz. a bright " aucuba " mottle on the lowest leaves. It also causes tuber blotch in British Queen, Champion, Dunbar Yeoman, Great Scot, Majestic and President.

Solanum Virus 14 is the cause of potato leaf roll, the chief cause of " degeneration " of potato stocks in many countries. In severe attacks the harvested produce may weigh less than the seed tubers planted. The virus is transmitted by several species of aphides. Newly infected plants show a stiff upward rolling of the topmost leaves. They may be yellowish or sometimes be tinged with purple on the under surface. This " primary leaf roll " is easily confused with rolling of the top leaves due to other causes (see p. 337), and when infection occurs late in the season the symptoms may be scarcely visible. Plants grown from infected tubers develop "secondary leaf roll " which is much easier to recognize. The invariable symptom in all varieties is a hardening and rolling upwards of the basal leaves. This is due to their becoming filled with starch which cannot be transported to the developing tubers. The rolled leaves may rattle when shaken, often they are pale and may have purple pigments or rusty-brown patches on them. No potato variety has been found to be immune from infection by this virus, but two old Irish varieties, Shamrock and Skerry Champion, show a remarkable capacity for escaping infection under field conditions. The reason for this is unknown as both varieties are readily infected with the virus by aphides, or by grafting under experimental conditions. The old continental variety Matador shows comparatively mild symptoms of the disease but even then infection with leaf roll may reduce the yield of tubers by 58% (McKay, R., and Clinch, P., 1944). Clinch *et al.* regard Arran Banner, Di Vernon, Great Scot, Ulster Monarch and Up-to-Date as the commercial varieties least affected by leaf roll infection. The last-named is, however, very prone to contract infection in the field. Varieties which suffer very severely when infected with leaf roll are: Arran Consul, Arran Crest, Arran Pilot, Dunbar Cavalier, Dunbar Standard, Golden Wonder, King Edward and Redskin. In Danish experience

Magnum Bonum suffers greatly; leaf-roll infection may reduce its yield to 20% of the normal, even under good growing conditions. Experiments at Lyngby in Denmark showed the varieties Birgitta, Deodora, Juli, King Edward, Preussen and Early Rose to be very susceptible to infection with leaf roll. Di Vernon and Majestic were less so while Ackersegen, Alpha, Bravo, Edeltrant, Erdgold, King George and Sigyn were somewhat resistant.

Cockerham (1943) considers that resistance to infection by the leaf-roll virus is an inherited character and that it may in time be possible to incorporate this property in new potato varieties of high commercial quality.

Solanum Virus 14 can also infect tomato, *Solanum nigrum*, *S. dulcamara*, *Capsicum* and *Datura* (thorn apple) (Dykstra, T. P., 1933), but it is doubtful if this takes place to any extent under field conditions.

Control of Potato-virus Diseases

Control of potato-virus diseases takes different forms in different districts. In the greater part of England conditions favour the spread of aphis-borne viruses, and avoidance of loss from these diseases depends mainly on purchase of certified healthy " seed " every few years from a suitable " seed "-producing region such as Scotland or Ireland. Small areas in the Lake District, Wales and South-west England are also suitable for production of healthy potato " seed." In the main they are exposed districts with a cool, wet summer climate. Districts near large towns, where there are extensive market gardens, are not suitable for potato " seed " production. This is because the aphides which spread leaf-roll virus and *Solanum* Viruses 2 and 3 overwinter largely on cruciferous green crops. Even in England, however, it is possible to select reasonably healthy crops in the first year after new " seed " has been obtained from Scotland or Ireland, and the Ministry of Agriculture maintains a scheme for the certification of the best of such " once-grown seed." In addition to the primary infection brought with the " seed " secondary infection may come from diseased ground-keepers that have survived throughout the rotation from the previous potato crop.

In Scotland and Ireland the responsible Departments of Agriculture operate rigorous inspection schemes for the certification of healthy potato crops suitable for use as " seed." In the Scottish scheme three grades of health are recognized. In **stock seed**, not more than 4 plants per acre infected with leaf roll or severe mosaic or showing the " wilding " condition are permitted, and only up to 0.25% of the crop may show symptoms of wild mosaic. Other rigorous conditions are laid down to ensure the high quality of these stocks, which are intended for multiplication in a " seed "-growing area rather than for sale directly to ware-producing regions.

Grade A stocks may contain not more than 0.5% of plants infected with leaf roll, severe mosaic and the " wilding " condition and not more than 1% of all virus diseases and "wildings."

Grade H stocks may not have more than 2% of leaf roll, severe mosaic and " wildings " and not more than 10% of mild mosaic. All grades must be 99.5% true to name, a condition originally introduced to guard against contamination of stocks of wart-disease-immune varieties by wart-susceptible rogues.

Health of " seed " stocks in " seed "-producing districts is maintained by early and drastic roguing. All plants showing severe virus disease are removed and their tubers dug out of the drill. In avoiding the onset of severe virus diseases there are obvious advantages in not planting side by side varieties which are universally infected with *Solanum* Virus 1 and *Solanum* Virus 3 which, in conjunction, cause crinkle. There is also an advantage in growing varieties like Craig's Defiance, which is field-immune to infection with *Solanum* Viruses 1, 3, 4 and 5. In such crops one need rogue only for infection with *Solanum* Viruses 2 and 14.

A scheme has also been set on foot by which tested virus-free tubers of commercial potato

varieties will be multiplied in isolation in the hope of building up completely virus-free stocks healthier even than the stock-seed grade. This scheme may prove of particular value in eliminating leaf-roll infection from high-grade stocks.

It is not practicable to control the spread of aphides into and within a growing potato crop. As, however, *Solanum* Viruses 2, 3 and 14 may be spread by aphides feeding on the sprouts in the chitting house it is important to inspect the sprouting " seed " periodically and to fumigate the house with nicotine as soon as any aphides are found.

Inherited, Non-contagious Variations

A potato variety is a clone, i.e. all plants belonging to it are the product of vegetative reproduction by a single seedling. Because of this absence of sexual reproduction the inherited constitution of all plants of a given variety should be exactly the same and they should all be exactly alike, apart from superficial differences brought about for one season only by such factors as manuring or temperature. Nevertheless, permanent changes in appearance and productivity do arise from time to time in certain plants within the variety, in addition to the changes brought about by virus infection. It is generally assumed that these non-contagious variations arise by mutation, i.e. as " bud sports," but their origin is not adequately understood. Once they have arisen they are perpetuated by all the tubers of the plants concerned. Three types of inherited variation are objectionable, viz:

(1) **Bolters.** These differ from the true varietal type in their greater height, stiffer and less luxuriant foliage, more profuse flowering, later maturity, longer stolons and coarser tubers. The most conspicuous character is the late maturity, and hence they are particularly noticeable and objectionable in early varieties and are seldom, if ever, seen in late maincrop varieties. Among earlies Ninetyfold is noteworthy for freedom from bolters. Intermediate types between the true bolter and the normal plant also occur, especially in early varieties. Selection from these may lead to the development of a tall-growing, later-maturing stock of some valuable early variety.

(2) **Wildings.** The true wilding is fairly easily recognized by its numerous slender stems and comparatively simple leaves in which the primary leaflets are fewer and rounder than in normal plants and the number of secondary leaflets is greatly reduced. Wildings are often a little shorter than the rest of the crop and seldom, if ever, flower, even in varieties which normally flower freely. Their objectionable character is the production of numerous stolons bearing only very small tubers. Stolons may arise even from the axils of the lowest leaves. Wilding tubers tend to produce spindling sprouts. Wildings occur in all varieties.

(3) **Feathery wildings.** These are like true wildings in being stemmy and producing small tubers but in other respects they represent a variation from normal in quite a different direction. Their leaves have narrow leaflets, especially at the top of the plant (" feathery tops ") and they flower more freely. Feathery wildings may occur in any variety but are much more difficult to detect than true wildings.

Control. Wildings are objectionable to the grower of ware potatoes because they reduce the yield of marketable tubers, while bolters, in early varieties, may mature inconveniently late. There is, however, nothing he can do to prevent them occurring; control rests with the " seed " grower. The only method of control lies in rigorous roguing of the " seed " crop throughout the growing season. Study of inherited variations has been undertaken mainly in Scotland by officers of the Department of Agriculture, and the best readily accessible account of them, by Dr. T. P. McIntosh, will be found in that Department's journal of 1945.

Leaf Scorch (Potassium Deficiency)

Potatoes that lack potassium produce a low-growing, rather dark, open haulm. About midsummer the leaves become curled, tip and margin curve downwards, and the blade

becomes arched between the veins, the margins and arched portions turn yellow, dry, become bronzed and finally die. This scorching appears first on the lower leaves and gradually spreads up the plant as the older leaves are drained of potassium for the benefit of the younger.

Tubers from potassium-deficient plants tend to turn black when cooked but similar blackening may occur in phosphorus- or calcium-deficient tubers also. Care should be taken to supply enough potassium when there has been a heavy application of a nitrogenous manure. Bordeaux sprays have been thought to encourage blackening but the effect is probably indirect; the spraying leads to a heavier yield, and hence to a larger potassium requirement by the crop. The soil may contain enough available potassium for an unsprayed crop but not enough for the larger yield from sprayed plants. Potatoes do well with stable manure.

The tendency to blackening is intensified by rough handling, as during transport. It may be counteracted by peeling the tubers and leaving them several hours in water before they are cooked and by addition of 1–2 desert-spoonfuls of vinegar to the saucepan of water.

According to de Bruyn, potato tubers from plants grown on a potassium-deficient clay soil may have small rust-coloured spots in the flesh about $\frac{1}{16}$ inch below the skin.

A somewhat similar form of leaf scorch may be caused by deficiency of magnesium, especially on very sandy or very acid soils low in organic matter (Chucka, J. A., and Brown, B. E., 1938).

Other Deficiency Diseases

Manganese deficiency causes a peculiar bronzing of the top leaves in which the veins are outlined on each side by a row of small brown dots. The condition is usually associated with over-liming and is of little economic importance (see p. 39). Magnesium-deficiency and calcium-deficiency diseases have been described by J. G. Hunter and A. J. M'Gregor (1945). In the former, growth of the haulm is retarded, the leaves curl up slightly at the edges and the top and margins then turn yellow. Later this colour deepens to orange and spreads inwards along the veins, while the interveinal areas remain green, or acquire a purplish-brown hue. The yield of an affected crop is greatly reduced.

In acute calcium deficiency, such as may occur on very acid soils, especially on "marginal" land, growth is even more restricted; plants may be only six inches tall by August. The lower leaves become blotched with yellow and brown, starting at the tip and margins and spreading inwards between the veins. These leaves die and fall early, leaving the pale-green top leaves. The yield is very poor and the tubers small. In an experiment in Perthshire the trouble was controlled by applying 5 cwt. shell lime per acre in the drill. Better results would probably have been obtained by incorporating ground limestone with the soil before the drills were made.

Dead or Defective Shoots

During sprouting some sprouts may be found to have blackened dried-up tips. This defect may be prevented by so arranging the sprouting boxes that all the tubers receive a little light; the boxes should be moved round in the middle of the sprouting period so that all the tubers get the same amount of light (Bates, G. H., and Dillon Weston, W. A. R., 1936). Lack of ventilation may also cause death of sprouts or death of the central bud in the eye. The result is formation of small tubers on the eyes, or merely a delay in sprouting while the lateral buds mature. Occasionally the sprouts proliferate to form a dense cluster like a small cauliflower, not to be confused with wart disease. Such tubers give normal plants and yields. The condition has been attributed to overheating during storage. Sometimes planted tubers produce no shoots but bear instead a number of small new tubers in clusters or chains. Such premature tuber formation is most common in early varieties. According to Dutch workers the trouble arises when tubers which have

been stored in a warm place, and have therefore had to be repeatedly desprouted before planting, are planted in cool soil, with a temperature below 50° F. Hence it tends to be prevalent when a mild winter is followed by cold weather in April and May. The trouble can be prevented by sprouting the tubers in the light (Moore, W. C., 1943).

Another defect which may arise during sprouting is the production of long thin thread-like sprouts (see spindling sprout, p. 336).

Sometimes potato tubers do not sprout at all and the " seed " is then said to be " blind ". Often the trouble is death of the eyes by skin spot (see p. 331) or decay of the tuber by dry rot (p. 330). In other cases it is attributable to removal of sprouts before planting. This is apt to occur especially with Arran Pilot, which tends to sprout early and from all the eyes at once. If these first sprouts have to be removed or are knocked off the tubers may not sprout again. Premature sprouting is encouraged by storage of the seed in clamps, and Moore (1948) suggests that Arran Pilot " seed " should be boxed on arrival at the farm. Tubers sometimes form no sprouts at all, though not obviously infected with skin spot, but remain hard and glassy. Presumably some unsatisfactory storage condition is responsible but the cause is not yet understood.

Internal Rust Spot and Spraing

These are tuber diseases of unknown origin characterized by the production of rusty-brown marks in the flesh. In internal rust spot the marks are small spots or blotches scattered throughout the flesh. In spraing they take the form of narrow brown areas or bands of tissue, the edges of which may form dimly visible elliptical lines on the skin of the uncut tuber (Fig. 206). Both disorders appear to be associated with particular soils, especially gravelly or sandy soils, but until their cause is understood no effective control measures can be suggested. Green manuring is said to reduce the amount of internal rust spot.

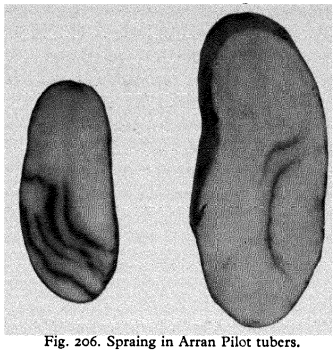


Fig. 206. Spraing in Arran Pilot tubers.

Pit Rot

More or less circular sunken brown spots, usually with a lenticel in the centre, often occur in the skin of stored potatoes. The spots seldom exceed ½ inch across unless several have joined together. The damaged tissue usually extends only ⅛ inch or so into the flesh and is delimited by a layer of wound cork. Various fungi may be found in the dead spots, notably *Cylindrocarpon radicicolum*, species of *Fusarium* and *Phoma* and sometimes *Corticium solani*, but none of these seems to be the primary cause of pit rot. The spots may, however, be confused with those caused by *Alternaria* blight (p. 317) or gangrene (p. 331).

Some such spots may appear on disinfected tubers when too strong a solution (corrosive sublimate and hydrochloric acid or formalin) has been used. Others may be the result of a lack of oxygen, in which case there may be dead tissue in the heart of the tuber as well. McKay (1940) has shown that spots round lenticels of potato tubers may be caused by rat urine.

Black Heart

The central portion of the flesh in stored potato tubers is sometimes found to be more or less blackened and this is usually traceable to heating in the clamp. Experiments in keeping potatoes cool but deprived of oxygen

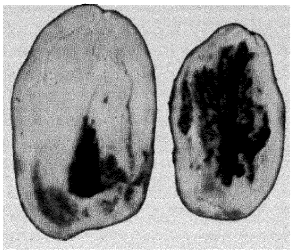


Fig. 207. Black heart of potato tubers (*S. p. F.*).

have shown that the real cause of the trouble is a lack of oxygen, i.e. suffocation. The discolouration passes through shades of grey, brown or red and finally becomes black; in fresh sections the colour often changes quickly (Fig. 207). Similar discolouration may, however, occur in connection with exposure of the tubers to hot sunshine, to frost, or to attack by species of *Pythium* or *Fusarium*. Hence unless the precise history of a consignment is known it may be difficult to be sure of the exact cause of black heart.

If bacteria are present the tubers may rot, or the suffocated black heart may acquire a leathery texture.

Heat damage without oxygen deficiency may occur in very hot or dry soil, as in light land on a slope facing south, or when freshly dug tubers are exposed to strong sunshine. Black heart of this kind, initiated before the tubers are dug, was prevalent in the South of England after the hot, dry August of 1933 and again in 1947 (Moore, W. C., 1949). The first symptom is a golden or brown colouring round the vascular bundles; later there may be large sunken blisters on the upper surface or a blackening of the heart. Deficiency of oxygen without heating may occur as a result of flooding. The usual cause of black heart, however, is lack of oxygen induced by heating. High temperature accelerates respiration which involves rapid oxygen consumption, and if ventilation is inadequate the damage occurs.

Black heart can be produced by keeping tubers in an enclosed space for 12 days at 70° F.; 18–20 hours' exposure to a temperature of 95–110° F. may lead to black heart in the following 3–8 days (Stewart, F. C., and Mix, A. J., 1917; Bennet, J. P., and Bartholomew, E. T., 1924).

If the temperature of the heap does not exceed 40° F. potatoes may be stacked to a depth of 2 yards in a well-ventilated cellar, but if the temperature approaches 50° F. the lower layers are exposed to considerable danger.

It is therefore important to allow for ventilation in the clamp and still more when tubers are stored in buildings or cellars. Respiration is especially active just after lifting and if tubers are warmed after being kept some time at a low temperature. " Sweet " potatoes (see p. 346) are comparatively easily suffocated.

Potatoes suffering from heating sprout irregularly and are unfit for planting.

Cold and Frost

Night frosts may cause yellowing of the first leaves or may even destroy the first shoots. The new growth grows away from the damage so quickly, however, that the effect is seldom noticeable later on, though

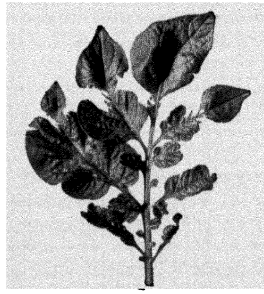


Fig. 208. Frost damage to potato leaves.

occasionally the haulm acquires a peculiar crumpled appearance with torn yellow leaves (Fig. 208). If potatoes have to be grown on boggy ground or in frost pockets the damage may be avoided by growing early varieties and

planting them late. One hard late frost may undo all the advantage gained by sprouting the tubers before planting.

The effect of cold and frost on the lifted tubers has been extensively studied. Storage at 40–45° F. reduces losses from respiration and heating to a minimum. At temperatures below about 38° F. the starch in the tuber becomes converted into sugar, often to such an extent that they taste unpleasantly sweet. But when stored for a few days at 50–54° F. this sweet taste is lost.

The nature and extent of damage by frost depends, among other things, on inherited characters of the variety, on conditions of growth, on the duration of the frost and on whether freezing and thawing occur suddenly or gradually. Tubers subjected for a short time to too low a temperature may suffer only discolouration of the vascular bundles. A more or less regular network appears in the flesh where the cortex meets the pith, and in certain cases within the pith also. The colour of the network is first an opaque white, then reddish, brown or black. The discolouration may spread into rather larger spots. The eyes seem the most resistant parts. With severe frost miscroscopic cracks occur in the tissues. If it occurs before the tubers are lifted, their outermost layers become frozen and later turn soft and rot in the clamp. Individual frosted potatoes or frozen lumps of soil may damage others in the clamp. If, therefore, there has been a sharp night frost, lifting should be postponed until the ground has thawed completely.

Longer freezing kills the tuber, its flesh immediately becomes chalky, then flabby and wet, and the colour after thawing turns reddish, brown or black. Bacteria and species of *Fusarium* quickly bring about rotting of the dead tubers.

In tubers that are cooled slowly the conversion of the starch into sugar dissolved in the cell sap will lower its freezing point which otherwise lies at about 30° F. Only a little below the temperature at which ice forms in the cells comes the point at which the tuber is killed and frost damage becomes apparent. Tubers that have suffered severe cooling without being frozen to death suffer more readily at the next frost. It has been estimated that the damage is done by exposure to 26.5° F. for several hours, to 23° F. for 2 hours or to 14° F. for 1 hour but in practice it is hard to be so precise. A potato that lies undisturbed may survive cooling to 23 or 22° F., but if it receives a blow or a wound, ice forms at once and death by freezing occurs. The practical observation that frosted potatoes may sometimes be saved by leaving them alone to thaw slowly depends on the occurrence of this supercooling without actual ice formation. Potatoes actually frozen stiff are killed in any case.

Prevention. Potato clamps should be provided with ventilation channels at the base and along the ridge. At first they should be lightly covered with straw and a little soil. Tubes containing thermometers may be inserted in the sides and care taken that the temperature does not rise above 45° F. When the potatoes have dried and the weather becomes chilly a thicker covering of soil should be applied. If severe and prolonged frost occurs a further temporary covering of straw or stable manure may be packed round the clamp.

Consignments of tubers that have been frosted should be picked over as soon as possible after thawing and preferably again a few days later. If all soft, wet tubers are cooked or added to silage they can be used for fodder, otherwise they rot and may set up soft rot in neighbouring tubers as well.

Attempts can be made to salvage particularly valuable lots of frosted seed tubers in the following ways: (1) all soft or leaking tubers should be picked out as soon as thawing is completed; (2) some time later all the remainder should be cut, large tubers halved, small ones sliced at the heel, and all rubbery or discoloured ones rejected. The survivors seem to have a specially strong growth in soil. After long severe winters tubers may be slow in sprouting and hence be usually exposed to severe attacks of stem canker and other diseases.

Protection against cold and frost has been discussed in more detail on p. 24.

Summary of Potato Diseases

I. Leaves *page*

A. Leaves with brown or blackish markings:

(1) Spots covered with mould in damp air:

(a) Large spots mostly starting at tip or edge of leaflet:

i. Dark-brown spots, white mould round their margins on lower surface ..... Blight 312

ii. Hairy grey mould ..... Grey Mould 59

(b) Spots scattered over the leaf surface:

i. Round brown spots, marked with concentric lines, bearing olive-brown mould *Alternaria* Leaf Spot .. 317

ii. Angular pale-green spots turning brown with purplish-grey mould ..... *Cercospora* Leaf Spot .. 318

(2) No mould on the brown parts:

(a) Leaves blackened, translucent, then shrivelled ..... Frost 345

(b) Growing point dead, youngest leaves black spotted ..... Top Necrosis 338

(c) Brown dead edges and spots on leaflets, cause not determinable without additional information about local conditions ..... Wind, Cold, or Hail Damage ..... 20 / Potassium Deficiency .. 342

(d) Brown to black streaks on under-side of leaf veins and petioles, sometimes angular spots round fine veins ..... Leaf Drop Streak 337

B. Leaves variegated or parti-coloured:

(1) Leaves with light-yellow bands, sometimes torn Frost 345

(2) Leaves mottled with light and dark green:

(a) Leaf surface flat ..... Mild Mosaic 337

(b) Leaf surface ruffled or undulating ..... Severe Mosaic 337

(3) Leaves whitish-yellow spreading from the edge between the veins, margin sometimes curled up or down; on soils very poor in lime ..... Magnesium Deficiency 343

(4) Bright yellow leaf spots, no mould, no distortion of surface ..... *Aucuba* Mosaic 337

(5) Yellowish tips to leaflets, leaves all shrivel .... *Verticillium* Wilt 328

(6) Light-green leaves, lines of fine bronze dots outline the larger veins ..... Manganese Deficiency 343

(7) White powdery coating ..... Powdery Mildew 318

C. Leaflets curled, distorted or deformed:

(1) Leaves rolled up, especially the lowest, stiff and hard, seed tuber often preserved until autumn . Leaf Roll 336

(2) All the leaves rolled, or only the young ones, soft and flaccid:

(a) Leaves yellowish, base of stem black and slimy ..... Black-leg 333

		page
(b) Brown lesions on stem below soil level	Stem Canker	326
(c) Brown vascular bundles in stem	*Verticillium* Wilt	328
(d) Stem normal	Drought	17
(3) Leaflets curved downwards, sometimes bronzed, edges dry and brown, sometimes spotted	Lime Deficiency	343
(4) Leaves crinkled and distorted with wavy edges:		
(a) Brightly mottled light and dark green	Crinkle	337
(b) Less mottled, very distorted stunted plants	Chronic Severe Mosaic	337
(5) Leaf with mealy eruptions which break down into holes, sometimes wavy-edged—on potatoes under glass	Œdema	392
II. Stems		
A. Underground portions:		
(1) Blackish-brown, wet, slimy, finally killed	Black-leg	333
(2) Dry brown wounds (brown fungal threads seen with lens)	Stem Canker	326
(3) Dark-grey, soft cortex (black dots seen under lens)	*Colletotrichum*	330
B. At the collar, about soil level:		
(1) Grey-brown mould or a powdery white coating	Stem Canker	326
(2) Cauliflower-like tumours, whitish-brown in the dark, green in the light	Wart Disease	318
C. Above-ground portions:		
(1) Blackish-brown lines and dots	Leaf Drop Streak	337
(2) Stems flattened and distorted over a patch in the crop, pith dead	Lightning	25
(3) Stem snapped but living and resuming growth	Hail	20
(4) Translucent, soft, cracked areas on the stem:		
(a) Hairy grey mould with flat black sclerotia	Grey Mould	59
(b) Dense white mycelium with round sclerotia, first white then black	*Sclerotinia*	77
(5) Green aerial tubers in the axils of leaves.....	Stem Canker	326
	Mechanical Injury to Base of Stem	335
III. Sprouts		
A. During chitting before planting:		
(1) Thread-like shoots	Spindling Sprout	336
(2) Dead brown tips	Lack of Light; Heating	343
B. After planting:		
(1) Sprouts "nipped off" or with brown sunken lesions, often replaced by numerous side shoots..	Stem Canker	326
(2) Sprouts feeble or wanting:		
(a) Tuber decayed early:		
i. Soft wet rot	Black-leg	333
ii. Shrivelled tuber	Dry Rot	330
(b) Tuber hard.....	Leaf Roll	336
	Skin Spot	331

IV. Tubers *page*

A. Disorders of the flesh:

(1) Colour of flesh normal:

(a) Hollow heart or internal cracks	Too Rapid Growth	19
(b) Flesh rubbery	Drought	17
(c) Flesh with a soft cheese-like consistency	Frost	345
(d) Flesh glassy, easily leaking	Second Growth	19

(2) Flesh an abnormal colour but not rotten:

(a) Flesh blackish grey after cooking	Lack of Potassium, etc.	342
(b) Brown vascular bundles or brown spots in the bundles. Final diagnoses require supplementary information re growing conditions and storage	Frost	345
	Heating	344
	Net Necrosis Phase of Leaf Roll	338
	Verticillium Wilt	328
	Fusarium Wilt	329

(3) Rust-coloured spots or lines, not along the vascular strands:

(a) More or less circular scattered spots	Internal Rust Spot	344
(b) Rusty lines more or less in concentric arcs	Spraing	344

(4) Grey-brown or black patches:

(a) In the pith of stored tubers	Heating	344
(b) Vague grey-brown areas under the skin	Frost	345
	Heating	344
	Potassium Deficiency	342

(5) Flesh rotten:

(a) Soft wet rot, in the last stages dry and chalky:

i. Beginning at the heel, tuber may become hollowed out, no particular bad smell	Black-leg	333
ii. Rot spreading into the flesh at various parts, usually with an evil smell	Wet Rot	335
	Watery Wound Rot	333

(b) Fairly firm rot, leaking when squeezed, flesh turns pink when cut	Pink Rot	332
(c) Soft flabby tissue at the heel end, sometimes falling off to leave a scar	Jelly-end Rot	333

(d) Fairly dry firm rot:

i. Leaden or brown patches on skin, slightly sunken, underlying flesh penetrated by rusty-brown lines and spots	Blight	312
ii. Skin shrivelled, with concentric wrinkles and bearing round pustules of mould, usually bluish or white, occasionally pink	Dry Rot	320
iii. Irregular sunken spots with hard dead brown tissue beneath (not yet known in Britain)	*Alternaria* Blight	317

		page
iv. Small round sunken spots with hard dead tissue beneath	Pit Rot	344
v. Cauliflower-like swellings that soon decay	Wart Disease	318
B. Flesh normal, skin diseased or abnormal:		
(1) Conspicuous swellings:		
(a) Cauliflower-like tumours, first hard and white, then turning brown and decaying..	Wart Disease	318
(b) Shining white, soft pustules, formed over the lenticels under damp conditions	Lenticel Proliferation..	19
(2) Skin unbroken but marked with crusts, discoloured patches or blisters:		
(a) Brown or black crusts, easily scraped off	Black Scurf	326
(b) Purple-brown crusts or network of fibres, not easily removed	Violet Root Rot	262
(c) Lead-grey pimples with sunken margins	Skin Spot	331
(d) Discoloured patches with a silvery sheen	Silver Scurf	332
(3) Skin damaged:		
(a) Network of fine cracks, intervening skin corky and flaky	Excess Moisture	19
(b) Big straight cracks, flesh normal	Cracked Tubers (Excess Moisture)	19
(c) Rather faint curved furrows connected with brown arcs in the flesh	Spraing	344
(d) Scabby wounds:		
i. Flat scabs, slightly raised or slightly sunken, with an earthy smell	Common Scab	322
ii. Small saucer-shaped hollows full of brown powder and surrounded by torn skin	Powdery Scab	324
iii. Large irregular sores and cankers, mainly on second growth	Canker Phase of Powdery Scab	325
C. Both flesh and skin of tuber normal but shape abnormal, yield poor:		
(1) New tubers few, small or none:		
(a) " Seed tuber " still hard at lifting time	Leaf Roll	336
(b) " Seed " tuber decayed	Drought	17
	Various Diseases of the Haulm	343
(2) New tubers of abnormal form:		
(a) Tuber swollen at the rose end or branched	Second Growth	19
(b) New little tubers on the sprouts or within the flesh	Little Potato	343

page

V. Whole Haulm Deformed

Examine the root system. Also note distribution in the field, whether scattered, in patches or concentrated in one corner.

A. Haulm luxuriant and tall:

(1) Dark-green leaves	Excess Nitrogen	40
(2) Olive-green leaves	Excess Phosphoric Acid	41
(3) Scattered tall late-maturing plants	Bolters	342

B. Haulm short or stunted:

(1) Pale green all over, leaves tend to point upwards	Nitrogen Deficiency	40
(2) Pale-green leaves, the lower ones rolled and stiff	Leaf Roll	336
(3) Dark-green, slender, upright growth	Phosphoric-acid Deficiency	41
(4) Dark-green open growth, leaves later bronze-coloured with dead brown margins	Leaf Scorch (Potassium Deficiency)	342
(5) Crinkled or mottled leaves, ripening early	Crinkle and Severe Mosaic	337

C. Haulm yellowish green, often with brown or black dots along each side of the veins ..... Manganese Deficiency 343

D. Haulm limp, yellowish, dying:

(1) Base of stems black and slimy	Black-leg	333
(2) Dense white mycelium on the stem with black sclerotia	*Sclerotinia*	77
(3) Hairy grey mould on the stem, and black sclerotia	Grey Mould	59
(4) Stems grey and soft below soil level	*Colletotrichum*	330
(5) Top leaves curled, wilting in hot weather then recovering, brown lesions at soil level or below	Stem Canker	326
(6) Haulm wilts permanently, brown vascular bundles	*Verticillium* Wilt	328

PUMPKIN

The pumpkin is not very susceptible to disease but under favourable circumstances it will probably be found to suffer from most troubles which affect its close relatives, cucumber and melon.

Mildew (*Erysiphe cichoracearum* DC.)

Is perhaps the disease most often seen on pumpkins in Denmark (p. 275). Elsewhere they have been found to suffer from leaf spots (p. 275) gummosis (p. 273), mosaic (p. 278) and blotch (p. 274).

RADISH

Summary of Radish Diseases (for details see under cabbage)

		page
I. Seedlings		
Rotting at soil level	Damping Off	68
II. Roots of Larger Plants		
A. Large irregular swellings	Club Root	246
B. Uneven, rough, scabby places	Scab	322
C. Decay, associated with white mycelium and big black sclerotia	*Sclerotinia* Rot	77
III. Seed Crops		
Leaves and seed stalks deformed, with pure white pustules of spores	White Blister	285

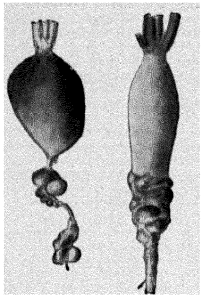


Fig. 209. Club Root on radish (after R. H. Larsen, 1934).

RED PEPPER (Capsicum)

Leaf Spot (*Cercospora capsici* Heald & Wolf)

This disease is general wherever capsicums are grown in America but has yet to be found in Denmark or Britain.

The spots are at first watersoaked but soon dry out and are then white with a dark-brown margin. Their size varies between $\frac{1}{8}$ inch and 1 inch. When many spots occur on one leaf it turns yellow and drops. Spots may sometimes occur also on the leaf-stalks and fruit stalks. The fungus cannot penetrate the skin of the fruit but may grow into it through the stalk and then sets up a " stalk rot."

Control is the same as for *Colletotrichum nigrum.*

Colletotrichum nigrum Ell. & Halst.

This disease has been known in America for many years, and Chupp (1925) believes it to occur wherever red pepper is grown. It is likely to appear sooner or later even in Denmark and Britain, where the plant is only grown on a small scale as a glasshouse crop.

Spots appear on the fruit, starting as small translucent or dark-red depressions, which quickly grow larger and gradually become lighter in colour. The pustules of the fungus form a dark coating on the diseased tissue and exude pinkish masses of conidia, which are spread by rain splashes or on clothes or tools of workers.

Control measures include destruction of diseased fruit, crop rotation, spraying with Bordeaux mixture, which is not always effective, and seed disinfection. The last-named practice is difficult because the seed is easily damaged. The following treatment has given good results in U.S.A.: the seed is set to soak in water for 6–15 hours, is then taken out and allowed to drain before being dipped for 5 minutes in a copper-sulphate solution, $\frac{1}{4}$ oz. in 1 pint of water. It is then dusted with air-slaked lime and sown immediately.

Sap Warts (Oedema)

When pepper is kept wet, especially if the atmosphere is nearly saturated or when great changes in humidity occur, it easily develops oedema. This takes the form of a large number of very small grey-brown warts on the underside of the leaf. One should try to avoid excessive humidity or much change in the moisture supply to the plant (see tomato, p. 392).

Grey Mould (*Botrytis cinerea* Fr.)

In Denmark it has often been noticed that red-pepper fruits have developed large decaying spots covered with grey mould.

We are not sure whether *Botrytis* is the primary cause of the damage or not. Quite likely the fruit suffered first from blossom-end rot and the grey mould followed and increased the damage. Leaves and stalks may also be attacked.

The plants must not be kept too dry or the fruit will develop blossom-end rot, or too damp or *Botrytis* will be encouraged. Indeed the art of growing this, like other greenhouse crops, consists in supplying the plants with enough water in a damp enough atmosphere without letting them become moister than is necessary (see grey mould, p. 59).

Blossom-end Rot

Capsicum fruits may develop blossom-end rot like tomatoes. The first sign of the disease is a more or less sunken area at the tip, or close to it, on the half-grown fruit. The spot is usually a little paler than the surrounding healthy tissue but it may be dark-green and translucent. Gradually the spot grows larger but it does not cover the whole of the blossom half of the fruit as may happen in tomato. The spots may, however, become infected by fungi or bacteria, which set up a rot that may involve the whole fruit.

The spots are not primarily due to a parasite, however, but to a physiological disturbance. Higgins has studied the problem closely in Georgia, both with controlled watering and under natural conditions out of doors.

In all cases it was shown that drought caused the appearance of the spots. In the open air they become infected by bacteria or fungi in the course of a few days. Of these only one, a species of *Alternaria*, was able under very favourable conditions to infect healthy fruit on which it caused spots only $\frac{1}{25}-\frac{1}{12}$ inch across.

In fairly young spots neither fungi nor bacteria were found. Only as the spots increased in size did these organisms invade them and thrive on the already damaged tissue of the fruit. The spots start by collapse of the large, thin-walled cells round the ends of the vascular bundles.

Control. Red-pepper plants should not be exposed to drought, especially when the fruit is half-grown and has been growing vigorously.

Virus Diseases

The virus of cucumber mosaic (*Cucumis* Virus 1) may infect capsicum and cause curling and mottling of the leaves. The older leaves may become abnormally narrow and the whole plant very stunted. There may also be dark-green swellings on the fruit.

Several other plant viruses have been found to attack capsicum in North America including those of tomato ring mosaic (*Lycopersicum* Virus 2), and sugar-beet curly top (*Beta* Virus 1).

For control see p. 278.

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

This causes rotting of fruit and stems with the usual cottony white mycelium and large white, then black, sclerotia (see p. 77).

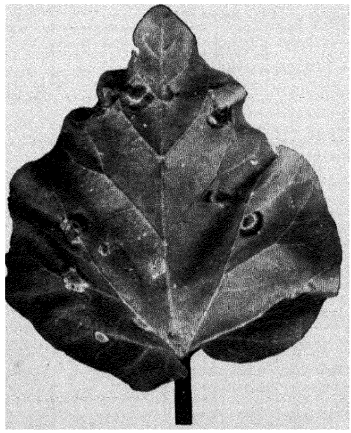


Fig. 210. Rhubarb rust on upper side of leaf.

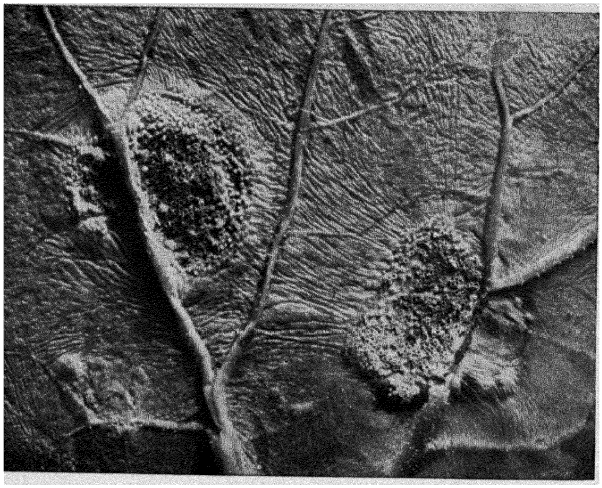


Fig. 211. Rhubarb rust, clusters of aecidia on under-side of leaf, as seen under a lens.

RHUBARB

Rust (*Puccinia phragmitis* (Schum.) Koern.)

The cluster-cup (*aecidium*) stage of this rust occurs on rhubarb leaves. It causes carmine-red spots (Fig. 210) on the under-side of which are the tiny whitish cups (Fig. 211) with delicately fringed edges. The same stage occurs on leaves of sorrel and dock. It is common in the vicinity of swamps and ditches but not elsewhere.

The uredospore and teleutospore stages of this rust occur in summer and winter on the common reed (*Phragmites communis*) where they cause yellowish-brown and black oblong pustules respectively.

Downy Mildew (*Peronospora jaapiana* Magn.)

This disease was first found in Denmark at Copenhagen in August 1918 (Ferdinandsen). It appeared on the varieties Linnaeus, Victoria and Vine. Since then it has been found to be widespread but does little damage and is therefore seldom noticed by rhubarb growers.

Discoloured spots appear on the leaves and eventually turn pale with a red margin. Their size varies greatly. The usual layer of downy mildew conidiophores appears on the lower surface. On young spots they may form a continuous mauve coating but on old spots they occur only as scattered white specks.

If the disease seems to be spreading the leaves should be sprayed or dusted with a copper fungicide. As a general rule there seems no necessity for any control measures to be undertaken.

Leaf Spot (*Ramularia rhei* Allesch.)

This fungus causes circular brownish sharply defined spots on the leaves, with a fine whitish mould on their under surface. If it appears to be on the increase a fungicidal spray or dust may be applied. The disease is only occasionally reported in Britain.

Grey Mould (*Botrytis cinerea* Pers.)

Grey mould may be associated with large grey-brown spots on rhubarb leaves and with decay of the crown.

Its presence is usually indicative of poor growth of the plant or of excessively wet conditions (see p. 59).

Gummosis

Sometimes small oblong spots appear on rhubarb stalks, in which form clear masses of gum. Affected stalks tend to crack.

The condition is suggestive of hail damage but this is not usually the cause as the leaves are usually unaffected.

Apparently it is due to some physiological disturbance but of what kind is still unknown.

Crown Rot

It fairly often happens that rhubarb rots at the crown, either starting in the crown and working a little way up the leaf stalks or, more often in Denmark, starting in the crown and spreading down into the roots. The leaves of such plants turn red or wither completely.

Often the diseased tissues are covered with a layer of grey mould (*Botrytis* sp.) but it does not always follow that grey mould was the primary cause of the trouble (see p. 59). Other fungi or bacteria may have been the primary parasites which opened the way for the *Botrytis*.

In North America *Phytophthora parasitica* var *rhei* Godfrey and *P. cactorum* Leb. & Cohn have been found associated with crown rot of rhubarb.

Verticillium albo-atrum may also attack this crop, causing the plants gradually to wither and die (see p. 60).

In Britain the trouble is usually ascribed to *Bacterium rhaponticum* Millard, which causes the roots to become soft and almost chocolate-coloured (Metcalfe, G., 1940). The problem is complicated by the activities of the eelworm *Anguillulina dipsaci* Kühn which is commonly associated with *Bacterium rhaponticum* in the rotting crowns and is apparently able to cause decay of the tissues on its own account (Johnson, L. R., 1939).

When a new rhubarb bed is made the mother plants should be very carefully selected and no part of a plant with any sign of crown rot should be used for planting. Also rhubarb should be replanted straight away after being dug up, not, as is so often done, left for some time on top of the soil.

Mosaic

A mosaic disease of rhubarb, which causes rather a bright yellow and green mottling of the leaves, seems to be fairly common in Britain but has not been critically investigated.

SALSIFY AND SCORZONERA

Leaf Spot (*Ramularia scorzonerae* Jaap)

In 1937 we found spots caused by this fungus on scorzonera leaves at Lyngby. They were round or rather angular, brownish, often with a light centre and margin. On this occasion the outbreak was of no importance because it came so late. If it begins early the attack should be checked, if possible, by a fungicidal spray. The remains of a diseased crop should be destroyed.

White Blister (*Cystopus cubicus* (Strauss ex Unger) Lév. = *Albugo tragopogonis* (Pers.) Schroet.)

White blister occurs on both salsify and scorzonera. It causes more or less twisting or distortion of all above-ground parts of the plant and breaks out in numerous pustules containing white spore powder which becomes pale yellowish with age. It also attacks the related weed goatsbeard (*Tragopogon pratensis*). In severe outbreaks it is recommended that the tops be cut down and the new growth sprayed once or twice with Bordeaux mixture 16 : 8 : 100 or some other copper fungicide.

Mildew (*Erysiphe cichoraçearum* DC.)

The superficial mycelium and conidia of this mildew may form numerous and extensive powdery white patches, which often join up to cover the foliage (see mildew, p. 63).

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

Rotten areas surrounded by masses of white cottony mycelium with black sclerotia appear on the growing plants or on the stored roots (see p. 77).

Smut (*Ustilago tragopogi-pratensis* (Pers.) Roussel)

Infected salsify plants develop more rapidly than healthy ones but the young flower heads are filled with purplish-black spore powder of the smut. The diseased plants should be burnt as soon as discovered. This smut also infects goatsbeard (*Tragopogon pratensis*). The head smut of scorzonera (*U. scorzonerae* (Alb. & Schw.) Schroet.) is very similar but attacks only that host.

SHALLOT

See onion, p. 304.

SORREL

Leaf Spot (*Septoria acetosae* Oud.)

Small greyish spots with brown edges and dotted over with tiny black pycnidia appear on the leaves. Infected plants should be cut right back and sprayed or dusted twice with a short interval, using a copper fungicide. The attack will then be checked while the leaves are still small. One should not treat leaves a short time before they are to be used.

Downy Mildew (*Peronospora rumicis* Corda)

The mycelium of this fungus overwinters in the root-stock and thence invades the new shoots in spring, causing their leaves to remain small and stiff. The whole under-side of these leaves as well as the deformed inflorescence is covered by a greyish or lilac mould.

Infected plants should be destroyed and so, possibly, should wild sorrel in the vicinity. The remaining plants should be cut back and sprayed or dusted as recommended for leaf spot.

SPINACH

Anthracnose (*Colletotrichum spinaciae* Ell. & Halsted)

This fungus causes olive-green angular leaf spots that may turn yellow or almost white and dry up. They may join together especially along the leaf edges and cause extensive withering. *Colletotrichum spinaciae* seems to be as yet unknown in England but was found on spinach in Denmark in 1941. According to Neergaard (1940) it is not uncommon on spinach seed and may be very injurious to germination. In Holland seed-borne infection up to 60% of a sample has been found. Fungicidal treatment by shaking up the seed with an organo-mercury seed disinfectant should be tried. It may also be useful to spray the seed crop with Bordeaux mixture or similar fungicide.

Leaf Spot (*Heterosporium variabile* Cooke)

This disease is seldom reported but Moore (1945) thinks it probably widespread in Britain. The early stages of attack consist of small, rounded, white spots, about $\frac{1}{10}$–$\frac{1}{10}$ inch across scattered over spinach leaves, mainly over the apical half. In later stages the spots are more numerous and may join up and are more or less covered with a fine olive-green or blackish mould. The first spots are usually on the upper surface of the leaf but in later phases of the disease the fungus is equally abundant on both surfaces. No control measures have been suggested.

Downy Mildew (*Peronospora effusa* (Grev. ex Desm.) Rabenh. = *P. spinaciae* Laub.)

Irregular, ill-defined yellowish patches appear on the upper surface of the leaves, while on the corresponding parts of the under-surface there is a close, downy, grey mould that may acquire a lilac hue. Severely infected leaves are unsaleable (Plate 9, Fig. B).

The fungus may overwinter as oospores in the soil and these have also been found mixed with the seed (Richards, M. C., 1939). A high degree of humidity, at least 85%, is needed for production of conidia and these germinate only in the presence of surface moisture. Infection may occur at temperatures between 40° and 75° F. with an optimum at about 48° F. At temperatures of 60–65° F. infection may take place within 3 hours of inoculation.

Control. Spinach should not be grown on land that has already carried a diseased spinach crop and it is advisable to sow the spring crop

at some distance from overwintered spinach. With indoor crops the temperature should be kept fairly high. On seedlings one may try spraying or dusting with a copper fungicide, taking care to cover both sides of the leaf. This is impracticable with older plants whose leaves are ready for table.

Some differences in varietal susceptibility have been noted. In Denmark Victoria is very susceptible and De Haudry much less so. Disinfecting spinach seed by soaking in 1% copper-sulphate solution for 2 hours has been recommended as a preventive of downy mildew (*Plantenziektenkundige Dienst*, 1939). In the past downy mildews on common weeds, notably goosefoot (*Chenopodium*) and orache (*Atriplex*) were regarded as the same as that on spinach. Cross-inoculation experiments, however, have shown that this is not the case and therefore destruction of these weeds does not help to prevent mildew of spinach (Richards, 1939).

Virus Diseases

Spinach Blight (cucumber mosaic, *Cucumis* Virus 1)

This common virus causes a very destructive disease of spinach. Young leaves become yellowish, thickened, brittle and remain small with the edges inrolled and wrinkled. In severe cases some leaves may be reduced to a fine ribbon and others twisted, distorted and mottled. The outer leaves later turn yellow, then brown and shrivel until only a central cluster of yellow malformed leaves is left. When seedlings are attacked they become completely yellow or die.

The virus may be transmitted mechanically with the sap or, as occurs in nature, by aphides. The incubation period is only about a week in summer, 3–4 weeks in colder weather. This virus has a very wide host range (see p. 278). and is said by Böning (1930) to infect docks (*Rumex latifolius* and *R. crispus*). In England infection of winter spinach is worst when the crop is sown early. Infection probably comes mainly from marrows in the vicinity; seed-borne infection has not been observed (Storey, I. F., 1939). The disease has also been detected in Skaane, Sweden. A mosaic disease of spinach in Holland and Belgium has also been ascribed to a strain of the cucumber mosaic virus (Roland, G., 1939b). A cross with the Manchurian spinach "Virginia Savoy Blight Resistant" has been found resistant to the disease in North America and in Germany.

Mosaic (*Beta* Virus 2)

Mosaic of spinach caused by infection with the common beet mosaic virus from beetroot or sugar-beet is well known in North America and has been produced experimentally in Europe. The first sign of infection is a downward arching of the young leaves, accompanied by the appearance of numerous bright golden-yellow spots. The yellow colour gradually spreads, then the outer leaves die back beginning at the tip. The virus is spread by the beet aphis and other species and can be transmitted artificially by sap inoculation. The incubation period is 1–3 weeks. White goosefoot or fat hens (*Chenopodium album*) may also be infected by this virus. It develops isolated yellow spots and the leaf edges curl under. The sow thistle (*Sonchus arvensis*) may also become infected. These weed hosts may play a part in enabling the virus to survive between beet and spinach crops.

Sugar-Beet Yellows Virus (*Beta* Virus 4)

Can infect spinach, in which it causes yellowing and curling of the leaves, which become deformed with twisted petioles, and general stunting of the plant. This virus is common in the sugar-beet fields of North Europe and is widespread in Great Britain (Moore, W. C., 1943).

The common **tobacco mosaic virus** (*Nicotiana* Virus 1) may spread to spinach, for example from tomato plants, and yellowing and mottling of the leaves and a check to growth results (see further, p. 383).

Aster Yellows Virus (*Callistephus* Virus 1)

(see p. 428)

Causes dwarfing and yellowing in spinach in North America but is as yet unknown in Europe.

Control. Control of the aphides that spread these viruses may prevent infection of spinach. Diseased plants should not be left but should be pulled up and destroyed when the rest of the crop is picked. Cleared areas should be re-dug or ploughed as soon as possible. The later one sows winter spinach the more likely is it to escape infection. It is also desirable to plant spinach some way away from likely reservoirs of virus such as marrows, cucumbers, beet or sugar-beet and to keep down weeds such as white goosefoot.

Summary of Spinach Diseases

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STRAWBERRY

Mildew (*Sphaerotheca humuli* (DC.) Burr. = *S. macularis* (Wallr.) Jaczewski)

Symptoms. Mildew may attack strawberries out of doors or under glass. When the leaves are attacked they bear only a thin coating of the floury white spores among the hairs on their under surface. On the corresponding parts of the upper surface dark purplish blotches appear and the leaves roll upwards, exposing their under surface. On the fruit mildew produces a similar sparse white powdery coating and if they are infected before they ripen they do not turn a good red colour but become dry and poorly coloured. Their market value is thus impaired. Deutsch Evern seems particularly susceptible, so are Sir Joseph Paxton and British Queen.

Strawberry mildew is always prevalent in the South of England and was particularly severe in 1933, 1937–9 and 1948 (Moore, W. C., 1943, 1948).

Control. Wide variations in temperature appear to encourage the disease and should be avoided under glass and in frames. The usual sulphur fungicides may be applied until the fruit begins to ripen and after it has been picked. F. H. Moore (1937) advocates a 1–2% lime sulphur spray or a sulphur dust on fruiting plantations in April and early May and on runner beds and maiden plantations in the first half of July. Burning the beds as recommended against leaf spot may also be helpful.

Leaf Spot (*Mycosphaerella fragariae* (Tul.) Lindau = *Ramularia tulasnei* Sacc.)

Symptoms. Reddish-purple spots appear on the leaves which become more or less circular, about $\frac{1}{8}$ inch across and turn white at the centre, which may die and fall out (Fig. 212).

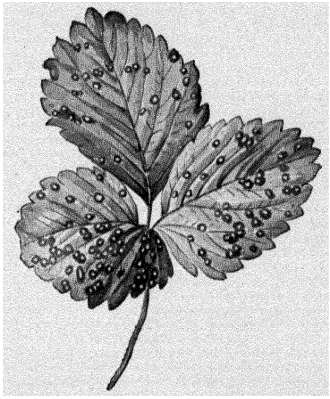


Fig. 212. Strawberry leaf spot, purple spots with a white centre.

In severe infections the spots may be small and angular, but crowded together so that large parts of the leaves turn brown and shrivel.

Cause and development. During the summer a white mould composed of bunches of short hyphæ bearing conidia of the *Ramularia* stage appears on the centre of the spots. On overwintered leaves the tiny black *Mycosphaerella* perithecia may develop, sunken in the leaf tissue. They are, however, seldom seen.

Importance. The disease is very widespread in Denmark and in England and probably occurs wherever strawberries are grown. When there are only scattered spots on the leaves it is of no real importance, but when they are numerous they may kill the outer leaves and weaken the whole plant.

Varietal resistance. Abundance may suffer severely from leaf spot, otherwise there is little information about varietal susceptibility in Denmark. In Germany, Noack made the following observations:

Highly resistant: Lucida Perfecta, Weisse Ananas.

Fairly resistant: Flanders, Deutsch Evern, Späte von Leopoldshall.

Very susceptible: Rheingold, Producktiv, Royal Sovereign, Konsum, Sensation.

Control. Though the fungus can be found in almost all old strawberry beds in Denmark, direct steps to control it are seldom taken. Where it does prove troublesome the beds may be burnt over with the straw after all the fruit is picked, so as to destroy the old leaves which carry most of the infection. Or the leaves may be cut with a scythe, raked up and burnt. When runners are being set out any leaves bearing leaf spots should be picked off. A Bordeaux spray may also be tried until the fruit begins to ripen.

Leaf Blotch (*Zythia fragariae* Laibach = *Phyllosticta grandimaculans* Bubák and Krieg)

This disease starts as small purple spots like those of leaf spot but never develops the white centre so characteristic of that disease. The spots gradually increase in size up to an inch or more and may involve an entire leaflet. The large spots are dark-brown, often paler at the centre, with a dark-purplish edge, sometimes fringed by a zone of yellowish leaf tissue. On the spots there appear minute brown pycnidia and in artificial culture a perithecial ascus stage has been obtained. This belongs to the genus *Gnomonia*. Wild strawberry, geum and cinquefoil (*Potentilla reptans*) are also susceptible to the disease (Wormald, H., 1944). Leaf blotch was first described in

central Europe and was first found in England at Long Ashton in 1931. It is now known to be widespread, especially on Royal Sovereign.

Leaf Scorch (*Diplocarpon earliana* (Ell. & Everh.) Wolf = *Marssonina fragariae* (Sacc.) Kleb.)

This disease starts as small dark purplish spots like those of leaf spot. As they increase in size, however, they do not develop the white centre. Individual spots become about ¼ inch across but several may unite to cover a large part of the leaflet and turn dark brown, hence the name "scorch." Infected leaves often turn yellow and the outer ones may wither altogether while the central leaves have few or no spots. Spots may also occur on the fruit stalks and check development of the berries. Tiny black conidial fructifications of the fungus develop on the upper surface of the spots. Control measures are the same as for leaf spot.

Somewhat similar leaf spots, brown with a reddish-purple border and often a yellowish zone outside that, may be caused by another pycnidial fungus, *Septogloeum fragariae* (Lib.) v. Höhnel, which also causes hard rot of the fruit.

Grey Mould (*Botrytis cinerea* Fr.)

Grey mould is a menace to the strawberry fruit crop under glass and in wet weather out of doors. On a single sultry July day a great many berries can be spoilt and covered by this hairy grey mould (Fig. 213). Excessive foliage favours the attack, for which reason it is advisable to avoid heavy manuring with nitrogen. The second and third years' crops are most likely to suffer. On low wet ground the fungus may also attack and kill the root stock and destroy the whole plant (see further, p. 59). On plants under glass all mouldy berries and any diseased or dying leaves should be picked off. Supporting the fruit on short forked twigs helps it to escape infection. Preventive spraying with Pomarsol has been tried.

Out of doors one should avoid too close planting and should spread straw or chaff under the trusses after flowering. The variety Deutsch Evern is said to be particularly susceptible to grey mould.

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

Verticillium wilt in strawberries has been studied in California by Thomas (1932). In England it appears most often in the fenland districts round the Wash but scattered outbreaks have been reported from other parts of England and Wales. Usually it attacks scattered plants in maiden beds but in 1943 severe outbreaks were recorded, especially on heavy soils (Moore, W. C., 1948). Infected plants may turn a reddish-yellow colour, and if they develop new leaves these may remain small and yellow or turn yellow between the

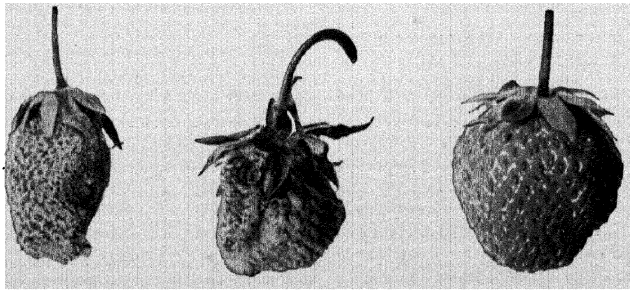


Fig. 213. Grey mould on strawberries, sound fruit on the right.

veins which are often banded with a faint reddish tint.

Sometimes the outer leaves wither first, turning brown and dry, or develop a brown margin. In other cases the whole plant withers at once and dies quickly. In milder attacks it may linger for a long time and develop a few narrow leaves and weak runners that soon die.

Usually there is no discolouration in the root stock until the disease is fairly well advanced, when small dark spots can be found in the flesh. Later on the roots turn dark brown. One can isolate the fungus from the roots much less readily than from the root stock. Usually one can obtain it from the upper part of the root stock and even in the petiole. *Verticillium* wilt is not the usual cause of black roots (see p. 362).

Runners should be taken only from healthy plants and not from those adjacent to wilted ones. It is, indeed, best not to take any runners from beds in which disease is present. Strawberries should not be planted on land known or suspected to contain *Verticillium* from another susceptible crop such as potato, tomato, raspberry or stone fruit (see p. 60).

Leathery Rot (*Phytophthora cactorum* (Leb. & Cohn) Schroet.)

In this disease the developing fruit turns brown and, instead of becoming soft and rotten as with grey mould, it becomes tough, leathery and eventually shrivels. As the rot proceeds there is a sharp dividing line between healthy and diseased parts of the berry (Fig. 214).

The disease was first recognized in Switzerland (1912) and North America (1922). It was found in the West of England in 1930 and in Denmark in 1935, though it had probably been present there in 1933. The fungus may attack a great many host plants and there is no reason to suppose the late date of its discovery indicates it to have been a recent introduction into England. Heavy rain favours the disease. Infection comes from the soil and is no doubt spread by splashing during rain. It is therefore apt to be more severe when the beds have not been properly strawed.

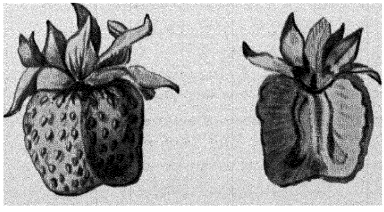


Fig. 214. Leathery rot of strawberry.

In the Tamar valley strawberry fields, Cornwall, H. W. Abbiss (1931) obtained some control of leathery rot by fungicidal dusts. A copper lime dust applied 5–6 weeks prior to ripening, followed by a second and third, the last very light to avoid poisoning the fruit, gave best control, but a sulphur dust was also beneficial and less objectionable for use on a soft-fruit crop.

"Running Out" or "Decline" of Strawberry Crops

It is a common experience that after a time strawberries cease to "do" at a particular place and the soil is then often said to have become "tired" of the crop. As in the case of other "tired soils" the phenomenon is not a simple one but may be due to several causes, operating singly or in combination. In the case of strawberries these include cultural factors, virus diseases (p. 364), root rots (p. 362), red core disease (p. 363), and insect pests. Strawberry plants suffering from several diseases and pests tend to develop much the same type of "degeneration," small, weak plants with short-stalked, red-edged leaves, few runners and low cropping capacity. The roots may be damaged by beetle larvæ, especially the June bug (*Melolontha vulgaris*), clay-coloured weevil (*Otiorrhynchus singularis*), and vine weevil (*O. sulcatus*). Where these insects are plentiful the plants wilt and die, especially in June and July. Nematode (eelworm) infection produces the disease known as red plant. The mite *Tarsonemus pallidus* is often troublesome in the South of England in hot, dry summers. The mites infest the leaves which remain small, brittle and yellowish and

in extreme cases turn red. Not only do they weaken the plant but the damage they cause masks the symptoms of virus diseases.

Soil conditions in many places are unsuitable for strawberry cultivation. Very heavy soils should be avoided, so should very light sands, except in areas of high rainfall. Strawberries require humus, and a direct relationship may be traced between the organic-matter content of the soil and the vigour of the plants. Manurial experiments have often shown good results from the use of stable manure. At Long Ashton manuring with waste products of the woollen industry has led to production of high-quality fruit. If a heavy dressing of farmyard manure (20–30 tons per acre) has been applied before planting, application of artificial manures seems superfluous, though a little nitrate may be given at the time of blossoming and a little more plus superphosphate after the crop has been picked. When dung is not available strawberries respond to phosphatic fertilizers, especially bone meal or superphosphate, plus a small quantity of nitrate of soda or sulphate of ammonia. Phosphorus-deficient strawberries develop purple and bronze tints in their leaves. Heavy potash manuring is not required except on definitely potassium-deficient soils. If there is chlorine in the fertilizer it may be definitely harmful, so too is sodium. Acid soils of pH 6–7 are said to be best but experience at East Malling shows that strawberries may do well on soil rich in lime, with pH 7.5–8 (Rogers, W. S., 1942). Presence of lime may, however, lead to minor element deficiencies and in Denmark excellent results have been obtained by micro-nutrient manuring on limed soils. It may be necessary to supply manganese, iron and copper. Boron deficiency has been demonstrated in sand-culture experiments but not yet in the field.

Methods of cultivation too are not always suited to the requirements of the strawberry plant. Its new roots are formed mainly in late summer and autumn and arise higher up on the root stock than the old ones. If the natural growing conditions of the plant are to be reproduced it should be surrounded with a layer of dead leaves or similar material every year. At Long Ashton good results have followed surface hoeing in August, after which the soil was pressed firmly against the plants. The practice of allowing weeds to grow in the the beds after the fruit has been picked and digging them in in autumn is undesirable as the digging destroys the new roots of the strawberries as well as the weeds. Runners take much nourishment from the parent plant and should be removed from a fruiting bed. Runner production should be carried on in separate beds and preferably as a separate industry (see p. 366). It is usually best to pick the blossoms from plants in their first year after planting and to allow them to fruit for the first time in the second year.

A plentiful supply of moisture is necessary for a good crop of fruit and in dry summers irrigation may be a profitable practice. On the other hand ill-drained soils are unsuitable and encourage root rots and red-core disease. Frost may damage many plants, and low-lying areas should not be chosen for strawberry cultivation. The practice of covering the beds with straw helps to protect against frost and drought. To protect the blossoms from late frosts straw should be placed above the plant at night and raked away during the day. It should not be strewn on the ground below the blossoms until frost is no longer to be feared or frost damage may be intensified (Cornford, C. E., 1938).

In Denmark covering the plants with powdered peat in winter has given good results. The peat is hoed into the soil in spring. Strawing under the plants after the fruit has set tends to reduce losses from grey mould and other fruit rots and increases the yield of first-class fruit. It may slow down ripening somewhat as the sun is less able to warm the soil (Oldham, C. H., 1937).

Brown roots. At the lower end of the root stock on any strawberry plant there are always some of last year's roots that have turned blackish-brown. The new roots that arise mainly in autumn higher up on the root stock and from the runners should be white or yellowish throughout, not dark brown or

black. A host of fungi is to be found on and in the brown and black roots, many of which are common inhabitants of diseased roots in many plants. Some of them have been shown to be capable of attacking healthy strawberry roots under certain soil conditions. The presence and extent of brown root rot in strawberries depends on the inter-relationship of fungi and soil conditions. In this connection we may mention Hildebrand and Koch's experiments in Canada with the "Wisconsin Tank," an apparatus in which the plants can be grown at a number of controlled temperatures and amounts of soil moisture. In this way it was demonstrated that there appeared to be no simple relationship between fungal activity and major nutrients, minor nutrients or soil reaction, but that soil moisture and temperature exercised an important effect. At a soil temperature of 45–50° F. and at 60% soil water-holding capacity the strawberry roots remained quite white, but as the temperature rose to 60° F. and moisture content to 80% browning of the roots steadily increased.

Well-defined brown lesions in the cortex of strawberry roots, followed by rotting and blackening, can be produced by the fungus *Leptosphaeria coniothyrium* (Fuckel) Sacc., best known in its conidial stage *Coniothyrium fuckelii* Sacc. on rose and raspberry canes (see pp. 524, 211). Berkeley and Lauder-Thomson (1934) in England have shown that this fungus isolated from raspberry canes can cause the brown lesions on strawberry roots. Similar observations have been made in U.S.A. Similar lesions can be produced by the small discomycete (cup fungus) *Discohainesia oenotherae* (Cke. & Ell.) Nannfeldt, formerly known as *Pezizella lythri* Shear and Dodge or, in its conidial state, as *Hainesia lythri* (Desm.) von Höhnel (Shear, C. L., and Dodge, B. O., 1921).

In North America the common soil fungus *Corticium solani*, the cause of black scurf in potato and damping off of many seedlings, has been shown to cause brown and black root rot in strawberry. The root stock itself may also be attacked by this fungus. We have seen a single instance of its attack on strawberries in pots, where it was cured by watering with Uspulun solution. *C. solani* has been found causing strawberry root rot in Somerset (Moore, W. C., 1948).

Small (1926) found a species of *Diplodina*, perhaps *D. lycopersici* (see p. 373), causing brown root rot of strawberries under glass in England.

Cylindrocarpon radicicola Wr., a common soil mould, has also been shown to be able to attack strawberry roots in some circumstances. This fungus is associated with brown roots in many plants, including narcissus, but is probably a very feeble parasite attacking tissues damaged by other agencies.

C. obtusisporium Wr. is a very similar fungus found on diseased strawberry roots in Utah.

Control. Brown root rot of strawberries is to be controlled mainly by maintaining good growing conditions with a well-drained soil. Only runners with clean healthy roots should be selected for propagation. In laying out new strawberry beds it is desirable to avoid land that has carried either strawberries or raspberries within the last 4–5 years. Planting in early autumn encourages strong growth. Control of foliage diseases encourages healthy root production.

Red Core (*Phytophthora Fragarioe* Hickman)

This is a serious root disease caused by *Phytophthora fragariae* Hickman. It was first recognized in the Clyde valley in Scotland about 1921, hence it was first called "Lanarkshire disease." In 1931 it was discovered in the South of England and is now known to occur in areas scattered throughout the country (see map in Moore, W. C., 1948). More recently it has been found in the United States (Demaree and Darrow, 1937; Bain and Demaree, 1938). The finer rootlets are first destroyed so that the main roots alone remain, looking like "rats' tails" when the root system is washed free from soil (Fig. 215). These main roots are then attacked at the tip and the fungus spreads upwards through the vascular cylinder, which it stains reddish-brown, the "red core." The root cortex remains white

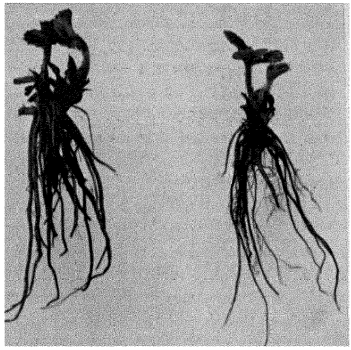


Fig. 215. Red core of strawberry (*Photo Dept. Agric. Scotland*).

and healthy for a time but eventually turns black and decays. The fungus has been isolated in pure culture by Hickman (1940), who was able to reproduce the disease by inoculating *P. fragariae* to healthy plants. Red core is worst on soils with a high water content but cannot be controlled by draining infected land. It is probably spread largely by the sale of infected runners and perhaps locally by infected drainage water as the fungus belongs to the group commonly called water moulds. Research sponsored by the Department of Agriculture for Scotland has led to the breeding and selection of a number of highly resistant strawberry varieties of good commercial quality. These are known as the Auchincruive seedlings, from the place where they were bred, the different varieties being distinguished by numbers (Reid, 1944). No method of soil treatment has proved effective against red-core disease, and the growth of resistant varieties offers the only hope of successful strawberry cultivation on infected land.

Cauliflower Disease (*Corynebacterium fascians* (Tilford) Dowson)

This disease was formerly confused with that caused by eelworm infection but has since been shown by Miss Lacey (1942) to be due to the same bacterium as causes leafy galls on chrysanthemums and fasciation in sweet peas.

Affected plants have small red leaves, those at the centre being often reduced to mere short, swollen leaf-stalks with rudimentary twisted leaflets. The tissue of the crown swells into an irregular gall. In severe attacks the plant may be killed; in others lateral crowns develop crowded together somewhat like a cauliflower, hence the name of the disease. Infection experiments have shown that the bacteria are able to cause the disease in the absence of wounding by any other agency.

Control measures have not been worked out but infected plants are worthless and should be destroyed. In nature infection presumably comes from the soil.

Virus Diseases—Yellow Edge and Crinkle

One of the main causes of degeneration in strawberry stocks is infection with one or more viruses, which leads to progressive loss of vigour and cropping capacity. Two kinds of virus disease can be distinguished in strawberry, yellow edge and crinkle, though the symptoms of each differ considerably in different varieties. Strawberry varieties can be divided into two groups: the virus-tolerant, such as Huxley's Giant, Oberschlesien, and Madame Lefebvre, and the virus-sensitive, such as Royal Sovereign and Sir Joseph Paxton. Tardive de Leopold and Western Queen occupy a somewhat intermediate position as regards reaction to virus infection. Unfortunately the tolerant varieties, though not clearly expressing the symptoms of virus disease, still suffer loss or vigour after virus infection and are liable after an indefinite period to show a " break-down " in tolerance. Some strains of the wild American species *Fragaria chiloensis* seem to possess complete and permanent tolerance to virus infection. This species is one of the parents of the cultivated strawberry, and there has been a tendency on the part of some writers to refer to virus-tolerant strawberry varieties as belonging to the " chiloensis type," and to virus-sensitive ones as the " virginiana " type.

Virus-disease symptoms are most clearly expressed by the variety Royal Sovereign and the following descriptions apply primarily to that variety.

Yellow edge. The first symptom of yellow edge is a flattening of the plants, followed by dwarfing owing to the leaves remaining small, curled and short-stalked. Runner production is also considerably reduced. In the typical phase of the disease the stunted leaves turn yellow at the edges. Expression of symptoms

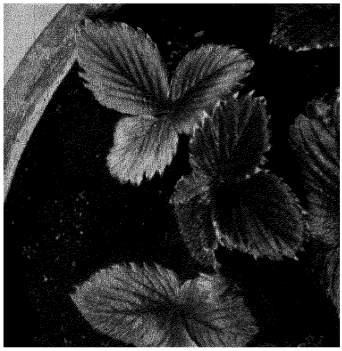


Fig. 216. Strawberry yellow edge (*Photo Dept. Agric. Scotland*).

depends not only on the strawberry variety but also on the temperature. In Royal Sovereign the symptoms begin to show up about a fortnight after the air temperature has risen to over 60° F. if soil moisture remains plentiful. Such periods, when the symptoms are easily detected and roguing is possible, usually occur in June and again in September and October (King, N. E., and Harris, R. V., 1942) (Fig. 216).

Crinkle. This is generally regarded as a less destructive disease than yellow edge but it occurs in several forms of different severity. These differences are now known to be due to the number of different viruses present in the plant. Leaves of an infected plant have a number of tiny, pale spots scattered over them. These are at first yellowish but soon turn red

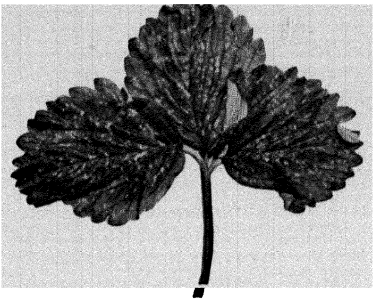


Fig. 217. Strawberry crinkle (*Photo Dept. Agric. Scotland*).

and do not grow. Hence the leaflets become distorted and crinkled.

Viruses concerned. It was soon realized that the great variation in symptoms even in a single variety probably indicated that yellow edge and crinkle were not simple virus diseases but complexes due to simultaneous infection with a number of viruses. Great difficulty has been experienced in separating and distinguishing the individual viruses, partly because no suitable differential hosts have been discovered. The only host known other than the cultivated strawberry is the wild strawberry *Fragaria vesca*. Of late years considerable progress has been made after the discovery that the aphis vector *Capitophorus fragariae* could be made to transmit different viruses according to the length of its feeding period on the infected plant. It is now known that one virus, that of mild crinkle, is carried without symptoms even by Royal Sovereign, though not by *Fragaria vesca*. This virus is one of the components of the virus complex which causes yellow edge. A second component, which by itself causes only very mild yellowing, has also been isolated from plants showing typical yellow edge. When these two components are recombined in a healthy Royal Sovereign plant typical yellow edge is produced. The second component may itself consist of more than one virus and it is clear that there is still much work to be done in identification and isolation

of strawberry viruses (Prentice, I. W., 1948). Severe crinkle is due to a combination of the mild crinkle virus and yet a third virus (*Nature*, clviii, p. 24). Other types of virus disease, presumably involving still further viruses, have been described in strawberries in North America.

Control. Once a strawberry plant has become infected with a virus it never recovers from it and all runners taken from the plant will be diseased. This applies even to runners taken at a time when, owing to temperature conditions, the symptoms are not fully expressed and the plant appears fairly healthy. All the strawberry viruses so far known in Europe are transmitted by the strawberry aphis *Capitophorus fragariae* and therefore one means of protecting plants from them is to control the aphis. This can be done to some extent by spraying with nicotine 8 oz., soft soap 8 lb., water 100 gallons, or more effectively by vaporizing nicotine over the bed, preferably towards the end of June. The latter process requires specialized equipment and is undertaken by contractors (Massee, A. M., and Greenslade, R. M., 1941). Strawberry runners should be dipped, roots and leaves, in a nicotine soap solution of the above strength before they are replanted. Roguing, or the removal and destruction of infected plants as soon as they are observed, is another important practice in maintaining the health of a stock. Because the disease symptoms are only clearly expressed when the air temperature is favourable roguing should be done at these times, viz. in June and September. The existence of virus-tolerant varieties introduces a difficulty in efficient roguing. Such varieties are a menace to any virus-susceptible variety in the neighbourhood. In fruiting beds of self-sterile varieties it is necessary to have a pollinating variety close by, but, especially in runner production, tolerant varieties should never be grown in the vicinity of sensitive ones. Thus, Tardive de Leopold needs a pollinator and for this purpose Huxley or Royal Sovereign is suitable, but in the latter case the Royal Sovereign plants cannot be relied on for fruit production. Hence there is today a tendency towards the development of a strawberry-runner-producing industry in districts well away from those where strawberry fruit production is carried on.

Virus-free stocks are available of certain varieties, notably Royal Sovereign M40, originally selected at East Malling from healthy plants discovered in Co. Mayo, Huxley M44 and Oberschlesien M42. To encourage production of virus-free runners on a commercial scale the Ministry of Agriculture in England and the Department of Agriculture in Scotland operate a Stock Certification Scheme. This scheme requires runners to be produced in small isolated blocks each consisting of 4 or 6 plants whose runners are allowed to become matted together, separated from the neighbouring blocks by a clear strip of cultivated ground 2 feet wide. The distance between plants in the block varies from 3–4 feet according to variety and locality. Full recommendations for this method of runner production have been published by W. S. Rogers (1947).

Under the existing certification schemes the certificates are offered for strawberry runners. The special stocks certificate is given only to those raised from tested pedigree stocks and is designed primarily to indicate stocks suitable for commercial runner production. The ordinary certificate is issued after an autumn inspection and signifies that the stock was found to be 99.5% true to type, free from substantial amounts of visible noxious diseases and pests and was growing at least 20 yards away from any obviously infected stock. These may be used for planting out beds for commercial fruit production.

Self-sterility

Plants with luxuriant foliage may disappoint the grower by flowering freely and setting no fruit. Some varieties have both male and female plants or female and hermaphrodite (bisexual) plants. In some varieties certain plants have poorly developed pollen and others sterile pistils. In propagating it is easy to take runners from the wrong plants and so to get only one type predominating. Varieties like Tardive

de Leopold and Oberschlesien are always self-sterile and another variety must be grown alongside to pollinate them. Huxley is suitable for this purpose. The relative proportions of the different kinds of blossoms may be changed by external conditions, especially the length of day.

Flower formation itself may be influenced by rainfall and watering. Rain checks flowering, irrigation does not.

Summary of Strawberry Diseases

		page
I. Roots and Root Stock		
A. Roots brown or black, poorly developed or killed	Brown or Black Root Rot	362
B. Fine roots destroyed, main roots dying from the tip with a red stain in the vascular strand	Red Core	363
C. Brown root stock associated with grey mould	*Botrytis*	360
D. Small dark-brown spots and streaks in the flesh of the root stock	*Verticillium* Wilt	360
II. Leaves		
A. Leaf spots:		
(1) Reddish-purple spots with white centres	Leaf Spot	359
(2) Reddish-brown or purplish spots, centre not white, edge sometimes yellow	Leaf Scorch	360
(3) Spots first small, reddish purple, spreading to an inch or more across or involving a whole leaflet, with a small brown centre	Leaf Blotch	359
(4) Yellowing between veins	*Verticillium* Wilt	360
B. Other discolourations:		
(1) Weak plants with reddened leaves	Decline	361
(2) Red-brown or bronze leaves (seldom seen)	Phosphorus Deficiency	362
(3) Leaf margins reddish purple, then shrivelling, leaves sometimes purple (seldom seen)	Potassium Deficiency	42
(4) Leaf edges shrivelled and brown, brown speck and lines in the flesh of the root stock	*Verticillium* Wilt	360
(5) Leaves reddish, youngest leaves in June and September yellowed at the margin and cup-shaped. Whole plant dwarfed and flattened	Yellow Edge	365
(6) Leaves reddish, rolled up, with a thin mealy coating	Mildew	358
(7) Leaves with tiny yellowish or purple specks, more or less distorted	Crinkle	365
(8) Leaves turn yellow and wither (on limed soil)	Manganese Deficiency	39
(9) Leaves yellow, under-surface reddish (on soil lacking lime)	Magnesium Deficiency	38
C. Leaves of an abnormal shape or size:		
(1) Leaves small, yellow-edged or slightly curled and cup-shaped	Yellow Edge	365
(2) Leaves distorted and curled, with dead specks, plant dwarfed or not	Crinkle	365
(3) Leaves small, yellow or reddish yellow, brown specks or lines in flesh of root stock	*Verticillium* Wilt	360

		page
III. Flowers		
Pistils and stamens blackened	Frost	22
IV. Fruit		
A. Lack of fruit	Frost	22
	Self-sterility	366
B. Small fruit	Potassium Deficiency	42
C. Mouldy fruit:		
(1) Grey, hairy mould	*Botrytis*	360
(2) Fine powdery white mildew	Mildew	358
D. Tough, firm rot	Leathery Rot	361
V. Whole Plant Weak, Dwarfed or Wilting		
A. Roots scanty and decaying:		
(1) Roots brown or blackish	Black Root Rot	362
(2) Fine rootlets lacking, main roots dead at tip with reddish vascular strand	Red Core	363
B. Brown specks and lines in flesh of root stock	*Verticillium* Wilt	360
C. Plant flattened, dwarfed, leaves cupped and yellow-edged or crinkled and spotted	Virus Disease	364
D. Stems stunted and swollen with a large number of buds crowded together	Cauliflower Disease	364

SWEET CORN (MAIZE)

Smut (*Ustilago zeae* (Beckm.) Unger)

Maize smut may attack all the young parts of the plant above ground, even the adventitious roots. It is least prevalent in the leaves. Large tumours are formed, sometimes as much as 7 inches long by 4½ inches broad, usually on the lower parts of the stem. To a lesser extent both male and female inflorescences may be partially replaced by irregular outgrowths full of black smut spores (Fig. 218). The shape of the plant may indicate infection with smut, for infected flower heads become so heavy that they bend down in some unnatural position. When the stem is attacked deformations may arise both above and below the smutted area.

The smut tumours may assume various peculiar shapes. At first they are covered with a grey, or whitish, shining skin made up partly of plant tissue, partly of a membrane

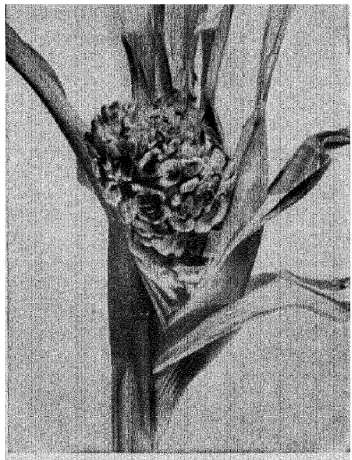


Fig. 218. Maize smut (*S. p. F.*).

formed by the fungus. Until it ripens the blackish-brown spore mass inside is moist and greasy but it rapidly changes to a dry powder which spreads the disease far and wide.

Cause and importance. Maize smut is caused by the fungus *Ustilago zeae* which does great damage in countries where maize is a staple crop. In Denmark it was first observed in 1917 on sweet corn in Farum. It was not found again until 1933, when a few ears were destroyed by it at Taastrup and Lyngby. Scattered attacks have been found almost every year since. Denmark seems to be at the northern limit of the fungus's distribution. It is very seldom found in North Germany, whereas ½–1% of smut is general in maize crops in South Germany. The great heat of July and August 1928 evidently favoured the disease, for 60% infection of maize crops was reported in Silesia and 40–50% in Baden. Smut has been observed from time to time in maize and sweet corn crops in England.

Control. The infected plants and parts of plants should be removed and burnt as soon as they are seen and all debris of the crop should be destroyed in the autumn. Infection is spread by maize debris in manure. It is stated abroad that the fungus is not carried by the seed, but its appearance in localities where maize has never been grown before indicates that this does occur to some extent. Possibly it is carried only on the surface of the seed.

Attempts to breed resistant varieties are in progress in countries where maize is an important crop.

Other Diseases

Rust (*Puccinia sorghi* Schw.) occurs occasionally on maize leaves in England. Stored cobs are sometimes damaged by grey mould (*Botrytis cinerea*) especially when harvested in wet weather or stored under damp conditions. Species of *Fusarium* may also cause decay of the cobs, and sometimes root rot of seedlings and growing plants (Moore, W. C., 1948).

TOMATO

Leaf Mould (*Cladosporium fulvum* Cooke)

Symptoms and development. Infection occurs mainly on the leaves, occasionally at the stalk end of the fruit. Leaf mould always appears first on the older mature leaves. The fungus enters through the stomata and the mycelium penetrates first between the leaf cells, afterwards into them.

The dark conidiophores emerge in bunches through the stomata on the under-side of the leaf and bear numerous tiny elliptical conidia, mostly two-celled. They form a greyish bloom on the spots that turns a purplish brown with age. No conidia are produced on the upper surface of the leaf but yellow blotches appear corresponding to the mould spots on the under-side (Plate 9, Fig. B). Gradually as the disease progresses the leaves turn yellow, curl and finally shrivel completely, thus greatly weakening the plants.

In North America infection of the fruit has been described but this has not been observed in Denmark. It takes the form of a black discolouration spreading outwards over the fruit from the end of the stalk but never covering the whole surface. The fruit may become lopsided and furrowed as a result of this attack. The fungus cannot attack the fruit directly but penetrates the stomata on the sepals or on the stalk and spreads through these into the young fruit or may prevent the fruit setting. Seed from infected fruit may carry the fungus and not only give rise to an infected seedling but act as a source of infection for neighbouring plants in the seed boxes. The seed coat containing the fungus is carried above ground on the cotyledons, conidia are produced on it and shed on to the surrounding seedlings.

In 1934 two instances were noted at Rostock in which outdoor tomatoes had a sunken rotting area covered with leaf mould fructifications round the style at the tip of the fruit (Reinmuth).

The conidia are extremely resistant. According to Hasper (1925) air-dried conidia stored for eight months still had a high germinative

capacity, and when stored dry out of doors they did not lose the power of germination until after three months' exposure to frost. A large proportion of the conidia were still able to germinate after being frozen in water for seven days, during which the temperature fell to 9° F. Conidia in water are killed by heating to 122–140° F. but some were still able to germinate after being exposed dry to a temperature of 156–158° F. for three hours.

The conidia are also very resistant to chemicals. This is evident by the difficulty experienced in devising satisfactory chemical control and has been further demonstrated by Rippel's (1932) laboratory experiments, in which conidia of tomato leaf mould were far more resistant to chemicals than those of the other fungi tested.

This is, however, much influenced by atmospheric temperature and humidity. Dry air checks conidial production (Lanford, A. N., 1937). According to Kalashnikoff (1936) the optimum temperature for growth and germination of the conidia is 68–78° F. and for infection 75–77° F. Small (1929) found the optimum temperature for growth of the fungus was 72° F. Under very damp conditions infection may take place at 50–54° F. but development is very slow below 60° F. Moisture not only favours the fungus but leads to the production of soft, sappy plants very susceptible to leaf mould. The harmful effect of humidity varies with the temperature. At the optimum for the fungus, about 72° F., a serious amount of infection takes place with only 80% relative humidity but only a little at 70%. When the temperature is below 66° F. primary infection and subsequent development of the disease are checked at 80% humidity. According to Newhall and Wilson the atmospheric humidity close to tomato leaves on warm sunny days may be 90 to 96%, because of the active transpiration, while a short distance away from the leaves it may be only 75%. During the summer 4 hours' excessive humidity at night is enough to bring about serious infection. Thorough ventilation during the day cannot overcome the damage done by dampness during the night. Spraying the leaves with water apparently does not favour the fungus directly, and a film of water on the leaf is not essential for conidial germination and infection.

The conidia seldom germinate at a temperature above 90° F. The incubation period for leaf mould under favourable conditions has been variously stated as 6–12 days, usually it is longer.

Opinions are divided as to whether the fungus grows best in light or darkness. Langford (1937) considered lack of light increased susceptibility; Guba (1938) found strong light may kill the conidia.

Volk (1931) found that mosaic-infected plants were more susceptible to leaf mould than healthy ones. Small (1930) found plants suffering from drought, or from starvation, were comparatively resistant. Tomato plants are relatively resistant to leaf mould when deficient in phosphates and nitrogen, or when supplied with excess potassium. Excess nitrogen and deficiency of potassium were conducive to the disease. The somewhat greater resistance of plants which receive excess phosphoric acid or potassium is due mainly to the consequent relative deficiency of nitrogen, for excessive application of these nutrients does not increase resistance if the nitrogen supply is increased at the same time (Schaffnit, E., and Volk, A., 1927). In Denmark and Britain the disease usually appears on the glasshouse crop but it has occurred occasionally out of doors, especially in warm, sheltered localities. The disease is most general from midsummer to the end of the tomato season but it may occasionally be seen in nurseries as early as 1st April.

The fungus may overwinter in the soil, on crop debris and on the glass and woodwork of hot-houses.

Varietal susceptibility. Tomato leaf mould has an unusual capacity for attacking different varieties of its host. Small tested 200 varieties and crosses at Cheshunt, but only Norduke, Stirling Castle and Up-to-Date showed any power of resistance. Unfortunately these are not good commercial varieties. In Danish experiments E.S.1 was attacked first and most

severely, Selandia and Potentate least. Several physiological races of the fungus have already been distinguished, differing in their capacity to attack resistant varieties. The wild species *Lycopersicum racemigerum* (= *L. pimpinellifolium*) or redcurrant tomato has a dominant factor conferring resistance to leaf mould (Alexander, L. J., 1934; Langford, 1937). Leaf-mould-resistant tomatoes of commercial quality have been raised, notably the Canadian variety Vetomold. Strains of the fungus able to attack this variety already exist, however, and much further work is required before there is any prospect of a completely satisfactory variety uniformly resistant to this disease.

Control. As the fungus overwinters in the soil and on the interior fittings of the greenhouse the house must be disinfected after a tomato crop. This is most easily done by burning sulphur (p. 600).

One should not pass straight from an infected house into one where the crop is still healthy, nor take tools, baskets, etc., from one to the other. By such means the conidia can easily be introduced into the healthy house.

It is of the utmost importance to prevent excessive atmospheric humidity. Therefore firing should be kept up at night throughout the summer and the house must be kept thoroughly ventilated. It is important to construct tomato houses so as to allow of plenty of side and end ventilation. An outbreak of leaf mould can be stopped altogether by heating and ventilating.

To help circulation of the air the lower leaves should be picked off as the trusses ripen and the fruit is picked. The upper shoots and leaves should be interfered with as little as possible, for some time always elapses before young leaves are attacked and it is important for the plants to retain a fair amount of healthy foliage.

Sulphur evaporation (see p. 600) may check the fungus very considerably, and some growers practise it at regular intervals of 8–14 days, but it appears that evaporation of sulphur may check fruiting.

Many spray mixtures have been tried. Bulbosan and Shirlan (see p. 599) seem to be best up to the present but even these have to be applied repeatedly if they are to be effective. Read (1936) obtained promising results with a combined spray of a colloidal copper compound in petroleum oil emulsion, to control both leaf mould and red spider. Any spray used should be started before the outbreak of disease is far advanced.

Any check in growth of the crop encourages leaf mould.

Alternaria Blight (*Alternaria solani* (Ell. & Mart.) Sor. sensu L. R. Jones & Grant)

Of late years in Denmark there have been one or two severe outbreaks of grey-brown, somewhat angular leaf spots on tomato. These are caused by the same fungus as *Alternaria* blight of potato (see p. 317), and under moist conditions an olive-brown mould formed by its conidia appears on the spots. Serious outbreaks occurred on the outdoor crop in South-east England in 1944 and 1945, causing both leaf spotting, stem spotting, fruit drop and rotting of fruit at the stalk end (Glasscock and Ware, 1944).

Leaf Spot (*Septoria lycopersici* Speg.)

This disease begins as small, dark, angular or round spots on the lower leaves. Gradually the spots dry up and shrivel, the skin over them becomes loose and they take on a silvery appearance. Scattered over the spots are the tiny dark pycnidia of *Septoria lycopersici*. The attacked leaves become yellow, curled, dry up and fall off and this loss of foliage reduces the cropping capacity of the plant.

The fungus can also attack various species of *Datura*, *Physalis* and *Solanum*. It does a great deal of damage in outdoor tomato crops in North America and has been found also in Germany. Moore (1943) knew of only five recorded outbreaks in England, where the disease has as yet been of little importance.

It may be controlled by repeated sprayings with Bordeaux mixture.

Canker, Fruit Rot and Fruit Spot (*Botrytis cinerea* Fr.)

Symptoms and cause. The common grey mould causes grey or grey-brown sunken patches on the stems, which under moist conditions become covered by the usual grey, hairy layer of conidiophores and conidia (Fig. 219). The fungus usually gains admission

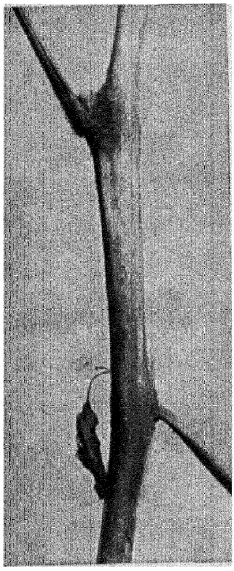


Fig. 219. Grey mould of tomato stem, the infected part is grey-brown.

through a wound and very commonly through the wounds left by defoliating mature plants, when conditions do not favour rapid healing of the scars. Often the part of the plant above the infected stalk wound is killed. This form of stem canker may be confused with bacterial canker but is easily recognized by the familiar growth of grey mould. *Botrytis* infection of this kind may be one of the most destructive fungus diseases in commercial glasshouses.

Control. Infection from the soil may be prevented by careful watering to avoid splashing the fruit or by covering the surface of the soil with straw, a practice often found satisfactory in Denmark.

Bewley (1923) states that the disease may be checked by spraying the soil and lower parts of the plants with Cheshunt mixture (see p. 593). Rosenbaum recommended spraying the soil with copper sulphate solution, $\frac{3}{4}$ oz. in 24 gallons. Bordeaux mixture might also be tried.

Grey mould may attack the fruit in various ways. Under very damp conditions when moisture accumulates round the stalk this end of the fruit becomes grey and soft, grey mould breaks out on it and the fruit drops off.

When the moisture remains only a short time small light rings may appear on the green fruit, almost always with a tiny dark dot in the centre. Similar pale rings may develop on the red ripe fruit (Fig. 220).

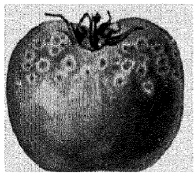


Fig. 220. Spotting of tomato fruit caused by *Botrytis*.

These rings (fruit spot or water spot) were known for a long time before their cause was discovered. The central dot looks like an insect puncture and aphides may cause a somewhat similar mark when they feed on the fruit, but true fruit spot may occur on plants quite free from aphides. It has also been suggested that they were due to drops of moisture on the fruit concentrating the sun's rays as does the lens of a burning glass.

In 1936–7 Ainsworth and Oyler studied this phenomenon at Cheshunt and found that *Botrytis cinerea* was the cause. Conidia of the mould drift about in the air and come to rest mainly on the stalk end of the fruit, which is normally uppermost as they hang in the truss. If the air is moist enough there will be a film of moisture on the fruit in which the

conidia germinate and penetrate the skin. If the surface of the fruit then dries the conidia die and no further penetration of the tissues takes place, but the germinative hyphæ which has perforated the skin secretes sufficient enzymes to affect the surrounding fruit cells and cause the ring round the dot.

It has been shown that infection occurs only at atmospheric humidities exceeding 90%. In experiments where fruit was sprayed with a suspension of *Botrytis* conidia and then kept in sealed jars, 4 hours in the moist atmosphere was insufficient for spot formation. After 4–5 hours some spots were produced, after 6 or more hours they were plentiful, and after 18 hours in a moist atmosphere rotting of the fruit set in.

Young fruit seems most susceptible to these spots. The varieties E.S.1 and Ailsa Craig were moderately attacked, Radio less so and Potentate very severely.

Control. As usual grey mould is checked by keeping conditions as dry as possible (see p. 59). It is also of great importance to make all cuts in such a way that they heal quickly. The wound surface should be left smooth, side shoots should be cut off up against the main stem, tops should be cut back at a leaf axil and if part of a leaf only is removed the cut should be made against the stalk of a pair of leaflets.

According to Bewley cankers that have not already girdled the stem may successfully be cut out and the wound smeared with a thick paste of lime or liver of sulphur. It has also been suggested that coating the brown tissue with a similar paste checks conidial production. Spraying with Pomarsol and dusting with Brassicol or Folosan may also be tried.

In the Cheshunt experiments spraying with Bouisol plus oil somewhat checked the development of fruit spot.

Stem Rot (*Didymella lycopersici* Kleb. = *Diplodina lycopersici* Hollos)

Symptoms and development. The disease occurs mainly on the stems of medium-sized and old plants and on the fruit. It may also appear on the stems of quite small plants and sometimes causes dark spots on the leaves

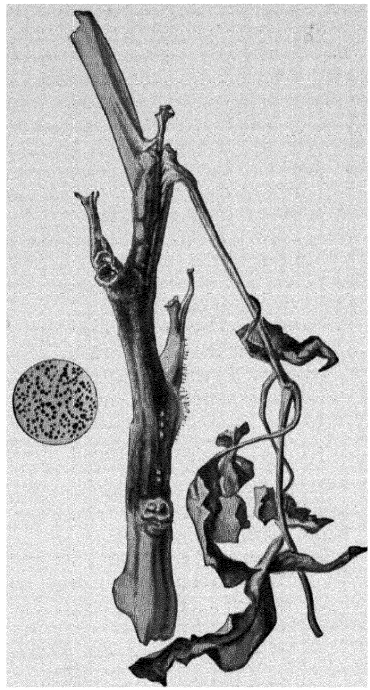


Fig. 221. Tomato stem rot. Inset, lens view showing pycnidia.

but this is exceptional. The rot on the stem forms a dark-brown area with a sharp boundary between it and the healthy tissue (Fig. 221). Dotted over the brown part are the tiny dark pycnidia. Infections can often be seen to be centred on wounds made by defoliation or other causes. In severe attacks the whole of the plant dies above the infected part.

According to Liesau (1932) infection occurs not only through cracks and wounds but also directly through the uninjured skin. The incubation period is 4 days or more.

The fruit is generally attacked only in one place but the rot may spread all over it. The spot usually appears when the fruit begins to turn red. It is then much the same colour as

the rest of the skin but has a granular appearance due to the crowded pycnidia. The latter are usually very close together over the whole surface of the spot and eventually turn black so that the diseased area as a whole looks blackish. Under moist conditions pink globules of conidia ooze out of the pycnidia. Infection at the base of the fruit may cause it to fall.

Dennis (1946) has shown that many of the dark spots and slow rots of this type on ripening tomatoes at the end of the season are due not to *D. lycopersici* but to related pycnidial fungus like *Phoma solanicola* Prill. & Delacr., which are unable to cause stem rot.

Schoevers (1929) has demonstrated that *D. lycopersici* may be transmitted with the seed and then attacks the stem of seedlings causing them to develop a brown rot. Pycnidia may be found on these stems and on the seed coat. Heavily infected seed does not germinate, however, and seedlings are less susceptible to infection than older plants, so that seed-borne infection is probably not very important (Orth, H., 1939).

On living tomatoes the fungus is found only in its pycnidial state formerly known as *Diplodina lycopersici*. Klebahn (1921) has shown that the perithecia of *Didymella lycopersici* develop on the overwintered dead stems.

According to Liesau the fungus grows best at 68° F. and the conidia germinate best between 68° and 75° F. Orth (1939), however, regards 62–66° F. as the optimum for both mycelial growth and conidial germination and found the conidia to be killed by exposure to 111–118° F. for 5 minutes. Both mycelium and conidia are resistant to frost (Liesau). Orth thought the disease less prevalent in glasshouses than out of doors because the temperature there often exceeds 90° F., the maximum for growth of the fungus.

As already mentioned the fungus may be spread with the seed but soil infection is much more frequent. The disease is always worst in cold houses and out of doors and where tomatoes have already been grown for several years. The fungus can grow in soil and thus remains alive in soil that has carried an infected crop. Old string and canes may also be sources of infection.

D. lycopersici may attack tobacco, potato and various species of *Solanum* (Liesau) but is of no economic importance on these hosts.

Control. In houses where the disease has appeared it is advisable to undertake sterilization of the soil or its complete renewal. When it occurs out of doors the plot concerned should not be used again for tomatoes.

As the fungus penetrates largely through wounds defoliation should be done as early as possible. Stubs of leaf stalk should not be left behind and the surface of the wounds should be kept as small and smooth as possible, so that they may heal quickly. Parts of stems that have been attacked should be cut clean away and the wound coated with lime, coal tar, or 50% fruit tree carbolineum. Orth recommends coating the base of the stem with a clay gruel containing 3% Ceresan or Uspulun. Other proprietary compounds of mercury, such as Sanagran or Aretan, may also be tried. Where the diseased area is near soil level it is advisable to pack soil round and over it after the above treatment to encourage the plant to put out new roots above the lesion. Diseased fruit should be picked and removed and so should all infected plants which cannot be treated.

In houses where an attack is to be feared the string tied low down on the plant should not be tied directly round it but to a stick in the ground or to a wire running level with the soil the length of the bed. Old string and canes should be disinfected before being used again (see p. 580).

Spraying with Bordeaux mixture is ineffective. Bewley (1923) recommended spraying the soil and lower parts of the plant with Cheshunt compound. Orth suggested watering once or twice during the summer with about a pint of 1:1000 corrosive sublimate solution per plant. As this is a concentrated poison it should only be tried when care can be taken to prevent any touching the leaves. The air should be kept as dry as possible.

Seed should only be saved from healthy

plants. If the seed available is suspected of being infected it may be disinfected (see p. 576).

Stable manure favours growth of the fungus in the soil and should be avoided in localities where stem rot has previously occurred.

Root Rot, Foot Rot and Damping Off

Symptoms and causes. The young plants become limp, the stem shrivels at soil level and they fall over. If the damage is caused by a species of *Phytophthora* or *Pythium* the shrivelled stem is greyish; if the damage is done by *Corticium solani* it is rather browner, but it is not always possible to distinguish the two forms of the disease without microscopic examination. Tomatoes are more often attacked by *Phytophthora* and *Pythium* than by *Corticium*.

According to Bewley *Phytophthora parasitica* Dast. does most damage to tomato seedlings under warm conditions because its optimum temperature is 80° F. That of *P. cryptogea* Pethybr. and Laff. is 75° F. and that of *Corticium solani* is 65° F. None of these fungi causes appreciable damage at temperatures below 50° F. (see also p. 68).

Control. All soil-borne fungus attacks can be prevented by sterilizing the soil by heat (see p. 577). *Pythium* and *Phytophthora* may also be controlled by formalin treatment of the soil before the seed is sown (see p. 599). Watering with Cheshunt compound (see p. 593) can also be done to disinfect the soil before sowing and to check an outbreak of *Phytophthora* damping off in the early stages. Solutions of corrosive sublimate and saltpetre and proprietory preparations containing mercury may be used in the same way against *Corticium solani* (see p. 73). Drying the surface soil by raking helps to check *Phytophthora* but not *Corticium*.

Toe Rot (*Phytophthora verrucosa* Alcock & Foister)

This disease of tomatoes has been widespread in Scottish tomato houses since 1934. It was first described by D. V. Howells (1936) but no description of the fungus appeared until 1940 (Foister, 1940). A few outbreaks have been reported in England but the disease is, no doubt, usually confused with other root rots.

Affected plants wilt by day and recover at night. Usually their foliage is darker than normal and the under-side of the leaves often has a purplish tint. The stem is thin and hard and the leaves are small and pointed. When the plant is dug up or knocked out of its pot the main root is found to be dead with a curiously pointed tip. Usually the core of the root is brown and hard. New rootlets are formed higher up, only to be destroyed in their turn, and eventually adventitious roots arise from the stem above soil level.

Infection may occur in the seed box but it is seldom detected at this stage as the only symptom is blackening of one or more of the roots. In pot plants the main root is usually destroyed and the hard appearance of the tops may develop, but often the trouble is masked by profuse development of secondary roots. Usually toe rot is first noticed after the plants have been set in the border and made considerable growth. Then, when conditions favour rapid transpiration, the damaged root system is unable to supply the necessary water and daytime wilting sets in. Cold soils encourage the disease.

Control measures as for foot rot and damping off.

Foot Rot (Collar Rot) of Older Plants (*Corticium solani* (Prill. & Delacr.) Bourd. & Galz = *Rhizoctonia solani* Kühn)

Symptoms. The plants are attacked at soil level where a dark, sunken, decaying patch appears on the stem. The side roots are usually dead or brown before this occurs. The main root is also often brown and rotten, its soft parts decay and only the woody core remains. The disease may resemble stem rot but the lesions of that disease are bounded by healthy tissue above and below, whereas those made by *Corticium solani* start in the root and spread upwards so that they are bounded by healthy tissue at the top only.

Cause. The disease is due to the fungus *Corticium solani* which lives in the soil. For further details see under potato, p. 326.

Control. If outbreaks have occurred in previous crops the soil should be sterilized by heat or by corrosive sublimate treatment (see pp. 577 and 579).

When older plants are attacked by foot rot they can sometimes be saved by packing fresh soil round the base of the stem. If possible soil free from the fungus should be used. The stem then puts out new roots above the diseased part. To prevent the fungus from growing into the fresh soil it should be watered occasionally with solution of corrosive sublimate and saltpetre or with a proprietary compound of mercury. Neighbouring plants should also be watered with the same solution. Damage by this disease occurs particularly during cold springs and to weak plants, hence if the disease appears in a greenhouse the temperature should be raised. Care should be taken to see the plants make the normal rate of growth.

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

Plants attacked by *Verticillium* wilt sometimes become stunted in growth because the stem ceases to elongate, thus causing the upper part of the plant to become rosette-like.

At first the plants wilt only during the day and recover their turgidity during the night. The lower leaves develop light-orange spots, and gradually this discolouration spreads up the plant and the leaves first affected wither. Infected plants may die quickly or may remain in an unthrifty condition for a considerable period. In the latter case they may ultimately die or may recover from the attack.

The vascular bundles of the stem are stained brown (Fig. 222).

Usually the fruit is not attacked but occasionally the fungus causes a soft rot (see *Verticillium* wilt, p. 60).

Wilting of tomatoes on hot days may also be due to root trouble, including eelworm infestation.

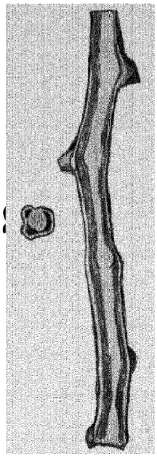


Fig. 222. *Verticillium* wilt of tomato. Stem cut longitudinally and transversely to show the brown vascular strands.

Brown Root Rot

For several years increasing attention has been paid to this disease in indoor tomato crops. Brown or brown-spotted roots appear in association with poorly developed tops. It has not been proved that the brown roots are the cause of the poor development above ground and the low yield of fruit, but circumstantial evidence makes this highly probable. There is often a small amount of browning on the roots even of plants that are fruiting well.

Brown roots are often seen also on melon, and the complex black root rot of strawberry (see p. 362) seems to be a comparable condition. In North America a widespread disease of tobacco is regarded as identical with that in tomato. We are much indebted for knowledge of this trouble to the researches of J. Johnson (1939), who worked with tomato plants. Johnson found the following crops particularly susceptible to brown root rot: garden beans, garden peas, sunflower, soy bean, tobacco, tomato, cornflower

(*Centaurea cyanus*), snapdragon (*Antirrhinum majus*) and a vetch (*Vicia villosa*); fairly susceptible were: alsike clover, potato, bird's foot trefoil, lucerne, lettuce, red pepper and egg plant as well as china aster, garden nasturtium (*Tropaeolum majus*) and *Euphorbia* sp.

Symptoms. In Danish tomato houses obvious brown roots may be found as early as April, about two months after planting out, when the plants are 18 inches to 3 feet high. If plants with healthy roots are planted in " sick " soil distinct brown roots may be found after 3–4 weeks (Johnson). Even earlier microscopic examination discloses spots in which the root cells have yellow thickened walls while their contents are brown and granular. The boundary between these spots and healthy tissue is clearly defined.

The brown spots or belts begin on the outer surface of the root, and gradually penetrate more deeply until they reach the vascular bundles, along which the brown discolouration spreads. When the roots are dug up one generally finds zones of brown, swollen and cracked cortex on the thicker roots (Fig. 223) alternating with zones of healthy cortex having the normal white or a light-brown colour. In more severe attacks the roots die until only a little tuft of weak fibrous roots remains at the base of the stem; the top is very backward, the whole plant thin and lanky and the fruiting very poor.



Fig. 223. Brown root of tomato.

Conditions of cultivation. Many observations indicate that appearance of brown root rot is closely bound up with conditions of cultivation. In experiments at Cheshunt the disease was worst on overmanured, wet and compact soil and it was concluded that great variations in soil moisture should be avoided. In North America the brown discolouration apparently appears most strongly after wide variations in temperature. In Danish experiments the best roots occurred with the smallest amount of water.

Brown root rot may be very prevalent in a new tomato house, and with continued cultivation of tomatoes the brown discolouration may decrease with successive crops until it almost disappears as soil conditions improve. Replacing the soil of an affected greenhouse often helps greatly but the question as to whence this soil should be taken, which previous crop is most favourable to tomatoes, requires closer investigation. Possibly soil from a beet or cornfield would be better than that from meadowland?

We have seen striking examples of the way in which particular soils retain the capacity for inducing brown root rot. Fig. 224 shows how soil from a tomato house in Funen still produced very sick plants after having been transported to Lyngby and decanted into experimental pots. Soil from another tomato house in Funen was brought in boxes to Blangstedgaard and on it in the following year we grew plants that were severely affected by brown root rot. On the other hand soil from the same house after being dried for 1½ years produced normal plants. In Johnson's experiments quick drying of the soil prevented brown root rot but slow drying had no effect.

In general the brown discolouration is worst when the plants are set out in the soil of the tomato house and much less marked when

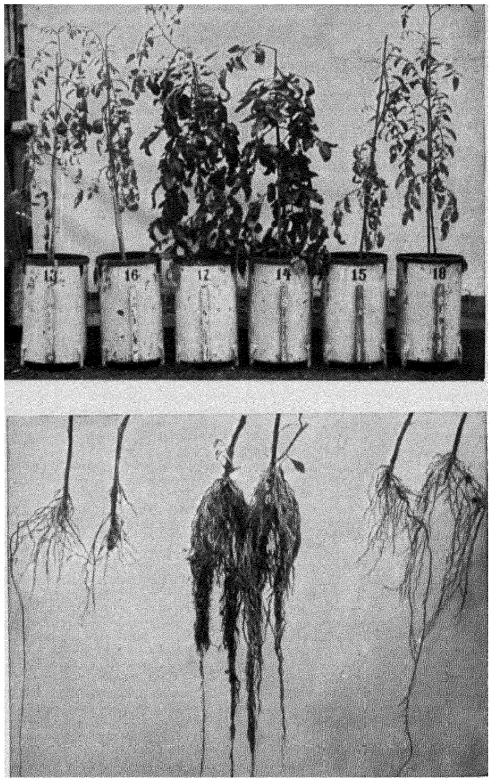


Fig. 224. Brown root of tomato. Tomato plants in experiment with soil from Drivhus, 7 years under tomato; they were weak and had severe brown root. Two pots of each treatment, from left to right: Untreated soil, baked soil, chemically disinfected soil (250 g. Kresol Soap × 5 g. corrosive sublimate in 40 litres per sq. metre). The chemical disinfection has to a great extent controlled brown root but both root and top growth are still weak. After baking, both roots and tops are normal and strongly developed (*S. p. F.*).

they are grown in pots. We have seen examples of tomato plants with roots healthy in the pot but brown where they have grown out through the bottom into the greenhouse soil beneath. This difference between the roots in the pot and those in the outside soil may persist until the autumn; the roots may be healthy in the clump and brown outside or vice versa. If the soil round large tomato plants is covered with a layer of stable manure and fresh soil or with fresh soil alone the plants may show brown roots in the old soil and fine white roots in the fresh covering layer. It has often been noticed that the tomato roots were much more vigorous and healthy along the walls of the greenhouse, apparently because the

soil became much less compacted there than in other parts of the house. Tomatoes grown on benches do not escape brown root rot.

Stable manuring seems in general to have a good effect on yield of fruit but does not necessarily prevent brown root rot. The manure should not be too fresh or strawy. Artificial manure may make the condition worse. Some observations seem to indicate that the large amounts of salts in ordinary commercial fertilizers are more unfavourable than highly concentrated nitrophoska. Phosphoric acid determinations in soils where there were severe attacks have varied from 5–6 to 60 and thus this seems not to be the decisive factor. Potassium levels have varied correspondingly from 10 to 50 without bringing about a decisive increase in the trouble. Various indications, however, suggest that very heavy potassium manuring is injurious. In individual severe attacks conductivity figures have been between 1 and 2, thus not high. Nor does the soil reaction, pH 6–7, give any indication, and in Johnson's experiments great changes in soil reaction (by adding hydrochloric acid, sodium hydroxide or chalk) had no effect on the disease. By addition of sulphur ($3\frac{1}{2}$ lb. per sq. yd.) the soil reaction was reduced to 3.1 and by adding coke-slag ash it was increased. Both treatments gave weaker plants. In one experiment addition of gypsum ($3\frac{1}{2}$ lb. per sq. yd.) gave somewhat more vigorous plants.

Additions of micro nutrients (boron, zinc, copper) have not been beneficial, indeed they have checked growth. Manganese is certainly often lacking in tomato houses. We have found manganese number 0, but supplying manganese has not helped to prevent brown root rot.

In Danish experiments digging in 9 lb. of fresh grass per sq. yd. in autumn gave the best yield of fruit from greenhouse tomatoes. Digging in vertical layers of straw has proved very effective at Cheshunt when the necessary nitrogen for decay of the straw was supplied as well (*Cheshunt Expt. Stn. Ann. Rept.*, 1933). In experiments on Fyn, digging in straw had a beneficial effect (C. Fich). In the State greenhouse experiments, on the other hand, adding chaff produced the brownest roots. Top dressing with peat, 9–14 lb. per sq. yd., has been recommended against "tomato sickness." In Danish experiments, however, we have not seen consistently favourable results, and where tomato roots have grown through the larger lumps brown discolouration may be found in the middle of the lump. The effect of peat seems to depend greatly on the quantity of nutrient available; the peat can tie up some of it. Addition of a large amount of clay has had a somewhat useful effect over one or two years in one of our experiments.

Fungi are found in great numbers on and in the brown roots. Amongst them are the water moulds *Pythium* and *Phytophthora*, *Verticillium* (see p. 376), *Didymella* (see p. 373), several species of *Fusarium* (including *F. culmorum* so common on cereals) and *Colletotrichum atramentarium* (see p. 330). In Holland, where "corky roots" are well known, a sterile grey fungus has been isolated in pure culture and used in inoculation experiments, but it did not attack healthy roots. A similar fungus has been found in Denmark. Finally we have several times found damping-off fungi (pp. 68 and 375) and a species of *Cylindrocarpon* (see pp. 417 and 489). Most of these fungi may be able to produce some kind of root disease on their own account but their importance lies particularly in completing the destruction of roots already weakened by other causes. Hence soil disinfection has only a limited usefulness against brown root rot.

Cause. Deficiency of nutrients or attacks by fungi are hardly the primary cause of brown root rot. The structure of the soil obviously plays a very great part but there must also be some other special factor which directly causes the trouble. It has been shown above that the cause can be transported with the soil but particular interest attaches to the diseased roots themselves. Johnson has shown that the disease factor is carried over with tomatoes that have just been dug up, but only to a slight

degree with air-dried roots and not at all with steam-sterilized roots. If partly dug affected roots are macerated in water the disease factor remains on the roots and does not dissolve in the water. The factor causing the disease is not destroyed by the oxygen of the air nor by sunlight. In our experiments the damping-off fungus (*Rhizoctonia*) was once carried over with diseased root material but it did not produce typical brown root rot. Amongst other experiments the one shown in Fig. 225

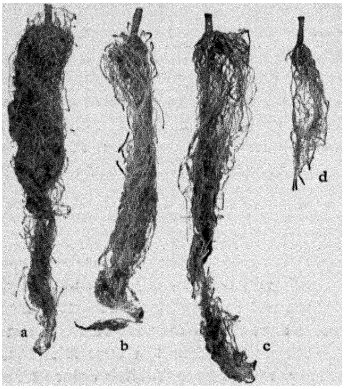


Fig. 225. Tomato roots can transmit brown root.
(*a*) Steam-sterilized garden soil from Lyngby.
(*b*) Same but with diseased tomato roots added.
(*c*) Steam-sterilized soil from tomato house with brown root.
(*d*) Same but with diseased tomato roots added (*S. p. F.*).

made with chopped diseased roots shows that they may have a deleterious effect in one soil and be almost harmless when added to another.

Soil disinfection. As the cause of brown root rot may be carried with the soil it is obvious that one should try prevention by soil disinfection. In this connection the good effect of baking the soil, heating to 203–212° F. and cooling in the course of 24 hours, is particularly noteworthy. A great many fungi and bacteria are killed in the baked soil but the soil structure is also completely changed. Certain substances may be completely destroyed by baking and others are liberated. In Johnson's experiments it was sufficient to warm the soil to 113° F. for 24 hours or to 118° F. for 12 hours.

The steam disinfection commonly used on greenhouse soils generally stimulates tomato growth. Observation of roots in and outside the disinfected root masses may indicate a beneficial action against brown root rot. Soil frozen at −13° F. for 10 days no longer induced brown root rot, on the other hand soil that had been frozen for 21 days at 23° F. still did so (Johnson). Experience in the severe winters 1939–42 showed that frost has no appreciable effect.

Chemical disinfection has often been tried but usually without success. The Benzol compounds Bayer P (¾ oz. per sq. yd.) and Brassicol (1¾ oz. per sq. yd.), however, have had a preventive effect in several instances; planting can be done the day after treatment.

Cresol soap (8 oz. per sq. yd.) in our experiments gave no effect but produced slightly more vigorous plants. Cresol (12 oz. per sq. yd.) has been recommended in Scotland. We have had feebly positive results with Cresol and corrosive sublimate. In Dutch greenhouses watering with Carbolineum has had some result. In our experiments Terracrimp (3 or 6 pints per sq. yd.) had some effect but the treatment should be done at least 6 weeks before planting.

Formalin (20–50 oz. per sq. yd.) gave better growth in Holland but brown roots were still present. This treatment, too, should be done 6 weeks prior to planting.

Carbon disulphide, used to a great extent against root-knot eelworm, does not prevent brown root rot and may even make it worse. The Dutch have had negative results with it (at 1 and 2 lb. per sq. yd.). We have obtained more vigorous plants with carbon disulphide and Cresol soap than in the control but the effect may have been due to the Cresol soap. Johnson had positive results with 3½–4½ pints carbon disulphide per square yard.

Corrosive sublimate (about ⅛ oz. per sq. yd.) dissolved in water has injured the plants. Dahmit, 1 pint to 8 square yards, gave a little larger root system than on unwatered plants.

Watering during winter with 60–100 gallons per square yard at 4–5 operations will wash out some of the injurious salts and thus, among other things, improve the condition of the soil.

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

The plants wither, sometimes the whole plant dies suddenly, in other instances the lower leaves shrivel first. A rotten area, often covered by white mycelium, is to be found on the stem near soil level. Threads of the white mycelium may grow out into the surrounding soil. Wrapped up by the mycelium or enclosed by the stem cavity from which the pith has disappeared are large sclerotia, at first soft and white but ultimately hard with a black rind (Fig. 226).

Green tomatoes picked to ripen off may be destroyed by the same fungus if they come from infected plants and especially if they have come in contact with infected soil, or been placed on a bench where others had decayed before. They are rapidly converted into a rotten mass overgrown by dense white mycelium containing black sclerotia.

Sclerotinia rot may be destructive both out of doors and in houses once the soil has become infected and if conditions are moist enough (see also p. 77).

Potato Blight (*Phytophthora infestans* (Mont.) de Bary)

This disease as it occurs on potato has been fully described on p. 312. It has been known on tomato in Denmark since 1888. On this crop infection occurs mainly on the green fruit, on which large grey-brown patches appear, covered as by a veil by the transparent skin (Fig. 227). At temperatures between 60 and 68° F. the incubation period is 3–5 days. Infection of the leaves is rare in Denmark or Britain. When it does occur dark-brown spots are formed, bordered under damp conditions by a whitish zone of conidia, as on potato leaves. Infection of the stems has been described in Italy and North America, both on newly planted tomatoes and on old plants (Ramsey and Bailey, 1931). If seed is saved from diseased fruit it often germinates badly (Bondartzeva, V. N., 1926), and one instance of the infection being transmitted to the seedling has been reported (Boyd, O. C., 1935).

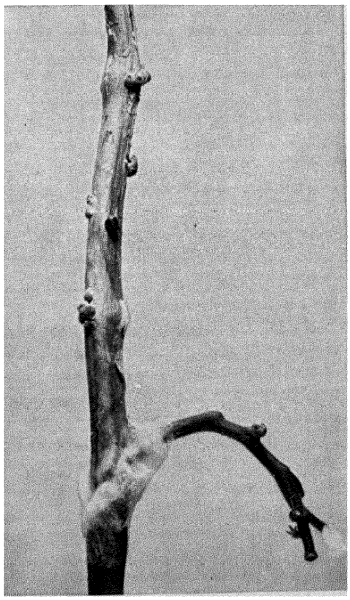


Fig. 226. Tomato stem attacked by *Sclerotinia sclerotiorum*.

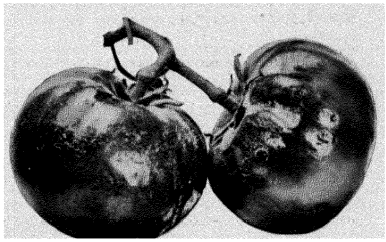


Fig. 227. Potato Blight on tomato fruit (*Photo Dept. Agric. Scotland*).

Intensive work on the physiological races of potato blight has led to the recognition of a race (A) widespread throughout the world and another closely related race (T) which has a particular capacity for infecting tomato leaves. Other races produce only larger or smaller spots on tomato leaves (Müller, K. O., 1926; Berg, A., 1926). Röder (1935) tested 81 tomato varieties and found that all were susceptible.

Control. In our experiments the yield of fruit has been increased and the number of diseased fruits reduced by spraying outdoor tomatoes with Bordeaux or Burgundy mixture as soon as blight is first seen on the potato crop, repeating the spray every 12 to 20 days thereafter. In American experiments good results have been obtained with copper lime dusts (Fromme, F. D., 1922; Poole, R. F., 1924). Where outdoor tomatoes are picked to ripen indoors spraying definitely improves their keeping quality.

Fusarium Fruit Rot (*Fusarium scirpi* var. *acuminatum* (Ell. & Everh.) Wr.)

Symptoms. Infection begins at the stylar scar at the blossom end of the fruit as a small, sunken, soft spot that gradually increases in size and turns dark. From its surface break out pink conidial pustules of the *Fusarium*, often arranged in concentric rings. Ultimately the entire fruit becomes permeated, turns yellow, shrivels and dries up. The disease occurs on the outdoor crop. It was serious at Lyngby, Denmark, in 1925, and at the beginning of this century in Berlin.

Cause. The attack is by the above-mentioned species of *Fusarium* which penetrates the fruit through the tiny wound left by the fall of the style. It cannot penetrate the unbroken skin but on picked fruit it easily enters by the stalk scar and may cause serious loss of outdoor tomatoes ripening in store.

The fungus needs a high temperature and high atmospheric humidity for active development.

Control. According to C. A. Jørgensen spraying with Bordeaux mixture effectively checks the disease.

Buckeye Rot (*Phytophthora parasitica* Dast. & *P. cryptogea* Pethbyr. & Laff.)

Symptoms and cause. The two species of *Phytophthora* which cause foot rot and damping off of tomato seedlings can also attack the unripe fruit. This develops a greyish spot marked with concentric brown rings round the point of entry of the fungus (Fig. 228).

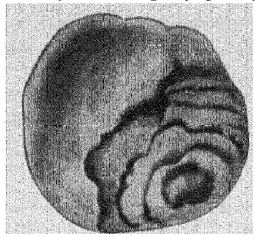


Fig. 228. Buckeye rot of tomato.

The disease almost invariably occurs on the lower trusses of fruit. The fungi concerned inhabit the soil and attack fruit in contact with the ground, or those on to which they have been splashed during watering.

Buckeye rot is well known in many countries where tomatoes are grown and has caused great loss from time to time in Denmark.

Bacterial Canker (*Corynebacterium michiganense* (E. F. Sm.) Jensen)

This disease was first recognized by E. F. Smith in 1909, in North America, where it has since been found to be widespread and destructive. In 1927 Kotte found it in Baden (Germany) and in 1939 there was an outbreak in Holland. It was first identified in England in Sussex in 1942 and in subsequent years numerous additional attacks were reported, mainly in south-east England (Moore, W. C., 1948).

Infection may occur at all stages from seedlings to large plants bearing ripe fruit. Long yellow or brown streaks appear on the stems and may crack to form canker-like wounds. The lower leaves wither and the damage gradually spreads upwards, though it may be

restricted to one side of the plant. Sometimes the leaflets on one side of a leaf wither before those on the other side. There may be yellow spots on the leaflets between the veins. In severe attacks the whole plant dies. The bacteria spread along and destroy the vascular bundles, thus upsetting the transport of food and water within the plant. One of the most reliable symptoms of the disease is the brown horseshoe-like mark made by the diseased vascular bundles on the scar left when a leaf is cut from the stem. In the early stages of infection the parasite is confined to the phloem strands but later it spreads into the cortex and sets up the canker phase of the disease.

There may also be round white spots on the fruit. At first they are flat and can be rubbed off, but gradually they develop a round, raised, yellow-brown centre, with one or several cracks in the outer skin. Often they are surrounded by a pale ring. The spots, not including this ring, never exceed $\frac{1}{8}$ inch in diameter.

The bacteria may be transmitted with the seed or may persist in the soil. Effective seed disinfection is difficult and consequently seed should only be saved from absolutely healthy plants. Infection is easily spread on the pruning knife and it is therefore advisable to break off the side shoots while they are still young to avoid use of the knife (Kordes, H., 1937). Bacterial canker is encouraged by an ample supply of nitrogen (Walker and Kendrick, 1948).

Bacterial Spot (*Bacterium vesicatorium* Doidge)

The first symptom of this disease is the appearance of numerous dark-green, translucent, circular or angular spots on the underside of the leaves. At first the spots are about $\frac{1}{10}$ inch across but several may unite. They soon turn grey or reddish brown and then become visible on the upper side of the leaf as grey spots with a brownish edge. Outside this is a lighter zone, best seen by holding the leaf up to the light. In severe attacks the leaves become rolled. Similar spots may appear on leaf stalks and stems.

On the fruit the bacteria cause greenish-white blisters which, with age, acquire an irregular flat or sunken brown centre on which the skin becomes cracked. These spots may be $\frac{1}{8}$ inch across.

The disease is transmitted with the seed and may be controlled by soaking it for 5 minutes in 1:3000 corrosive sublimate solution, followed by washing for 10–15 minutes in running water. Hot-water treatment for ten minutes at 122° F. has also been recommended (Gardner, M. W., and Kendrick, J. B., 1921).

This disease may be serious in both the outdoor and indoor tomato crop in North America but is as yet unknown in Denmark or Britain.

Virus Diseases of Tomato

Common Tomato Mosaic (*Nicotiana* Virus 1)

Tomato mosaic has been known in U.S.A. since 1901 and in Europe since 1905. It was discovered in Holland in 1907 and one of the earliest papers on this disease is that by Westerdijk (1910). Appreciable damage by mosaic to the greenhouse crop around Copenhagen was observed in 1911, and in 1912 it was found in greenhouses on Fyn. No precise information is available regarding its distribution in Denmark but it may safely be said to be widespread. The severity of the attack varies greatly and in mild outbreaks the loss caused is small. Selman (1941), however, has shown that even apparently mild forms of mosaic may cause 20% reduction in yield if the plants are infected early. The loss is due largely to failure of flower buds to differentiate and to set fruit.

Symptoms vary from ill-defined, light-green spots, mainly between the veins of the leaflets, to quite a bright, clearly defined, light and dark-green mottle. The surface of the leaves may also be slightly uneven or even curled. Tomato variety, nutrition and the time of year all affect the expression of symptoms; thus, during the sunny warm days of summer the mottling is most obvious, and during spring and autumn the curling becomes apparent. At low temperatures the symptoms

may be almost entirely lacking but the plants remain infected and infectious to others. Flowers of infected plants may be deformed, sterile and fall off. There may be slight streaking of the stems and pale, slowly ripening spots on the fruit.

Infection from tomato plants. The invisible infectious matter, or virus, occurs in the sap of almost all the cells of an infected tomato plant, including the cells of its numerous hairs. These are easily broken and it is a familiar experience that one cannot handle a tomato plant without getting sap on one's hands, tools and clothes. If one touches an infected plant and then a healthy one a mere trace of sap transferred from one to the other is enough to transmit the infection. Hands, knives and other tools are the most important agents of spread. Aphides do not spread this disease. The incubation period is from 5 to 20 days or 28 days in our experiments out of doors. Once introduced into the plant the virus travels through leaves and stems at the rate of about 1¼ inches in 24 hours. It persists for a very long time in dead plants and purified virus preparations can remain viable in the laboratory for 5 years. The virus is not killed until heated for 10 minutes at 194° F. or until treated for 24 hours with 60% alcohol, but it is sensitive to soap. Probably tomato sap dried on clothes can remain infectious for a long time. Virus liberated from a plant into the soil can remain infectious for a long time if the soil is moist; freezing and drying the soil renders it non-infective (Hoggan and Johnson, 1936). Virus in the plants and their roots is destroyed when they rot (Johnson, J., 1937).

Seed-borne infection. Over 50,000 tomato plants have been raised from seed obtained from a mosaic-diseased crop and not one of them has shown symptoms of mosaic. Yet in practice there is often circumstantial evidence that seed-borne infection has occurred (compare streak and yellow leaf pp. 385, 386).

Infection from other plants and products. Common tomato mosaic is caused by the same virus as common tobacco mosaic (see p. 83), which is common in all tobacco-growing countries. Fermented, commercial, smoking or chewing tobacco may retain the virus in an infectious condition. Hence chewing tobacco is a common source of infection in commercial tobacco production. Besides species of *Nicotiana* this virus can infect most plants belonging to the family *Solanaceae* and also species in other families, notably asclepias, snapdragon, larkspur, zinnia, beans, spinach and possibly celery. Potato, black nightshade and woody nightshade (bittersweet) show no symptoms, but they are carriers of infection.

From these hosts the virus can be returned to tomato. The nightshades afford a means of carrying it over winter as also do susceptible greenhouse ornamentals like *Petunia*, *Solanum* and others. There seems little danger to the tomato crop from mosaic-infected potatoes though there is evidence of one such a transmission out of doors in Denmark.

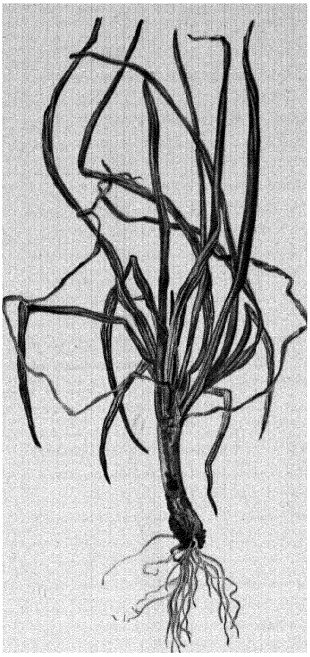
Growing conditions. It may be supposed that the virus multiplies more luxuriantly in vigorously growing plants. The statement that a crop can be "manured out of mosaic" only means that suitable manuring may overcome the consequences of infection; the virus in the plants is not destroyed. When nitrogen is very deficient the mottle becomes invisible; if nitrogen is then supplied it reappears (Volk, A., 1931). The influences of light and temperature have been mentioned above. Attacks are often found to start near doors and ventilators, which can scarcely be due to draughts but rather to infection from outside the house. In other cases mosaic is first seen in the warmest parts of the house. For control measures see p. 388.

Spotted Wilt (*Lycopersicum* Virus 3)

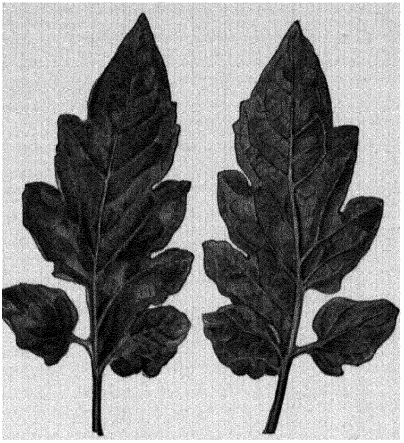
Spotted wilt was first noticed in Australia in 1915 and has become a very widespread and destructive tomato disease there. In the British Isles it has become very prevalent since its discovery there in 1932.

The first symptom is a slight thickening of the veins in the youngest leaves, sometimes accompanied by one or two concentric yellow rings in the leaf tissue. Almost at once the leaves curl slightly downwards and inwards.

PLATE 9



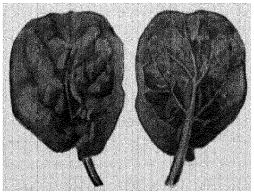
A. Shallot mosaic.



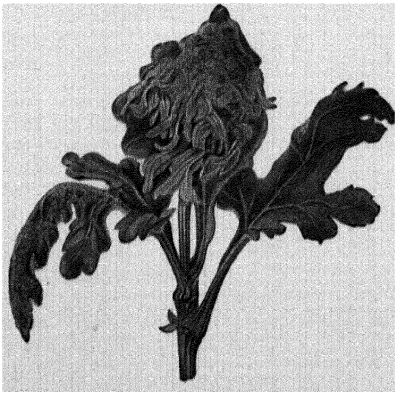
B. Tomato leaf-mould.
Left: upper side of leaf.
Right: lower side of leaf showing the patches of mould.



C. Penicillium rot in tulip bulb.



D. Spinach downy mildew.
Left: upper side of leaf.
Right: lower side showing the mauve mould.



E. Grey mould on chrysanthemum.

Then a large number of tiny bronze-coloured spots appear either in rings or scattered over the whole leaf surface. Growth stops for a time so that infected plants appear stunted. Young seedlings may be killed but more often the plant resumes growth after a time, but its leaves become distorted and have a rather conspicuous yellowish mosaic mottling. The fruit may be unaffected or it may bear pale-red, yellow or almost white markings, often in concentric rings.

The virus is sap-transmissible but not to the same degree as tomato mosaic. It is not spread by aphides but only by thrips. Curiously enough it is only in the larval stage that thrips can take up the virus from an infected plant, but once they have done so they remain infective throughout their life. *Lycopersicum* Virus 3 is not very resistant, it remains viable only a few hours in expressed tomato sap, is inactivated by drying, by oxidizing agents and by exposure for 10 minutes to a temperature of 108° F.

It has a very wide host range and commonly infects many popular greenhouse perennials which then form reservoirs of infection for the next tomato crop. Most of these show little sign of infection or bear only a few rather inconspicuous ring marks on their leaves. Amongst the known hosts are:

Amaryllis, *Anemone*, *Antirrhinum*, *Apium* (celery), *Aquilegia*, *Begonia*, *Browallia*, *Calceolaria*, *Callistephus*, *Calendula*, *Campanula*, *Capsicum*, *Chrysanthemum*, *Cineraria*, *Clivia*, *Coreopsis*, *Cosmos*, *Dahlia*, *Datura*, *Delphinium*, *Gerbera*, *Gloxinia*, *Hippeastrum*, *Hyoscyanus*, *Isotoma*, *Lactuca* (lettuce), *Lathyrus* (sweet pea), *Lobelia*, *Lupin*, *Lycium*, *Nicandra*, *Nicotiana*, *Paeonia*, *Papaver*, *Penstemon*, *Petunia*, *Physalis*, *Pisum* (culinary pea), *Plantago* (wild plantain), *Polygonum* (black bindweed), *Primula malacoides*, *P. sinensis*, *Saintpaulia*, *Salpiglossis*, *Salvia*, *Scabiosa*, *Schizanthus*, *Silene*, *Solanum* spp. especially *S. capsicastrum* (winter cherry), *Streptocarpus*, *Streptosolen*, *Trachelium*, *Tropaeolum* (garden nasturtium), *Vicia* (broad bean), *Zantedeschia* (arum lily) and *Zinnia*.

Of these arum lilies, begonias, chrysanthemums, hippeastrum and winter cherry are particularly common sources of infection for greenhouse tomatoes.

For control see p. 388.

Tomato Yellow Leaf (*Nicotiana* Virus 1B)

This disease was found in English indoor tomatoes in 1935 (Smith, K. M., 1935). It starts as a well-marked mosaic mottle on the youngest leaves. The older leaves turn yellow, except along the main veins which remain dark-green. As the plant grows the yellow spreads until only the topmost leaves remain green. The whole picture resembles nitrogen starvation. Some evidence of seed transmission has been obtained. In properties the virus closely resembles that of common tomato mosaic.

The aster yellows virus (*Callistephus* Virus 1) can also infect tomato and causes a certain amount of yellowing and a bushy type of growth, as it stimulates development of leafy buds and shoots in the axils of the leaflets as well. This virus is as yet unknown in Europe.

In the western United States the curly top virus of sugar-beet (*Beta* Virus 1) causes a tomato disease called yellows or western yellow blight. Fortunately this virus too is unknown outside America.

Aucuba Mosaic (*Nicotiana* Virus 1C)

This disease is caused by a strain of the common tomato mosaic virus and must not be confused with aucuba mosaic of potato, caused by quite a different virus. Bright yellow spots often appear on the leaves of tomato plants infected with common mosaic. If these are punched out and used for inoculation the aucuba mosaic disease is produced. It is also sometimes found by itself in commercial tomato houses. About 5 days after inoculation the youngest leaves curl down and their surface becomes rough, wrinkled or corrugated. By the seventh or eighth day yellow spots appear and become gradually larger, until in extreme cases the whole leaf surface is yellow, with a few raised islands of

dark-green standing out on it like blisters. More often the yellow and green areas are of about equal extent and intimately intermingled. Plants are not killed by the virus but their growth is checked and the fruit may be mottled.

Properties of the virus are the same as for common tomato mosaic. There is some evidence that seed-borne infection can take place in a very small number of seedlings, perhaps by virus dried on the seed coat.

Tomato Distorting or Enation Virus (*Nicotiana* Virus 1A)

This is another strain of the common tomato mosaic differing in that, in addition to causing mottling of the leaves, it stimulates the plant to excessive and unnatural growth. The leaflets may be increased so that a fern-like effect is produced with very many small leaflets crowded together, gradually diminishing in size and ending in a corkscrew-like tendril. In some cases small outgrowths (enations) are formed on the under-side of the midribs. There is a tendency to excessive growth and production of giant plants crowded with foliage. The fruit is often conical and corrugated (Ainsworth, G. C., 1937).

Bushy Stunt (*Lycopersicum* Virus 4)

This is a distinct and apparently rather uncommon virus which is easily transmitted by sap but has no known insect vector. In seedlings it causes yellow and purple colouration of the leaves, yellow spotting and death of the young leaves and often a sunken brown lesion at soil level, causing the seedlings to fall over. In older plants it causes cessation of growth by the terminal shoot and stimulation of the side shoots, hence the name " bushy stunt." The lower leaves turn yellow or purple and often there are concentric rings of bright yellow or purple, or purple lines along the veins of a yellow leaf. Ultimately the lower leaves shrivel and drop off (Ainsworth, G. C., 1936). This virus is particularly interesting because it has been purified and obtained in the form of minute cubical crystals.

Combined infection with *Nicotiana* Virus 1 and *Cucumis* Virus 1 (see p. 278 and fern leaf, p. 387) leads to a short, compact plant in which the stalks of the upper leaves become twisted and erect. The young leaflets are curled, distorted and thickened, sometimes thread-like, while older leaves roll up at the margins and have a mild yellow-green mottle. The flowers are usually malformed and sterile and any fruit set is ridged and misshapen.

Big Bud (*Lycopersicum* Virus 5)

Is due to a virus whose insect vector is unknown. It is not sap transmissible but is easily transmitted by grafting. Fortunately it is unknown in Western Europe, but it is widespread in North America and Australia. First symptoms are seen in the young fruit trusses which become erect and have their buds pointing upwards. The calyx segments remain united and swell into a bladder shape. As the disease progresses the leaves become smaller and smaller until dense rosettes of leaves only $\frac{1}{2}$ to 1 inch long are produced. The stems gradually become abnormally swollen and branched. Any fruit already present becomes hard and woody. A similar disease occurs on tomato in Russia.

Tomato Streak

Tomato growers all over the world are familiar with this disease, the most obvious symptoms of which are the dark-brown, grey or blackish stripes on the stems and leaf stalks, caused by bands of dead cells in the cortical tissues (Fig. 229). The disease has been known in Denmark since 1912 but its incidence varies greatly from year to year. It is a far more destructive disease than common mosaic as it checks the growth of plants and reduces their yield and often disfigures such fruit as does ripen.

Streak was first erroneously ascribed to a bacterium (*Bacillus lathyri*) commonly found in the damaged tissues. The bacteria, however, are destroyed by the usual bacterial poisons and by heating to 122° F., but the sap expressed from tomatoes infected with streak remains infective after treatment with 1: 1000 corrosive sublimate, heating to 176° F. for 10

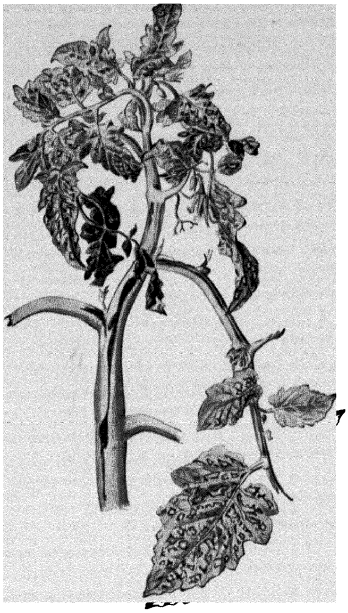


Fig. 229. Streak disease of tomato.

minutes or keeping for years in a sterile container. The common cause of the trouble is a virus closely allied to that of common tomato mosaic, viz:

Lycopersicum Virus 1.

The disease caused by this is known as "single virus streak" to distinguish it from the almost identical condition caused by a combined infection with *Nicotiana* Virus 1 and *Solanum* Virus 1 (see below).

Infection is very easily spread by sap and by touch but no insect vector is known. Seed transmission occurs in a very small number of cases if at all. Other known host plants are species of *Nicotiana*, *Datura*, *Solanum*, *Physalis* and *Capsicum*.

Mixed Virus Streak

Is due to simultaneous infection of the tomato with *Nicotiana* Virus 1 and *Solanum* Virus 1 (see p. 339). On stems and leaves the symptoms are indistinguishable from those of single virus streak but it is said that the two diseases can be recognized by the symptoms on the fruit. In single virus streak the markings on the fruit are sunken and discoloured (Fig. 230), in mixed virus streak they are slightly raised spots or flecks (Moore, W. C., 1943).

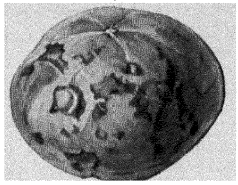


Fig. 230. Streak on tomato fruit.

Experience in Denmark and in England has shown that heavy dressings of stable manure make the symptoms of tomato streak worse, while ample doses of potassium fertilizers tend to reduce them. The damage is usually worst in warm houses or in the warmest parts of a house. Often plants are infected late in the season and the streaks occur only in the top growth. In Holland streak symptoms have been seen on black nightshade (Van Koot, Y., 1939). Dutch experiments have shown that the virus may survive from one tomato crop to the next in infected debris in the soil and may thence infect the young tomatoes as soon as they are planted out. A thick layer of sand applied all over the soil just before planting out protected the plants from infection.

Fern Leaf

Thread-like leaflets, better known as "fern leaf," were described in Holland by Westerdijk in 1910 in connection with tomato mosaic. Later American investigators have shown that the virus from mosaic-diseased cucumbers (*Cucumis* Virus 1) can be transmitted to tomato by the common aphis *Myzus persicae* and causes fern leaf, especially at low temperatures about 65–72° F. (Mogendorf, N., 1930). Fern leaf should not be confused with distorting

mosaic (see above). At higher temperatures tomatoes infected with cucumber mosaic suffer little and produce normal foliage. Thread-like leaflets, associated with poor growth and malformed flowers may also be caused by an inherited condition arising by mutation from normal plants (Schiemann, E., 1932).

Fern leaf occurs occasionally in Denmark but is regarded as of no importance there. It was particularly prevalent on the outdoor tomato crop in England in 1944.

Control of Tomato Viruses

Tomato virus diseases cannot be cured. Heavy manuring with potassium fertilizers may lead to considerable improvement which, however, may be too dearly bought (see pp. 28 and 379). Forcing growth by applications of nitrogen, by watering or by heating may make the symptoms worse. There is little prospect of breeding resistant varieties.

Much evidence shows that though seed-borne infection is not general it can occur to some extent with the mosaic and streak viruses. Hence, as even one infected seedling in a batch will soon infect all the others with these highly contagious viruses, it is unsafe to save seed from infected plants. It should preferably come from houses free from virus disease. The viruses can remain infective in soil that has not been dried or frozen. This is yet another reason for sterilizing all soil used in seed boxes and for potting. Batches of seedlings should be looked over critically at frequent intervals and any suspected plants removed and burnt.

Weeds such as nightshades, plantains, thorn apple and black bindweed that may carry infection should be destroyed. It has been pointed out that spotted wilt virus may be harboured by an enormous number of perennial ornamental plants which often themselves show little or no sign of disease. Hence this disease tends to be worst in small greenhouses where ornamentals share the house with tomatoes, or in nurseries where a chrysanthemum crop alternates with tomatoes. On a commercial scale the tomato crop should always be kept well away from ornamental plants and there should be regular fumigation with nicotine to keep down thrips.

Hands and tools of workers in tomato houses should be washed with soapy water at frequent intervals. Or the following solution kinder to the hands may be used: boil 1 lb. tri-sodium phosphate and ½ lb. soap in 1 pint of water until dissolved. Use 1 part of this solution with 5 parts of water. It is handy to have two basins, one containing the solution and the other water so that one can quickly dip one's hands in the former and then rinse in the latter (Van Koot, 1939).

Chewing or smoking tobacco may also be a source of infection when handled but it may be disinfected by heating in an enclosed place to 212° F. for two minutes (Thornberry, 1938).

It is doubtful if it is worth while to rogue out mosaic-diseased plants as they may bear fairly well and in any case considerable transmission from them is likely to have already taken place before they are detected. The first plants found infected with more serious diseases should, however, be at once destroyed.

The remains of an infected crop should be burnt as most of the viruses remain infective in them for long periods.

Poor Setting

Poor setting of tomato fruit may be due to various causes. The flowers may fall because of insufficient moisture in the air. Very dry air is particularly harmful when it is very warm. Cold may also check setting. In the tomato pollination takes place between stamens and pistil of the same flower. As it occurs best in sunshine and rather moist air it is a good thing to spray the plants lightly with water about 11 a.m. on sunny days and then ventilate the house.

If weather conditions during the pollination period remain unfavourable the grower should help the plants by shaking them, as by beating them or, better, the supporting strings or canes, so as not to injure the stems. Pollination is best effected artificially by painting the flowers lightly with a fine dry brush or a rabbit's tail.

Excessive nitrogenous manuring may hinder fruiting and should be avoided.

Mosaic infection may cause flowers to be deformed, sterile and unable to set (see also abnormal growth, p. 393).

Misshapen Fruit

The tendency to production of ridged and misshapen fruit is an inherited one, as shown by Pape. It is also, however, influenced by conditions of cultivation. Thus, Kondine Red is a variety rather prone to bear ridged fruit but it only does so when the plants are set very far apart and richly manured (see Fig. 20). Varieties of the E.S.1 and Ailsa Craig type also have a tendency to produce ridged fruit.

To avoid this disfigurement seed should be saved only from carefully selected plants with well-shaped fruit. The seed crop should be grown under conditions conducive to misshapen fruit so that any inherited tendency to do so becomes evident. In normal commercial fruit production spacing and manuring should be that found by experience to suit the variety grown.

Blossom-end Rot

Symptoms. The most striking symptom is a dark, more or less sunken, area at the blossom or style end of the fruit, i.e. farthest from the stalk. The trouble begins as a water-soaked spot at or near the base of the style, or the whole of that end of the fruit may look as though bruised. The affected part soon becomes leathery and usually turns black (Fig. 231). As the rest of the fruit continues to swell the blossom end becomes slightly sunken or remains abnormally flattened.

Cause. Various bacteria and fungi are commonly to be found in the affected tissues and have from time to time been described as the cause of blossom-end rot. They are, however, purely secondary invaders, living in tissue already damaged by a physiological disturbance. Especially if the surface of the fruit remains moist these secondary organisms may enter and set up an extensive rot. Tomato varieties in which the style remains attached for a long time seem particularly susceptible

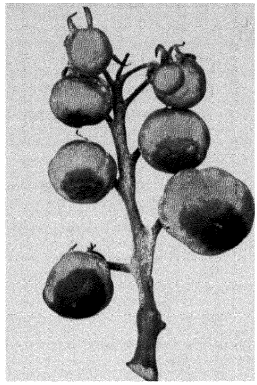


Fig. 231. Blossom-end rot of tomato (*S. p. F.*).

to blossom-end rot, perhaps because fungi and bacteria become established on the dead style and are hence ready to invade even a small spot of true physiological blossom-end rot.

By experiments over 5 years Brooks (1914) showed that the disease is not primarily due to parasites but to a physiological disturbance. Quite a large area round the style may be damaged without any help from micro-organisms.

The osmotic pressure in tomato leaves is 1–2 atmospheres greater than that in the fruit, so that in periods of drought the leaves are able to withdraw water from the fruit or at least prevent the fruit from obtaining enough water.

Hence the disease is to be attributed to unfavourable conditions of growth and especially to variations in water supply. These conclusions by Brooks have been confirmed by English and Danish experience. The disease most often appears 2–4 weeks after a dry period. Richly manured plants growing vigorously and accustomed to plenty of water suffer most. The condition is particularly serious on plants grown in pots or boxes which easily dry out.

In Denmark serious blossom-end rot has been noted in plants severely attacked by root

eelworm, which interferes with the water absorption by the roots.

Control. The most important preventive measure is regular watering to avoid alternate drying out and soaking of the soil.

It may be worth while to pick off the dead corolla and style on varieties that retain them long after the fruit has set. Otherwise fungi or bacteria established in the dead tissue may set up secondary rots following blossom-end rot. The variety Comet seems particularly susceptible to this trouble.

Hollow Fruit

Fruits divided by a single septum into two compartments are particularly prone to develop empty spaces between the septum and the fruit wall. Such fruit when shaken often makes a rustling or rattling sound. A larger or smaller part of the seed and flesh inside may be black and dead.

This unfortunate phenomenon, which appears especially towards the end of the summer, is not due to attack by any parasite. It is due especially to a period of drought during the swelling of the fruit but other growing conditions may possibly cause it. In such circumstances when there is a heavy crop only part of it will contain normal contents. The variety Radio is particularly prone to hollowness; so, in Danish experiments, were E.S.1. and Carrich.

Greenback

Greenback appears as a hard green or yellow area at the top of the fruit, surrounding the stalk. When ripening starts this part does not turn red like the rest but remains green and then slowly goes yellow or orange. It never becomes red and soft like the rest of the flesh.

Greenback, like blotchy ripening, is due to a deficiency of potassium, especially in fruit exposed to strong sunlight. Tomatoes forced to ripen at a temperature exceeding 90° F. do not turn red but yellow. Lycopin, the red colouring matter, is not formed at such high temperatures.

Greenback always occurs on the uppermost part of the fruit as it hangs on the truss, which is, of course, the part most exposed to the sun.

When defoliating tomatoes one should be careful to leave enough leaves to shade the trusses during the hottest part of the day. If the foliage is scanty, side shoots should be allowed to grow out and produce a couple of leaves before being stopped. Glasshouses should be shaded during the hottest periods. Care should be also be taken to supply enough potassium and water.

Kondine Red and Tuckwood are very prone to greenback, Selandia much less so.

Blotchy Ripening

In blotchy ripening larger or smaller parts of the fruit surface do not change colour with the rest as the tomato ripens. When it begins to turn red they remain green for a time but gradually turn yellow, develop a waxy appearance and a turnip-like flavour. The vascular bundles in the blotches are often dark and show faintly through the skin as dark lines (Fig. 232).

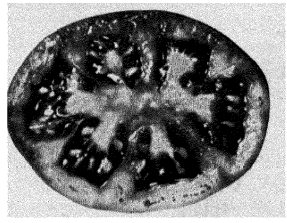


Fig. 232. Cross-section of tomato with blotchy ripening, showing dark vascular strands.

The blotches may occur on any part of the fruit, a feature which distinguishes them from greenback which occurs only round the stalk.

Blotchy ripening is seen mainly in June and July. Tomato varieties differ greatly in susceptibility to it; Kondine Red is one of the most susceptible.

Blotchy ripening is due to cultural factors or to diseased roots. In manurial experiments at Cheshunt (Bewley and White, 1926) it has been demonstrated that the greatest number of fruits with blotchy ripening occurred on unmanured plots and on plots deficient in potassium and nitrogen.

Later investigations by White (1938) have shown that light also plays a considerable part. In plots deficient in potassium, increasing the potassium supply or the light gave a greater weight of fruit and a lower percentage with blotchy ripening. On plots deficient in nitrogen or fully manured, the influence of light was much less.

Seaton and Gray (1936) in America thought the blotches due to the fruit being deprived of water for a period of increased transpiration about 2–5 days before the fruit ripened.

If tomato roots are diseased, if they receive too much water or too little, they cannot absorb enough nutrients and this may lead to blotchy ripening. The dampness of the subsoil should therefore be taken into account. It may well happen that the top 6–9 inches of soil are fairly moist but that the deeper levels are too dry. Tomatoes seem to develop most blotchy ripening in heavily limed soil. In Danish nurseries blotchy ripening is often prevalent where there is a large amount of phosphoric acid in the soil. If a heavy dressing of manure is given the roots may be damaged and thus less able to absorb nutrients so that the plants may starve on over-manured soil. Blotchy ripening is usually worst in new houses where growth is unusually vigorous. In summers with plenty of sunshine less potassium and more nitrogen should be supplied than in dull, cloudy seasons.

In Danish experiments less blotchy ripening developed in Carrich and Kondail than in other varieties tested.

Cracked Fruit

Cracking of tomato fruit occurs in two chief forms, single large cracks (Fig. 233) and many small ones; both types occur mostly on the top half of the fruit.

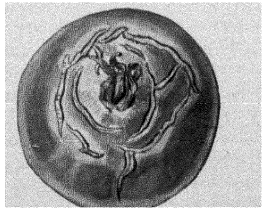


Fig. 233. Cracked tomato fruit.

The large cracks happen especially when a dry period is suddenly followed by a damp one. They are therefore usually found in the outdoor crop, where the grower has least control over the moisture relations of the plant. Cracking may also result from a sudden acceleration of growth for other reasons, as after manuring. The skin makes poorest growth under cool conditions and then has difficulty in keeping pace with rapid expansion of the flesh.

The numerous small cracks on tomato fruit occur especially in greenhouses where there are great fluctuations in atmospheric humidity. Growers should therefore be careful not to stop night heating too early.

In Montana, U.S.A., it has been found that tomato plants watered with 0.2 gram potassium permanganate per plant at replanting produced fewer cracked fruits than those not so treated. In other experiments where the root development was investigated it was found that plants watered in this way had more fibrous roots and grew bigger (Iverson, V. E., 1938). It is probably the increased root development that counteracts the effect of a fluctuating water supply. Potassium permanganate is usually used for watering, etc., at a strength of 1 : 1000.

Rolling of the Leaves

Tomato leaflets sometimes roll up so much that the upper surface becomes completely overarched by the upcurled margins which may meet above the midrib of the leaflet. Such leaves feel very hard and stiff. Varieties and individual plants may differ somewhat in the degree of leaf rolling.

would have gone to those that have been removed. Hence they become gorged with sap and stiff. Rolling is most pronounced under dry warm conditions but it seems to have no ill-effect on the plant. No diminution in yield of fruit has been traced to this condition. The phenomenon is seen mostly in outdoor

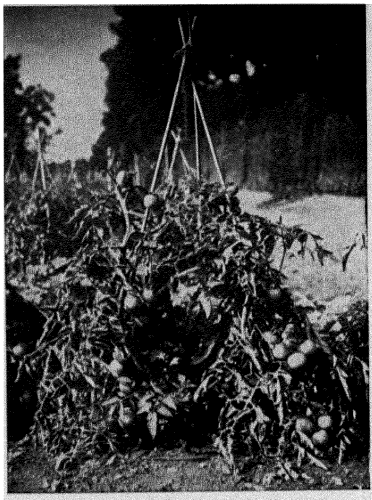


Fig. 234. Outdoor tomatoes showing rolling of leaves.



Fig. 235. Œdema on under-side of tomato leaf veins.

tomatoes, perhaps because they are more extensively stripped than the glasshouse crop.

The rolling of tomato leaves has no connection with the leaf roll disease of potatoes, which is due to virus infection (see p. 336).

If one wishes to avoid rolled leaves, and there is no great point in doing so, one should defoliate gradually, removing only a little at a time, and should leave the growing terminal shoot or allow a side shoot to take its place.

Rolling up of the top leaves may occur on young, actively growing plants. It occurs mainly when the crop has been heavily manured so that there is a high concentration of nutrient elements. Such plants have dark-green foliage and the top may have a violet tint somewhat reminiscent of spotted wilt.

Œdema

Œdema, named from a Greek word meaning swelling, first appears as a whitish thickening, especially on the under-side of the leaf veins (Fig. 235), but it may also occur as larger or smaller swellings on the leaf stalk and stems.

It occurs mainly in spring, particularly on tomatoes but also on other greenhouse plants such as pelargoniums and early forced potatoes. The outer cells become gorged with water, swell and burst the skin. The swellings then have a white powdery appearance but gradually turn brownish as the broken tissue falls away. Affected leaves have a tendency to roll.

Œdema occurs when the roots are absorbing more water than the leaves are able to transpire. A warm, moist soil increases the rate of water absorption but damp air, poor ventilation and shade check transpiration from the leaves. The condition may also become serious when, after a cool period with slow growth, the temperature rises and supply of water and nutrients to the foliage is suddenly increased. Such plants have relatively few leaves unable to cope with the large increased water supply resulting from the altered conditions.

To check œdema the soil should be kept fairly dry and there should be thorough ventilation.

It has been noticed in England that œdema became much more prevalent after the adoption of petroleum oil sprays against red spider. The film of oil checks transpiration from the leaves.

In Denmark œdema occurs mainly in the spring and never seems to cause any permanent injury to the crop. It has also sometimes been noticed some time after severely pot-bound plants have been planted out. Once the roots have grown out into the new soil they are able to absorb more water than can be dealt with by the rather stunted tops.

Hollow Stems

Some time after planting out the stems may become soft and flaccid; in bad cases the plants fall over without any actual rot taking place. When such a stem is cut open the pith is found to be wholly or partly wanting. Plants that have fallen over in this way may remain green for a long time but make no growth.

This condition arises in plants that have received too much water or excessive nitrogen or have experienced a sudden change in conditions when planted out.

Abnormal Growth Forms

The grower often finds a few plants which early attract notice by remaining short because the stem between successive leaves is shorter than in normal plants. These should be rogued out before the crop is planted out as such plants are partially or completely unfruitful.

Another objectionable inherited variation is the giant plant with abnormally vigorous growth. As a rule there are only a few of these in each batch of seedlings. Like the short-jointed plants they are practically sterile.

These varieties seem to be due to inherited defects. They occur scattered amongst normal plants and should be weeded out as early as possible.

Summary of Tomato Diseases

		page
I. Roots		
A. Brown roots:		
(1) Bark cracked and scaly	Brown Root Rot	376
(2) Roots decayed, brown threads visible under lens	*Corticium solani*	375
(3) Damping off, fine roots decayed	Damping Off and Foot Rot	375
(4) Slight decay, shoot tips rolled, yellow and dying	Boron Deficiency	32
B. Nodules and swellings on roots:		
(1) Irregular swellings (quite common)	Root-knot Eelworm	380
(2) Pea-size nodules on root or collar (rare)	Wart Disease	318
(3) Smaller nodules on fine roots	Powdery Scab	324
II. Stems		
A. Stem of seedlings or small plants brown at soil level, possibly shrivelled so that plants fall over	Stem Rot	373
	Damping Off	375
	Corticium solani	375
B. Plants " sleepy," brown vascular bundles	*Verticillium* Wilt	376
C. Abnormal growth:		
(1) Large, coarse plants	Abnormal Giant Plants	393
(2) Soft, hollow stems	Hollow Stem	393
(3) Short internodes	Abnormal Plants	393
(4) Slender pale plants	Nitrogen Deficiency	40
	Phosphoric Acid Deficiency (Rare)	41
(5) Slender, dark-green, rather short plants	Excess Phosphoric Acid	41
(6) Thin blue-green stems with bluish spots (rare)	Phosphoric Acid Deficiency	41

		page
(7) Top leaves curled, dark-green, then tinged bronze	Spotted Wilt	384
(8) Lower leaves yellow and curled. Growth in height checked, growth of side shoots stimulated	Bushy Stunt	386
(9) Young leaves rolled, yellow, shoot tip dying	Boron Deficiency	32
(10) Top much branched, foliage yellowish (not in Europe)	Aster Yellows Virus	385
D. Outgrowths:		
(1) Small whitish thickenings, later turning brown	Œdema	392
(2) Cauliflower-like growth on stem	Crown Gall	81
(3) Pea-like nodules at soil level (rare)	Wart Disease	318
E. Striped stems:		
(1) Canker-like wounds, yellow-brown streaks	Bacterial Canker	382
(2) No open wounds:		
(a) Light-green stripes	Common Tomato Mosaic	383
(b) Brownish streaks (seldom seen)	Potassium Deficiency	42
(c) Dark-green to blackish-brown or grey streaks	Streak	386
F. Spots, wounds or rot:		
(1) Large grey or grey-brown lesions covered with grey mould	*Botrytis*	372
(2) Extensive rotting, plentiful white mycelium and large sclerotia	*Sclerotinia*	381
(3) Sharply defined dark-brown lesions (dotted with dark spots under lens)	Stem Rot	373
(4) Brown spots	Potato Blight	381
III. Leaves		
A. Leaf edges abnormal:		
(1) Yellow-brown spots near leaf edge, dark spots between veins, margins dry out	Potassium Deficiency	42
(2) Shrivelled edges, then whole leaves shrivelled	Boron Deficiency	32
(3) Leaf edges rolled back, leaves spotted	Aucuba Mosaic	385
B. Leaf surface abnormal:		
(1) Leaflets threadlike, leaves may be distorted	Distorting Mosaic	386
	Aucuba Mosaic	385
	Fern Leaf	387
(2) Leaves curved downwards, finally hanging; leaf edges spotted and withering	Potassium Deficiency	42
(3) Leaf stiff, rolled in, veins normal	Leaf Rolling	391
(4) Veins with white or brownish eruptions on lower surface, leaflets may be rolled	Œdema	392
(5) Veins of youngest leaves more prominent, leaves curled back, surface covered with bronze dots or rings	Spotted Wilt	384

		page
(6) Leaves slightly curled or uneven, with faint mottle	Common Mosaic	383
(7) Leaves rough, often twisted, with white or bright-yellow spots alternating with dark-green	Aucuba Mosaic	385
(8) Leaf stalks brittle, leaves with purple tint, shoot tips yellow and dead	Boron Deficiency	32
C. Leaf surface spotted, discoloured or abnormally coloured:		
(1) Blue-green or dark-green leaves:		
(a) Vigorous, branched growth, terminal shoot possibly weak, leaves possibly curled and spotted	Excess Nitrogen	40
(b) Leaf edges spotted, withered, leaflets may have purple tint	Potassium Deficiency	42
(c) Small, sickly plants, roots may be brown, leaves dark-green. Not an uncommon condition	Excess Potassium	42
(d) Slender blue-green stems, upper leaves dark-green or purplish, lower leaves yellow or blackened (rare)	Phosphoric Acid Deficiency	41
(2) Leaves uniformly yellow, yellow-green or pale-green:		
(a) Slender plants	Nitrogen Deficiency	40
(b) Stronger plants on limed soil	Iron Deficiency	36
(c) Thick, firm leaves with purple under-side, stems stiff, slender and woody (rare)	Sulphur Deficiency	42
(d) Yellow or purple leaves, sometimes yellow-mottled on young plants. Older plants with lower leaves yellow and curled. Growth in height checked, many side shoots	Bushy Stunt	386
(3) Leaf edges yellow or pale:		
(a) Leaves yellow-green from the edge inwards, perhaps only the veins left green (on soil deficient in lime)	Magnesium Deficiency	38
(b) Leaves yellow except the veins, young leaves mottled	Yellow Mosaic	385
(4) Spotted leaves:		
(a) Light and dark-green mottle:		
i. Leaves possibly a little curled or uneven, no streaks of brown on stalks	Common Mosaic	383
ii. Stalks and main veins streaked brown	Streak	386
(b) Bright yellow or white and green mottle, leaves may be uneven or deformed	Aucuba Mosaic	385
(c) Leaves yellow all over with raised green spots (rare)	Aucuba Mosaic	385

		page
(d) Youngest leaves mottled. Older leaves yellow except for the green veins (rare)	Yellow Mosaic	385
(e) Yellow rings in youngest leaves, surface later bronzed	Spotted Wilt	384
(5) Spotted or discoloured leaves:		
(a) White or reddish spots in middle of leaves of young plants	Sun Scald	27
(b) Light-yellow dry spots between veins	Manganese Deficiency	39
(6) Light-yellow discolouration between veins:		
(a) Reddish veins, lower leaves shrivelled, whole plant yellowish green	Nitrogen Deficiency	40
(b) Grey-brown or mauve mould on under surface	Leaf Mould	369
(c) Main veins marked by brown line, small dark brown spots between veins	Streak	386
(d) Light-yellow or orange spots, first on lower leaves	*Verticillium* Wilt	376
(e) Brown spots with white mould beneath (rare)	Potato Blight	381
(f) Numerous small bronze-coloured spots	Spotted Wilt	384
(g) Small dark dry spots, ultimately glinting silvery	*Septoria* Leaf Spot	371
(h) Dead spots between veins and along leaf edges	Potassium Deficiency	42
(i) Dead spots (rare)	Phosphoric Acid Deficiency	41
(j) Blackish-green or black leaf spots	Ammonia Injury	43
(k) Dark-grey, translucent spots, later reddish or grey with a translucent edge, $\frac{1}{10}$ inch across	Bacterial Leaf Spot	383
(l) Angular dark dry spots	*Alternaria* Leaf Spot	371

IV. Flowers and Fruit

A. Fruit lacking or nearly so:		
(1) Luxuriant blue-green plants with excess foliage	Excess Nitrogen	40
(2) Large coarse growth	Abnormal Giant Plants	393
(3) Short internodes	Abnormal Plants	393
(4) Flowers misshapen, sterile, falling	Common Mosaic	383
(5) Flower trusses misshapen, calyx swollen	Big Bud	386
(6) Poor setting with no other distinctive feature	Sterility	388
B. Fruit misshapen or cracked, not rotten:		
(1) Grooved and ridged	Misshapen Fruit	389
	Distorting Mosaic	386
(2) Cracked skin	Cracked Fruit	391
(3) Grooved and split, fruiting poor	Excess Nitrogen	40
(4) Hollow between placenta and fruit wall, seeds and flesh may be blackened	Hollow Fruit	390

		page
C. Rotting fruit:		
(1) Blossom end dark, firm, sunken slightly	Blossom-end Rot	389
(2) Large spots, blackish brown, dotted all over and rough	Stem Rot	373
	Phoma Fruit Rot	374
(3) Large grey-brown spots and lines	Potato Blight	381
(4) Large greyish spots with concentric brown rings	Buckeye Rot	382
(5) Dark rot spots covered with dark layer of conidia (rare)	Leaf Mould	369
(6) Soft rot at stalk end, grey mould on surface	*Botrytis*	372
(7) Wet rot with white mycelium and black sclerotia	*Sclerotinia* Rot	381
(8) Sunken dark soft spots with pink conidial pustules	*Fusarium* Rot	382
(9) Dark spots, dark sunken vascular bundles	Boron Deficiency	32
(10) Pale spots and a peculiar smell	Bacterial Spot	383
D. Abnormal colouring:		
(1) Hard green or yellow patches at stalk end	Greenback	390
(2) Green or yellow waxy spots anywhere on fruit, taste like turnip	Blotchy Ripening	390
(3) Pale spots, ripening slowly	Common Mosaic	383
(4) Round white spots that can be rubbed off, then raised brown spots with cracked centres, spots not exceeding $\frac{1}{8}$ inch	Bacterial Canker	382
(5) Green or white blisters, then tiny brown cracked spots	Bacterial Spot	383
(6) Small pale rings with a dark dot at the centre	*Botrytis*	372
(7) Greenish-yellow irregular spots on the red skin	Aucuba Mosaic	385
(8) Yellow concentric rings	Spotted Wilt	384
(9) Dark spots and irregular lines	Streak	386

TURNIP AND SWEDE

Turnips and swedes suffer from most of the diseases that effect cabbages and their allies (see p. 260). Two, however, are distinctive enough to be dealt with separately, viz.:

Brown Heart (Boron Deficiency)

This disease, known as "Raan" in parts of Scotland and by a name meaning "marbling" in Denmark, affects only the flesh of the root. When this is cut across, either the whole central part or a zone about halfway between the centre and the rind is seen to be marked with irregular discoloured spots. At first they are rather grey and watery but in later stages of the disease they turn brown. Ultimately the affected tissue may become light and spongy. Affected roots have an abnormally low sugar content and a bitter flavour. They are also tough and woody so as to be unfit for table (Fig. 16).

Brown heart tends to be worst in the biggest roots. It is caused by a deficiency of available boron in the soil. In some districts, notably parts of South-west and North-east Scotland and North-west England, the soils are often truly deficient in boron, but in other districts brown heart may be the result of temporary

boron deficiency brought about by excessive liming. In such cases use of an acid fertilizer like sulphate of ammonia in place of sodium or potassium nitrate may have a beneficial effect. Brown heart can be easily and cheaply prevented by supplying boron to the soil, preferably before the seed is sown, in the form of borax or boric acid. Because boron is poisonous to plants when present to excess the dressing should not exceed 20 lb. borax or 15 lb. boric acid per acre, or 1 oz. to about 15 and 20 sq. yards respectively, and must be thoroughly and evenly mixed with the soil. It may also be applied mixed with the fertilizer, though preferably not with sulphate of ammonia.

Mosaic

In some seasons in Denmark turnips have suffered from a very infectious mosaic disease. This was easily spread by touching a diseased leaf and then one of a healthy plant. Diseased leaves may bear small pale spots, be strongly variegated or be yellowish-green with isolated dark-green islands scattered over them.

In the Danish experiments the virus was easily transmitted between turnips, radishes, mustard and charlock, but not to swede or cabbage.

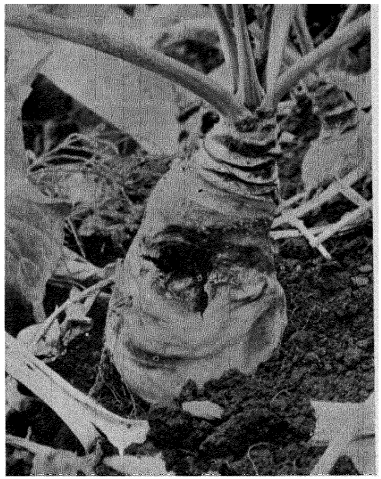


Fig. 236. Dry rot of swede (*Photo Dept. Agric. Scotland*).

Dry Rot (*Phoma lingam*)

This disease (Fig. 236) is caused by the same fungus as canker of cabbages (see p. 249). It is common and destructive in agricultural crops but seldom does much damage to garden turnips.

Summary of Turnip and Swede Diseases

		page
I. Root		
A. Irregular outgrowths	Club Root	246
B. Rough patches on the surface	Scab	322
C. Watersoaked or grey-brown mottling of the flesh	Brown Heart	397
D. Sunken grey-brown rotting patches, dotted with tiny black pycnidia	Dry Rot	398
II. Leaves		
A. Surface mottled light and dark-green	Mosaic	398
B. White floury coating	Mildew	252
C. Small dark spots with translucent edges	Bacterial Leaf Spot	255

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IV

DISEASES OF ORNAMENTAL PLANTS AND TREES

4 DISEASES OF ORNAMENTAL PLANTS AND TREES

ABIES (FIR)

Scorched Needles (*Rehmiellopsis bohemica* Bubak & Kab.)

This disease was first described on silver fir in Czechoslovakia in 1910 but it had been present in Scotland at least since 1905 and is now widespread in some districts. The young needles are attacked soon after they expand; they become reddened, then dark-brown or blackish, and shrivel. In severe attacks almost all the needles on a young shoot are infected and the shoot is often twisted and curved. The diseased needles do not fall at once but remain on the twigs for about a year, towards the end of which they may be somewhat bleached. The disease is caused by the fungus *Rehmiellopsis bohemica*, which forms fructifications like black dots protruding slightly from the upper surface of the dead needles. They are of two kinds which can only be distinguished by microscopic examination. Pycnidia (*Phoma bohemica*) usually appear first, followed by perithecia which usually ripen in the April or May of the year following infection (Wilson, M., and Macdonald, J., 1924).

Severely affected branches sometimes die during the summer but it is not always clear how far this is due to the fungus and how far to infestation with *Chermes*, which is commonly associated with it. Though best known as a disease of *Abies pectinata*, *Rehmiellopsis* can also attack *A. cephalonica*, *A. nobilis*, *A. pindrow* and *A. pinsapo*.

No control measures have been suggested beyond destroying infected young trees and cutting out the diseased branches from older ones. Planting pure stands of *Abies* is undesirable as it encourages the spread of the disease.

Rusts

Calyptospora goeppertiana Kühn is a rare rust with teleutospores in swellings on stems of the cowberry (*Vaccinium vitis-ideae*) and aecidia occurring as small orange-coloured pustules in a row on each side of the midrib on the lower surface of *Abies pectinata* and *A. nordmanniana* needles. *Melampsorella caryophyllacearum* (Johnst.) Schröt. forms rather similar aecidial pustules on *A. pectinata* needles and has its uredo- and teleutospore stages on species of *Cerastium* (mouse-ear chickweed) and *Stellaria* (stitchwort). In both its hosts the mycelium is perennial, so that once infection has taken place the disease will break out on the plant year after year, even in the absence of the alternate host. On the fir infected buds produce close clusters of small leaves which are arranged spirally and fall in their first year, about August. They occur in small erect bunches of twigs like small witches' brooms. Control measures include cutting out the witches' brooms and eradicating the alternate hosts.

Aecidia on *Abies* needles are also caused by other rusts, such as *Melampsorella symphyti* Bub., with uredospores on Comfrey, *Puccinastrum abieti-chamaenerii* Kleb., with uredospores on Rosebay (*Epilobium angustifolium*), and many fern rusts, notably *Hyalopsora aspidiotus* Magn., *Milesina kriegeriana* P. Magn. and *M. blechni* P. Magn., with uredo- and teleutospores on *Polypodium dryopteris*, species of *Aspidium* and *Blechnum spicant* respectively. The aecidia of these fern rusts can scarcely be distinguished from one another on the fir, and are known collectively as *Aecidium pseudocolumnare* Kühn. Infected needles occur scattered sparingly over the shoots, on which

they are conspicuous by their pale green colour. The aecidia occur in a row on each side of the midrib of the lower surface and each has a delicate white cylindrical sheath about $\frac{1}{16}$ inch long, with a torn apex, projecting from the needle surface (Wilson, M., 1924; Hunter, L. M., 1936).

ABUTILON

Variegation

The brilliant white variegation of the leaves of *Abutilon thompsoni* is familiar to gardeners, but it is perhaps less well known that this is due to infection by a virus. Baur discovered long ago that it could be transmitted to healthy *A. striatum* by grafting and that in fact *A. thompsoni* is only a mosaic-infected plant of *A. striatum*. The virus cannot be transmitted by sap inoculation, and no insect capable of spreading it has been found in Europe or North America. Recently, however, it has been stated that a species of white fly in Brazil can transmit mosaic of *Sida*, which appears to be the same as abutilon mosaic. This appears to be an instance of a virus which has been introduced from its original home, tropical America, into Europe and propagated there artificially because of its decorative effect on the host plant and because it is unable to spread in Europe in the absence of its insect vector (Kunkel, L. O., 1947).

ACACIA

See *Robinia*, p. 518.

ACER (Maple, Sycamore, Norway Maple)

Coral Spot (*Nectria cinnabarina* (Tode ex Fr.) Fr.)

The branches die and the cinnabar-red or blood-red fruit bodies appear on them (see p. 555).

Mildew (*Uncinula aceris* (DC.) Sacc.)

Leaves and shoot tips are covered by a white, floury fungus coating and may be deformed. This mildew may attack many species of *Acer* but is particularly common on the English maple (*Acer campestre*), both in nurseries and in hedgerows (see mildew, p. 63).

Tar Spot (*Rhytisma acerinum* (Pers.) Fr.)

Large black spots with a bright yellow edge are often to be seen in late summer on the leaves of sycamore (*Acer pseudoplatanus*). If they are very numerous the leaves may fall early, otherwise the disease appears not to damage the tree (Fig. 237). The fungus overwinters on the fallen leaves, in which the perfect ascus stage matures. Hence, if the fallen leaves are removed or composted in autumn the infection should be less next year. English maple and Norway maple are less often attacked.

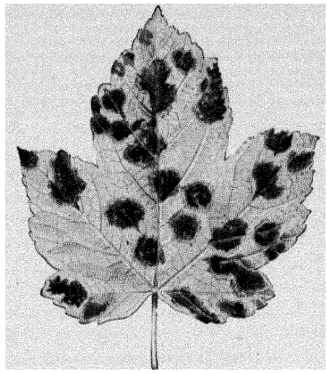


Fig. 237. Tar spot of sycamore, October.

ACHIMENES

Light Spots on Leaves

Achimenes leaves, like those of other members of the family *Gesneraceae*, may develop pale ring-like spots, said to be due to the lens action of drops of water in bright sunshine (see under *Saintpaulia*, p. 528). Thrips, however, often cause leaf spots on *Achimenes*.

ACONITE, WINTER

See *Eranthus*, p. 455.

ACONITUM (Monkshood)

Mildew (*Erysiphe polyoni* DC. = *E. nitida* (Wallr.) Rabenh.)

This fungus forms a white floury coating that spoils the appearance of the plants and checks their development (see mildew, p. 63).

Smut (*Urocystis anemones* (Pers.) Rabenh.)

This causes dark swollen spots on the leaves, which break open to expose the soot-like spores (see anemone, p. 411).

AGAVE

Leaf Spots

The leaves develop rotting spots where they are exposed to dripping water. The following fungi also cause leaf spots: *Cladosporium herbarum* Link f. *agave echeveriae* Sav., *Coniothyrium concentricum* (Desm.) Sacc. var. *agaves* Sacc., and *Colletotrichum agaves* Cav. (Fig. 238).

All diseased leaves should be removed and the plants kept as dry as possible or perhaps sprayed with a copper fungicide.

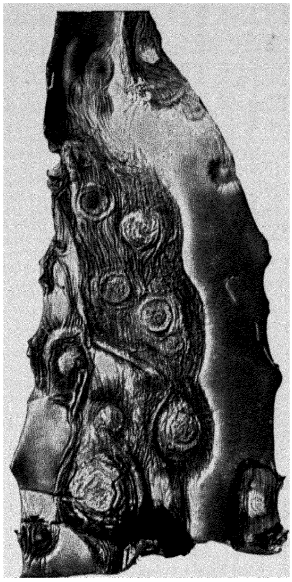
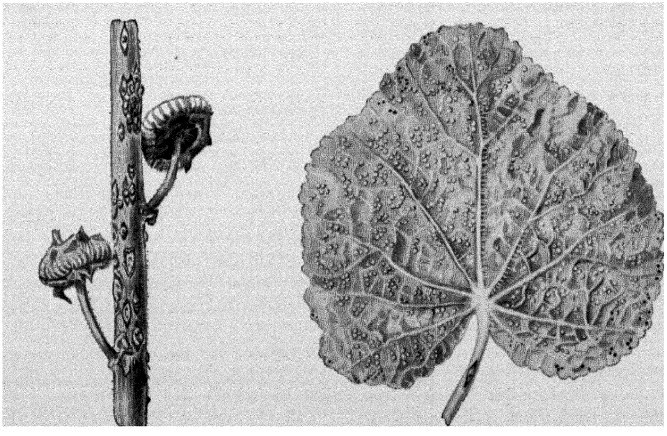


Fig. 238. Agave leaf spots caused by *Colletotrichum agaves*.



(*a*) (*b*)

Fig. 239. (*a*) Hollyhock rust on the stem. (*b*) Hollyhock rust on the under-side of the leaf.

ALTHAEA (Hollyhock, Marshmallow)

Hollyhock Rust (*Puccinia malvacearum* Mont.)

Symptoms. This rust appears on hollyhocks and many other members of the mallow family. It may attack any green part of the plant. On stems, leaf stalks and midribs the pustules are usually oblong; on the underside of the leaf blades and on calyxes and fruits they are round (Fig. 239). At first the pustules are orange-yellow but they soon become grey-brown as the spores mature. There is only one spore form in this rust, the teleutospore, which germinates in the pustule to bear four tiny basidiospores. These give the old pustules a greyish appearance.

This rust is extremely common on hollyhocks, where it does much damage by causing the leaves to wither prematurely. Hence the flowers do not develop fully and in the worst cases the plant may be killed.

History. Hollyhock rust is an instructive example of the speed with which a fungus may spread.

It is a native of Chile, from which it was described in 1852. In 1869 it was first observed in Europe, in Spain. In 1873 it did great damage around Bordeaux, where very dark-red hollyhocks are extensively grown for colouring the wine. By 1874 it had reached Denmark (Rostrup 1902) where it was found in many places both on hollyhock and on the common wild mallow (*Malva silvestris*) on Fyn. Within a few years it was to be found everywhere on hollyhocks, on cultivated species of *Lavatera* and on all native members of the *Malvaceae*.

At first it was very destructive to hollyhock. In some places it was so serious that the species of mallow were killed and disappeared from the district, while cultivation of hollyhocks had to be abandoned. The fungus is still important but not as destructive as in the early years of its invasion of Europe. Presumably the most susceptible plants were all killed out by it and those that still exist are descended from the more resistant plants.

One reason for the very rapid spread of the fungus may be that it produced such vast numbers of teleutospores. Rostrup counted over 2,000 rust pustules on a medium-sized leaf of the common wild mallow (*M. silvestris*). In an average pustule he found 4,000 teleutospores each of which would normally produce 8 basidiospores. Therefore a single mallow leaf may bear 64,000,000 basidiospores, each of which may give rise to a new infection if it falls on a plant belonging to the mallow family and finds suitable conditions for germination there.

Control. Very heavily infected plants should be removed and burnt. Seed should only be saved from healthy plants. In autumn all parts of hollyhocks above soil level should be cut off and destroyed. In spring the first leaves should be removed even if no sign of rust appears on them and the following leaves should also be cut off if there is the slightest sign of any attack on them. The plants should be sprayed or dusted several times with a copper fungicide.

In a private garden near Copenhagen good results were obtained by these hygienic measures and the use of a Burgundy spray.

Other diseases recorded on hollyhock in Britain include *Sclerotinia* rot (see p. 77) and leafy gall (*Corynebacterium fascians* (Tilf.) Dowson) (see p. 484).

ALYSSUM

Downy Mildew (*Peronospora galligena* Blumer)

The white downy coating of the fungus occurs in small blister-like spots on the underside of the leaves, which may also be curled, deformed or even gall-like, and growth of the plants is checked (Fig. 240). This disease was first found in England in 1946 (Moore, W. C., 1949).

Control by cutting off the diseased tops and spraying or dusting with a copper fungicide should be tried.

AMARYLLIS

See *Hippeastrum*, p. 470.



Fig. 240. *Peronospora galligena* on alyssum leaves.

ANEMONE

Smut (*Urocystis anemones* (Pers.) Rabenh. = *Tubercinia anemones* (Pers.) Liro)

Irregular blisters and streaks appear on the diseased plants, out of which the sooty black spore powder falls when they break open. Several members of the *Ranunculaceae* may be affected by this disease, including the common wild buttercup, but it is probable that physiological races exist specialized to each host. Among garden plants the smut occurs also on *Aconitum*, *Eranthus* (winter aconite), *Hepatica* and *Trollius* (globe flower). It has been noted several times in Denmark and England.

Diseased plants should be rooted out as soon as recognized, care being taken not to scatter the spores in the process.

Black Rot (*Sclerotinia tuberosa* (Fr.) Fuck.)

This fungus occurs especially on the wood anemone (*A. nemorosa*) and its varieties and slowly kills them. In the dead rhizomes sclerotia may be found which greatly resemble the corms of *A. coronaria*.

Brown cup-shaped apothecia are produced in great numbers from the sclerotia in spring (Moore, W. C., 1946). The fungus also attacks *Anemone coronaria*, *A. apennina*, *A. blanda* and *A. ranunculoides*.

Diseased plants should be dug out completely with the surrounding soil and destroyed.

Downy Mildew (*Plasmopara pygmaea* Unger)

This fungus causes patches of fine whitish mould on the under-side of anemone leaves. It may attack many plants belonging to the *Ranunculaceae* and may do considerable damage to various species of anemone and hepatica.

It is advisable to try to check an outbreak in the early stages by spraying or dusting. Copper fungicides are likely to be most effective.

A powdery mildew (*Oidium* sp.) was reported on leaves of St. Brigid anemone (*A. coronaria*) in Cornwall and Devon in 1937.

Anemone Rust (*Puccinia fusca* (Pers.) Wint. = *Tranzschelia fusca* (Pers.) Diet.)

This rust is common on the native wood anemone (*A. nemorosa*) in Denmark and Britain. It has only one spore stage, the teleutospore, which occurs in dark chocolate-brown pustules on the under-side of the leaves, especially towards their edges. The mycelium permeates the whole plant and overwinters in the rhizome. Affected plants are conspicuous because their leaves are taller and more erect than healthy ones and the leaf blades are smaller. The pustules may be so numerous as to resemble the sori of a fern leaf.

If it is desired to check the spread of the disease the infected plants should be dug out and destroyed.

Cluster Cup Rust (*Puccinia pruni-spinosae* Pers. = *Tranzschelia pruni-spinosae* (Pers.) Diet.)

This rust has its uredo- and teleutospore stages on plum leaves (see p. 194), and only the aecidial or cluster-cup stage occurs on anemones. This covers the whole under-surface of infected leaves with its bright orange cups and was formerly called *Aecidium punctatum* Pers. The upper surface of the infected leaves bears only the little brown or black dots made by the spermogonia.

Plum rust occurs mainly on *Anemone coronaria*, *A. fulgens* and *A. ranunculoides*, but *A. japonica* is said to be resistant (Ducomet, V., 1924).

Diseased anemones seldom flower and should, if possible, be destroyed, especially if there are plum trees in the vicinity.

Rowan Rust (*Ochropsora ariae* (Fuck.) Ramsb.)

The third rust of anemone has its alternate stage on the rowan (mountain ash) and related trees. These have small yellow spots on the leaves, bearing whitish pustules of uredospores or flesh-coloured teleutospores on the under-side. They occur especially on seedlings or on the lower part of older trees. Basidiospores are formed in autumn and are washed down by rain into the ground where they infect the buds on the rhizomes of wood anemone (*A. nemorosa*). The shoots arising next spring from the infected buds are taller, paler and more slender than healthy ones. Numerous spermogonia appear on the leaves and bracts, and later white cluster cups break out from the lower surfaces.

The mycelium persists from year to year in the rhizomes of the anemones which throw up diseased leaves each spring. This rust is frequent in the woods in Denmark and impossible to eradicate there.

Probably several different races of it exist, each restricted to some member of the *Pomaceae*, including the crab apple.

This rust seems to be rare in Britain.

Synchytrium anemones Woron.

Small dark red warts caused by this fungus sometimes occur on leaves, stems and even the flowers of wood anemone.

The fungus lives inside the individual cells of the epidermis causing them and the surrounding cells to become enormously enlarged, thus forming the warts.

The only known control is to rogue out diseased plants.

Leaf spots of *Anemone coronaria* caused by *Septoria anemones* Desm. var. *coronariae* Grove sometimes occur. Both flowers and corms may also be destroyed by the common grey mould *Botrytis cinerea*.

ANTIRRHINUM (Snapdragon)

Snapdragon Rust (*Puccinia antirrhini* Diet. & Holw.)

Symptoms. In severe outbreaks the rust occurs on leaves, calyxes, stems and capsules. As a rule the pustules are on the under-side of the leaves (Fig. 241) while the upper surface

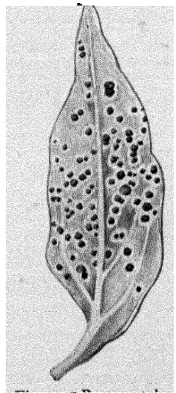


Fig. 241. Rust pustules on under-side of antirrhinum leaf.

is marked by corresponding light-green rather vague spots. The pustules start as small yellow pimples, generally round on the leaves and oblong on the stems. These soon burst, exposing the chocolate-coloured dusty mass of spores, surrounded by a yellow ring of leaf

tissue that has lost its green colour. In severe attacks the leaves die and the flowers do not develop or are weak. In Denmark uredospores are mainly found (Buchwald, N. F., 1934). In Germany Pape (1934) also found mainly uredospores, but teleutospores occurred on the stems. No cluster-cup stage is known.

History. This fungus has spread rapidly through Europe in the last two decades. It has probably been known in California since 1879 (Peltier, G. L., 1919); now it occurs throughout the United States, southern Canada and Bermuda. The first record in Europe came from France in 1931. It appeared in England in Kent in July 1933 and is now widespread also in Germany, Denmark and Sweden. In the North of England it occurs only sporadically and seems not to have established itself in Scotland.

Control. In 1934 the Royal Horticultural Society asked all snapdragon growers in England to root up and burn all their snapdragons, whether known to be diseased or not, before the 30th November. It was hoped thus to prevent overwintering of the spores but it is difficult to obtain general co-operation in such a scheme and in any case shrivelled infected leaves will be left on the ground or covered by the surface soil. Certainly the attempt failed (Green, D. E., 1934b).

Propagation by seed seems to give more resistant plants than propagation by cuttings. Apparently the infection is not seed-borne but it is inadvisable to save seed from diseased plants. If cuttings are required they should never come from rusted plants.

Snapdragons should not be grown on soil which carried rusted plants the preceding year.

As far as possible watering should be confined to the soil beneath the plants, avoiding wetting the leaves.

As the optimum temperature for germination of the rust spores is 50° F. it is advisable to keep the night temperature of houses where snapdragons are grown above this, in case the disease appears.

In England good control of rust has been secured by 6–8 sprayings with Burgundy mixture between 1st July and 1st October. 0.8% Bordeaux mixture gave rather inferior control.

In British Columbia two sprayings with 1½% Bordeaux mixture plus a spreader worked well. Copper sprays are preferable to others on flowering plants as they cause less spotting of the blooms.

In America sulphur dusting has been recommended in greenhouses if the day temperature can be kept up to 72° F. for 11–12 hours and the night temperature up to 60° F. for two days after the treatment.

Snapdragon varieties differ greatly in susceptibility to rust. It seems probable that a breeding programme for production of good resistant varieties would meet with considerable success (Emsweller, S. L., and Jones, H. A., 1934).

Shot-hole (*Heteropatella antirrhini* Buddin & Wakef.)

This disease starts as small round pale-green spots, less than ¼ inch across, on snapdragon leaves. Gradually the centre dries up, turns whitish and may drop out, leaving a " shot-hole." There may be a distinct purplish margin to the spot. In severe attacks spots develop also on the stems, which may be swollen and distorted (Cayley, D. M., 1920). Under moist conditions conidia of the causal fungus form a pale pinkish bloom on both surfaces of the spots. These were first called *Cercosporella antirrhini* Wakef. (*Kew Bull.* 1918, p. 233), but subsequently pycnidial fructifications were found on overwintered dead snapdragon stems. These were proved to belong to the same fungus and as they are regarded as a " higher " form of reproduction than the conidia the name was changed to *Heteropatella antirrhini*. If an ascus or " perfect " stage is discovered the name of the fungus will have to be changed again.

Wilt (*Myrothecium roridum* Tode ex Fr.)

This fungus causes rotting of the cortical tissues at the base of the stem, leading eventually to wilting and death of the plant. The decaying tissue becomes covered with a thin white mycelium, amongst which are numerous

small black fructifications. Under a lens these are seen to be disc-like with a distinct white rim. They bear conidia of the fungus *Myrothecium roridum* which causes a similar disease in several other ornamental plants, including lupin, viola, sweet sultan, godetia, matthiola, clarkia, petunia, gilia and callistephus (Brooks, F. T., 1945).

Leaf Spot and Stem Rot (*Phyllosticta antirrhini* Syd.)

Symptoms. This disease is well known in America and Germany, occurs from time to time in Britain and did serious damage at Vordingborg in Denmark in 1927. The leaf spots are yellowish brown with dark edges and may join together sufficiently to kill the leaves. Dotted over the spots are the tiny black pycnidia. Similar spots appear on the shoots and may girdle them so that all the part above dies. On young plants the stem may be attacked and girdled at the base and the whole plant killed. Spots also occur on flower stalks and capsules.

The fungus may overwinter on the dead plant parts and on fragments of the capsules among the seed.

Control. Only seed from healthy plants should be saved. If necessary the seed should receive disinfectant treatment (see p. 576). Cuttings should only be taken from healthy plants. Snapdragons should not be sown or planted again where diseased ones have grown. Too much moisture should be avoided and indoor plants should be kept well ventilated. Fungicidal sprays or dusts may also be used.

Downy Mildew (*Peronospora antirrhini* Schroet.)

In the spring of 1943 a severe outbreak of downy mildew occurred on snapdragons in a nursery near Copenhagen. The leaves, especially the young ones, were small and somewhat rolled inward and their under surface had a greyish coating of mould. The seedlings had been left too long before being transplanted, which may have rendered them more susceptible. Their growth was obviously checked by the fungus.

This downy mildew had been seen before in Denmark on the wild *Antirrhinum orontium* but not on the cultivated *A. majus*. It occurred on the latter in Ireland in 1936, in England in 1937 and subsequently in Wales in 1940 and in Sweden in 1943.

In Denmark the disease appeared first on the variety Fakkel then spread to other varieties, but neither in 1943 nor in 1944 was it found in other nurseries.

Two sprays with Bordeaux mixture seem to have checked the disease quite satisfactorily.

An unidentified powdery mildew has occurred occasionally on snapdragons in England (Moore, W. C., 1943).

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary.)

Shoots and flower heads rot and become covered by the white mycelium of the fungus, with which are the large white, then black, sclerotia. It is thought that infection takes place through the style, to which ascospores are carried by the wind or on the bodies of insects, especially honeybees and bumble bees (see *Sclerotinia* rot, p. 77).

Foot Rot (*Phytophthora cactorum* (Leb. & Cohn.) Schroet.)

This is a serious disease of snapdragon in California and has occasionally occurred in England. The fungus is present in Danish soil but has not yet been observed there attacking antirrhinums. Foot rot has been fully investigated by Harris (1934).

Symptoms. At soil level or a little higher a light yellow, translucent area appears on the stem. As the damage spreads the older part of the lesion turns brown and finally almost black. As a rule the patch spreads until it has girdled the stem and extends a considerable way up it. The soft cortical tissues decay and only the woody core remains. In early stages of the attack the plant wilts in the middle of the day but recovers at night; later on it wilts permanently, turns brown and dries up.

Varieties differ greatly in susceptibility to foot rot.

Cause. Bacteria and the mould *Cephalosporium acremonium* Corda have been isolated from the diseased tissue but in all cases *Phytophthora cactorum* or some other species of *Phytophthora* has been found closely associated with it. *Phytophthora* species are common causes of foot rot and root rot in many plants.

Control. It appeared that the plants were infected in the transplanting boxes, and when steam-sterilized soil was used the disease no longer appeared. When it does break out its further development may be checked by watering with Cheshunt mixture.

AQUILEGIA (Columbine)

Leaf Spots

Ascochyta aquilegiae Roum., *Gloeosporium aquilegiae* Thüm. and *Marssonina aquilegiae* Rostr. have been described causing leaf spots on columbine. The latter is said to be fairly general in Denmark. Grove (1937) regards these names as all applying to the same fungus which he calls *Actinonema aquilegiae* Grove. It causes irregular or rounded whitish or grey-brown spots ¼–¾ inch across, with a narrow brown border. The spots are covered with a whitish bloom caused by radiating fibres on which are seated the tiny brown dot-like fructifications of the fungus. As general control measures one may recommend spraying or dusting with a fungicide whenever the spots seem to be spreading to an unusual degree.

Leaf Blotch (*Haplobasidium pavoninum* Von Höhnel)

In this disease conspicuous, irregular, buff-coloured blotches, ¼–1 inch across, appear on the leaves, mostly spreading inward from the margins. Often there is a broad purplish band between dead and living tissue but sometimes a whole leaflet is killed. A fine short grey mould, formed by conidiophores and conidia of the fungus *Haplobasidium pavoninum*, occurs in lines along the smaller leaf veins, especially on the lower surface. The disease was first described in Austria and has since been seen in Spain and Cornwall (Moore, W. C., 1939a).

Rust (*Puccinia agrostidis* Plowr.)

In the spring small patches of orange cluster cups may be found on the under-side of columbine leaves. This is the aecidial stage of a rust which has its uredo- and teleutospores on the common bent grasses, *Agrostis stolonifera* and *A. tenuis*, on which it overwinters. The infected columbine leaves may be twisted or distorted.

Mildew (*Erysiphe polygoni* DC. = *E. aquilegiae* DC.)

Columbine leaves are often seen coated with a white powdery mildew. For control measures see mildew, p. 63.

Mosaic (*Cucumis* Virus 1)

This virus, common in cucumbers and many ornamental plants may cause pale-coloured veins, mottled leaves and stunted growth in columbine. Flowering is checked (see cucumber, p. 278).

ARABIS

White Blister (*Cystopus candidus* (Pers. ex Chev.) Lév.)

This fungus can attack any part above ground, in which it causes thickening and malformation. The spores are formed in white pustules which break open to expose the chalk-white spore powder. It is common also on wild species of *Arabis* (rock cress) and other cruciferous weeds (see horseradish, p. 285).

Mosaic and Ring Spot

Cabbage ring spot virus may cause mottling of *Arabis* leaves (see p. 258). K. M. Smith (1944) has described another virus disease of *Arabis hirsuta* in which dark-green lines and rings appear on the leaves.

ARMERIA (Thrift, Sea Pink)

Rust (*Uromyces armeriae* Lév.)

All stages of this rust, aecidia, uredo- and teleutospore pustules, occur on the leaves and flower stalks of sea pink. The uredospores are

the stage most often seen and occur in light-brown pustules on small purplish spots on either side of the leaf (see rust, p. 74).

Leaf Spot (*Septoria armeriae* Allesch.)

This was described originally on sea pink in Greenland, but what is probably the same fungus has been found on leaf spots of *Armeria cephalotes* and *A. plantaginea* in England (Moore, W. C., 1948).

ARUM LILY

See *Zantedeschia*, p. 562.

ASH

See *Fraxinus*, p. 460.

ASPARAGUS

Yellowing

Yellow colouring in the tops of *Asparagus plumosus* and *A. sprengeri* is often due to over-watering during the short days of winter. This weakens the roots and causes the tops to lose their green colour.

Drought may have a somewhat similar effect but then the tops become thin by shedding of the needles. An excessive amount of lime in the soil may also cause yellowing.

Thrips attack on *Asparagus sprengeri* not only causes light silvery spots on the green tissue but makes it greatly deformed. This helps to distinguish thrips injury from that due to growing conditions.

Rust

See p. 232.

Crown Gall

See p. 81.

ASPIDISTRA

Leaf Spots

Spots of different shapes on aspidistra leaves are often associated with various fungi, species of *Phyllosticta*, *Ascochyta* and *Colletotrichum*, but the primary cause is usually unfavourable growing conditions. The plants should not stand in strong sunlight nor permanently in deep shade. They can tolerate being kept well away from the window but the darkest corner of a room may be too dark even for them. If they are watered too much the roots are damaged and the tops turn yellow.

When fungus leaf spots occur one may try spraying with a copper fungicide.

ASTER (Michaelmas Daisy)

Mildew (*Erysiphe cichoracearum* DC.)

Many varieties of Michaelmas daisy are often severely attacked by mildew, which forms a white floury coating over leaves and shoots (see Fig. 32).

The most susceptible varieties should not be grown (see mildew, p. 63).

Verticillium Wilt (*Verticillium Vilmorinii* (Guég.) Westerd. & Van Luijk)

Symptoms. Wilt of Michaelmas daisies has been observed in England, Holland and Norway. In England Dowson (1923) noted that at the end of May the leaves began to hang down, became lemon or orange-coloured and finally turned brown and dried up. Early formation and continued growth of suckers is another characteristic sign of the disease. When attacked early the plant does not flower. The mycelium of the fungus occurs particularly in the wood of the stem, where it tends to block the water-conducting vessels with a rubbery substance.

Cause. Dowson described the fungus he found as *Cephalosporium asteris* Dows. Later Westerdijk and Van Luijk (1924) reinvestigated the disease and found the fungus was the same as the earlier-described *Acrostalagmus Vilmorinii* Guég The genera *Cephalosporium*, *Acrostalagmus* and *Verticillium* are not very clearly distinguished from one another.

Control. According to Dowson suckers may give healthy plants. Preston (1938) has shown that top cuttings from diseased plants remain healthy. Hence diseased plants should be dug up and burned and replaced by cuttings taken in this way.

ASTER, CHINA

See *Callistephus*, p. 427.

ATROPA BELLADONNA (Deadly Nightshade)

Foot Rot (*Phytophthora erythroseptica* var. *atropae* N. L. Alcock)

The base of the stem is attacked in mature plants, causing a foot rot followed by wilting. The fungus may also cause damping off of seedlings (Alcock, N. L., 1926a).

Control. See tomato, p. 375.

Virus Disease

K. M. Smith (1943) has described a virus disease of this crop in which there was a patchy mottle of the leaves, associated with necrotic spots and severe necrosis of the stems. The virus also attacks henbane (*Hyoscyamus niger*), in which it causes a rather faint mottle. No insect vector is known.

This disease was apparently rather prevalent in Surrey from 1941 onwards.

AZALEA

See also *Rhododendron*, p. 517.

Leaf Scorch (*Septoria azaleae* Vogl.)

Symptoms. Brown dead patches develop on the leaves, starting at the tip, at the margin or as spots on the surface. They may be bounded by the veins and therefore be angular. The diseased parts soon become very dark brown and the pycnidia of the fungus occur on them as small black dots on both surfaces of the leaf. As a rule the leaf falls off when about half its area has become affected. The attack starts on the older leaves so the thinning of the foliage starts at the bottom. This shedding of the leaves not only spoils the appearance of the plants but may weaken them sufficiently to prevent flowering. In some cases the buds scarcely open; in others, like the deep-rose-coloured Madame Petrick, if the flowers do open, instead of being deep rose they are dirty white or pale pink. Madame Petrick is one of the most susceptible varieties but others are also attacked.

Damp and other unfavourable growing conditions can also cause leaf fall.

Control. Plants which are apparently healthy when forcing starts may become severely infected before they bloom. Probably they were slightly infected and the disease spread both in the leaves where it was already present and to others.

If the slightest sign of the disease is seen one should at once apply a fungicidal spray or dust and repeat several times at intervals of a few weeks.

If the nurseryman raises his own azaleas he should spray or dust them regularly throughout the summer for it is better to avoid the disease altogether than to have to check it during forcing, when spotting the leaves with chemicals is to be avoided if possible.

Bordeaux mixture and other copper fungicides are recommended but Burgundy mixture has caused a great deal of spray damage in Germany.

According to Pape (1939) the following varieties are particularly susceptible to this disease: Madame Petrick, Petrick Superbia, Weisse Petrick, Madame August van Damme, Schepens Kerstperel, Roosevelt, Paul Schäme, Theodore Findeisen and John Haerens.

Azalea Wilt (*Cylindrocarpon radicicola* Wollenw.)

Symptoms. Withering of azalea plants was first noticed in Denmark in February 1927. The plants looked as though short of water but this was not the case and their condition was not improved when the pots were stood in water long enough thoroughly to saturate the soil. In some cases the plants made no growth for some time before they became limp and withered; in others the withering appeared suddenly without any preliminary indication of disease. The foliage was often poor but sometimes was normal. As a rule the disease appeared when the flower buds began to show colour, though some plants withered just before the buds burst, and one not until it was in bloom. The disease is centred in the main stem which turns brown to a greater or lesser extent near soil level. It is easily noticed when the bark is removed (Fig. 242). It is mostly the stock that is affected but the browning may also extend some distance into the scion.

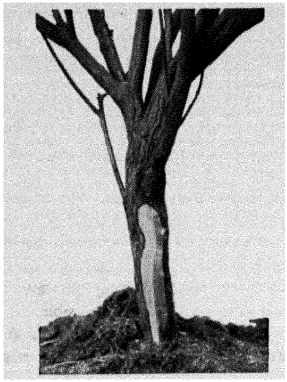


Fig. 242. Azalea wilt, discoloured wood at base of stem (*S. p. F.*).

Cause and distribution. In 1927 and on several subsequent occasions we have isolated from the brown wood a fungus since identified with *Cylindrocarpon radicicola*, described by Wollenweber in 1928. This is common on the roots of many woody and herbaceous plants and can also cause rotting of apples and other fruit if introduced through wounds (see cyclamen, p. 444; lily, p. 489; narcissus, p. 497).

In 1927 we could find no mention of the disease in any foreign literature on plant pathology. We then made enquiries of five foreign plant pathological institutes but the disease was known only at that time in Belgium and had only recently been seen by them. In 1928 the English writer Miles, in an article on azalea cultivation in Belgium, spoke of a very injurious disease of azaleas exactly corresponding to ours and ascribed it to a species of *Ramularia*. That was not surprising as prior to Wollenweber's work the species of *Cylindrocarpon* were usually ascribed to *Ramularia*. The account of the extensive damage caused was somewhat surprising but it is evident that the Belgian azalea growers knew of the disease long before it came to the attention of their plant pathologists. In 1933 the Belgian azalea growers communicated with us about the disease so it was well known to them then.

Almost every year since 1927 we have seen plants attacked by the disease or have received reports of extensive loss caused by it.

When the plants that develop wilt have only been in the nursery since the spring it is highly probable they were infected when received, but that the fungus only obtained sufficient hold of them to impede the water supply after forcing had begun. As the fungus is very common in soils everywhere and occurs on the roots of so many plants, it is almost impossible to prove the plants did not become infected after arrival at the nursery. The first year we saw the disease in Denmark the circumstantial evidence left us in no doubt it came with the plants. We examined plants from four Copenhagen nurseries and one provincial nursery and of these the plants in two of the Copenhagen nurseries and the provincial nursery came from the same Belgian firm. One of the nurseries also had a consignment of plants from another Belgian firm and these were all healthy.

Control. Miles wrote that in Belgium plants grafted on *Azalea concinna* are more susceptible to the disease than those on *A. phoenicea*. *A. concinna* has stronger growth than *A. phoenicea*, but the latter is more used as a stock because of its greater disease resistance and for plants where slow development is desirable, as in those for collections or shows. Miles also noted that too deep planting and overwatering, as well as great fluctuations in temperature during the growing period, seem to make the disease worse. No direct control measures are known.

Leaf Fall and Dieback

Shedding of the leaves may occur very extensively in azalea and may be caused by the leaf scorch fungus (see p. 417). Other fungi may also occur on azaleas and rhododendrons, notably *Pestalotia macrotricha* Kleb. and *Phyllosticta maxima* Ell. & Everh., with its perfect state *Venturia rhododendri* Tengw. *Pestalotia* has been shown to be able to attack leaves and shoots already weakened by some

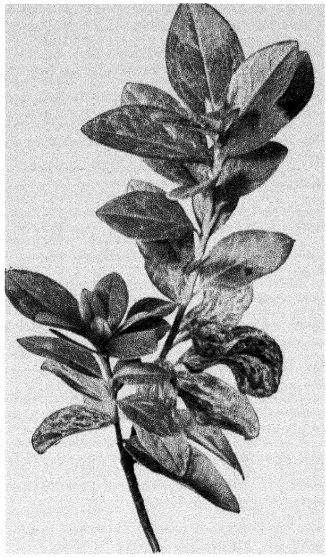


Fig. 243. Azalea plant in which the lower overwintered leaves are spotted with white.

other agency. *Phyllosticta* can attack healthy leaves but seems to play only an insignificant role in practice.

Azalea leaves produced during cold conditions may be spotted with white (Fig. 243).

Lihnel, in Sweden, has studied the form of leaf fall and dieback seen also in Denmark on imported azaleas. The leaves fall off, sometimes quite suddenly and very extensively. It is the leaves on the innermost branches that fall first. Their appearance varies greatly: they may be green and fairly turgid when they drop and only gradually turn brown with lighter or darker spots, but this discolouration may often begin before they fall. In some varieties, for example Paul Schäme, Apollo, etc., leaf fall is often associated with an extensive yellowing of the leaves. In plants of Mme. Petrick the leaves often turn dark-green or brownish purple. Black leaf tips may also occur.

After the leaves have fallen the branches begin to die back from the tips. This may occur all over the crown or only on individual branches.

Under certain conditions drought, frost or overwatering may cause similar symptoms without the help of the fungi mentioned above. In imported azaleas, however, Lihnel thinks the chief cause is a form of asphyxiation during transit. The plants are so tightly packed that no fresh air can reach them and as they are unable to assimilate carbon dioxide under these conditions it accumulates to an excessive amount, as a result of respiration by the plants and the various soil organisms accompanying them.

Lihnel made experiments in Sweden which confirmed his theory even though he could not make experiments on plants in transit.

As many plants do not suffer in transit in spite of close packing it is evident that conditions must vary considerably, especially time involved, packing materials and temperature on the journey. Even if the actual journey from Belgium was a quick one the plants may have been packed some time in advance so that the total period of their close confinement may be too long, a circumstance unknown to the consignee. The risk of poisoning is greatest when it is warm; at 40–45° F. there is very little risk. Even if the crates or baskets used have been provided with ventilators they are often stuffed so full that no ventilation occurs.

Brown Buds

It happens not infrequently that various buds on azaleas turn brown, stop growing and do not flower. If the roots of such plants are examined they are nearly always found to be much damaged.

When imported azaleas arrive they are often found to have become completely dried out on the journey, so when one attempts to water them the water escapes between clumps of dry soil and the pot. It is very difficult to expel the air from such completely dried-out soil. Roots often suffer permanent injury from such extreme drought.

To minimize the damage the pots should be carefully examined as soon as they are received and the very dry plants should be

stood for some hours with the whole pot immersed in water.

Bad roots and the consequent shrivelled buds may perhaps sometimes result from other causes but the matter has not been more fully investigated. There is no doubt drying out is a frequent cause.

Gall (*Exobasidium vaccinii* (Fuck.) Woron = *E. japonicum* Shir.)

Symptoms and importance. Whitish nodules are not infrequently seen on greenhouse azaleas towards flowering time. These are leaves attacked by the fungus *E. vaccinii*. First the leaves become light-green, thick and juicy, then the malformation extends and the fungus appears all over the surface of the affected parts, making them chalky-white and floury (Fig. 244). Sometimes only part of a leaf is affected, sometimes a whole leaf and sometimes several or all the leaves of a shoot may be attacked and the terminal bud as well. The leaves may be larger than normal in all directions but in some varieties, e.g. Hexe, the leaf area may be increased while the thickness remains about normal. The deformed leaf surface may, however, be so blistered and curled as to look like a nodule. Whole flowers or individual petals may also be attacked. If the diseased parts are left they gradually turn brown, shrivel and begin to rot.

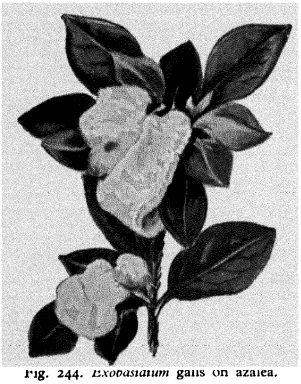


Fig. 244. *Exobasidium* galls on azalea.

The thickenings are due to the fungus stimulating the host cells to multiply; their chlorophyll disappears.

According to Pape (1939) the variety Prof. Wolters is particularly susceptible but Ernest Eeckhoute, Jules Roehrs, Mme. John Haerens, Ernest Thiers, Hexe, Apollo, Niobe and Deutsche Perle are also quite susceptible. The following are less so: Mme. Van der Cruyssen, Eclaireur, Paul Schäme, Temperance, Simon Mardner, Raphael, Präsident de Kerchove, Mme. Petrick and Vervaeneana.

Rhododendron ferrugineum, R. hirsutum, R. wilsoniae and other species are attacked by a race of the same fungus, sometimes regarded as a distinct species under the name *Exobasidium rhododendri* Cram. Leaves, shoot tips and sometimes the flowers may be attacked and are often turned into round galls which may be crowded together. Especially out of doors, for example in the Alps, the galls become red by development of the colouring matter anthocyanin in the cells, hence the names " Alpine pink apples " or " sap apples."

In North America *Exobasidium azaleae* Peck has been described as turning the shoot tips and leaves of *Azalea nudiflorum* into light-green or bluish fleshy round galls 1–2 inches across. It is not certain if this is the same as *E. vaccinii* of Europe.

Laubert (1925) listed about 50 species of *Exobasidium* parasitic on *Ericaceae* and other plants but the distinction between many of the species is uncertain. In England the azalea and rhododendron gall fungi are generally regarded as races of the common *E. vaccinii* found on the native whortleberry and cowberry. On the Continent they are often regarded as distinct species.

The gall on *Rhododendron hirsutum* was found in Denmark in 1893 in the garden of the Agricultural College. Galls on azaleas were reported from Belgium, Holland, England, Italy and Germany by 1908. In 1916 Lind, writing in a gardening magazine, said the disease was probably well known to growers of azalea and rhododendron. Now it is almost always found where azaleas are grown. Sometimes only single galled leaves occur in a whole

consignment and the disease is of no importance. On other occasions it is prevalent enough to deform many leaves and spoil the commercial value of the plants.

Control. The fungus has a long incubation period and it is extremely probable that the galls found in Denmark occur on leaves already infected when the azaleas were imported. The deformed leaves should be picked off so that they do not spoil the appearance of the plants and if possible they should be removed while still green. Once they turn white they are producing spores which will infect and cause galls on new leaves before flowering. Plants to be used for propagation should be quite free from the disease and all galls in the same nursery should be removed before they turn white and shed spores.

BALSAM

See *Impatiens*, p. 477.

BEECH

See *Fagus*, p. 456.

BEGONIA

Bacterial Wilt (*Xanthomonas begoniae* (Takimoto) Dowson)

The first symptoms are minute glassy spots on the under-sides of the leaves, especially near the margins and in lines running parallel to the main veins.

These increase in size and appear as pale watersoaked areas which may join up and cover the greater part of a leaf. Ultimately they turn brown and die. Meanwhile the smaller and larger veins become dark-brown or black, and similarly coloured rotting areas develop on leaf stalks and stems. In some varieties, like Optima and J. Mensing, the stems may be attacked first and the whole plant wilts. If the stem of a diseased plant or midrib of an affected leaf is cut across a sticky yellow slime oozes out, composed of the bacteria which cause the disease. The organism was found attacking Gloire de Lorraine in Denmark and occurs on many winter-flowering *Begonia socotrana* hybrids in England, though varieties differ greatly in susceptibility. The bacterium found by Buchwald in Denmark and that isolated by Dowson in England (1938) are the same, but in Holland Wieringa found a slightly different organism, *Phytomonas flava Begoniae* Wieringa, responsible for a very similar disease. Gram and Weber suggest that the leaf spot and vascular wilt diseases are distinct but Dowson found *X. begoniae* causing leaf spot without any infection of the vascular system in *Begonia tuberosa* in Portugal. It seems therefore that the different expression of symptoms depends mainly on the variety of begonia attacked. An entirely different organism, *Bacterium flavozonatum* McCulloch (1937), causes small leaf spots on begonia in North America. It causes scattered small spots, mostly less than $\frac{1}{4}$ inch across, at first pale green, then brown with a translucent yellow halo.

Control. Severely infected plants should be removed and burnt. Those only slightly affected may be saved by cutting out the diseased parts. If this is done the knife should be dipped in Lysol or other disinfectant after each cut or the bacterial slime will only be transferred to other plants on the knife or hands. Cuttings should only be taken from healthy plants and handled only with disinfected hands and tools. They should be planted in sterilized soil and kept well away from infected plants, preferably in another house. Pots or boxes used for them should also be disinfected. Water tanks may become contaminated if dead begonia leaves or other debris falls into them. Hence water from a tank in an infected house should not be used for healthy plants or cuttings, nor should watering cans be taken from an infected house to a healthy one. High atmospheric humidity favours the disease. Houses should be kept as dry as possible and well ventilated. Wounding the plants during tying up or watering should be avoided.

It is possible that tuberous begonias may have the disease in a mild leaf-spot form, easily overlooked, and may act as a source of infection to the susceptible winter-flowering hybrids.

Mildew (*Oidium begoniae* Putt.)

Mildew on begonia has been known in Denmark for many years but was first described by Buchwald in 1930 on the hybrid Gloire de Lorraine. It is now not uncommon and occurs on other varieties as well. It was found on Baardse's Wonder in England in 1938 (Moore, W. C., 1943).

Symptoms. The disease first appears as small indistinct brown spots, which gradually increase in size and become covered by a white powdery layer of mycelium and conidia. We have seen dark perithecia on the mycelium but they have never been ripe enough to indicate the systematic position of the fungus. In Sweden Hammarlund found a single perithecia containing asci and suggested the name *Erysiphe begoniae* Hammarlund but his material was too scanty to be adequately described.

Leaves, petioles and flower stalks may all be affected. The spots may join up to form large mildewed patches and the disease may be quite virulent during the winter months.

Control. Diseased plants should not be used for propagation (see also mildew, p. 63).

Brown Root Rot (*Thielaviopsis basicola* (Berk. & Br.) Ferraris)

Roots of young and old begonia plants, both in greenhouses and out of doors, may become so brown and decayed that only the woody cylinder remains. In tuberous begonias the tubers also may be attacked. Affected plants appear pale and deformed and may ultimately die.

The diseased roots contain the fungus *Thielaviopsis basicola* which lives commonly on decaying plant remains in the soil and is widespread in leaf mould. It may attack roots of many different plants, e.g. cyclamen, primula and viola, and is well known as a serious parasite of tobacco in warm climates. Usually it attacks plants that are not under the most favourable conditions. It is favoured by damp, compact soil.

Plants should not be set in soil where the disease has previously occurred unless the soil has been disinfected or renewed, e.g. groups of tuberous begonias in outdoor beds (see p. 577).

Care should be taken that the leaf mould is well rotted and the soil not too heavily pressed round the roots, also that it is not kept too moist for the plants. They should be kept growing steadily and not be weakened by careless treatment.

Foot Rot (*Corticium solani* (Prill. & Delacr.) Bourd. & Galz.)

Begonia plants not infrequently become brown at the collar and a little way up the stem. The trouble may be due to attacks by *Corticium solani* that sends fine brown hyphae over the diseased tissue and through the soil. From brown stems we have also isolated *Verticillium* sp., *Fusarium* sp., *Botrytis*, etc. It is often difficult to decide what has been the primary cause of the attack as it is not necessarily the fungus most abundant at the time of examination.

By watering with a disinfectant solution it is possible to check spread of the disease to other plants and possibly to check its spread on those already diseased.

One of the mercury-containing fungicides may be used or a solution of 1 oz. of corrosive sublimate and 9 oz. of chile saltpetre in 11 gallons of water. The disinfectant solution should be applied to the base of the plant and not on the leaves. After watering the plants should be lightly sprayed with clean water. For grey mould on the stalks see below.

Grey Mould (*Botrytis cinerea* Fr.)

If begonias are kept too damp they are easily attacked by grey mould. Large brown spots appear on the leaves, especially on the variety Rex, but others may also be attacked. Stems and leaf stalks may also be rotted. The infected tissue always turns brown, and gradually, under damp conditions, becomes covered with the hairy grey mould.

If the disease is prevalent it is a sign that the plants are kept too moist (overwatering, lack of ventilation, drip), have unhealthy roots, or the whole plant is growing poorly.

When these faults in cultivation have been corrected development of the fungus is checked (see further under grey mould, p. 59).

Propagation Fungus (*Moniliopsis Aderholdi* Ruhl.)

Begonia propagation is often difficult because the cuttings are attacked by this fungus, which makes them brown and rotten just above and below the level of the substance in which they have been planted. In leaf cuttings the attack spreads from the base of the stalk on to the leaf surface (Fig. 245). It seems to be

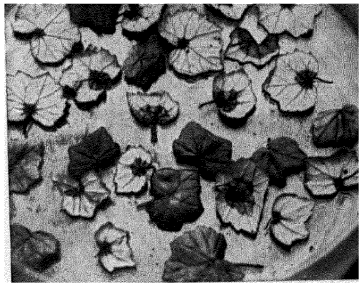


Fig. 245. Propagation fungus attacking begonia leaf cuttings (*Photo Pape*).

more prevalent on leaf cuttings than on stem cuttings (Pape, H., 1933) (see further, p. 69).

Ring Spot (*Lycopersicum* Virus 3)

The virus of tomato spotted wilt often causes yellowish or brown rings up to about $\frac{1}{2}$ inch across on begonia leaves. They cause little damage to the begonias and may disappear at times, but a watch should be kept for them as infected plants are a menace to tomatoes in the vicinity.

Abnormal Leaves

Over a period of years a considerable amount of information has been accumulated regarding the causes of various abnormalities in begonia. Thus recurved leaf surfaces can be ascribed to excessive heat. Red-edged leaves with arrested growth are caused by too strong sunlight (possibly heat as well), and improve when given shade.

If the soil is too rich in lime the leaves turn pale. For small plants the soil reaction should not exceed pH 6.5; if this occurs and damage results, one may try watering with a solution of iron and manganese salts (see p. 31). Dirty green or brown leaf edges may be the result of unbalanced nitrogenous manuring; possibly the real cause is potassium deficiency (see p. 42).

Soft, easily snapped leaf stalks may result from ammonia scald arising from too fresh dung.

BERBERIS (Barberry)

See also *Mahonia*, p. 492.

Bacterial Leaf Spot (*Pseudomonas berberidis* Thornb. & And.)

Thornberry and Anderson (1931) described a bacterial disease of *Berberis thunbergi* in North America. Cross-inoculations showed that *B. vulgaris*, the common barberry, was also susceptible.

In 1933 the disease was found on *B. vulgaris*, *B. vulgaris* var. *atropurpurea*, *B. canadensis*, *B. crataegina* and *B. chinensis* in the garden of the Danish Agricultural High School. Round or angular spots, at first translucent then purplish red-brown, were scattered about the leaves (Buchwald).

Rusts of Barberry

Two host-changing cereal and grass rusts have their cluster cup (aecidial) stage on species of barberry.

Black rust of cereals and grasses, *Puccinia graminis* Pers. (see p. 74), causes thickened bright orange spots on the leaves, the lower surface of which is covered by the small aecidial cups. It is common on *B. vulgaris* but also attacks *B. chinensis*, *B. canadensis* and many other species of barberry. On the other hand *B. thunbergi* and most evergreen species are resistant and hence are not a menace to cereal crops.

A closely related rust, *Puccinia arrhenateri* (Kleb.) Erikss., apparently infects only tall oat grass in its uredo and teleutospore stages

and in its aecidial state causes small witches' brooms on barberry. These have deformed leaves covered on the under-side by a layer of crowded, tubular cluster cups, the spores from which infect tall oat grass (Eriksson, J., 1896). The rust can, however, persist for some time on barberries alone, as the mycelium overwinters in the witches' broom.

Eradication of barberries is easiest after leaf fall as the red clusters of berries then make the bushes easy to find. In digging them out it is difficult not to leave roots or small shoots behind to make further growth. It is safer to kill the bushes by spreading cooking salt over the ground beneath the branches, packing it well round the individual stems. Small bushes can be killed with 2 lb. of salt. Those with a diameter of 12 inches at soil level need 11 lb. To kill scrubby growth one should allow 6–8 lb. of salt per square yard. After the salt has been applied it may be covered with a little soil to prevent it being scattered by animals.

BETULA (Birch)

Rust (*Melampsoridium betulinum* (Pers.) Kleb.)

Common birch seedlings in nurseries are often severely attacked by rust. The yellow pustules of uredospores may be so crowded as to cover the lower surface of the leaves. *Betula verrucosa* is less susceptible, but this and other species of birch are sometimes attacked. Diseased plants may suffer severely from the premature death of their leaves.

In woods the disease is common in young plantations and on shoots arising from old stumps but is less destructive than in nurseries. Brown scab-like patches of teleutospores form within the tissues of the fallen overwintering leaves, and on these the basidiospores are produced in spring. These infect larch and cause a small blister-like aecidial stage on the needles. The larch stage, however, has never been seen in Denmark and seldom in Britain, where birch is native and larch is not. Attacks on birch usually originate from rust that has survived the winter in the buds of young birches (Liro, J I., 1907).

In nurseries birch rust may be controlled by spraying with Bordeaux mixture 8: 4: 50 when the buds burst and once or several times later. Dusting with sulphur from the time the buds break and every fortnight after till the middle of September also has a good effect.

Witches' Broom (*Taphrina betulina* Rostr.)

Large trees of the common birch often carry a number of conspicuous tufts of branches formed by slender twigs growing out in all directions. In the spring these twigs bear pale leaves covered with asci. The annual growth and buds of the witches' broom are permeated with mycelium of the fungus that overwinters there.

If the witches' brooms grow too big or too numerous they may be cut off; it will be some years before new ones form.

The witches' broom of common birch can be found on other species of *Betula* but Rostrup observed that when *B. pubescens* and *B. pendula* (=*B. verrucosa*) were growing together only the former had witches' brooms. He concluded that brooms on *B. pendula* were caused by a distinct fungus, *T. turgida* (Sadeb.) Giesenh. (Rostrup 1896). Recently, however, Mix (1949) has shown that *T. betulina* and *T. turgida* are indistinguishable. Witches' broom of birch may also be caused by gall mites.

Heart Rot

Birch trees throughout Britain are commonly attacked by the bracket fungus *Polyporus betulinus*, which causes the wood to decay until the tree dies or its trunk becomes so weak as to be snapped in a gale. The flat bracket-like fructifications, buff-coloured and smooth above, white at the margin and underneath, are a common sight on old birch trees. In the highlands of Scotland another fungus, *Fomes fomentarius*, destroys the wood of birches. It has hard, dark grey, hoof-shaped fructifications with a flat porous undersurface. On the Continent, but not in Britain, a race of this fungus is a common cause of decay in beech trees.

BLUEBELL

See *Scilla*, p. 531.

BROOM

See *Cytisus*, p. 444.

BUXUS (Box)

Various fungi may be found on leaves and branches of box but there is some disagreement about their relationship and importance.

Rostrup (1902) described *Pseudonectria* (*Nectriella*) *Rousseliana* (Mont.) Wollenw. thus: it parasitizes only box and often causes dieback of the branches. All leaves on a branch at the same time take on a pale-green colour and later become yellow and shrivelled. The lower surface of affected leaves is covered by a pink layer of small oblong conidia. Later flesh-coloured pustules of spindle-shaped spores also appear on the under-side of the leaves. This is the imperfect state, called *Volutella buxi* (Cda.) Berk., and finally, after leaf fall, larger globose perithecia appear, at first reddish yellow, later of a greenish hue. The fungus is found on clipped box which it may entirely destroy. Apparently it penetrates through wounds, of which there are plenty owing to the clipping. Diseased branches should be removed and burnt at once.

B. O. Dodge (1944) has shown that the fine white mould *Verticillium buxi* (Schm. ex Fr.) Awd. & Fleisch., which sometimes occurs on the under-side of dead or dying box leaves, is a distinct fungus, and not a stage in the life history of *P. rousseliana*, as has been thought. The latter can infect living box leaves, *V. buxi* apparently cannot. Weiss (1940) considers that neither of these fungi is an active parasite but that they attack boxwood damaged by freezing of unhardened shoots or unseasonably active cambium.

The only control measure recommended is to cut out the damaged twigs and branches, and shake off the dead leaves, collect and burn them. If spraying is to be attempted it should be with Bordeaux mixture 8:8:100 or lime sulphur 1:50 in spring before the growth of new foliage is complete.

According to Weiss poor colour, weak growth and premature dropping of old leaves in box are usually associated with failure to become well rooted after transplanting or with the bush being grown on hard infertile or badly drained soil or with the root being too deeply covered.

Guignardia buxi Desm. was recorded causing leaf fall of box in Sjaelland, Denmark, in 1931. It causes yellow spots on the under-side of the leaves, which crack open exposing the dot-like fructifications of the fungus. According to Juel (1926) *Macrophoma mirbelii* V. & V. is the worst fungus parasite of box in Sweden. It forms black pycnidia on fading and dead leaves and twigs and is common in Britain but usually regarded as of little importance.

Rust (*Puccinia buxi* DC.)

This rust has only one spore form, the teleutospore. The young leaves are infected in spring and during summer the infected spots become thickened. Large dark-brown or purplish-brown spore pustules break out from them on both sides of the leaf from September onwards. The disease is common but of little importance.

CACTI

Glassiness

In the softer kinds of cactus dark-green translucent spots may appear which from pale or dark green ultimately turn black. In virulent cases the parts of the plant above the affected zone die but in milder cases cork forms on the wounds. The diseased tissue cracks and forms sunken discoloured places. The disease is frequently followed by bacteria which cause the affected tissue to fall to pieces.

An investigation of the diseased tissue by Ulbrich (1921) showed it to consist of abnormally long cells filled with glucose and poor in starch. Starch is completely lacking in the most severely affected plants, while healthy cells contain much starch.

Glassiness attacks cacti standing in very moist warm air, as when they are grown amongst other tropical plants. The cacti recover when removed to a drier, well-ventilated place.

Dropping of Flower Buds

It quite often happens that cacti, especially Christmas cactus, lose their buds. Many different conditions may bring this about, e.g. overwatering, drought, great fluctuations in temperature, draughts or damage to the roots by too strong manure water. All these faults should be avoided by the grower.

Corkiness and Œdema

Corkiness appears in irregular patches with a corky or rusty appearance, in which ultimately sunken places or even holes may arise. The first signs of the disease are pale yellow-green spots, often somewhat translucent, which are easily overlooked. Corkiness may become bad enough to destroy the branches and even in mild cases it may reduce flowering. The outer layers of tissue, comprising several layers of cells, become corky and small wart-like outgrowths appear (Fig. 246). The



Fig. 246. Corking of opuntia stem.

outermost cork cells dry and the skin cracks. In milder attacks the spots may not crack but retain a smooth, greyish-white surface.

According to Ulbrich (1921) and others the disease is caused by too much moisture in the air and is made worse by shading. Though we think this is often the case we have also seen cacti in rooms where the air was not particularly damp but corky spots nevertheless appeared. Hence we think the condition may arise under other unfavourable growing conditions as well. Böhme thinks they appear as a result of unfavourable nutritional conditions and that similar spots may arise if the plants are suddenly exposed to strong sunshine. Particularly sensitive to sun scald, as he calls it, are cacti taken from shady places or those newly replanted, with poor root systems or growing in soil easily dried out.

Excessive atmospheric humidity and strong sunlight are to be avoided as well as sudden changes in growing conditions and great changes in nutrition.

Basal Rot (*Phytophthora* spp. etc.)

Cuttings may rot at the base before they have struck root and older cacti may rot at the collar. Probably the commonest cause of the trouble is *Phytophthora cactorum* (Leb. & Cohn) Schroet. but other fungi may also be concerned such as *Pythium aphanidermatum* (Eds.) Fitz., *Botrytis* sp., *Fusarium* spp. and *Sclerotium cacticola* Beyma.

Possibly soaking the lower ½–1 inch of the cuttings for 24 hours in potassium permanganate solution may do good; ½ oz. in 1–3 gallons of water may be tried. Striking of roots is often encouraged by such treatment and the most sensitive period is shortened. Treatment with root hormones may also be tried. Clean or disinfected soil should be used for cuttings and kept as dry as possible.

Watering with Cheshunt compound may help against rotting caused by *Phytophthora* or *Pythium*.

CALCEOLARIA

Grey Mould (*Botrytis cinerea* Fr.)

Under damp conditions flowers and leaves are easily attacked by grey mould which causes large brown patches covered by the usual grey mass of conidiophores and spores (see grey mould, p. 59).

Foot Rot (*Phytophthora* spp.)

The plants rot at the base. An attempt may be made to check spread of the disease by watering with Cheshunt compound (see p. 593).

Corticium solani may also cause stem rot and *Thielaviopsis basicola* causes black rot of the roots (see p. 422).

Lycopersicum Virus 3, the cause of tomato spotted wilt, often infects calceolaria, causing mottling and distortion of the leaves (see tomato, p. 384).

CALENDULA (Marigold)

Leaf Spot (*Entyloma calendulae* (Oudem.) de Bary)

On marigold leaves this fungus causes numerous circular spots about $\frac{1}{4}$ inch across, at first dark green and somewhat translucent, then whitish and finally brown (Fig. 247).

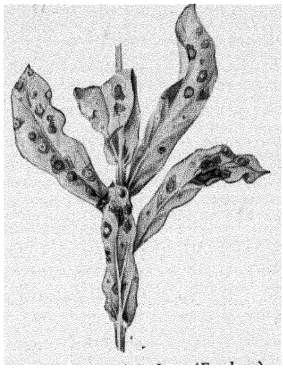


Fig. 247. Calendula leaf spot (*Entyloma*).

They may be so numerous as to join together. When plentiful they spoil the appearance of the plants and check their growth.

The disease is common throughout Central Europe, also in Denmark and Britain, especially in the South-west. It is most prevalent when marigolds are allowed to seed themselves and come up year after year in the same place. One should therefore sow them in fresh soil each year.

Grey Mould (*Botrytis cinerea* Fr.)

Under damp conditions grey mould can cause rotting of extensive patches on leaves, stems and flowers or even of the whole plant. These are covered by the usual layer of hairy grey mould. The American Ball types are comparatively resistant (see grey mould, p. 59).

Mildew (*Sphaerotheca fuliginea* (Schlecht.) Salm.)

This mildew may almost completely cover the leaves and stems of marigolds with its white powdery coating (see mildew, p. 63). Perithecia are very seldom seen so the precise identity of the fungus is sometimes doubtful.

Rust (*Coleosporium senecionis* Fr.)

This rust, which very commonly causes bright orange pustules of spores on the underside of groundsel leaves, occasionally attacks calendula in Britain and in Switzerland (Terrier, C. A., 1948).

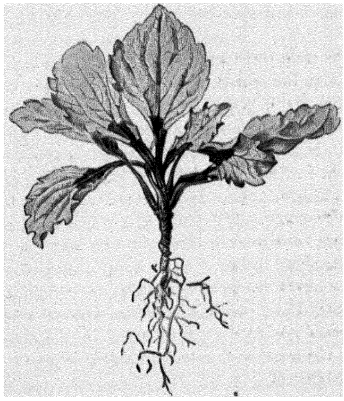


Fig. 248. Foot rot of aster seedling.

CALLISTEPHUS (China Aster)

Foot Rot and Wilt (*Phytophthora cryptogea* Pethybr. & Laff. and *Fusarium conglutinans* Wollenw. var. *callistephi* Beach)

Symptoms. These two diseases are much alike. Young asters when attacked turn yellow, rot at the base and die (Fig. 248). In older plants the leaves, buds and flowers become



Fig. 249. Foot rot of old aster.

limp, hang down, the plants turn brown and die. The upper part of the stem may remain green for a considerable time. The foot of the stem turns brown and decays, the woody tissue being dark greenish-brown or blackish (Fig. 249).

Cause. *Fusarium conglutinans* var. *callistephi* has been shown to be the common cause of aster wilt in Sweden and in England (Ogilvie and Mulligan, 1934). *Phytophthora cryptogea* has been commonly found causing foot rot from June onwards in England but not yet in Denmark. It can also attack many other plants but only becomes serious under wet conditions. Both these diseases become serious when asters are grown several years in succession in the same bed, unless the soil is renewed or disinfected.

Verticillium wilt (*Valbo-atrum*) may also attack asters (see p. 60).

Control. According to Wollenweber and Reinking (1935) if asters have been grown in a bed within the last four years the grower should either plant wilt-resistant varieties, renew the soil or disinfect it. In Sweden good results have been obtained by disinfecting the soil with formalin (see p. 579).

In America much work has been done in raising wilt-resistant asters and these have proved successful also in England.

Virus Diseases

In England china asters sometimes become dwarfed with mottled yellowish leaves. Their flower heads are small with malformed florets. Often there are short side shoots with abortive flowers low down on the stems. Such plants are infected with the virus of common cucumber mosaic (*Cucumis* Virus 1, see p. 278) which is spread by greenfly. A similar condition is known in Denmark, Holland and Germany.

Aster yellows (*Callistephus* Virus 1) is a very widespread and destructive virus disease in North America, fortunately unknown in Europe. The first symptom of the disease in asters is a paling of the leaf veins. Later developed leaves are yellowish-white, not mottled, while the crowns become short and green. As a rule the internodes are short, making the plant bushy, and flowering is feeble. Sometimes only one side of the plant is infected. The virus is spread by the leaf-hopper *Cicadula divisa* Uhl. (= *Cicadula sexnotata* Falt.). Curiously enough it takes about 10 days after sucking an infected plant before the leaf-hopper can transmit the disease. But after that the insect can go on transmitting the virus for about two months after a single feed on a diseased plant. The virus may overwinter in two-year-old or older plants and also in many weeds. It is transmitted by grafting but not by expressed sap nor by the leaf-hopper's eggs or excrement. The aster yellows virus can infect at least 120 species of plant, belonging to 30 families, but is specially prevalent in the *Compositae*. Peach yellows, beet yellows, spinach yellows, etc., have no connection with aster yellows.

Among the susceptible vegetables may be mentioned carrot, celery, dill, lettuce, parsnip, spinach and tomato; among ornamentals: adonis, begonia, calendula, cheiranthus, chrysanthemum, erigeron, myosotis, nicotiana, tagetes, zinnia; and among susceptible weeds: sow thistles, dandelions, daisies and plantains.

Control. Where infected plants are discovered they should be taken away and burnt, including those only suspected of harbouring

the disease. In North America the seed beds are kept sprayed with nicotine and surrounded with screens of unbleached calico 1½–2 yards high, as the leaf-hoppers always fly close to the ground.

Attempts are being made to breed resistant varieties. China asters may also be infected by the virus of tomato spotted wilt, which causes breaking of the petal colour (Severin, 1947) (see p. 384).

CAMELLIA

Shedding of Buds

Camellia buds are apt to be shed as a response to any unfavourable change in conditions of growth such as drought, overwatering, too high or too low a temperature, draughts, lack of light, excess nitrogen or injury to the roots by artificial manures.

Leaf Spots (*Pestalotia guepini* Desm. and *Phyllosticta camelliae* West.)

These two fungi occur in Denmark and elsewhere on spots of camellia leaves but are of little importance.

Pestalotia guepini is found on large yellow spots with dark edges. Doyer (1925) thinks it is not a parasite but colonizes spots due to other causes.

Phyllosticta camelliae occurs on circular or irregular brown spots.

Growers should endeavour to maintain the most favourable conditions of growth for camellias to prevent dead spots from developing on the leaves. Especial care should be taken to maintain a healthy and vigorous root system; the roots are easily damaged by overwatering, by too much lime or by strong manure. The fungi then colonize dead spots on the leaves and may enlarge them. If the spots appear to be spreading one may try to check them with a fungicidal spray or dust.

Camellias are also apt to develop yellow spots if the soil reaction is too high; they do best at pH 4–4.5. Frequent watering with a fairly weak solution of sulphate of ammonia suits them.

CAMPANULA (Harebell)

Leaf Spot

Various leaf-spotting fungi occur on leaves of species of campanula. The only one noticed in Denmark is *Ramularia macrospora* Fresen. which occurs also in Britain. This may cause a serious disease of *Campanula isophylla*, on which the leaf spots are more or less circular and brown on the upper side of the leaves and covered with a fine whitish mould on the under-side (Fig. 250). Spraying



Fig. 250. *Ramularia macrospora* on leaf of *Campanula isophylla*.

or dusting with a fungicide should be tried as a control measure.

In England two pycnidial fungi, *Ascochyta bohemica* Kabat & Bubak and *Septoria* sp., also occur on campanula leaves (Moore, W. C., 1940a).

Rust (*Coleosporium campanulae* (Pers.) Lév.)

All our native species of campanula and many cultivated ones are susceptible to infection by this rust, which has its cluster-cup stage on needles of various kinds of pine (see p. 510). The uredo and teleutospores form yellow and reddish-yellow pustules on the under-side of campanula leaves.

The rust can, however, overwinter in the uredo state on campanula, and may therefore persist even in the absence of pines from the neighbourhood.

Another rust, *Puccinia campanulae* Carmich., forms only brown pustules of teleutospores on campanula leaves. It has no alternate host.

Other diseases recorded on species of campanula in Britain include *Sclerotinia* rot (*S. sclerotiorum*) on *C. pyramidalis* and canterbury bells (see p. 77), *Phytophthora* foot rot (see p. 68), crown rot caused by *Botrytis cinerea* (see p. 59), *Verticillium* wilt (see p. 60), and mildew (*Erysiphe cichoracearum* DC.) (see p. 63).

CARNATION

See *Dianthus*, p. 448.

CEDRUS (Cedar)

Dieback (*Phomopsis pseudotsugae* Wilson)

Terminal parts of the main shoot die back for a considerable distance and soon shed all their leaves. The small black pycnidia of the fungus appear in cracks on the dead bark and from them under damp conditions exude whitish tendrils of spores. Branches may also be attacked at any point on their length. The mycelium spreads all round the branch and causes a sunken dead area completely girdling it. The part of the branch above the constriction then gradually loses its leaves and dies (Wilson, M., 1930). Cross-inoculation experiments have shown that the fungus on cedar is the same as that on Douglas fir (see p. 515).

CHEIRANTHUS (Wallflower)

Bacterial Stem Rot

A bacterial disease of wallflowers in which the base of the stem was constricted and turned brown was described in Holland by Van Hall as early as 1902. In 1928 we observed a severe outbreak of wallflower root rot which closely resembled soft rot of cabbage in texture and smell (see p. 257). After dusting the stem base with lime and repotting in soil mixed with slaked lime nearly all the plants recovered.

Brown veins in the leaves, associated with brown vascular bundles in the stem and possibly with small leaf spots, are supposed to be due to black rot (see cabbage, p. 256).

Moore (1948) records the leafy gall organism (*Corynebacterium fascians* (Tilf.) Dows.) causing leafiness and malformation at soil level in wallflower (see p. 484).

Grey Mould (*Botrytis cinerea* Fr.)

Wallflowers are liable to attack by grey mould, especially when overwintering under glass. The leaves and shoots develop brown patches which are gradually covered by the hairy grey fungus. Its presence is an indication that conditions are too moist and this should be corrected (see grey mould, p. 59).

Downy Mildew (*Peronospora parasitica* (Fr.) Tul. = *P. cheiranthi* Gäumann)

On the upper surface of the leaves this causes pale yellowish patches, while the corresponding parts of the under-surface are covered with a greyish or white mould. Stems and flower heads may also be infected and are then swollen and often twisted. Infected flower buds do not develop.

It is customary in Britain to regard the downy mildew of wallflower as a specialized race of *P. parasitica*, similar to those found on many other cruciferous plants. On the Continent it is more usual to treat it as a distinct species, *P. cheiranthi*. The difference in name implies merely a slight disagreement among mycologists as to what constitutes a species and has no practical significance.

The disease is best controlled by copper fungicides.

Black Mould (*Alternaria cheiranthi* (Fr.) Bolle)

This black mould has been noticed rather extensively in Denmark during the last few years on wallflower seed crops (Neergaard, P., 1938). It attacks the leaves and under damp conditions may cause them to wither, especially if the plants have been weakened in any way. The mould may also attack the pods and infect the seed. It may also attack stock and Neergaard has been able to infect red cabbage and Yorkshire cabbage heads with it.

Wallflowers should not be grown where stocks or other cruciferous plants have been planted in previous years. Seed disinfection by soaking for ½–1 hour in ¼% solution of Germisan or other organo-mercury fungicide may be tried.

White Blister (*Cystopus candidus* (Pers. ex Chev.) Lév.)

This causes white swellings on leaves, stems and flower heads, which burst to expose powdery white masses of conidia. The infected parts become thickened and misshapen (see horseradish, p. 285).

Club Root (*Plasmodiophora brassicae* Woron.)

This commonly causes swellings on wallflower roots like those on cabbage. Infected plants are stunted (see cabbage, p. 246).

Mildew (*Erysiphe polygoni* DC.)

Powdery mildew sometimes covers wallflower leaves with its white spores, mostly on the upper surface (see mildew, p. 63).

Damping Off and Foot Rot (*Corticium solani* (Prill. & Delacr.) Bourd. & Galz. and *Phoma lingam* (Fr.) Desm.)

These fungi cause seedlings to turn brown and soft at soil level so that they fall over and die (see damping off, p. 68). On older plants they cause stem rot and wilting (see *Corticium solani*, p. 68.)

Breaking of Flower Colour

Two different viruses may cause " breaking " of flower colour in wallflower, i.e. irregular yellow spots and lines on the petals of self-coloured varieties. These are, *Brassica* Virus 1, common in cabbages, etc. (see p. 258), which causes also some curling and twisting of the leaves, and *Matthiola* Virus 1 which comes from stocks (see p. 494) and causes a faint mottle of wallflower leaves. Both are aphis-transmitted and may, of course, spread from wallflower to wallflower. These viruses often completely spoil the appearance of bedding wallflowers in parks and public gardens, especially in the South of England.

The only control measures appear to be to try to keep down aphides and to destroy infected brassica crops in the neighbourhood (Smith, K. M., 1935).

CHERRY (Ornamental) and CHERRY LAUREL

See *Prunus*, p. 514.

CHRISTMAS ROSE

See *Helleborus*, p. 470.

CHRYSANTHEMUM FRUTESCENS (Marguerite)

Leaf Spot (*Ramularia bellunensis* Speg.)

Greyish-brown spots appear at the tips and edges of marguerite leaves and later turn brown with a darker margin. They may spread until the whole leaf is destroyed. Colourless conidia are produced on the dead tissue. Infection occurs only under damp, shady conditions, such as occur in winter and early spring. The disease should not be troublesome in a well-lit ventilated house (Oyler, E., 1938).

Black Foot Rot (*Phytophthora cryptogea* Pethybr. & Laff.)

The stems at soil level and upwards turn black and decay. Eventually the leaves wilt and the plant dies. Oyler (1937) found the cause of this disease to be the common damping-off fungus *Phytophthora cryptogea*. For control see tomato, p. 375.

CHRYSANTHEMUM INDICUM (Chrysanthemum)

Blotch (*Septoria chrysanthemella* Sacc.)

Symptoms. Blackish-brown spots appear on the leaves, usually first on the lower ones, and may gradually increase to about $\frac{1}{2}$ inch across, often marked with darker more or less concentric lines (Fig. 251). Several spots may join up and so destroy a large part of the leaf. The pycnidia of the fungus appear as tiny dark dots on the surface of the spots.

This disease must be carefully distinguished from the more destructive leaf spots caused by the chrysanthemum eelworm. The latter are also brown but angular, because they are limited by the main veins, and bear no pycnidia.

Cause and history. Chrysanthemum blotch is caused by the fungus *Septoria chrysanthemella* which has been known in Denmark

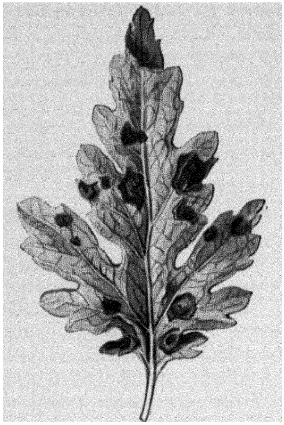


Fig. 251. *Septoria* leaf spot of chrysanthemum.

since Rostrup, in 1897, described it under the name *S. chrysanthemi*. Later it was called *S. rostrupii* Sacc. & Syd. The same or a very similar fungus causes brown spots on the lower leaves of the wild moon daisy (*C. leucanthemum*) and the shasta daisy (*C. maximum*).

Varietal susceptibility. There seems to be some difference in susceptibility of different chrysanthemum varieties. In Denmark the disease has been specially noticed on Delaware, Flory King, Framfield, Goldfinder, Ivy Gay, Massee, Normandie, Queen Mary, Rayonnant, Romance, Sax's Export and Winter Cheer, but it may also occur on many others.

Control. Soil containing diseased chrysanthemum leaves should not be used. The mother plants should be stripped clean of diseased leaves before the cuttings appear. Klougart obtained good control by spraying with Bordeaux mixture 8: 4: 50 and somewhat less with Sulsol and Pomasol ¾: 100. The latter had the advantage of causing less spotting. Dusting with sulphur was fairly effective.

Chrysanthemum Rust (*Puccinia chrysanthemi* Roze)

In 1895 chrysanthemums in England were attacked by a hitherto unknown rust, the origin of which was eventually traced to Japan. It spread quickly over Europe, reaching Denmark in 1897, and is now known throughout the world as *Chrysanthemum indicum*. Rusts on other species of chrysanthemum are quite distinct fungi, e.g. the groundsel rust *Coleosporium senecionis* Fr. occasionally attacks *C. carinatum* and *Puccinia leucanthemi* Pass. occurs occasionally on the moon daisy, *C. leucanthemum*.

Symptoms and development. Rust attacks are usually noticed in late summer, when yellowish-green spots appear on the upper surface of the lower leaves. On the under-side are chestnut-brown pustules of uredospores (Fig. 252). If

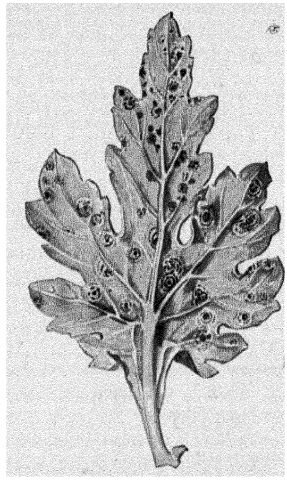


Fig. 252. Rust pustules on under-side of chrysanthemum leaf.

the weather is damp or the locality sheltered several generations of uredospores quickly develop. When the plants are moved into the greenhouse and crowded together the rust may destroy a good part of the leaves. The uredospore stage overwinters as inconspicuous attacks on the mother plants. Infection of next year's plants may also come from spores on old leaves or leaf stalks in the potting soil.

A few two-celled teleutospores sometimes occur mixed with the uredospores, and sometimes whole teleutospore pustules at the end of the season, but they seem to play no particular part in Europe. In Japan teleutospore pustules are fairly common. No cluster-cup stage is known. A fungus that normally lives on scale insects, *Verticillium coccorum* (Petch.) Westerd., sometimes parasitizes the rust pustules and penetrates the leaf tissue through them, but is unable to attack a healthy chrysanthemum leaf (Kotthoff, P., 1937b).

Varietal susceptibility. When a number of chrysanthemum varieties are grown side by side some may be seen to be severely attacked by rust and others scarcely at all. Various lists of susceptible and resistant varieties exist (N. Flenstead-Andersen in MS., Moore, W. C., 1948). Comparison of these and personal experience shows that the relative susceptibility of varieties differs greatly from place to place. Some are said to be completely resistant, such as Alice M. Love and October Sun. Even if they have themselves no commercial value they may prove useful in a breeding programme for raising rust-resistant varieties. Among fairly resistant varieties may be mentioned: Buckingham, Golden Seal, Hatzuwarai, In Memoriam, Kyokko, Robert Collins, R. T. Blyth, Sirius, Thalia and Yoko. The following are often severely attacked: Ambassador, Flory King, Goldfinder, Glow, Harvester, Mona Davis, Roy, Titan and Welcome.

Control must be based on preventing infection. When the mother plants are selected the basal summer shoots are removed and some of the potting soil is replaced by disinfected soil. We have found it well to expose the mother plants to some frost during the autumn. When the cuttings are taken the two or three lowest leaves, most likely to be infected, should be picked off and when they are repotted the lower leaves should again be removed.

Plants with good firm growth seem to suffer least. Remembering the risk of infection from old leaves the grower should burn all rubbish and change the plants' summer quarters from year to year. As far as possible water on the ground, not by spraying. After they have been taken indoors dew formation should be avoided.

Spread of the disease may be satisfactorily prevented by spraying with lime sulphur 1: 100 three or four times between late summer and the moving of the plants indoors. A spreader should be added to the spray.

There are so many varieties to choose from that there is no need to grow those most susceptible. New varieties should be kept apart for a time until their susceptibility is discovered. Newly bought plants of any variety should be kept apart under observation until one is sure they are not introducing infection into the nursery.

Chrysanthemum Mildew (*Oidium chrysanthemi* Rabenh.)

This forms a floury white coating over the leaves (Fig. 253). It is worst in greenhouses that are not well ventilated (see mildew, p. 63). Varieties differ considerably in susceptibility.

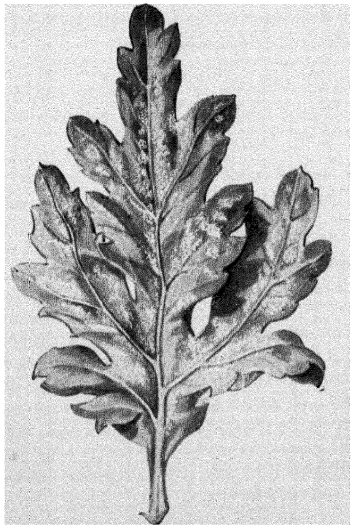


Fig. 253. Chrysanthemum mildew.

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

This disease has been studied in Washington by Huber and Jones (1934) and in Holland by Van der Meer (1924). It is widespread in England.

The symptoms start by a yellowing of the leaves which gradually turn brown and die, starting at the base of the plant and spreading up the stem. Growth becomes stunted and the flowers are small. In severe attacks the plant dies before blooming. The fungus enters from the soil by way of the roots and spreads through the vascular tissues that convey water through the plant. Varieties differ greatly in susceptibility to wilt.

Cuttings should not be taken from diseased or suspected mother plants. Infected soil should be replaced or sterilized (see *Verticillium* wilt, p. 60).

Leaf Curl

The leaves become large, stiff, brittle and curled, particularly on one-stemmed chrysanthemums that have received too much nitrogenous manure. Severe disbudding may also accentuate this malformation.

Growers should not manure too heavily on varieties known to be prone to leaf curl. Disbudding should be done with discretion and not all at once.

Cracked Neck

Symptoms. This phenomenon has been observed in Holland and Massachusetts as well as in Denmark. Deep cracks girdle the flower stalk and in the worst cases the buds are destroyed. The deepest crack is usually just under the bud and as well as a few or several cross cracks there may be one longitudinal one. Cracked neck tends to be serious on disbudded plants where only 1 or 2 buds remain on each stem. If all the buds are left to mature and produce a cluster of flowers the trouble does not appear. In Holland the variety Mountain Greenwood is particularly susceptible, William Turner rather so.

Cause. The cracking occurs when the temperature of the house drops suddenly so that the air becomes saturated with moisture while the soil remains warm and the roots continue to absorb water freely. The flower stalks then become charged with water which cannot be given off in transpiration and accordingly split.

Control. Watering should be done early in the day, especially in cloudy weather, and the temperature of the house should be kept up at night.

Dead Flower Centres

Particularly late in the season flowers appear lacking the central crown which has turned black. The damage may be very bad in Mona Davis but Mrs. R. C. Pulling and others also have a tendency to this malformation.

The reason for it has not been clearly demonstrated but there is evidence to show that the phenomenon is encouraged by very high temperatures combined with heavy manuring. Connected with the heat is, perhaps, dryness. Instances have been seen where it was quite obviously the plants that flowered first that had the worst affected flowers.

The condition is not necessarily connected with early forcing but is perhaps due to these flower buds having been at a particularly susceptible stage when exposed to the heat. In the autumn of 1939 two experiments were carried out in minor-element manuring but no obvious effect on this trouble could be detected.

Green Flower Centres

Some varieties, such as Mrs. R. C. Pulling and Mona Davis, may have a green heart to the flowers (" green button "). It is thought that this may result from leaving the wrong bud at disbudding, disbudding too late, delaying development too long or keeping the plants too dry.

Virus Diseases

Chrysanthemums are very commonly infected with the virus of tomato spotted wilt (*Lycopersicum* Virus 3) but they often show little or no sign of disease. At most there may

be rather ill-defined bronze spots and rings on some of the leaves (see p. 384).

Infection with cucumber mosaic virus (*Cucumis* Virus 1) causes mottling of the leaves.

Cold Chlorosis

Cold chlorosis is often seen in Edith Cavell; after a cold night larger or smaller zones quite devoid of green colour may develop along the main vein.

This kind of chlorosis must not be confused with the white or yellow leaf edges which may appear on chrysanthemums after spraying or fumigating with nicotine (see p. 50).

Summary of Diseases of Chrysanthemum Indicum

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B. Older plants covered with hairy grey mould. Mother plants rot in boxes	*Botrytis*	59
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C. Flower centres abnormal	Green Flower Centre	434
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D. Flowers covered with small brown spots or covered by hairy grey mould	*Botrytis*	59

CHRYSANTHEMUM MAXIMUM (Shasta Daisy)

Blotch (*Septoria leucanthemi* Sacc. & Speg.)

Symptoms. The fungus causes brown spots with dark edges. On the leaves examined at Lyngby the spots were not individually more than ¼ inch across but several often joined together to destroy large parts of the leaves (Fig. 254). In older spots some of the dead tissue broke away leaving irregular holes in the leaf. In England Wormald (1927) found leaf spots ½–¾ inch across, apparently infection had occurred earlier than in our plants.

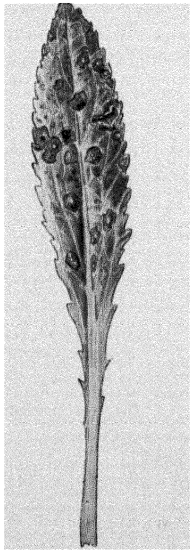


Fig. 254. Leaf blotch of Chrysanthemum maximum.

It is not certain that this fungus is distinct from *S. chrysanthemella*.

Control. Control experiments have not been made but probably picking off the infected leaves in autumn and spraying with a fungicide would have some effect.

CINERARIA

See *Senecio*, p. 532.

CISSUS (Russian Vine)

Leaf Spots and Withered Leaves

Cissus plants frequently have withered leaves or dry, often clear, somewhat angular, leaf spots with no evidence of any particular disease or insect damage. The cause may be unfavourable growth conditions, for example overwatering or too strong light.

Neergaard (1943) records infection of the shoots of an indoor *Cissus* plant by the fungus *Gloeosporium physalospora* Cav. The small black fructifications of the fungus formed pimples on the stem and the shoot wilted. When such an attack is discovered the diseased shoots should be cut out and the plant sprayed with a fungicide.

CLARKIA

Downy Mildew (*Peronospora arthuri* Farlow)

The fungus first attacks the lower leaves and then spreads upwards. On the under-side of the leaves there is a fine downy mould, at first white, later grey with a faint mauve tint, ultimately greybrown on the dead leaves. On the upper surface the leaves show vague, pale-yellowish spots. They slowly wither and fall so that the stalks are left naked below. Neergaard (1943) saw severe attacks on seed crops of *Clarkia elegans* in 1940 and 1942 while *C. pulchella* growing alongside remained healthy.

The disease seems particularly injurious to seed crops.

As control measures we would recommend rotation of crops, seed disinfection by soaking it for ½ hour in ¼–½% Danish Tillantin and spraying the plants with Bordeaux mixture or other copper fungicide. The spraying should commence in good time before flowering and be directed particularly against the under-side of the leaves.

CLEMATIS

Leaf Spot and Dieback (*Ascochyta clematidina* Thüm.)

Small watersoaked spots appear on the leaves, these later dry out, turn brown, become somewhat marked with rings and develop a red margin. Brown somewhat sunken spots appear on the stems and sometimes girdle them, causing the parts above to die. Stems may be attacked right down to the base. There may also be spots on the leaf stalks (Gloyer, 1915).

This disease has only once been observed in Denmark. It was found in Devon in 1937 (Moore, W. C., 1943).

The diseased parts should be removed and the plants sprayed or dusted with a fungicide. According to Gloyer *Clematis paniculata* cannot endure much sulphur.

Mildew (*Erysiphe polygoni* DC. = *E. nitida* (Wallr.) Rabenh.)

Clematis, particularly the large-flowered varieties, are apt to suffer from a white floury coating of mildew on the leaves and sometimes on the petals as well (see mildew, p. 63).

Non-parasitic Dieback

Many growers of large flowering clematis have experienced the sorrow of seeing vigorous, apparently healthy plants suddenly wither and die.

This phenomenon has been known for over 40 years. Sorauer described it in the last century without being able to offer any satisfactory explanation. Nor, unfortunately, can we. (But cf. *Ascochyta clematidina* above.)

In 1939 Pape wrote that this form of dieback was worst in wet years on very moist or heavy soils and after sharp night frosts. Broek and Schenk found it may occur at any time through the summer, but particularly after sharp night frosts in spring or on easily frozen soil.

In Denmark we have seen the disease in the middle of a dry summer on a dry situation and it seems particularly to affect clematis on south walls. *C. jackmannii* is particularly susceptible and Russell (1933) thought dieback in that species was due to excessive exposure to the sun.

Dieback of potted clematis due to contamination of the crocks with chlorate has also been described (Hunt, I. V., 1939).

So little is known about clematis dieback that reliable control measures cannot be suggested. We can, however, advise planting on good, warm, well-drained soil and planting deeply. Walls with an east or west aspect are to be preferred to south walls. According to Markham (1939) clematis on its own root is much less prone to dieback than grafted plants. Hence he recommends propagation by layering.

CLIVIA

Leaf Spot

Clivia often develops larger or smaller, reddish or brown leaf spots, often starting at the margin. As a rule they are due to unfavourable growing conditions, such as the air of the

room being too dry. Spraying the leaves with lukewarm water as soon as the spots begin to appear may be advised. In the case of plants in a living-room their position may be changed to one where they receive more or less sun than before.

The mould *Colletotrichum cliviae* Oud. may cause small, irregular, red or brown spots which bring about premature withering of the leaves. One should avoid excessive moisture and perhaps try spraying with a copper fungicide.

Small reddish spots with a translucent edge are also sometimes seen, suggestive of a bacterial infection.

COLCHICUM (Meadow Saffron, Autumn Crocus)

Smut (*Urocystis colchici* (Schlecht.) Rabh.)

This fungus forms small blisters containing black smut spores in the outer skin and outermost layers of the white corm tissue. In exceptional cases the patches of smut spores occur deep in the flesh of the corm.

The leaves may be covered with long rows of similar smut blisters.

This disease was prevalent in Holland in 1939 especially on the varieties Bornmulleri and Lilac Wonder as well as on ordinary *Colchicum autumnale*. It is as yet unknown in Britain.

Control experiments undertaken in Holland have as yet yielded no useful results.

Corm Rot (*Pythium ultimum* Trow)

Infection occurs from the soil before the corms are lifted but the rot is mainly seen in stored corms. At the top or side of the corm is a crater-like hole filled with soft, wet, rotting tissue. The same fungus causes rotting of potato tubers (see watery wound rot, p. 333), tulip bulbs, etc. (Moore, W. C., 1940b).

COLUMBINE

See *Aquilegia*, p. 415.

CONVALLARIA MAJALIS (Lily of the Valley)

Leaf Spot (*Dendrophoma convallariae* Cav.)

The leaf spots are red-brown, later dark brown and may cover the greater part of the leaf surface. Very small black pycnidia occur on them. This fungus has frequently proved very destructive in Denmark (Rostrup, 1902).

Leaf spots may also be caused by two species of *Septoria* and one or more of *Gloeosporium*.

Repeated sprayings with a copper fungicide may be tried as a control measure.

Grey Mould (*Botrytis paeoniae* Oudem.)

Symptoms. The fungus causes brown spots on leaves and stems. On these are produced the grey mould and also black oval sclerotia up



Fig 255. Grey mould of lily of the valley.

to about $\frac{1}{16}$ inch long. In the affected parts the leaf tissue is often completely destroyed, with the exception of the skin and veins. The parts of the plant above the diseased places naturally die also (Fig. 255). As infection often occurs at the base of the plant the result is often death of the whole above ground portion.

The parts below ground are not affected so the plant grows up again the following year, but is naturally weakened by its loss and has only poorly developed flowers.

The flowers are also easily attacked, especially in greenhouses. Here the disease appears as small yellow spots which gradually enlarge and turn yellow-brown and dark brown. The tissues shrink and become overgrown by the grey mould which easily spreads to further plants in the house.

If the conidia fall on blooms being packed for market and the blooms are then kept shut up and damp for some time they too become spotted with yellow and spoiled.

Cause. The disease is caused by the grey mould *Botrytis paeoniae*. According to Ritzema Bos (1898) the fungus from lily of the valley can attack paeony, and Hahmann (1930) produced spots on tulip flowers and leaves by cross-inoculations with the fungus isolated from lily of the valley. We have seen lily of the valley develop brown spots on the flowers when growing next to tulips affected by grey mould while others growing farther away from the tulips were unaffected.

Control. The diseased plants should be dug up and destroyed as soon as possible, with the surrounding soil which may contain sclerotia. Close planting and planting in moist places should be avoided. Under glass, too, excessive moisture should be avoided (see also grey mould, p. 59).

Failures in Forcing

Failures in forcing may be due to attack by grey mould or by *Sclerotium denigrans*. They may also, however, arise when the shoots are exposed to drying out after being dug up instead of being carefully replanted.

Rust (*Puccinia sessilis* Schneider)

Small groups of orange cluster cups (*aecidia*) are found on the leaves, which then wither prematurely. These may occur also on *Majanthemum* (May lily), *Paris* and *Polygonatum* (Solomon's Seal). The alternate uredo and teleutospore stages are found on the reed grass, *Phalaris* (*Digraphis*) *arundinacea*, which grows in marshes, ditches and along river banks. Lily of the valley and the other liliaceous hosts are infected from this grass each spring.

Black Lily of the Valley (*Sclerotium denigrans* Pape)

This disease has long been known in Germany where the sale of forced roots is of some importance. Great losses may occur during storage in the boxes prepared for export. Apparently the disease also occurs in Denmark. Pape (1943) has studied it thoroughly in Germany It is practically impossible to detect it when the roots are taken up in autumn but the disease may become apparent during packing or in transit. The buds develop spots or turn quite black. At the outset only the covering scales are attacked while the buds themselves remain white and healthy. In more severe attacks the buds may rot completely and break off easily at a touch. The blackening may extend far into the tissues. Infection begins in small grey spots which spread in all directions. Sometimes healthy buds are found between diseased ones but often all the buds on a plant are attacked. The spots may also appear on the underground stems and roots and on these parts of the plant they may be an inch or more long. Mycelium or sclerotium-like bodies may be found in the blackened areas and under moist conditions a fine web of mycelium may occur between the affected parts. Often all the roots in a particular package are attacked and the disease may spread from a single focus to all the roots in 8–14 days.

If severely attacked roots are forced they naturally produce no flowers but if only the covering scales are affected more or less

unsatisfactory shoots may appear. Hence if the grower sorts out the apparently healthy roots and forces them he may be able to salvage something from a diseased consignment if the disease has not already attacked them all.

Infected roots introduce the disease into outdoor plantations and give rise to blanks in the row in the first year. The blanks often extend over the area planted from one or more packages, the roots from which have been planted one after the other. In older beds the blanks have a different distribution because the disease gradually spreads from the roots first affected.

This disease seems particularly prone to occur on mossy soils and on old garden soils rich in humus. It is less prevalent on soils kept loose and open. Plentiful manuring, especially with organic nitrogenous manures, seem to make it worse.

Repeated cultivation of lily of the valley on the same soil definitely favours the disease.

It is advisable to put the roots into sand as soon as possible. Storing in cool places at 18–25° F. checks the disease. The remains of forced lilies of the valley should never be put on the compost heap but should be burnt.

CRASSULA

Mildew

See p. 63.

Shoot Rot (*Fusarium avenaceum* (Fr.) Sacc.)

The shoots of *Crassula rubicunda* may become pale green and wither. They are constricted at the base and permeated by mycelium and often bear pink pustules of *Fusarium* conidia.

The plants should be kept in a well-ventilated dry place and diseased shoots should at once be removed. Possibly it would be well to spray or dust them with a fungicide. As the species of *Fusarium* are sensitive to mercury there is reason to suppose that spraying with a fungicide containing mercury might be beneficial, especially if a spreading agent were added (see pp. 594 and 601).

CRATAEGUS (Hawthorn)

Mildew (*Podosphaera oxyacanthae* (DC.) de Bary)

Leaves and shoot tips become covered by a white, floury layer of conidia and are underdeveloped and stunted. In the spring a few completely mildewed shoots occur, which have come from buds in which the mildew overwintered. Later on it spreads over the bush, especially on the younger leaves.

Mildew does most damage to nursery stock by retarding its growth; on hedge plants it is merely unsightly (see mildew, p. 63).

Rust (*Gymnosporangium clavariaeforme* DC.)

Symptoms. On the leaves, young branches and fruits of hawthorn there develop during summer brownish thickened patches crowded with the cluster-cup stage of this rust, formerly called *Roestelia lacerata*. These cluster cups have rather stiff tall margins much cut up into narrow lobes. Severe attacks may deform the branches (Fig. 256).

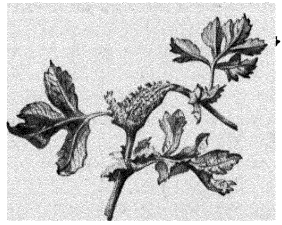


Fig. 256. *Gymnosporangium* aecidia on hawthorn.

The other host plant is the common juniper. On this the rust infects the branches, which swell up and become spindle-shaped. From the swellings the teleutospores of the rust burst forth in spring, stuck together to form slender yellow or orange horns that may be about ¾ inch long. As a rule these occur in great numbers on each swelling and are very conspicuous in damp weather. They disappear very quickly, however, leaving only small scars behind them (Fig. 257).



Fig. 257. *Gymnosporangium* horns on juniper stem.

Cause and development. The rust on both hawthorn and juniper is the same, *Gymnosporangium clavariaeforme* DC., described in 1788 by Jacquin as *Tremella clavariaeforme* on juniper and by Sowerby in 1801 as *Aecidium laceratum* on hawthorn. The connection between the two states was proved by A. S. Ørsted in 1867.

In the spring basidiospores from the juniper infect hawthorn and in late summer aecidiospores from hawthorn infect juniper. But the mycelium persists for many years in the infected juniper branches and the spore horns break out year after year from the same swellings.

Importance. This rust infects the common juniper, *Juniperus communis*, and also *J. hibernica*, *J. oxycedrus*, *J. nana* and *J. sibirica*. The infected branches die after some years. Most damage, however, is done to hawthorn, especially to high-stemmed thorn in nurseries, for the branches may be so much damaged that the crown becomes deformed. In England, where juniper is not common, the disease is not often seen but it occurs where the two hosts grow side by side, as on the North and South Downs, in Hampshire, Westmorland, etc.

This rust has a tendency to form physiological races. Eriksson (1919) recognized three races of which f. sp. *Crataegi* infected the common hawthorn, other species of *Crataegus*, quince, pear and various species of *Amelanchier*. The other two races occurred mainly on pear and *Amelanchier* but could occur on hawthorn.

Jørstad has observed serious infection of pear fruit in Norway and we have seen the same in Denmark.

Control may consist in eradicating one of the alternate hosts. Usually it is juniper that is destroyed but where it occurs wild this may prove impracticable. We know of one case where a nursery found it necessary to give up growing juniper for several years as hawthorn was the more profitable crop.

If a grower has only one or two diseased junipers with which he is unwilling to part he should cut away as far as possible the infected swollen branches. Spraying with Bordeaux mixture, lime sulphur, etc., is not always effective though it gave good results at one nursery in Jylland. It must not be forgotten that infection of the hawthorn depends largely on moist weather at the time in spring when transfer of spores from the juniper is taking place. Hence if spraying or dusting is being tried it should be done particularly in wet seasons from mid-April to mid-June.

Leaf Blotch (*Sclerotinia crataegi* Magn.)

This fungus in its conidial (*Monilia*) stage produces brown blotches on hawthorn leaves, on which grey cushions of spores appear under damp conditions. They give off a peculiar sweet odour. Its life history is like that of *Sclerotinia cydoniae* (p. 207) but it does not attack fruit trees. *S. crataegi* is not uncommon in some seasons in England and South Scotland, in others it is not seen at all (Wormald, 1937).

Leaf Spot (*Septoria crataegi* Desm.)

Numerous small reddish or brown spots may appear on hawthorn leaves. Gradually they turn pale at the centre and then minute black pycnidia become visible on them (Fig. 258).

The attack can apparently be checked by spraying with a copper fungicide.

CROCOSMIA

See *Gladiolus*, p. 463.

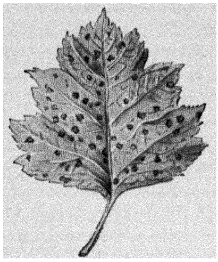


Fig 258. Hawthorn leaf spot.

CROCUS

Blind Buds

The shoots appear above ground normally and the leaves develop well during forcing but the flower buds cease to grow, and dry up or rot. While the shoot is still small and enclosed within the white scale a brown dot can be found deep inside it when it is cut through. Close examination shows this dot to consist mainly of the withered stamens that have turned brown.

Cause and development. The condition is of purely physiological origin. When the buds ultimately decay it is because they are already dead and therefore fall a prey to common moulds like *Botrytis*, *Penicillium* and *Verticillium*, but these are not the real cause of the trouble.

Unfavourable growing conditions, either drought or excessive heat, have caused the death of the flower bud. It does not take much heat to destroy them, especially if the corms are brought into the warm too soon.

Diseases of the Corm

Penicillium Rot (*Penicillium* sp.)

Especially during storage and when forced crocus is very liable to be attacked by *Penicillium* and the attack may become quite serious. The shoots rot at the base and the corms become soft, brown, almost mealy and often break up completely (see further under tulip, p. 545).

Sclerotinia gladioli Drayt. and *Septoria gladioli* Passer. may both attack crocus (see gladiolus, p. 466 and p. 465). So may *Sclerotinia bulborum* (Wakk.) Rehm (see hyacinth, p. 470) and *Sclerotium tuliparum* (see p. 542).

Fusarium Rot (*Fusarium* sp.)

Starts at the base of the corm or in the roots and causes premature yellowing and death of the leaves. It progresses in stored corms and eventually destroys them completely. According to Wollenweber and Reinking the fungi concerned are *F. oxysporum* and *F. bulbigenum* (see narcissus, p. 496).

Copper Web (*Helicobasidium purpureum* Pat. = *Rhizoctonia crocorum* (Pers.) DC)

Causes the plants to die in patches. It is a soil fungus with violet strands of mycelium, which form a felted mass on the outer surfaces of the dry scales surrounding the corm. On the inner surface are numerous tiny sclerotium-like bodies, first white then violet and ultimately brown. From these mycelium grows into the flesh of the corm and destroys it (see also carrot, p. 262).

CYCLAMEN

Black Root Rot (*Thielaviopsis basicola* (Berk. & Br.) Ferraris)

The plants languish and are easily pulled out of the soil because their roots are rotten. The corms, too, rot. Permeating the diseased tissue is the mycelium of the mould *Thielaviopsis basicola* (see begonia, p. 422).

Grey Mould (*Botrytis cinerea* Fr.)

Grey mould is one of the worst enemies of cyclamen. Not only does it attack the seedlings (see below, p. 444) but also the new leaves and flower buds down among the stalks of the older leaves on mature corms. They rot and are covered with grey mould which then attacks the older stalks as well (Fig. 259). The decay may gradually spread into the corm and kill the plant altogether. Large decaying patches covered with grey mould may also appear on the leaf blades.

There can hardly be a cyclamen grower who does not know grey mould but when it is abundant it is a sign that conditions are too

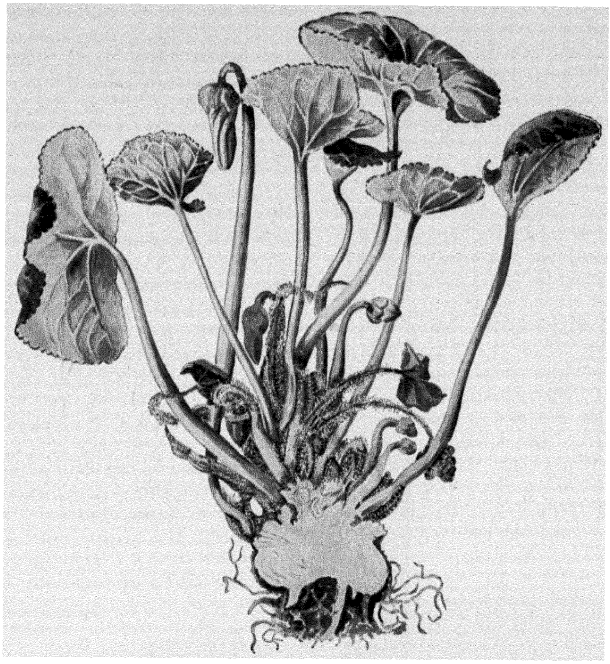


Fig. 259. Grey mould attacking leaf and flower stalks of cyclamen.

damp. Either the plants have been watered too often or it has been done carelessly. Watering should be done only when required, and as far as possible the stream should be directed beside the corm, not into the heart of the plant. Moreover the air should be kept dry by firing and ventilating. This may be difficult in a dull, wet autumn. It may even be worth while to consider raising the temperature and selling the plants as they come ready for market rather than trying to keep them for Christmas and risking losing them with grey mould. If much nitrogenous manure is supplied the tissues become soft and this indirectly encourages grey mould. Spraying cyclamen with Pomarsol has given promising results (see also grey mould, p. 59).

Mildew (*Oidium* sp.)

During the winter 1938–9 Wenzl in Germany observed a close whitish-grey floury mildew on cyclamen petals though the leaves and other parts were not attacked. The conidia were relatively large but the systematic position of the fungus could not be determined because no perithecia were found.

The flowers became pale and hung limply on their stalks. No control was attempted, but if an outbreak occurs the usual measures against mildew may be tried (see p. 63).

Seedling Diseases

Many young cyclamen plants die before they are big enough to be potted. One of the commonest causes of loss is the propagation

fungus *Moniliopsis aderholdi* Ruhl. This turns the above-ground parts of the plant brown and may rot the little corm so that the plant is completely destroyed. As a rule an outbreak starts at a few centres from which the fungus grows out radially, killing all the seedlings it reaches.

The damping-off fungus *Corticium solani* behaves in much the same way and is hard to distinguish from the propagation fungus. The latter usually makes a more conspicuous web of mycelium over the plants but many mycologists regard the two fungi as strains of one species.

On fairly young cyclamen plants which had small spots on the corms and more or less brown roots we have several times found the fungus *Cylindrocarpon radicicola* Wollenw. As this fungus can be found in many kinds of roots, bulbs, etc. (see azalea, p. 417; lily, p. 489; narcissus, p. 497), it seems likely it can also cause damage to young cyclamen, though Wollenweber (1935) has tried unsuccessfully to infect older healthy plants with it.

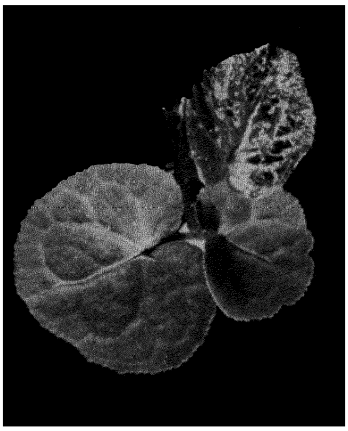


Fig. 260. Cyclamen plant grown in overlimed soil showing distorted leaf.
The normal leaves unfolded after it was replanted in acid soil (*S. p. F.*).

If they are kept too damp they may be attacked by grey mould (*Botrytis cinerea* Fr.), when they become brown, rotten and covered with the grey fungus.

If the soil is rich in lime the plants turn pale or yellow or develop small pale spots (Fig. 260). Cyclamen, especially when young, need a slightly acid soil pH 5.5–6.5. Hence amongst other things one should be careful that the gravel used does not contain lime.

As far as fungus diseases are concerned it is important that there shall be no remains of diseased plants in the soil and it is advisable to use sterilized soil. Steam sterilization is best but formalin treatment is satisfactory if the soil is well and thoroughly aired before it is used. Some growers treat the soil with corrosive sublimate solution, and cyclamen plants seem to do best of all then if no saltpetre is added to the solution, i.e. if only 1 oz. of corrosive sublimate in 12 gallons of water is used and nothing else. Practical experience indicates that sublimate treatment of soil is no use against *Cylindrocarpon radicicola*. In combating grey mould it is important that the plants are not allowed to become too damp. When patches of propagation fungus appear it is permissible to sprinkle them and the surrounding area with corrosive sublimate solution at the above strength. Heavy dusting with sulphur may also check development of this fungus, and dusting with Brassicol may be tried. With all these diseases it is important to remove the diseased plants as soon as possible and preferably also the soil surrounding them. For further details see propagation fungus, p. 69; damping off, p. 68; grey mould, p. 59; and soil disinfection, p. 577.

CYTISUS (Broom)

Leaf Spot and Dieback (*Ceratophorum setosum* Kirchn.)

According to Green and Hewlett (1949c) *Ceratophorum setosum* causes a black leaf spot, leaf fall and dieback of young shoots in *Cytisus scoparius* and *C. pallidus* (see also laburnum, p. 480, and lupin, p. 491).

DAHLIA

Leaf Spot (*Entyloma dahliae* Syd.)

Symptoms. Of late years this disease has been observed in Denmark from time to time as well as over much of Central Europe. It is widespread in Britain and was first noticed in South Africa in 1912.

During summer it forms circular or elongated pale spots on the leaves that gradually grow larger and more yellow. Usually they are from $\frac{1}{16}$ – $\frac{2}{5}$ inch across but several may become united.

The middle of the spots dries a blue-grey or grey-brown colour. As a rule the sharply defined spots have a yellow-brown margin (Fig. 261).

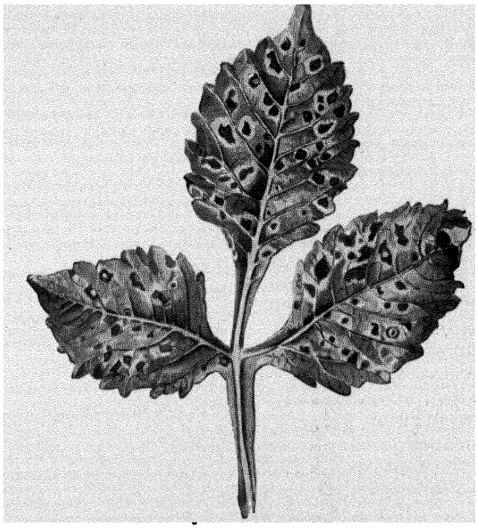


Fig. 261. Dahlia leaf spot.

They are best distinguished from other leaf spots by holding up to the light. Within the leaf tissue is a cluster of dark-coloured smut spores which are then seen as a dark spot. This fungus may also form oblong streaks on stems and petioles.

Cause. The spots are caused by the smut fungus *Entyloma dahliae*.

The smut spores germinate within the leaf, sending out hyphæ which penetrate to the surface and there give rise to small needle-shaped spores at their tips. These serve to spread the disease to other leaves or stems of dahlia.

Cactus dahlias are more severely attacked than the Pom-Pom varieties.

Control. The tops of diseased dahlias should be burnt in autumn and when the disease has occurred dahlias should not be planted in the same soil for several years. Growers should take care that no infected leaves remain attached to the tubers. If there are spots on the scales they too should be

Good control has been obtained by spraying with Bordeaux mixture 4: 4: 50 (Green, D. E., 1932).

Grey Mould (*Botrytis cinerea* Fr.)

In wet autumns grey mould may do a great deal of damage to dahlias. Leaves, stalks and especially the buds and flowers may be rotted

and covered with the grey mat of mycelium and spores. The fungus may also destroy tubers in store and cuttings before they strike (see grey mould, p. 59).

Sclerotinia sclerotiorum also sometimes attacks stems and tubers (see p. 77).

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

Verticillium infection causes leaves of hitherto healthy dahlias to wilt suddenly and then wither. The damage begins at the base of the plant and spreads upwards and is first seen in sunny weather. In the course of a few weeks all the leaves and young shoots shrivel up. A faint brown discolouration can be found in the vascular bundles of the tubers.

The disease was observed at Flensborg but not as yet in Denmark (see *Verticillium* wilt, p. 60).

Virus Diseases

At least three viruses are known to infect dahlia, though the diseases they cause are not always easy to distinguish.

Lycopersicum Virus 3

This, the cause of tomato spotted wilt, is very commonly found in dahlia, in which it causes light-green rings on the leaves. On some varieties these are clearly defined, especially on recently infected plants, and may be concentric with a dot in the middle. On other varieties and on old plants the rings are vaguer and may be mistaken for an ordinary mottle. They may even disappear but the plant remains infected and is a menace to any tomato crop in the vicinity. This virus is spread only by thrips (see tomato, p. 384).

Dahlia Mosaic (*Dahlia* Virus 1)

Is caused by a distinct virus which is spread by greenfly. There are no ring marks on the leaves of infected plants but the veins are banded with yellowish green. There may be distortion of the leaves, shortening of the internodes and stimulation of lateral shoots, giving bushy, stunted plants.

Cucumber Mosaic Virus (*Cucumis* Virus 1)

Also spread by greenfly, often causes mottling of the leaves in light and dark-green patches. On some varieties, however, it may cause ring and line patterns, less regular than the clear-cut concentric rings of the spotted wilt virus. Bishop of Llandaff shows no symptoms when infected but acts as a carrier of cucumber mosaic virus (see p. 278).

Control of virus diseases in dahlias can only be accomplished by starting on a small scale with virus-free foundation stocks and multiplying from these. When a virus-free stock has been obtained every care should be taken to keep it free from aphides and thrips which would introduce viruses. If possible the healthy stock should be kept in a separate glasshouse, regularly fumigated with nicotine. If grown out of doors they should be at least 100 yards from other dahlias and from any of the numerous ornamental plants and vegetables likely to harbour the viruses of tomato spotted wilt or cucumber mosaic (Smith, K. M., 1945).

DAPHNE MEZEREUM

Leaf Spot (*Marssonina daphnes* Magn.)

Irregular greenish spots appear on the leaves, which later turn brown and bear the minute pale conidial pustules of the fungus on both surfaces. If the petioles are attacked the leaves are often shed.

DELPHINIUM (Larkspur)

Mildew (*Erysiphe polygoni* DC. = *E. nitida* (Wallr.) Rabenh.)

Delphinium may be much attacked by mildew, which forms a white coating all over the plant. Not only is its appearance spoilt but the leaves wither prematurely, development of the flower buds is checked and they may wither without opening. The whole plant becomes small and deformed.

Control. Varieties differ greatly in susceptibility to mildew. Nurseries should discard varieties which experience shows to suffer greatly from it. In private gardens, too, it

may pay to remove the most susceptible varieties. According to Browarski and Förster the following are the most resistant varieties: Albrecht Dürer, Baldur, Berghimmel, Dein blaues Wunder, Enzianturm, Fernzünder, Havelland, Kirchenfenster, Morgenstrahl, Perlmutterspeer, Pfingstberg, Purpurritter, Rosenenzian and Sturmback.

If one wishes to save very susceptible varieties they should be treated frequently with a suitable fungicide from spring onwards (see mildew, p. 63). If they have been attacked they should be cut right down to the ground and the new shoots protected with a fungicide.

Mosaic (*Cucumis* Virus 1)

In England delphinium frequently suffers from a mosaic disease causing pale veins, stunted growth and reduction in flowering. The cause is the common cucumber mosaic virus which is spread by greenfly (see p. 278).

The plant also sometimes becomes infected by the virus of tomato spotted wilt which causes blackish spots and lines on the stems (see p. 384).

Black Blotch (*Pseudomonas delphinii* (E. F. Sm.) Stapp)

Delphinium leaves sometimes bear jet-black spots nearly an inch across (Fig. 262) and similar discolourations appear on stems and flower buds. The young spots are translucent. This disease is caused by bacteria which seem to overwinter in the soil and to be spread by rain splashes. If the plant is attacked early it is much damaged. In North America spraying the leaves and surface of the soil with Bordeaux mixture is recommended. Next spring the spraying is repeated as soon as the young shoots appear above ground, care being taken to wet the undersurface of the leaves. Infected plants should not be used for propagation.

Sclerotium delphinii Welch

This fungus causes the leaves to wither, the shoots die and at the soil level a lesion is found covered with greyish-white mycelium in which are small white sclerotia, which turn red-brown at maturity.

Diseased plants should be removed and destroyed along with the surrounding soil. Other control measures are as for *Sclerotinia* rot (see pp. 77 and 479).

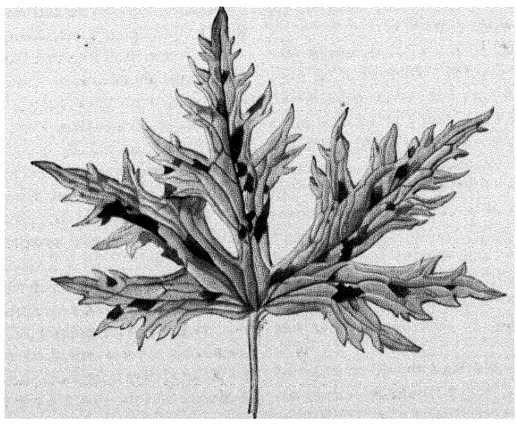


Fig. 262. Black blotch of delphinium.

DIANTHUS (Carnation and Pink)

Leaf Rot (*Heteropatella valtellinensis* (Trav.) Wollenw.)

This disease may occur on plants out of doors or under glass. It has caused much damage in Germany, Holland and England but not yet in Denmark.

Large discoloured spots appear on both sides of the leaves and sometimes on the stems as well. They are soft and brown or, when dry, glistening greyish-white marked with small dark dots. Leaves are attacked at the tips or in patches extending right across them. If the attack is very severe the plant may be killed.

Control. Only healthy plants should be used for propagation. They should be given plenty of light and air. Diseased plants should be removed and the rest sprayed or dusted several times with a fungicide.

Bud Rot (*Fusarium poae* (Peck) Wollenw.)

This fungus was formerly called *Sporotrichum poae* Peck until Wollenweber showed that it occasionally produces *Fusarium* spores and transferred it to that genus. One serious outbreak has been observed on carnations under glass in Denmark. The disease is well known in Germany and North America and has been fully studied there.

The buds rot, shrivel or swell up, or give only deformed flowers. When diseased buds are cut through they are found to be brown inside and also to contain tiny whitish mites belonging to the species *Pediculoides dianthophilus* Wolc. Apparently the mites carry the conidia of the fungus about with them and by its activities in rotting the buds it affords them better nutriment. The disease is therefore a complex one due to the combined activities of fungus and mites. They are both common on flower heads of grasses out of doors, especially on the meadow grasses (*Poa* spp.).

Control. Diseased buds should be removed and burnt. Dry, warm conditions check the fungus. Fresh turf should not be used in compost for carnations.

Dieback, Stem Rot and Wilt

It is a common experience in Denmark, England and elsewhere that carnation plants under glass become unhealthy and die. *Verticillium* wilt, *Fusarium* wilt, *Alternaria* and other fungi all occur in Denmark but opportunity has been lacking to study them in detail.

Investigations in England have shown that several different fungi are involved but the diseases they cause are so similar that they cannot always be distinguished without microscopic examination (Dowson, W. J., 1929; Wickens, 1935; White, 1936a, 1940).

Wilt (*Verticillium cinerescens* Wollenw.)

Symptoms. One or more shoots turn yellow and wither and finally the whole plant dies. In the vascular bundles of the part first affected there is a brown discolouration but no rotting of the stem tissues.

As a rule the disease first appears only on isolated plants but by the second year it has often spread extensively.

Cause. *Verticillium cinerescens* has been shown to be the commonest cause of wilt and death of carnations under glass in England. Van Beyma suggested in 1939 that the fungus should be renamed *Phialophora cinerescens* (Wollenw.) Van Beyma but it does not appear to fit well in either *Verticillium* or *Phialophora*.

According to White the fungus can penetrate unwounded roots. Soil infection can take place even after the top soil has been renewed. Infection from plant to plant above ground does not usually occur. Probably the disease is spread by cuttings more often than is generally supposed, for Wickens has shown that the fungus may be present in shoots which have no visible symptoms of disease inside or out.

Fig. 263 shows the results of examination of a plant which had a withered shoot at the topmost node of the main stem, resulting from inoculating with *Verticillium* at the base of the plant. No other shoots showed symptoms of disease. In the diagram the shoots with no outward signs of disease are shown by straight lines, the withered one by a curved line.

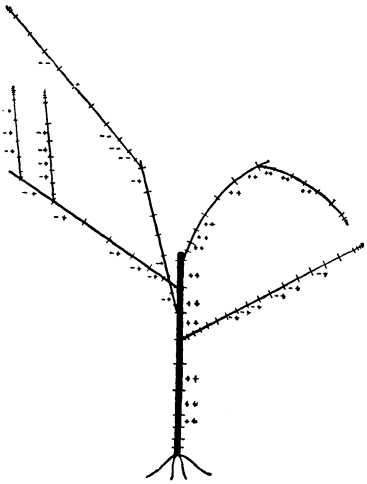


Fig. 263. Diagram of carnation plant inoculated with *Verticillium cinerescens*. Explanation in text.

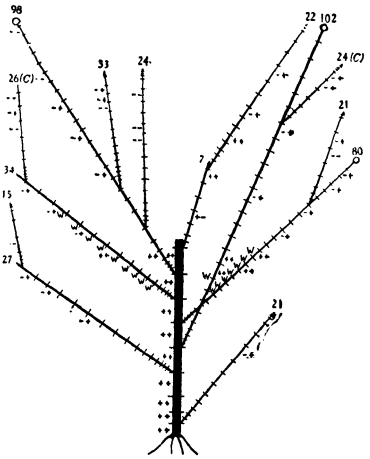


Fig. 264. Diagram of carnation plant naturally infected with *Verticillium cinerescens*. Explanation in text.

In Fig. 264 are shown the results of similar examination of a one-year-old plant of the variety Mrs. A. J. Cobb, naturally infected with *Verticillium* wilt and showing the first very clear indications of wilt. No shoot was completely dead but individual leaves had withered and the shoots bearing these are marked *W*. Numbers give lengths in centimetres. *C* indicates shoots that appeared quite suitable for cuttings.

In both diagrams changes in the vascular bundles apparent to the naked eye are marked by + outside the shoot concerned. A minus sign signifies no apparent change. The other sign indicates whether the organism was isolated (+) or not (—). The diagrams clearly show that the fungus spread much farther through the plant than the visible signs of disease.

White gives an equally impressive proof of the importance of taking cuttings from completely healthy plants. From a bed in a nursery where there were wilted carnations he took from apparently healthy plants 67 cuttings on one occasion and 72 on another. He found that actually 7% and 15% of these cuttings respectively were infected with *Verticillium*.

In inoculation experiments White found that the fungus grew about ¼ inch up the plant each day, more rapidly in summer than in autumn, and that a two-year-old plant would therefore wither completely about 3 months after infection.

He was unable to infect tomatoes, chrysanthemums or *Callistephus* with the carnation wilt fungus, though others have regarded the last-named as a host of the fungus.

When beds were replanted widespread symptoms of the disease appeared in 4 months; if the top soil had been renewed the disease first appeared after 10–14 months.

Control. Wickens obtained good, but not invariably complete, control by soil disinfection with 1 : 50 formalin solution at the rate of 6 gallons per square yard. The soil should be covered for 2 days with sacks soaked in formalin and then ventilated for 2–4 weeks until the smell can no longer be detected.

Steam sterilization of the soil has also not always been completely effective.

If the house is resoiled one is likely to obtain soil free from the fungus, but there can be no certainty of this. As mentioned above, the fungus may possibly occur on *Callistephus* and no doubt it has other hosts besides carnations.

One may try to prevent infection of the deeper layers of soil by laying down paper or chips of stone or by cementing the bottom of the beds. Carnations seem to do well with a very shallow layer of soil but obviously some provision must be made for drainage by inserting tubes or making holes of some kind in the isolating layer.

White thinks it possible to starve the fungus out by growing tomatoes in infected soil for several years in succession.

As the fungus can be found in apparently healthy cuttings and be carried with them from the propagating bed to the carnation house it is safest to keep the mother plants in pots. In spite of the trouble involved this should be done at least until the nursery is free from the disease. Newly bought plants used for propagation should always be kept in pots. Any mother plant that shows the slightest sign of disease should be destroyed at once.

White has pointed out that drainage water from the beds should not be allowed to contaminate the watering water and has stressed the risk of infection from tools. Workers should not work alternately in diseased and healthy houses.

Fusarium Wilt (*Fusarium dianthi* Prill. & Delacr.)

This fungus tends to block the vascular strands and causes a kind of wilt with yellowing and withering of the leaves. The brown stain in the vascular bundles is much more distinct than with *Verticillium* and extends as far as the fungus has penetrated. There is, therefore, no risk of spreading this disease by taking cuttings from plants that show neither wilting nor browning of the vascular bundles. It is easy to reject those showing these symptoms.

Fresh or disinfected soil should be used.

Stem Rot (*Fusarium culmorum* (W. G. Sm.) Sacc. and other species)

When cuttings are inserted in soil or sand infected by the above fungi they are apt to rot at the base. Carnation cuttings should only be struck in freshly washed sand which should be discarded after use. These fungi can also attack rooted plants and cause rotting of the stem so that it falls over. The cortical tissues are decayed but the vessels are seldom blocked as in *Fusarium* wilt.

When these species of *Fusarium* occur in older plants it is usually because they are already suffering from attack by *Verticillium cinerescens* or *F. dianthi.*

Carnation cuttings may also be rotted by *Corticium solani.*

Dieback (*Fusarium culmorum* (W. G. Sm.) Sacc.)

When the blooms are cut the cut ends of the stalks left on the plant may start to rot. They shrivel up and turn dark purplish-brown. Gradually the stem dies back and the shoots and leaves on it wither. One should, therefore, try to avoid leaving short bits of stalk behind. Wickens has demonstrated that *F. culmorum* can always be isolated from these bits of dead stem and that when inoculated into plants it produces the characteristic symptoms. The disease is common in carnation houses but is of little economic importance.

In inoculation experiments *Verticillium cinerescens* has caused a similar dieback but this seems less common in practice.

Black Mould (*Alternaria dianthi* Stev. & Hall, etc.)

The mould *Alternaria dianthi* apparently causes round or irregular raised spots on carnation leaves. Their centres become covered with a black layer of spores. Ultimately the leaves wither. From the base of the leaves the fungus often attacks the stalks, girdling them so that the part above wilts and dies.

Too much moisture is to be avoided and spraying with a copper fungicide may be tried.

Mildew (*Oidium dianthi* Jacz.)

Carnation mildew forms a mealy white or yellow coating, sometimes quite thick and cushion-like, on leaves, stem and calyxes of carnations under glass. In England it is seen specially on Lady Arlington, Bridesmaid and British Triumph. It seems to have been brought to Denmark with carnation cuttings in 1931. We have seen a case where it was almost confined to plants standing against the side wall of the house.

Control. One may try spraying with a copper ammonia solution (4 oz. copper sulphate + 4 oz. triplex liquid ammonia in 24 gallons water) or with 2:100 lime sulphur or dusting with sulphur (see also mildew, p. 63).

Rust (*Uromyces dianthi* (Pers.) Niessl)

Carnation rust has been known in Europe since 1789 when it was described by Schrank. One seldom sees a carnation crop which has not been attacked to some extent, at least in the dark months.

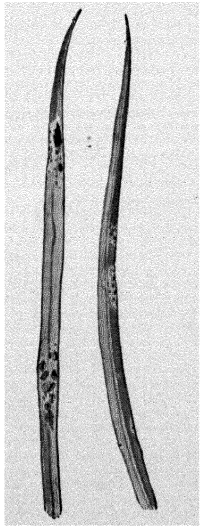


Fig. 265. Carnation rust.

Symptoms. The rust pustules appear on both sides of the leaves and sometimes also on the stems. The attack is apt to start on the lower leaves. First small yellow blisters appear, which then burst and expose a reddish-brown powder, the uredospores. Later on, when the teleutospores develop, the spore powder is dark brown. Surrounding the spore mass are the edges of the leaf skin and outside this again is a yellow zone of leaf tissue (Fig. 265).

Control. The fungus spreads easily and it is, therefore, important to avoid introducing it into a hitherto healthy crop. One should not only inspect newly bought cuttings and plants very carefully but should keep them away from the others for a few months after they are received.

Cuttings ought not to be taken from infected plants. If that is unavoidable the risk can be reduced by dipping the cuttings in a solution of 3 oz. potassium sulphide in 2½ gallons of water.

By growing the crop in suitable houses and keeping a good distance between the plants it is possible to ensure that they have plenty of air and light. By avoiding wetting the plants themselves, by watering in the morning, by ventilating and heating at night the above-ground parts can be kept dry. The soil should be dry on the surface but moist lower down. The lower leaves should be supported above the soil by a network of threads.

Growth should be good, firm but not too close. It should neither be soft because of too much nitrogen nor hard because of too high a temperature in relation to the light intensity.

Direct control by spraying or dusting is rather ineffective so all possible care should be taken to avoid introducing the disease.

Particularly susceptible varieties should be avoided. Pape (1939) mentioned as particularly susceptible Betty Lou, Cardinal, Early Dawn, Enchantress, Supreme, Harvester, Jubilee, Meta, Princess Marie Scott, and as very resistant: Donald, Eldora, Fairy Queen, Golden Glow, Ivory, Lawson, Liebling, Pink Abundance, Pink Eldora and White Matchless. Very different opinions have been

expressed regarding the susceptibility of Enchantress. In America it is regarded as somewhat resistant while in England and Germany it is considered susceptible.

Different physiological races of the fungus exist and thus a variety severely infected at one place may not be so at another. Hence one must not take statements about resistant varieties too seriously.

Control with fungicides has been recommended from time to time but very satisfactory results are not to be expected. One may try spraying with potassium sulphide ½ or 1% plus a spreader, or dust with sulphur. Spraying should be done below the flowers to avoid spotting them. In North America spraying with a solution of common salt has been recommended but the strength was not stated. 2 lb. in 20 gallons may be tried and the strength gradually increased if the plant can stand it.

Whatever treatment is tried it should be repeated at fortnightly intervals. Sulphur dust is apt to make the soil acid in time so its reaction should be checked from time to time. The soil reaction should preferably be pH 6.5 or a little higher and if necessary lime should be added to counteract excess acidity.

Soil in which a diseased crop has been grown should not be used again for carnations.

Ring Spot (*Didymellina dianthi* Burt = *Heterosporium echinulatum* (Berk.) Cooke)

This disease has caused serious damage from time to time in Denmark both in carnation houses and especially in the outdoor crop, particularly on Chabaud varieties. Another physiological race may attack Sweet William (*Dianthus barbatus*) (Burt, C. C., 1936).

In America the fungus attacks especially Matchless under glass, while Aviator is less susceptible and Enchantress and Supreme least of all. The less vigorously growing varieties are most susceptible.

Symptoms. Round or oval grey spots with a dark-reddish margin develop on all the green parts of the plant. Gradually the dark conidiophores of the fungus break through the skin, first in the middle of the spot, then in concentric rings, while the edge remains greyish (Figs. 266 and 267). The spots often join together and in severe outbreaks the leaves wither. The flowers develop poorly and the stalks may snap just above the spots. Beneath the skin of the spots the fungus forms a resting mycelium which can survive the winter.



Fig. 266. Carnation ring spot, young spots on flowering shoot, part of two leaves with older spots.

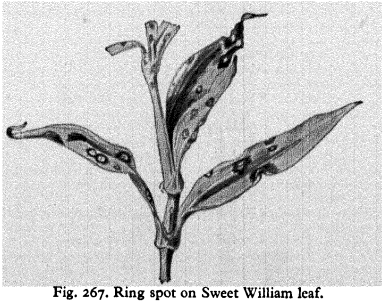


Fig. 267. Ring spot on Sweet William leaf.

Ring spot may spread very quickly and do a great deal of damage. It is favoured by moisture and warmth.

Control. Clean soil should be used and cuttings should only be taken from healthy plants. The houses should be kept dry and well ventilated and the plants should make firm growth. Spraying with Bordeaux mixture may be helpful but it is apt to spot the plants. Burgundy mixture has given good results in Denmark. Though scarcely as effective as Bordeaux mixture it is preferable because it causes less spotting. Spraying should be done just below the buds. Possibly other copper sprays that do not cause spotting could be tried.

Formalin 1:200 has been tried without success; it did not prevent production of fresh conidia on the sprayed spots.

Anther Smut (*Ustilago violacea* (Pers.) Fuck.)

Biological races of this fungus occur on very many species of caryophyllaceous plants, in Britain especially on lesser stitchwort and the common red and white campions. The only disease of economic importance, however, is that caused by the race which infects the perpetual-flowering carnation and causes anther smut. This disease has been known in Britain since 1907 and has been fully described by White (1936b).

Infection probably occurs, as in other races of the fungus, by spores falling on axillary buds. The fungus permeates the carnation tissues and stimulates the axillary buds to develop before the terminal bud opens. Infected stems thus have a characteristic bushy appearance. Infected flower stalks are stunted and the developing bud is thick and squat, often with a split calyx. The anthers are filled with blackish-purple dust instead of pollen and the ovaries are aborted. When the flowers open they appear sprinkled with the sooty smut spores.

Control. Once a plant is infected it apparently always remains so. Diseased plants should be removed before the buds open. Diseased buds should never be allowed to bloom or they will shed their spores and spread the disease. Cuttings should only be taken from healthy plants and struck in clean sand in propagating houses where there are no infected carnations.

Split Calyxes

Split calyxes occur especially in certain varieties when the plants are kept where it is too dark or are exposed to moisture and to fluctuations in temperature. They may also be caused by an overdose of nitrogen.

The deformity may be corrected to some extent by slipping a grey-green rubber ring inside the calyx lobes to hold the flower together.

Summary of Dianthus Diseases

I. Stems		page
A. Brown cortex, cuttings decay at the base	Stem Rot	450
B. Purplish-brown dieback where blooms have been cut	Dieback	450
C. Stem girdled at base of leaves, black mould seen under lens	Black Mould	450
D. No decay but leaves turn yellow and wither and vascular bundles are brown	*Verticillium* Wilt	448
	Fusarium Wilt	450
E. Stems spotted:		
(1) Mealy patches of white or yellowish mould	Mildew	451
(2) Brown powdery pustules	Rust	451
(3) Grey spots with dark rings and red edges	Ring Spot	452
F. Axillary buds breaking into leaf, stems bushy with leaves before blooming	Anther Smut	453

II. Leaves
- A. Yellow, withering on one or more shoots:
  - (1) No decay of stems but vascular bundles stained brown ..... *Verticillium* Wilt 448
    Fusarium Wilt 450
  - (2) Purple-brown decay of cut stumps ..... Dieback 450
- B. Spotting:
  - (1) White or yellow mealy patches ..... Mildew 451
  - (2) Yellow blisters opening to expose brown spore masses ..... Rust 451
  - (3) Leaves arched with a white spot on the highest part ..... Fume Damage 45, 50
  - (4) Grey spots (black mould visible under lens) .... Black Mould 450
  - (5) Round or oval spots greyish with dark rings and red edges ..... Ring Spot 452
  - (6) Tips or bands across leaf soft and brown, drying greyish white, dotted with black pycnidia ..... Leaf Rot 448

III. Flowers
- A. White or yellow mealy spots on calyx lobes ..... Mildew 451
- B. Buds swollen, shrivelled or decayed, often inhabited by mites ..... Bud Rot 448
- C. Calyx split ..... Split Calyx 453
- D. Anthers filled with sooty spores ..... Anther Smut 453

DIGITALIS (Foxglove)

Leaf Spots

Ramularia variabilis Fuck. causes spotting, especially of the lower leaves. The spots are angular, individually $\frac{1}{16}$–$\frac{1}{4}$ inch across, but often several join together. On the upper surface of the leaf they are snuff brown with redder edges, on the under-surface they are grey-brown.

Gloeosporium digitalis Rostr. causes large dark brown spots visible on both sides of the leaf.

Both these fungi occur on foxglove in Denmark, others cause leaf spots on it elsewhere.

Spraying or dusting with fungicides may be tried against them all.

DOUGLAS FIR

See *Pseudotsuga*, p. 515.

DRACAENA

Coniothyrium concentricum (Desm.) Sacc., *Phyllosticta dracaenae* Griff. & Maubl. and several other fungi may cause leaf spots of various colours and sizes. Copper fungicidal sprays may be tried against them.

Grey mould (*Botrytis cinerea* Fr.) causes extensive rotting. Keep the plants as dry as possible (see also grey mould, p. 59).

ECHEVERIA

Leaf Blotch (*Cladosporium herbarum* Link f. *agave-echeveriae* Savelli)

This fungus has been credited in Italy with causing yellowish spots covered with olive-coloured mould on leaves of *Echeveria* and *Agave americana*. Infected *Echeveria* leaves may be killed (Savelli, 1913).

We have not seen this disease but there have been several reports of growers abandoning cultivation of *Echeveria retusa* on account of some unspecified disease.

Rust (*Endophyllum sempervivi* (Alb. & Schw.) de Bary

In the spring this fungus forms two kinds of fructifications on leaves of *Echeveria* and *Sempervivum*. Minute, closed, orange-coloured spermogonia are scattered amongst much larger cup-shaped pustules of yellow-brown teleutospores. These resemble the aecidiospores of other rusts but germinate like teleutospores. The fungus overwinters as mycelium in the plant tissues and causes the leaves to be abnormally narrow, erect and yellow (Ashworth, D., 1935).

This rust occurs occasionally in Britain and is said to be common and often destructive in Germany.

Plants bought from abroad should be carefully examined and preferably kept in quarantine. Those severely attacked should be destroyed and the others dusted at frequent intervals with a fungicide (see rust, p. 74).

ELM

See *Ulmus*, p. 555.

EPIMEDIUM

Mosaic Disease

Epimedium in Denmark is often found to have conspicuously spotted leaves, with restricted growth and flowering (see virus, p. 83).

ERANTHUS (Winter Aconite)

Smut (*Urocystis anemones* (Pers.) Rabenh.)

May cause black blotches on winter aconite leaves (see *Anemone*, p. 411).

ERICA (Heath)

Wilt (*Phytophthora cinnamomi* Rands)

Symptoms. This disease has caused extensive losses of cultivated erica in England. The first symptom is a grey hue on the leaves. Then one or more branches begin to wither, often one side of the plant or even the whole. The leaves turn brown and the dead stems and roots are brown right into the wood. Wilt affects *E. hiemalis*, *E. nivalis* and *E. willmoreana*.

The disease does not appear in the propagating beds or boxes, nor in quite young potted plants, but usually breaks out 6–8 weeks after the plant has been planted out.

Cause. According to Oyler and Bewley (1937) the disease is caused by *Phytophthora cinnamomi*. Further studies have shown that *P. cactorum* may also cause it.

P. cinnamomi was found to attack also antirrhinum, calceolaria, schizanthus and beech seedlings and to rot apple and tomato fruits. Elsewhere it has been found on various shrubs (see rhododendron, p. 518).

The fungus has been found in water tanks and in peat litter which has become mixed with soil from pots containing a previous erica crop.

Control. Efforts must be made to prevent pollution of the water supply. The tanks should be emptied and thoroughly cleaned, after which they and their surroundings should be sprayed with 1% formalin solution. The tank should be filled with this solution to the level of the pipes, covered for two days with sacks and then emptied and washed with clean water.

Compost containing remains of diseased plants must not be used. It must be remembered that the fungus may be present on other plants besides erica.

Beech leaf mould may also be infected, for the fungus is able to attack beech seedlings.

Soil disinfection before planting out may be attempted by watering with 1% copper sulphate solution at the rate of a little over a gallon per square yard.

Heath Mildew (*Oidium ericinum* Erikss.)

The whitish or greyish coating of this fungus on leaves and young shoots may cause the former to turn brown and fall off. Badly affected plants may be killed (see mildew, p. 63).

Cultural Troubles

When erica does not flourish it is often difficult to assign a specific reason for its failure. The plants become stunted and wither, and sometimes the stem becomes dark-coloured.

In most cases unfavourable growing conditions are the cause of the trouble. According to Gleisberg the best soil reaction for erica is pH 3.5–4.5. If alkaline manure or gravel containing limestone is used the soil reaction may easily become too high, even if heath soil, peat litter or similar substances have been mixed with it. The reaction may also gradually be raised by watering with alkaline water.

Fahrenkamp thinks failure of erica is solely due to lack of nourishment, the plant has an especially high potash requirement. According to T. Bacher *E. hiemalis* does quite well at pH 6.

EUONYMUS JAPONICA

Mildew (*Oidium euonymi-japonicae* (Arcang.) Sacc.)

This fungus is very common on leaves of *Euonymus* hedges especially in the South and West of England. For control see mildew, p. 63.

EUPHORBIA PULCHERRIMA (Poinsettia)

Curl

In Germany Pape (1927b) has described a disease of *Euphorbia pulcherrima* in which leaves become curled, distorted and somewhat spotted; the stem may be curved and the red bracts small and twisted. The cause is probably a virus but which one was not determined. Plants so affected are often seen in Denmark. They should not be used for propagation.

EVERLASTING FLOWERS

See *Helichrysum*, p. 469.

FAGUS (Beech)

Damping Off (*Phytophthora cactorum* (Leb. & Cohn) Schroet.)

The cotyledons of beech seedlings growing under damp conditions may often be marked by large dark-brown blotches, often surrounded by a fine white zone of mould. The stem may also be attacked and the seedling is then killed. The sporangia of the water mould blown from diseased cotyledons germinate on other beech seedlings and in four days a fresh infection may be established. In nurseries, and in beech woods after a good mast year, the fungus may be very destructive if the early summer is wet. Seedlings that have been killed contain the resting oospores of the fungus which retain their power of germination for several years.

In nurseries an outbreak can be forestalled by spraying with Bordeaux mixture, repeating the spray at 10–15-day intervals. In woods dusting with a copper lime dust is easier.

The fungus on beech has been described as a separate species, *P. fagi* Hartig, but it seems to be really only the common *P. cactorum* which can attack so many different plants (see pp. 480 and 361).

Honey Fungus (*Armillaria mellea* (Vahl ex Fr.) Quél.)

The honey fungus is one of the best-known toadstools because it is common and world-wide in distribution and can attack such a large number of plants. It may attack any forest trees grown in Britain, many shrubs and occasionally herbaceous crops like potatoes. In Danish forests it has been destructive to young common spruce planted where beech woods have been cut down.

The name " honey fungus " and the Latin name *mellea* are given because of the honey-coloured, slightly scaly cap. The fungus has white gills, a conspicuous ring round the stem, and occurs in dense clusters, on and round infected and killed trees. Often extensive sheets of bark are loosened by it, and beneath them will be found a dark-brown or black network of rhizomorphs of the fungus (Fig. 268). They have been compared to leather laces. Higher up the trunk the mycelium spreads out in close white fan-shapes between the bark and wood. The crowns of infected trees die back and their foliage is scanty. If



Fig. 268. Rhizomorphs of *Armillaria mellea* under bark of apple tree (*Photo Plantenziektenkundige Dienst*).

the tree is cut, or falls in a gale, the wood is found to be dry, white and friable. Freshly broken pieces shine with a phosphorescent glow in the dark. The mycelium may shine similarly, especially in pure culture.

The rhizomorphs combine the toughness of sclerotia with the capacity of growing for considerable distances through the soil, as brown root-like bodies, paler inside and at their tips. On the gills of the toadstools spores are produced in millions, but the actual infection of a root seems always to be made by a rhizomorph.

The honey fungus is not an obligate parasite; it can thrive for years on dead stumps and roots decaying in the soil. The edge of a wood may be full of rhizomorphs. Young trees planted in such situations are often attacked and killed; the damage is often discovered only when the beech dies during the summer. When old trees are attacked the struggle against the fungus may last many years. We have seen trees swarming with fungi at the root regain a dense crown after being given light and air.

Tree roots become infected partly through wounds, partly where an unhealthy root is in contact with a sound one, but the rhizomorphs can also penetrate directly through normal root bark. It has been claimed that physiologic races of the fungus occur (Childs, L., and Zeller, S. M., 1929), but most observations indicate that it is not at all specialized and can attack many victims.

Relative susceptibility of hosts. Among coniferous trees common spruce is easily attacked but silver fir is very resistant. Larch and green Douglas fir are fairly resistant. *Thuja* and *Juniperus* may be attacked.

Beech is very prone to infection in old plantings, oak may be severely attacked when weakened by insects or mildew. Rowan (mountain ash) and poplar are particularly susceptible.

In parks and gardens old trees of many kinds are often attacked by the honey fungus. So are shrubs like rhododendron, azalea, euonymus, olearia, privet, the roots of which may be covered in rhizomorphs, and even the common heather (*Calluna*) (Alcock, N. L., and Wilson, M., 1927).

Fruit trees and bushes are especially exposed to infection when planted on the edge of a wood. We have seen apple trees die from the attack in the course of a couple of years. Pear trees are said to be fairly resistant, at least on certain subsoils. Plum may be severely attacked, when the wood of the roots develops cavities filled with gum (Thomas, H. E., 1934). Apricot, peach and almond apparently suffer extensively in France. Wøsterdijk has described attacks in young cherry trees which developed yellow leaves shed early in their second year and died in their third year. The fungus came from infected stakes used to support the trees. The cherry roots were brittle and there were toadstools attached to the collar though the trunk remained healthy. Walnut may be killed by *Armillaria* but *Juglans Hindsii* is said to be resistant. Vines have been attacked in Holland, France and England. Gooseberries, currants and

raspberries may also be destroyed. One should look for rhizomorphs in the case of unthrifty or dying fruit trees, though gnawing by rodents may often be the cause of failure.

Herbaceous plants are occasionally attacked.

Moore (1948) has described brown rot of potato tubers by *A. mellea* in a crop grown on land that had been cleared of wood two years before. Carrot, rhubarb, hop, strawberry, dahlia, lupin and paeony have all been attacked on a few occasions.

Control. When the attack is discovered it is usually too late to do anything. Attempts have been made to limit spread of the rhizomorphs by digging trenches, or making sunken walls of cement, but usually without much success. For possible control by carbon disulphide, see p. 593.

Clearing out stumps and main roots left by diseased trees will minimize the danger of infection spreading. Where fruit trees are to be planted on land cleared of wood all stumps should be carefully dug out. Where expense is a secondary consideration soil disinfection after clearance may be tried (p. 577). For replanting the most resistant species of trees should be chosen. Fences and stakes should be from resistant trees if possible or they may carry rhizomorphs.

Heart Rot (*Ganoderma applanatum* (Pers. ex Fr.) Pat.)

This very common bracket fungus, with a hard smooth brown crust on its upper surface, tough red-brown flesh and tiny white pores, turning brown with age, over the lower surface, is very common on beech trunks. It causes a white rot of the heart wood.

Root Rot (*Phytophthora cambivora* (Petri) Buism., etc.)

Phytophthora cambivora and its allies *P. cinnamomi* Rand and *P. syringae* Kleb. cause root rot and death of beech and sweet chestnut trees both in Britain and on the Continent. The parasites attack the trees from the soil, either by way of small roots or near the root collar. They live mainly in the cambium and inner cortex and spread through the roots and a short distance up the trunk. In the latter the fungus occurs especially between the root buttresses, where its probable presence may be indicated by dead or discoloured bark. On sweet chestnut the tree may be girdled at the root collar, the leaves wilt and the tree dies in the first year of obvious disease symptoms. In beech and often in sweet chestnut an infected tree shows early yellowing of the leaves, which then turn brown over part or all of the crown. Leaves on such trees are not appreciably dwarfed but at the base of the trunk a zone of dead or dying bark is found, spreading up from soil level. From this there may be exudation of fluid, blackish in the case of sweet chestnut, whence the name "ink disease" sometimes used. In beech the fluid is not black nor is the root bark stained blue-black as in diseased chestnut.

Phytophthora root rot is associated with moist, water-retentive soils, especially where a shallow, well-drained soil rests on subsoil with impeded drainage. Control measures include good drainage and avoidance of planting susceptible trees, especially sweet chestnut, on ill-drained soils. *Castanea crenata* is said to be much less susceptible than *C. sativa* (Day, W. R., 1938, 1939).

Root rot of beech on shallow chalk soils is often associated with the black, crust-like pyrenomycete *Ustulina vulgaris* (see *Tilia*, p. 537).

FICUS

Propagation Fungus (*Moniliopsis aderholdi* Ruhl.)

In a nursery in North Zealand many plants of *Ficus pumila* were attacked by *Moniliopsis* in 1934. The leaves withered and fell and in a favourable light one could see the fungus growing in a fine web between the plants.

In this case dusting with sulphur checked spread of the fungus (see further, p. 73).

FILICES (Ferns)

Leaf Spots

Outdoor ferns are attacked by a number of leaf-spotting fungi none of which do any real damage. One of the best known is *Corticium*

anceps (Bres. & Syd.) Gregor which sometimes kills bracken fronds in wet shady places. It forms sclerotia very like those of *C. solani* on the dead leaves. A great many rather primitive rust fungi have their teleutospore stages on ferns and their aecidial stages on firs (*Abies* spp.) (Hunter, L. M., 1936). They may be destructive to the firs but do little or no damage to the ferns. *Milesina blechni* P. Magn., *M. dieteliana* P. Magn. and *M. scolopendrii* Jaap are particularly common in Britain, causing conspicuous brown spots and blotches on the fronds of hard fern (*Blechnum*), common polypody (*Polypodium*) and hart's tongue (*Scolopendrium*) respectively. *M. Kriegeriana* P. Magn. causes blackening of fronds in buckler fern (*Aspidium dilatatum*) and male fern (*A. filix-mas*), especially in Scotland where its aecidial stage occurs on *Abies pectinata* and other firs. The lady fern (*Asplenium filixfoemina*) is occasionally attacked by the mould *Cercosporella filicisfoeminae* (Bres.) Von Höhnel.

Septocylindrium aspidii Bres. sometimes forms a white bloom over fronds of *Aspidium* spp. (Dennis and Wakefield, 1946).

Glasshouse ferns, though often attacked by leaf eelworm, very seldom suffer from fungi. Prothalli and sporelings may, however, be destroyed by the propagation fungus (p. 423) and damping-off fungi (p. 68). The fungus *Completoria complens* Lohde seems to be a specialized parasite of fern prothalli (Atkinson, G. F., 1894). It attacks small groups of cells which turn yellow, then brown and finally black. These groups may be dotted over the surface of the prothallus and give it a mottled appearance. Eventually they may drop out leaving it riddled with holes.

FIR

See *Abies*, p. 407.

FORGET-ME-NOT

See *Myosotis*, p. 494.

FORSYTHIA

Leaf Spot

Phyllosticta forsythiae Sacc. causes large, dead, brown-coloured spots and *Marssonina forsythiae* Lind large, dark, grey-brown spots on *Forsythia* leaves in Denmark. The former is known to occur in Britain. If necessary infected plants may be sprayed or dusted with a fungicide.

Dieback (*Sclerotinia sclerotiorum* (Lib.) de Bary)

Osterwald described this disease in Switzerland in 1905. In wet weather the previous year's branches withered, together with shoots of the current year, and on them appeared a white mycelium in which black sclerotia were gradually produced. From these he was able to obtain ripe apothecia and ascospores, thus identifying the fungus as *Sclerotinia sclerotiorum*. It is exceptional for this species to attack a woody plant. *Botrytis* appeared on the dead shoots but this was a secondary invader as inoculation experiments with it did not reproduce the disease.

A similar disease has occurred on *Forsythia* in Jylland, Denmark, but its cause was not investigated.

The only control measure recommended is to cut out and burn the diseased shoots (see also p. 77).

Bacterial Blight (*Pseudomonas syringae* Van Hall)

On young succulent shoots infection produces black lines and sometimes spotting of the leaves. The shoots may blacken and die. A brown stain appears in the wood, usually throughout a whole branch, which may die back. Affected branches fail to bloom if cut for forcing (Metcalfe, G., 1939) (see p. 535).

Stem Galls

Upright shoots of *Forsythia* are prone to develop prominent, irregular, woody nodules which may be so numerous as to render the plant unsightly. According to Thompson (1946) these are structures analogous to the burr knots of fruit trees, and give rise very rapidly to roots when submerged in water. In North America similar structures have

been ascribed to parasitism by species of *Phomopsis* (Brown, N. A., 1938).

FOXGLOVE

See *Digitalis*, p. 454.

FRAXINUS (Ash)

Canker (*Nectria galligena* Bres. etc.)

The same fungus that causes apple canker may produce cankers on ash. Large cankers occur on branches and on the main trunk, often more regularly ridged than those on apple trees. The red perithecia of the fungus are usually common on the dead bark (see apple, p. 113). The condition seems to be worse on poor wet soils. In manurial experiments with young trees lime had a favourable effect, potassium and nitrogen were particularly beneficial to growth, while phosphoric acid had no particular effect.

Cankers on ash may also be caused by the pycnidial fungus *Phomopsis scobina* (Cooke) Van Höhnel (Macdonald, J. A., and Russell, J. R., 1937). On young twigs this kills the terminal bud, stimulating growth of the laterals. If the twig is killed back to the main branch a canker is formed which may extend indefinitely. A related fungus *P. controversa* (Sacc.) Trav. is a feeble parasite causing only dieback of small twigs.

Bacterial Canker (*Pseudomonas savastanoi* var. *fraxini* N. A. Brown)

This canker is much more open, irregular and often deeper than that caused by *Nectria galligena*. It starts as a small crack in the bark with a thickened margin but may grow into an irregular fluted outgrowth several inches across, with an open cavity extending into the wood and increasing in size from year to year (Brown, N. A.). This form of ash canker seems to be the more common in Britain (Fig. 269).



Fig. 269. Ash canker.

Heart Rot (*Polyporus hispidus* Fr. and other fungi)

Polyporus hispidus Fr., a large shaggy reddish-brown bracket fungus, with spongy flesh and a thick layer of small pores on the under-surface, is very common on old ash trees. It causes a yellowish rot of the heart wood in trunk and branches. Less common is *Fomes fraxineus* Fr., a harder, smooth, bracket fungus with pale-yellowish, woody flesh, which causes a brittle white rot of the heart wood at the butt. Very common on dying branches is the black, hemispherical, pyrenomycete *Daldinia concentrica* which may be up to 2 inches across. This is a feeble parasite causing a blotchy white rot of the wood in the branches, marked with black specks and lines. The toadstools *Armillaria mellea* (see p. 456) and *Pholiota adiposa* Fr. also cause rotting of the butts of old ash.

FREESIA

Leaf Spot (*Didymellina macrospora* Kleb. = *Heterosporium gracile* Sacc.)

Small oval brown spots with a pale margin appear on the leaves (see iris, p. 477).

Other small oblong brown leaf spots are often seen on freesia, also withered leaf tips and other discolourations. The cause of these

is unknown but they are apparently connected with some unfavourable soil condition.

Corm Rot (*Fusarium bulbigenum* Cooke & Mass. etc.)

Six different kinds of *Fusarium* may attack freesia corms and cause rotting leading to yellowing, wilting and death of the foliage. The species most concerned in North America are *Fusarium bulbigenum* Cooke & Mass. (see narcissus, p. 496), *F. solani* var. *Martii* f. 1 Wollenw. and *F. moniliforme* Sheld.

Infected soil should be avoided and lifted corms carefully sorted and the sound ones stored dry. It is a good plan to put sand instead of soil over the corm when planting.

Mosaic Disease (*Freesia* Virus 1)

Not much work has yet been done on diseases of freesia and there is still much to be learnt about them. In 1927 Longford described a destructive disease which attacks the plant in many parts of the south of England. When the plants have developed 2 or 3 leaves one or more of them shows brown spots; in the course of a few days the spots spread and the whole leaf withers, becoming white and papery. Severely affected plants die before flowering, in others the blooms do not open or are distorted. No fungus was found to be associated with the trouble and its cause was not determined. Subsequently K. M. Smith (1937) concluded this to be a virus disease and attributed it to what he called *Freesia* Virus 1. Nothing is known about this virus, however, except that corms from diseased stock give rise to affected plants the following year.

Dry Rot (*Sclerotinia gladioli* Drayton)

This causes the corms to develop dark sunken spots and shrivel up. On growing plants the disease causes death of the foliage, accompanied by the appearance of minute hard black sclerotia, just visible to the naked eye, on the base of leaves and stalks (see p. 466).

Septoria gladioli (see p. 465) and *Penicillium gladioli* (see p. 465) may also attack freesia.

FRITILLARIA IMPERIALIS (Fritillary)

Rust (*Uromyces lilii* Fuck.)

Spots appear on the leaves, bearing pustules of pale-yellow, later brown, dusty spores (see rust, p. 74). This disease is not yet known in Britain.

Fritillaries may also be attacked by *Sclerotium tuliparum* (see p. 542) and *Sclerotinia bulborum* (see p. 470) as well as by lily mosaic (see p. 489).

FUCHSIA

Rust (*Uredo fuchsiae* Arthur & Holway)

Affected leaves show vague pale patches on their upper surface, while dotted over the corresponding parts of the lower surface are numerous pale-orange pustules of uredospores. The lower leaves are most affected and may wither and fall off. This rust occurs on fuchsia in Central America and has been encountered occasionally on indoor fuchsias in England and Wales (Smith, A., and Rees, J., 1932; Moore, W. C., 1943).

GAILLARDIA

This plant may be attacked by the downy mildew *Bremia lactucae* (see p. 288).

GALANTHUS (Snowdrop)

Smut (*Urocystis galanthi* Pape)

This disease seems to be known only in Germany. It causes lead-grey blisters on leaves and flower sheaths, which split to expose the blackish-brown spore powder inside.

Stagonospora curtisii (Berk.) Sacc. (see narcissus, p. 495) may cause rotting of snowdrop bulbs (Moore, W. C., 1939b).

Grey Mould (*Botrytis galanthina* (Berk. & Br.) Sacc.)

As soon as the shoots of infected snowdrops appear above ground they are covered by a close light-grey mould of *Botrytis* conidiophores and conidia (Fig. 270). The disease is worst on heavy soils, or where snow has lain, or the shoots have been damaged by alternating frost and thaw. On the rotting shoots, sometimes

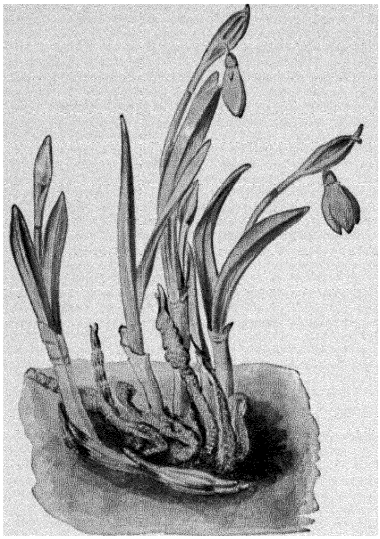


Fig. 270. Grey mould of snowdrop.

also on the bulbs, small black sclerotia form. These possibly give rise to the cup fungus *Sclerotinia galanthi* Ludw. (Keissler, K., 1917), but the connection between the *Botrytis* and *Sclerotinia* stages has not been proved.

Where the disease has appeared all the diseased plants and their neighbours should be carefully dug up and destroyed with the surrounding soil. After the disease has obtained a hold it may be impossible to check it. If it is desired to replant snowdrops in the same place the soil should be carefully renewed or 4–5 years should elapse between the appearance of the last diseased shoot and replanting with fresh bulbs.

Much smaller dot-like black sclerotia are common on the papery scales of snowdrop bulbs. These belong to a harmless fungus as yet unidentified.

GARDENIA

Chlorosis

Gardenias are particularly liable to yellowing of their leaves if the soil reaction is too high or the temperature too low.

Soil temperature should be at least 68° F. and the reaction about pH 5. The latter should be kept down by adding a little sulphate of ammonia to the watering water unless this is already acid.

Stem Canker (*Phomopsis gardeniae* Buddin & Wakefield)

Gardenia cankers may occur at the base of the main stem or on the branches. The latter originate from " snags " which have been left when flowers were cut and have died back. Cankers on the branches may be 3 inches long, exposing the wood and bordered by an extensive development of callus. Small twigs may be completely killed but large branches usually survive. Cankers at the base of the main stem cause wilting and death of the whole plant. The same fungus, *Phomopsis gardeniae* Buddin & Wakefield, that causes the cankers, causes also discoloured spots and blotches on the leaves, especially at their bases.

The fungus appears to be a wound parasite and infection occurs only through wounds. It is probably spread by the use of slightly infected cuttings (Buddin, W., and Wakefield, E. M., 1937, 1938). Control measures include care in cutting blooms not to leave " snags," avoiding unnecessary wounds and selecting cuttings only from healthy plants. The disease also occurs in America (Hansen and Barrett, 1938).

GERANIUM (Glasshouse)

See *Pelargonium*, p. 504.

GERBERA

Leaf Spot (*Ascochyta gerberae* Maffei)

Circular brown spots up to half an inch across, surrounded by a purple margin, occur on the leaves. Dotted over them are the tiny pycnidia of the fungus *Ascochyta gerberae* Maffei (Fig. 271). This was first described in Italy and has occurred on *G. jamesoni* in Cornwall and Devon (Beaumont, A., and Gregory, P. H., 1937).

Protecting the plants with Dutch lights

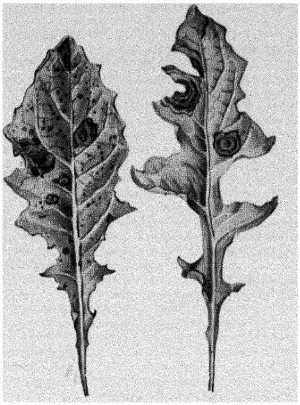


Fig. 271. Gerbera leaf spot.

has been suggested as a means of checking spread of the disease. Spraying with Bordeaux mixture may also be tried.

Foot Rot (*Phytophthora* spp.)

Symptoms. The leaves suddenly wither and lose their green colour, changing first to violet and then to brown. Plants of all ages are susceptible but the chief damage occurs at flowering. The fungus enters the stem at or just below soil level and spreads downwards into the roots. The invaded tissues become soft, watersoaked and turn blackish-brown but have no smell.

Cause. In U.S.A. both *Phytophthora cryptogea* Pethybr. & Laff. and *P. drechsleri* Tucker have been isolated from *Gerbera* and have been shown to reproduce the disease when inoculated into healthy plants. *P. cryptogea* is a common parasite of many ornamental plants and vegetables.

We have found species of *Fusarium* and undetermined bacteria in *Gerbera* showing the above symptoms but we have not found *Phytophthora*. How far either the *Fusarium* or the bacteria may be a primary cause of the disease we are unable to say.

Control. The disease is favoured by moisture and the plants should therefore be kept as dry as they can stand. Coarse sand or gravel may be packed round the base of the stem. In the case brought to our notice beneficial results followed the use of Uspulun or of potassium permanganate. Where *Phytophthora* is involved watering with Cheshunt compound may check spread of the disease to other plants.

GEUM

Downy Mildew (*Peronospora gei* Syd.)

Infected plants are rendered conspicuous by the large yellowish patches on the upper surface of their leaves. The corresponding parts of the lower surface are covered by a close light-grey felted mould composed of conidiophores and conidia. No control measures are known but spraying with a copper fungicide may check the disease if done as soon as the first spots are seen (Dennis, R. W. G., and Wakefield, E. M., 1946).

Leaf Blotch (*Zythia fragariae* Laibach)

Moore has described dark olive-brown blotches on leaves of cultivated *Geum* caused by the strawberry leaf blotch fungus (see p. 359). No control measures have been suggested (Moore, W. C., 1949).

GLADIOLUS

Botrytis Rot (*Botrytis* sp.)

Species of *Botrytis* may attack both tops and corms of gladioli but there has been much confusion about their identity.

If infected corms are planted, or if the soil is infected, they either fail to grow or give weak shoots that turn yellow and soon die. The corms themselves become soft and rotten.

Healthy plants may be attacked either at the base or higher up. Under damp conditions the decaying tissue becomes covered by grey mould but sclerotia are very seldom formed. Small light-brown spots with red edges may appear on the leaves. Spots may also occur on the flowers and are very conspicuous on red-flowered varieties, on which they have a whitish edge. In damp weather the spots may spread rapidly and become covered with grey mould.

Infection of the corms causes great losses during storage. They may be attacked in various ways. There may be small discoloured sunken spots beneath which a dry brown rot penetrates deeply into the flesh. More often the rot starts at the base of last year's flower stalk and penetrates right through the centre of the corm (core rot) (Fig. 272).

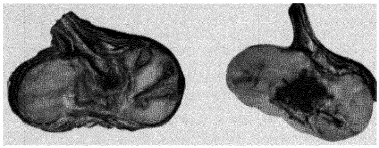


Fig. 272. Core rot of gladiolus corms (*Photo Dept. Agric. Scotland*).

The rot may be reddish-brown and moist, or dry and corky, in which case the decayed tissue may break up and fall out. On the surface of the corms, but usually beneath the covering scales, there may be black irregular sclerotia (Fig. 273). Corms may also rot from the base upwards. Early stages of corm rot are easily overlooked as they are concealed by the scales.

Klebahn (1930) described *Botrytis gladioli* Kleb., recognized by its very dark conidiophores, but it is not certain that this fungus is the cause of the corm rots. Wade (1946) thought it was. Timmermans (1941, 1942) thought the corm rot fungus a new species and called it *B. gladiolorum* Timm. Hawker (1946a) attributed corm rot to strains of *Botrytis cinerea*. The question is not purely academic for it is important to decide whether the fungus causing gladiolus corm rot is specialized and attacks only gladioli or related species, or whether various strains of the ubiquitous *B. cinerea* can destroy gladiolus corms.

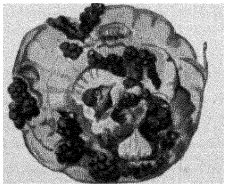


Fig. 273. *Botrytis* sclerotia on gladiolus corm.

There is little information about control of this disease. All forms of grey mould are favoured by moisture. Gladiolus corms should be well dried, preferably in a warm, well-ventilated room, and should be stored in a dry place. Hawker (1946a) recommended avoiding growing gladioli on wet, heavy soils, in shaded places or on infected ground. Lifting early in October gave less corm rot than later lifting. Early removal of adhering soil, roots, parent corms and old shoots and leaf bases from the harvested corms gave good control. Almost complete control was obtained by steeping for $\frac{1}{3}$–3 hours in a 0.1% solution of corrosive sublimate. This also controls hard rot, scab and to some extent dry rot but may damage the corms. Dusting with pentachloronitrobenzene dust before storage also gave good control. Application of this dust to corms at planting time was also beneficial.

Gram and Weber obtained good results by soaking corms for 6 hours in $\frac{1}{4}$% Aretan solution and drying before storage.

The fungus is a wound parasite but quite small wounds are enough to allow it access. At comparatively high temperatures wounds are rapidly healed and the fungus is unable to penetrate them.

Control measures include avoiding wounding the corms and storing them for 10 days at 84° F. to allow such wounds as are present to heal.

Fusarium Rot (*Fusarium oxysporum* Schlecht. var. *gladioli* Massey)

Corms become infected in the field but rot mainly during storage. When they are lifted they may bear small reddish-brown water-soaked spots. These increase in size during storage and become marked by prominent, irregularly concentric ridges. Normally the rot does not penetrate deeply but corms stored at high temperatures in a moist atmosphere may rapidly be completely destroyed and reduced to blackish-brown mummies.

Control measures include avoiding replanting gladioli where infected plants grew the previous year, sorting and rejecting infected corms at lifting, storing in a dry, well-ventilated place and dipping in ¼% Aretan solution.

Storage Rot (*Penicillium gladioli* McCull. & Thom)

On the side of the corm there appears one, or occasionally more, irregular, sharply defined reddish-brown, sunken spots, up to ½ inch across. On the central portion there may be groups of small, round, buff-coloured or very pale pink sclerotia, though these are sometimes concealed beneath the dead top skin. Under damp conditions white mycelium and a little blue-green mould may appear. Beneath the spot a rather dry, grey-brown rot penetrates the flesh and may ultimately destroy the whole corm.

Hard Rot and Leaf Spot (*Septoria gladioli* Passer.)

Symptoms. The corms first develop small, round, reddish-brown or blackish-brown spots, often not seen until the scales are removed. In severe outbreaks there may be spots on the scales as well. When the spots on the corms enlarge the rotting tissue sinks in, becomes hard and dry and turns black. The spots may join up but their margins often remain as ridges on the dead flesh. Corms may be completely destroyed in storage.

On the leaves the fungus causes small brown or purplish-brown spots, more or less circular, which gradually become larger and turn grey in the centre. They may unite to cover extensive areas of the leaf, especially along the margins. These must not be confused with small pale oblong spots caused on gladiolus leaves by potassium deficiency.

On the centre of the leaf spots are numerous tiny black pycnidia, and similar fructifications, often abortive, develop on the diseased corms. The fungus overwinters on the corms and on debris of diseased plants in the soil, where it may survive for several years. Apparently it does not grow directly from the corm into the young shoot but it is not clear how the leaf spots do arise in the first place. It has been suggested they start from mycelium splashed up from infected soil during heavy rain, or by the leaves bending over to touch infected soil. Hence though severely infected tubers may rot and give no plant, those slightly less affected may give healthy foliage.

Once the leaf spot stage has appeared in a bed it easily spreads from plant to plant. Infection of the new corm may come from spores washed off the leaves or by mycelium from the soil. The disease is worst in wet years. According to German authors hybrids of the *gandavensis* type are very susceptible, those of the *primulinus* type much less so. Blushing Bride and Queen of the Pink are very susceptible in Cornwall (Moore, W. C., 1939). Crocus and freesia may also be infected.

Control. Soil that has carried an infected crop should not again be used for gladioli for 4–5 years. One should try to avoid spreading soil infection by planting only healthy corms. All obviously diseased corms should be sorted out and destroyed at lifting.

Drayton (1929) recommended dipping corms for 15 minutes in 5% Semesan or Uspulun at 122° F. Hawker (1944) found steeping corms for 3 hours in 0.1% corrosive sublimate solution quite effective, especially if obviously infected corms were sorted out first. Aretan and Uspulun were also effective but dipping in 1% calomel suspension was not.

The leaf spot phase may be checked by spraying with Bordeaux mixture, 5:5:50.

It is a general experience of growers that the disease is worst on under-manured soil.

Scab and Neck Rot (*Bacterium marginatum* McCulloch)

Symptoms. On the leaves the disease starts as minute reddish-brown specks which gradually grow into circular or elongated dark-brown spots with a slightly raised and darker margin. They are most abundant on the lower parts of the leaves where they may join up to form an extensive soft rot which destroys all the tissue except the veins and affects especially

the inner leaves. In dry weather only one or two leaves die and the plant may flower, but in wet weather the rot spreads rapidly and the whole top falls over with neck rot.

On the corm scales there are pale-yellow oval spots, which gradually darken until almost black and soon split. Below these on the corm and especially towards the base are sharply defined circular spots, at first pale yellow and watersoaked, then dark brown or black (Fig. 274). Individual spots are about

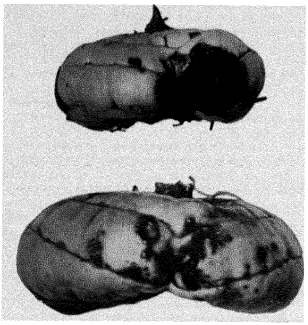


Fig. 274. Gladiolus scab on the corm (*Photo Dept. Agric. Scotland*).

¼ inch across, saucer-shaped with a prominent rim, and can be picked out cleanly with a knife, leaving a clean cavity in the flesh. In severe attacks the spots join up to form irregular corroded areas and deep cavities. They exude a gummy substance which dries brown, brittle and shiny. Particles of soil often adhere to the gum and so may the corm scales.

Cause. Scab and neck rot are caused by *Bacterium marginatum*. This is carried over on the corms and may also survive in the soil. The young shoot becomes infected from the corm, especially from lesions on the outer scales.

Control. Hawker (1946b) found that the mercury dip effective against other corm diseases controlled scab also. Others have recommended soaking 1–12 hours in corrosive sublimate solution 1: 1000, Germisan or Semesan 1: 100 for 1 hour, or calomel suspension 5: 100 for 5 minutes.

Gladioli should preferably not be planted on soil where a crop infected with scab has been grown. Leaf infection may be checked by spraying with Bordeaux mixture.

A different bacterial disease caused by *Bacterium gummisudans* McCull. occurs on gladioli in Finland, North America and Australia. This attacks mainly the leaves, on which it causes rectangular dark-green watersoaked spots that dry brown. A sticky film of bacterial exudate oozes from these spots and covers the leaves.

Dry Rot (*Sclerotinia gladioli* Drayton)

On the corms this fungus causes dark sunken spots like those of hard rot but usually smaller. Usually they are quite shallow but in severe attacks they may be so numerous as to cover the surface and the rot penetrates deeply into the flesh, especially along the conducting strands.

On the growing plant the first sign of infection is a yellowing and drying up of the leaf tips. Then the whole foliage turns brown and dies as a result of decay of the leaf sheaths just above soil level. This decay starts at the inside and works outwards. The dead foliage is easily pulled away and the parent corm will be found to be dead and rotten. Scattered over the dead leaf sheaths are innumerable tiny black sclerotia just visible to the naked eye. Occasionally a few of these occur also on the corm lesions. The disease seems to have been present in England for very many years. It has been known in North America since 1909 and in Denmark since 1928, also in Germany, Holland and France. The same fungus attacks montbretia, freesia, crocus, lapeirousia and acidanthera.

In artificial culture the fungus forms cup-shaped apothecia but these have not been found in nature.

Control measures include dipping corms in 10% Uspulun, Semesan or similar organo-mercury dip for 15 minutes at 122° F. This does not give complete control, however, and all visibly infected corms should be rejected.

Gladioli should not be planted for at least 4 years where an infected crop has grown. Growing plants showing the disease should be dug up and destroyed.

Smut (*Urocystis gladiolicola* Ainsworth)

On stems and leaves there appear elongated leaden-coloured stripes like those of onion smut (see p. 298). In time the skin bursts and blackish spore powder falls out.

Affected plants should quickly be removed and destroyed. Lifted corms should be carefully sorted and diseased ones removed. Gladioli should not be grown again on soil that has borne smutted plants.

According to Van Poeteren (1926) soaking corms for ½ hour in water at 110° F. kills the fungus.

Gladiolus smut occurs occasionally in Cornwall and Devon but has been much confused in the past with a minor disease of the corms caused by the sterile fungus *Papulaspora gladioli* (Reg.) Dodge & Laskaris (1941).

This forms powdery brown masses in cavities on shrunken corms, rather like powdery scab on potatoes. It probably occurs only on corms previously injured by other fungi.

Leaf Spot (*Didymellina macrospora* Kleb. = *Heterosporium gracile* Sacc.)

This common disease of irises also attacks gladiolus (see p. 477).

Rust (*Puccinia gladioli* Cast.)

This rust occurs on many species of gladiolus throughout the Mediterranean region but is very rare in Britain. It forms only teleutospores which occur in dark brown pustules on both sides of the leaves.

GLOXINIA

See *Sinningia*, p. 532.

GODETIA

Alternaria Stem Rot (*Alternaria tenuissima* (Fr.) Wiltshire var. *godetiae* Neergard)

Brown spots appear on the stems and gradually extend, both superficially and into the tissues. The pith dies and the stems become hollow, leaves wither and fall. If the weather is slightly damp infected plants become covered with an olive-coloured or sooty-black coating of mould. Varieties differ in susceptibility but Sybil Sherwood and Kelvedon Glory suffer severely (Neergaard, P., 1945).

Steeping the seed for ½ hour in ¼% solution of an organo-mercury disinfectant may be tried. With seed crops spraying with Bordeaux mixture may be beneficial.

Wilt (*Diplodina passerinii* Allesch.)

The stem becomes brown, especially towards the base, often starting at one of the nodes near soil level. After the lesion has encircled the stem the plant wilts and dies. On the dead stems the fungus, *Diplodina passerinii*, forms innumerable small dot-like black pycnidia.

The same fungus causes wilt of antirrhinum, clarkia and *Centaurea moschata* (sweet sultan) (Taylor, E., 1941).

GRASS

See turf, p. 553.

GYPSOPHILA

Stem Rot (*Sclerotinia serica* Keay)

This fungus causes rotting of the stems and leaves of *Gypsophila elegans* near ground level. It appears to attack mainly plants belonging to the same family, *Caryophyllaceae*, viz. species of *Lychnis*, *Silene*, *Spergula* and *Stellaria*, but would not attack *Dianthus* or *Cerastium*, nor does it parasitize Jerusalem artichoke or red clover, which are readily attacked by *Sclerotinia sclerotiorum* and *S. trifoliorum* respectively (Keay, M. A., 1937). Control measures would appear to include rotation of crops and those recommended against *S. sclerotiorum* (see p. 77).

Rust (*Puccinia lychnidearum* Link)

This rust, which may attack also species of *Arenaria*, *Dianthus*, *Lychnis*, *Stellaria*, etc., forms only teleutospores. These occur in rather light-brown cushion-like pustules, usually situated on conspicuous yellowish leaf spots.

HAREBELL

See *Campanula*, p. 429.

HAWTHORN

See *Crataegus*, p. 440.

HEATH

See *Erica*, p. 455.

HEDERA (Ivy)

Leaf Spots

Several fungi cause leaf spots on ivy but the commonest is probably *Phyllosticta hedericola* Dur. & Mont. It forms circular ash-grey spots, some half an inch across, surrounded by a reddish-brown margin with a translucent outer edge (Fig. 275). Similar spots are caused by *Mycosphaerella hedericola* (Desm.) Lindau, *Vermicularia trichella* Fr. (= *Amerosporium trichellum* (Fr.) Lind), *Gloeosporium helicis* (Desm.) Oud. and *Trochila craterium* Fr., all of which occur in Denmark and Britain. *V. trichella* has also been seen causing injury to ivy stems (Neergaard, P., 1943).

If the attacks are very severe the ivy should be clipped, provided the time of year is suitable. Otherwise spraying or dusting with a fungicide may be tried.

HELENIUM

Leaf Spot (*Septoria helenii* Ell. & Everh.)

This is a minor disease forming greenish to chocolate-brown spots up to $\frac{3}{4}$ inch across on helenium leaves. Often they have a narrow darker margin. The spots may become paler with age and the pycnidia of the fungus *Septoria helenii* appear as minute brown dots scattered over them. No control measures have been devised (Moore, W. C., 1941).

The plant may also suffer from *Verticillium* wilt (see p. 60).

HELIANTHEMUM (Rock Rose)

Leaf Spot (*Septoria chamaecisti* Vestergr.)

Reddish-purple spots, seldom more than $\frac{1}{10}$ inch across, occur on the upper surface of the leaves and occasionally on the sepals. Sometimes they are pale brown with a purple border. A few minute blackish pycnidia of *Septoria chamaecisti* occur on the spots (Moore, W. C., 1946).

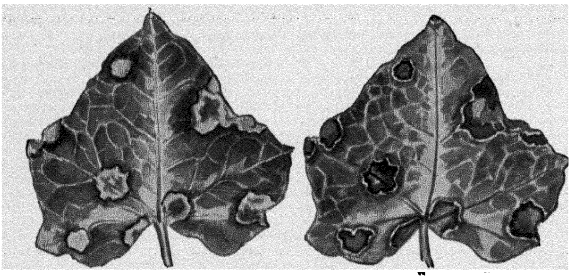


Fig. 275. *Phyllosticta* leaf spots on ivy.

Mildew (*Leveillula taurica* (Lév.) Arn.)

This forms rather sparse white mildew patches on rock rose leaves (see mildew, p. 63).

HELIANTHUS (Sunflower)

Grey Mould (*Botrytis cinerea* Fr.)

Grey mould can attack sunflowers very severely when the plants are crowded and conditions are moist. The stalks are attacked partly at the base, partly from the leaf axils, and all the soft tissues are destroyed so that the plant wilts and dies. The grey coating of mould with flat black sclerotia may also develop on the flower heads. *Sclerotinia sclerotiorum* is often present as well.

According to German experience excess nitrogen makes plants susceptible to attack.

Care should be taken to avoid replanting sunflowers where they suffered much from grey mould the previous year (see further, p. 59).

Mildew (*Erysiphe cichoracearum* DC.)

The leaves become covered with a floury white coating formed by the mycelium and conidia of this fungus (see mildew, p. 63).

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

Sclerotinia sclerotiorum may attack sunflowers severely, on both stems and flower heads. First appear brown spots covered with white mycelium, which on unripe seeding heads permeates between the seeds. The mycelium soon produces black sclerotia which may join up to form a continuous black network among the seeds. Grey mould is often associated (see further, p. 77).

Rust (*Puccinia helianthi* Schw.)

Is common on sunflowers in many countries though not in Britain. All four stages of the rust occur on the same plant so there is no alternate host (see p. 74).

Downy Mildew (*Plasmopara halstedii* (Farl.) Berl. & de Toni)

Is common in North America. It causes yellowish spots on the leaves which bear the usual whitish mould on the lower surface. Seedlings may be infected from the soil and killed. Rotation of crops and spraying older plants with Bordeaux mixture are recommended control measures.

HELICHRYSUM (Everlasting Flower)

Downy Mildew (*Bremia lactucae* Regel)

May attack this plant. For symptoms and control see lettuce (p. 288).

HELIOTROPIUM (Heliotrope)

Grey Mould (*Botrytis cinerea* Fr.)

If heliotrope is grown in too damp a place the above-ground parts of the plant easily become decayed and overgrown with grey mould (see p. 59).

The plant may also suffer from damping off caused by *Corticium solani* (see p. 68).

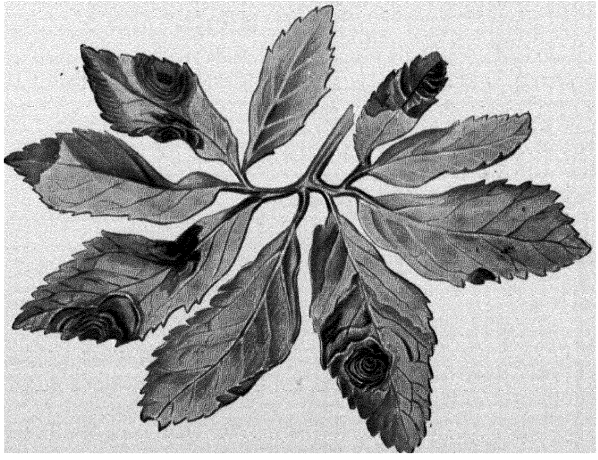


Fig. 276. Leaf spot of Christmas rose.

HELLEBORUS (Christmas Rose)

Leaf Spot (*Coniothyrium hellebori* Cooke & Mass.)

Round or elliptical black spots up to ¾ inch across appear on the leaves. They are usually marked by concentric zones (Fig. 276), and may run together to form large patches, spoiling the appearance of the plant. In very severe attacks the fungus may cause the leaves to turn yellow prematurely and die. It may also attack the leaf and flower stalks, forming sunken spots at which the stalk may snap. Small black spots may be produced on the flowers.

The disease is common in Denmark and sometimes very destructive. Severely affected plants should be dug up, all the above-ground parts removed, and the roots replanted in fresh soil. The plants should be sprayed with Bordeaux mixture or dusted with a fungicide at fortnightly intervals throughout summer and early autumn. Diseased leaves should be picked off and destroyed. Plants to be forced should be dug up and potted in September and their leaves protected from wetting.

Downy Mildew (*Peronospora pulveracea* Fuck.)

A greyish layer of mould appears on the under-side of the leaves, which may be smaller than normal and somewhat deformed. The fungus overwinters as mycelium in infected plants, which therefore remain permanent sources of infection to others. They should, therefore, be dug up and destroyed. Other plants in the neighbourhood may be protected by spraying or dusting with a copper fungicide.

A mosaic disease attacked Christmas rose in Soro and Skanderborg in 1926. The diseased plants were destroyed.

HEPATICA

See *Anemone*, p. 411.

HESPERIS (Dames Violet)

Mosaic (*Cucumis* Virus 1)

A conspicuous chlorotic vein-clearing followed by a coarse bright yellow or yellow-green mottle of Dames Violet leaves in North America has been shown to be caused by the virus of common cucumber mosaic (Pound, G. S., and Walker, J. C., 1948).

HEUCHERA

Leafy Gall

See *Lathyrus*, p. 484.

HIPPEASTRUM (Amaryllis)

Leaf Spot (*Stagonospora curtisii* (Berk.) Sacc.)

Leaf spot of *Hippeastrum* has been attributed to the fungi *Phoma amaryllidis* Kotth. & Fried., *Phyllosticta gemmipara* Zond., *P. amaryllidis* Bres., *Stagonospora crini* Bub. & Kab., and *S. curtisii* (Berk.) Sacc. (see narcissus, p. 495), which are apparently all the same thing. The spots are small and reddish brown and occur on all the green parts and on the bulb scales. They may cause malformation of the leaves (Smith, C. O., 1935).

The fungus may be controlled by keeping the air as dry and cool as possible. Water carefully to avoid wetting the leaves. Spray with Bordeaux mixture if necessary. Plants should be protected from drip as far as possible. Diseased bulbs should be pared clean and then dusted with charcoal.

Red Leaf Spot (*Lycopersicum* Virus 3)

Pale-yellow or white spots, often associated with small blood-red dead spots or red lines along the leaf edge, are due to infection with the virus of tomato spotted wilt. Individual leaves may be killed but infected plants are chiefly important as a menace to the tomato crop (see p. 384).

HOLLYHOCK

See *Althaea*, p. 410.

HONESTY

See *Lunaria*, p. 490.

HYACINTHUS (Hyacinth)

Black Slime (*Sclerotinia bulborum* (Wakk.) Rehm)

The plants languish, their leaves turn yellow and fall over and flowering may be prevented. The leaf bases are completely

decayed and the bulb scales are dark grey, the outer ones almost completely rotten. White mycelium permeates the bulbs and sclerotia, at first white, then smooth and black, irregular, flattened bodies, form between and within the decaying scales.

Ultimately the infected bulb shrivels up to a black mass and disintegrates, leaving the sclerotia in the soil. The following spring they germinate and bear one or more apothecia of the fungus. In bulb fields the disease occurs in patches, and for control it is necessary to remove not only the infected bulbs but also the surrounding soil, which contains mycelium and sclerotia of the fungus.

S. bulborum also attacks *Crocus*, *Muscari*, *Scilla* and *Fritillaria*.

Yellow Disease (*Xanthomonas hyacinthi* (Wakk.) Dowson)

This disease was first described in Holland in 1883. It is distributed with the bulbs and apparently has not become established in any other country, though it is frequently encountered in imported hyacinth bulbs. No other plant is attacked by this disease.

Symptoms. Superficial examination of an infected bulb at planting time discloses nothing wrong unless the disease is far advanced, in which case the inside may be rotten and the bulb feel soft when squeezed. If a slightly infected bulb is cut across, however, a number of small yellow spots are seen on the cut surface of the scales and a slimy yellow liquid oozes out if they are squeezed. In longitudinal section the slime is found to be occupying the vascular bundles and hence appears as thin yellow stripes (Fig. 277). The yellow slime is made up of myriads of the bacterium *Xanthomonas hyacinthi*. Later on they spread from the vascular bundles into the surrounding tissue and eventually the whole bulb is reduced to a rotting putrid mass, especially if other secondary bacteria and fungi cooperate to complete the destruction.

Above-ground parts of the plant become infected through wounds in the skin of leaves and flower stalks, or through the flowers. Watersoaked spots or streaks appear on them,

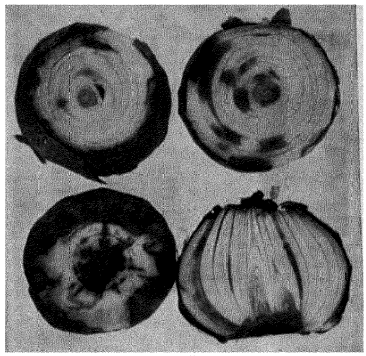


Fig. 277. Yellow disease of hyacinth bulbs (*Photo v. Slogteren*).

which spread and turn yellow or brown and dry up. The diseased stripe may run down the middle of a leaf, leaving the edge green, or the margins may be affected first (Fig. 278). Similar symptoms may appear on the flower stalks. The bacterium spreads down through the vascular system into the new bulb.

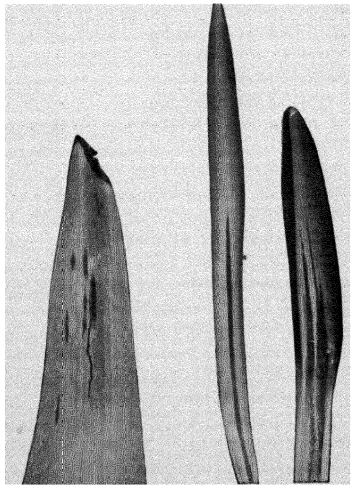


Fig. 278. Striping of hyacinth leaves caused by *Xanthomonas hyacinthi* (*Photo v. Slogteren*).

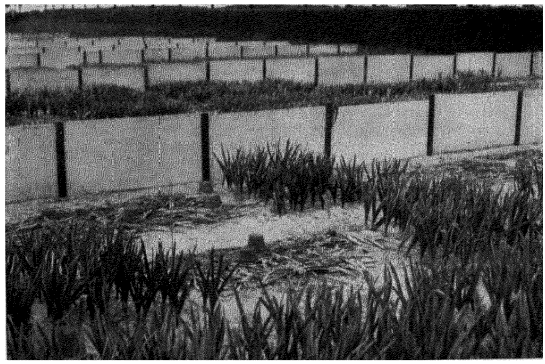


Fig. 279. Control of yellow disease of hyacinth in Holland (*Photo v. Slogteren*).

Centres of infection in the bulb fields are formed by slightly infected bulbs, into which the bacterium has not penetrated very deeply. These root well and produce leaves and flowers but they are marked with the symptoms described above, spreading from below upwards. Sooner or later the skin bursts, the bacterial slime oozes out and is splashed by rain, or distributed by various agencies, to the leaves and flowers of surrounding healthy plants. So far as is known the bacteria do not survive the winter in the soil.

Control. When the disease appears in bulbs bought for forcing nothing can be done beyond getting it confirmed by a competent authority and claiming damages from the supplier.

In Holland stringent measures are taken to control the disease. The bulb fields are constantly inspected and an earthenware pot is put over diseased plants as soon as they are detected. All the surrounding plants within a radius of about 1½ yards are killed by spraying with 5% formalin solution or 10% carbolineum. Screens of asbestos board or plaited reeds are placed round infected plots to hinder spread of the bacterium (Fig. 279). Diseased bulbs are not removed until all the healthy ones have been lifted, as there is risk of spreading the bacterium on the hands of workers.

Bulbs for export are subject to inspection. Serious cases are detected by the soft feel when squeezed. Samples of the firm ones are taken for testing by cutting off the tip of the bulb to see if the yellow slime oozes out.

Suspected stocks of lifted bulbs are stored at 86° F. from lifting to the end of August, then at 98–100° in September; then, after 3 weeks at this temperature, it is safe to raise it to 107–110° F. for a couple of days. The temperature is then allowed to drop gradually to 70° F. The object is to accelerate development of the disease, so that infected bulbs are soft and can be rejected at planting, which is delayed as long as possible, usually until November.

Excessive manuring with nitrogenous compounds makes the bulbs more susceptible.

Fire (*Botrytis hyacinthi* Westerd. & Beyma)

This disease is called "fire" (in Holland *Vuur*) because of the rapidity with which it may spread over the fields, especially in wet springs or if mists creep in from the sea.

Infection occurs at or near the leaf tips and spreads downwards, making the leaves brown and either rotten or shrivelled. Some or all of the flowers may also be attacked with a soft rot but the disease does not attack the bulbs. Fire appears particularly after frosts.

The fungus produces the usual *Botrytis*

type of grey mould on the affected parts and also small black sclerotia the size of a pinhead on the dead leaves. It can also attack lilies.

As the fungus is not spread on imported bulbs it does not trouble the forcer.

Soft Rot (*Bacterium carotovorum* (L. R. Jones) Lehm.)

This causes a soft, evil-smelling rot of the tissues at the base of the leaves and flower stalk in forced bulbs. Eventually the whole bulb is destroyed. L'Innocence, Gertrude and Queen of the Blues are very susceptible. Queen of the Pinks is more resistant (Stapp, C., 1933). Infection may occur in the field, especially in over-manured crops. In such cases the bacteria may lie dormant in the outer scales or round the neck of bulbs stored in good dry conditions and become active again when forcing begins (see also p. 257).

Penicillium Bulb Rot (*Penicillium hirsutum* Dierckx, etc.)

This, and probably also allied species of *Penicillium*, causes a soft brown rot of the inner scales of hyacinth bulbs. The scanty blue-green mould appears on the surface under favourable conditions. When hyacinths are grown in glasses species of *Penicillium* may be encouraged where the water reaches the bulb (see also tulip, p. 545).

Root Rot (*Fusarium culmorum* (W. G. Sm.) Sacc.)

This is a brown rot of the roots very like that associated with *Cylindrocarpon radicicola* on narcissus roots (see p. 497).

Yellow Spots

Due to some unfavourable condition in storage may occasionally occur in hyacinth bulbs. They may closely resemble those of yellow disease but do not yield a yellow slime.

Green-tipped Flowers

This is a malformation sometimes encountered in forced hyacinths. It is a non-parasitic disorder, apparently due to forcing too early or at the wrong temperature (Purvis, O. N., 1937).

Withering of Individual Flowers

Symptoms. Not infrequently individual blooms in a forced hyacinth spike wither, while the rest develop normally. The trouble starts by the buds drying up. They then fall an easy prey to fungi and bacteria and often decay. L'Innocence and especially bulbs with large flower spikes are prone to this unsightly disorder.

The disease is a physiological one but its precise cause is not clear. Beijer and Van Slogteren (1931) found that certain treatments of the bulbs making them suitable for forcing early tended to lead to this trouble. It is found especially in bulbs from greenhouses (*warenhuizbollen*) or in those raised out of doors in warmed soil (*pijpenvollen*). It occurs mostly on bulbs which have a strongly developed shoot when planted. If it is so far advanced as to protrude from the bulb at planting time brown-tipped flowers may appear in great numbers. They will be equally numerous whether forcing is early or late.

Possibly development of the bulb during the summer has an influence on this condition, so that if the bulbs ripen early and are lifted early the chance of their drying out is increased and the tendency to withered blooms increased. In one instance it appeared to us that rotten bulbs and rotten buds were due to the bulbs having been packed for a long time before being despatched from Holland. Root development was good.

Control. The tendency to the disease is in the bulbs when they are planted and cannot be counteracted during forcing. High humidity favours development of secondary fungi and bacteria, which attack the withered flowers and may set up a rot which spreads to healthy ones. Hence diseased flowers should be snipped off as soon as seen.

Loose Bud

Symptoms. This phenomenon, called *Spouwen* (= spewing) in Holland, results in the young flower spike being cast off the plant as soon as the leaves have opened sufficiently to release it (Fig. 280). The flower stalk is broken at the base, but the flower spike



Fig. 280. Loose bud of hyacinth (*S. p. F.*).

remains apparently normal until it is exposed and found to be lying loosely among the leaves.

Cause. The trouble is a non-parasitic one, due to cultural conditions. It has been suggested (Ingelström, E., 1938) that storage at low temperature is the cause and perhaps also the change in temperature when the bulbs are taken indoors where the temperature may be 60° F. higher.

In the two cases where we saw most damage from loose bud, low temperature was the cause.

In the first case out of a consignment of Locarno one batch of bulbs lost many blooms, the next lost none. This may have been due to cold when the plants were taken up. In another case 10% of the flowers of various varieties out of doors were shed, after there had been a cold period when the buds were scarcely above the soil.

Control. In particularly cold winters the stores should be particularly well covered and the bulbs should be protected against cold when taken up. The temperature should not be too high immediately after they are moved into the house. Regular watering should be the aim and great variation of temperature during forcing should be avoided.

Cold Chlorosis

In outdoor hyacinths those parts of the leaves which were touched by frost often remain permanently yellow because chlorophyll is not formed in them.

If low temperatures occur repeatedly while the leaves are growing they may bear several pale cross stripes which subsequently disappear (see Fig. 8).

White Slime and Gummosis

Affected bulbs become soft, root well but form no shoots. Instead a dirty white, bubbly mass of slime oozes out of the nose. The disease is occasionally seen in forced hyacinths. Formerly, when it was the practice to put the lifted bulbs in earth pits for a few weeks before storing on shelves, it was much more prevalent. According to Wakker (1889) it is an advanced stage of gummosis, a much more common trouble in which drops of light-brown transparent gum exude from the nose of the bulb. Bulbs affected only by gummosis usually flower normally.

Summary of Hyacinth Diseases

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(2) Vague pale stripes on leaves	Mosaic	546
(3) Black sclerotia in rotting bulbs	Black Slime	470
F. Grey mould present	Fire	472

HYDRANGEA

Mildew (*Microsphaera polonica* Siem. = *Oidium hortensiae* Jørstad)

Hydrangea leaves not uncommonly bear brown spots, often reddish at the margin and more or less covered with a whitish mildew. The fungus is found mainly on the under-side of the leaves. It may also attack the flowers. The perithecia are very rare and have never been seen in Britain.

There have been conflicting statements about varietal susceptibility. In one instance Etincelant was particularly severely attacked in Denmark. Pape (1939) cited as highly susceptible: Blauer Prince, Deutschland, Eclaireur, Elmar, Fortschritt, Holstein, Kølner Jungfrau, Lanzelot, Loreley, Matador, Tischers Silberblau; and as comparatively resistant: Elbe, Gertrude Glahn, Goliath, La France, La Marne, Merveille, Molière, Niedersachen, Rheingold, Schöne Dresdnerin, Sonnengruss and Wicking. Dark-coloured, smooth-leaved varieties are generally less susceptible than those with light-coloured, rougher, more uneven leaves.

Control. According to Pape, confirmed by a Danish experiment, dipping the tops of the plants in 1: 20 lime sulphur solution before planting has a beneficial effect. Before the plants are put away for the winter all diseased leaves should be removed (see mildew, p. 63).

Bacterial Leaf Spot

In 1934 we first saw bacterial spots on hydrangea leaves.

The leaves might be dark or light green but always had a light edge, particularly evident in transmitted light. Along the edges of the leaf were numerous small spots (see Fig. 281). The

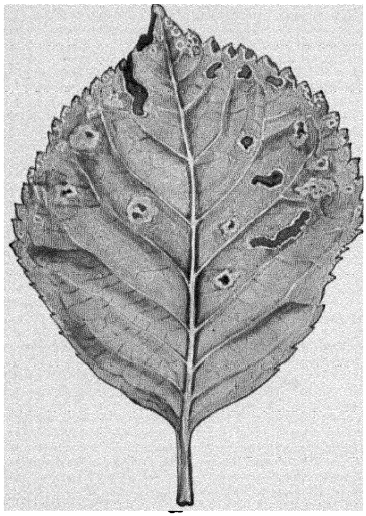


Fig. 281. Bacterial leaf spot of hydrangea.

leaves may be deformed, so, exceptionally, may petioles and shoots. Bacteria are very numerous in the spots.

This disease seems to have become more widespread and is now not uncommon.

As far as we know no direct control has been attempted. Diseased leaves should be picked off. It would be advisable to keep the leaves as dry as possible and to avoid drops of water running from leaf to leaf during watering.

Varieties differ greatly in susceptibility. Hamburg seems particularly prone to attack.

Other Leaf Spots

Various fungi, species of *Ascochyta*, *Phyllosticta* and *Septoria*, have been described causing spots on hydrangea leaves. Of these *Septoria hydrangiae* Bizz. makes brown spots with a blood-red border on hydrangeas in Britain (Pethybridge, G. H., 1934).

Failure of Cuttings (*Corticium solani* (Prill. & Delacr.) Bourd. & Galz.)

This common damping-off fungus, and its ally or strain *Moniliopsis aderholdi* Ruhl, may attack hydrangea cuttings and cause them to decay at the base (Fig. 36).

Hydrangea cuttings cannot endure soil which has been treated with corrosive sublimate as usually prescribed to control these fungi. It is therefore important to raise them in soil which has never come in contact with diseased plants or which has been sterilized by heat.

An attack which has just started may possibly be checked by removing the infected cuttings and dusting the rest heavily with sulphur or Brassicol (see damping off, p. 68).

It may also be pointed out that very sour peat litter (pH 3–3.5) may check callus formation and cause the base of the cuttings to turn black. The same happens if they are plunged too deeply.

Grey Mould (*Botrytis cinerea* Fr.)

During overwintering hydrangea buds are apt to be attacked by grey mould and rotted, especially if the plants stand under the staging in a greenhouse and are hence exposed to drips. If there is no place indoors where the plants can be kept dry and well ventilated they should be bedded down under a single layer of soil out of doors and protected against frost by straw.

An additional reason for destruction of the buds is that the plants are not properly ripened in autumn.

If the conditions for overwintering are particularly bad and the plants are poorly

ripened the shoots themselves may be destroyed (see p. 59).

Chlorosis

Hydrangeas often develop yellow leaves, usually because they are growing in a soil containing too much lime. They should only be grown on soils with a low lime content and sulphate of ammonia should be used as the source of nitrogen. Even in a lime-rich soil the new leaves may turn green if watered with a weak solution of ferrous sulphate, or sometimes of manganese sulphate (see lime-induced chlorosis, p. 31).

Mosaic

In 1935 a mosaic disease was observed in hydrangeas in a Copenhagen greenhouse. The trouble was not investigated.

Red Leaves and Brown Leaf Edges

When hydrangeas are exposed to too much sun, wind or variations in temperature, they are apt to react by reddening of the leaves. The plants should not, therefore, be exposed too much or too early when on staging, and when removed out of doors they should not be put in an exposed situation.

Brown leaf edges apparently sometimes result from the above conditions but there may probably be other undiscovered factors involved as well. Hydrangea varieties differ greatly in their liability to this disfigurement.

HYPERICUM (St. John's Wort, Rose of Sharon)

Rust (*Melampsora hypericorum* Wint.)

This forms very numerous tiny orange pustules of spores on the under-side of leaves in many wild and cultivated species of St. John's wort. Vague yellow spots may appear on the corresponding parts of the upper surface. No control measures are known.

IMPATIENS (Balsam)

Leaf Spot (*Ascochyta weissiana* Allesch.)

Large dark-brown spots, paler at the centre, appear on the balsam leaves and become dotted with the tiny blackish pycnidia. No control measures have been suggested.

IRIS

Bacterial Rot (*Bacterium carotovorum* (L. R. Jones) Lehm. and *B. aroideae* (Towns.) Stapp)

Both in Europe and North America a soft wet rot of rhizomes, stalks and the lower parts of the leaves has been repeatedly described. The leaf tips turn yellow and wither, or they may fall over at soil level. Under dry conditions the rhizome may become mealy; under moist conditions it is transformed to a putrid pulp enclosed in the loose corky skin. The rotten pulp swarms with bacteria, usually the common soft rotting organisms *B. carotovorum* and *B. aroideae*, but a third species, *Pseudomonas iridis* Van Hall, has also been described (van Hall, 1903) (see pp. 257 and 563).

The disease may be controlled by shallow planting of the rhizomes in dry, well-drained sites. All rotting shoots and rhizomes should be cut away, but the knife should be dipped repeatedly in Lysol, or other disinfectant, to avoid spreading infection.

Rhizomes from which rotten parts have been cut should be dipped for 10 minutes in 1: 1000 corrosive sublimate solution. Liming appears to promote the disease but sprinkling superphosphate around the plants and washing it in has proved beneficial.

Leaf Spot (*Didymellina macrospora* Kleb. = *Heterosporium gracile* Sacc.)

Symptoms. This attacks all kinds of iris, especially rhizomatous forms, also gladiolus, freesia, hemerocallis, narcissus and allied plants. Small elliptical brown spots appear on the leaves, surrounded at first by a yellow translucent margin, best seen by transmitted light (Fig. 282). These later become grey-brown with a dark edge and become dotted at the centre with the grey spore tufts of the fungus. These are the mould *Heterosporium gracile* which has for its perfect stage the pyrenomycete, *Didymellina macrospora*. *Heterosporium pruneti* Nich. & Agg., causing similar symptoms

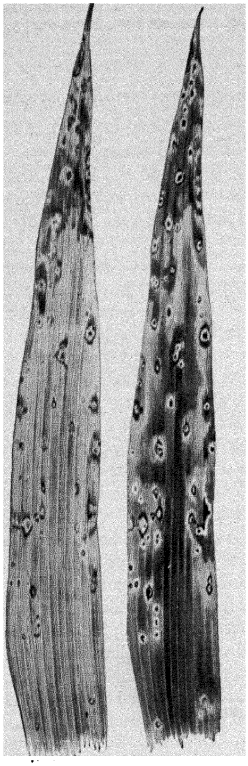


Fig. 282. Iris leaf spot: left, by reflected light; right, by transmitted light.

on *Iris germanica* in France, is a closely related species or perhaps the same.

Control. The diseased leaves should be removed in autumn. Spraying is only effective if a wetting and sticking agent has been added. Bordeaux mixture (4:4:50) is satisfactory if 5% neutral soft soap is added to the spray. Dusting with sulphur may also be tried.

Ink Disease (*Mystrosporium adustum* Massee)

This causes black crust-like patches or streaks, like splashes of ink, on the outer

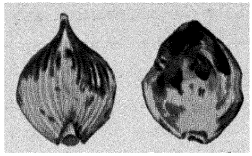


Fig. 283. Ink disease of *Iris reticulata* (*Photo Plantenziektenkindige Dienst*).

reticulate scales of *Iris reticulata* bulbs (Fig. 283). When these are planted the fungus penetrates the flesh and gradually reduces the bulb to an empty skin containing a black powder. The fungus may also cause black spots and blotches on the leaves of English and Dutch iris and occasionally occurs on the bulb scales of these and of *Iris tingitana*. It may also attack montbretia, and Green and Hewlett (1949b) have described brown spots caused by *Mystrosporium* on leaves of lachenalia.

The only control is to remove and burn the blackened outer scales and destroy all bulbs in which the underlying fleshy scales are also attacked. Green (1936) obtained no control by dipping the bulbs in 2% commercial formalin for 1–4 hours.

Botrytis Rot (*Sclerotinia convoluta* Drayt. = *Botrytis convoluta* Whetz. & Drayt.)

This disease occurs on rhizomatous irises derived from *Iris germanica*, *I. pallida* and *I. plicata* in North America and apparently occurs also in Europe. It has been studied by Whetzel and Drayton (1932).

Diseased plants either produce no leaves in spring or, if they do, the leaves turn yellow and wither. The rhizomes and bases of last year's leaves become covered with a grey or purplish-brown felt formed of mycelium and conidia of *Botrytis convoluta*. In the rotting tissue and surrounding soil arise convoluted black sclerotia up to $\frac{3}{4}$ inch across. The roots are rotten, the rhizomes shrivelled and the plants are easily pulled out of the soil. In partially attacked rhizomes there is a dark line forming a sharp boundary between healthy and decayed tissue.

Infection takes place through wounds.

The only known control measure is to remove diseased plants with the surrounding soil. Damaging the rhizomes should be avoided as far as possible and they should be planted high.

Grey Bulb Rot (*Sclerotium tuliparum* Kleb. = *Rhizoctonia tuliparum* Whetz. & Arth.)

The bulbs decay and brownish sclerotia develop on them. This is best known as a disease of tulip, but Klebahn found *Iris hispanica* was just as easily attacked (see p. 542).

Rust (*Puccinia iridis* (DC.) Wallr.)

This rust forms dusty brown pustules of uredospores on yellow leaf spots during the summer. Later, blackish-brown pustules of teleutospores occur. There is no alternate host. In Britain rust occurs mainly on the wild irises and on cultivated varieties of the *I. germanica* type. In North America physiological races of the rust have been distinguished (see also rust, p. 74).

Crown Rot (*Sclerotium delphinii* Welch)

S. delphinii is a sterile sclerotial fungus first described in North America but now known to occur also in England, France and Holland. It attacks the leaf bases of bearded and bulbous irises and the flower stalks, rotting them so that they fall over. The decayed parts become coated with white fan-like mycelium, in which develop the sclerotia, at first white, then cream-coloured and finally dark reddish-brown, up to ¼ inch across.

Diseased plants should be dug up, cut clean and dipped for 10 minutes in 1 : 1000 corrosive sublimate solution before being replanted in clean soil.

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

The leaf bases and younger parts of the rhizomes of *Iris germanica* and *I. pumila* may sometimes be attacked by this fungus. They become covered with fluffy white mycelium containing large black nodular sclerotia. The rot spreads very rapidly.

Diseased plants should be dug up as soon as noticed, to avoid sclerotia being left in the soil, and the soil should be well soaked with 2% formalin solution (see further, p. 77).

Iris Mosaic (*Iris* Virus 1)

This affects mainly the bulbous irises, but has been seen occasionally in *I. tuberosa*, *I. pumila* and *I. reichenbachiana* (Moore, W. C., 1939b). It has been known on bulbous irises in Denmark since 1935, in England since 1928, and its virus origin was demonstrated by Brierley and McWhorter (1936) in North America in 1934. The leaves may bear a light and dark green mottle, especially prominent on the young leaves and bud sheaths, or, in early winter, the leaves may be striped with yellow. Yellow leaf stripes may also, however, be due to low temperature. The flowers may be marked with dark spots or patches.

The virus is transmitted by two species of aphides.

Wedgewood is a particularly susceptible variety, so is Imperator in Denmark. Lily is not susceptible and the virus could not be transmitted to turnip, tomato or tobacco.

The only control measures are to dig out diseased plants and then to keep down aphides by spraying or dusting with a suitable insecticide.

IVY

See *Hedera*, p. 468.

IXIA

This plant is sometimes attacked by *Sclerotium tuliparum* (see p. 542), and sometimes by *Botrytis cinerea* on the leaves (see p. 59).

JUNIPERUS (Juniper)

Rust

Various species of juniper are teleutospore hosts of rusts which have their aecidial stages on other plants (see apple, p. 128; hawthorn, p. 440; pear, p. 185).

KALANCHOË

Mildew (*Oidium* sp.)

In 1935 some plants of *Kalanchoë blossfeldiana* were sent to the Danish State Experimental Station with a severe outbreak of mildew. The spots were black in the centre where the leaf tissue had been destroyed, while round the edge of the spots was a border of mycelium and conidia (Fig. 284). The

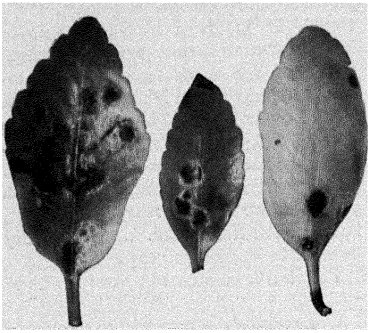


Fig. 284. Mildew of *Kalanchoe blossfeldiana* (*S. p. F.*)

plants had obviously been grown under unfavourable conditions, probably with too much moisture and too little light, and had been overfed. In subsequent years *Kalanchoë* mildew has been fairly frequent.

In Sweden Hammarlund has transferred the *Kalanchoë* mildew to *Bryophyllum tubiflorum*, *Crassula coccinia* and seven kinds of *Sedum*. Only two of all the *Crassulaceae* tested were not attacked by the fungus and several became so heavily infected as to suffer from severe leaf fall.

The plants should be so treated as to make firm growth and the usual control measures against mildew should be applied (see p. 63).

Stem Rot (*Phytophthora cactorum* (Leb. & Cohn.) Schroet.)

Neergaard first observed this disease on *Kalanchoë blossfeldiana* in 1935 and it has occurred several times since. It starts as a watersoaked rot usually at soil level and soon turns dark brown, spreading up the stems and down into the roots. We have also seen an attack commence some way above the soil surface.

Phytophthora cactorum is quite a common soil fungus and can attack many plants.

It seems best to keep the plants dry with plenty of light and not to give them much nitrogenous manure. Spraying with Bordeaux mixture, watering with Cheshunt compound or with a $\frac{1}{4}\%$ solution of an organo-mercury fungicide may be tried to check an outbreak at the start.

LABURNUM (Golden Rain)

Leaf Spots

The fungi *Phyllosticta cytisi* Desm., *Septoria laburni* Pass., *Phyllosticta cytisorum* Pass. and *Coniothyrium laburnophilum* Oud. have all been credited with causing laburnum leaf spot in Denmark. The first-named is common in Britain, causing irregular or round grey-brown spots with a narrow brown border. According to Grove it is probably a stage in the development of *Ascochyta kabatiana* Trot.

The *Septoria* found on laburnum leaves in Britain has been called *S. cytisi* Desm. with which *S. laburni* Pass. may be identical.

If these leaf spots become prevalent a fungicidal spray may be tried but Grove comments that *S. cytisi* commonly follows attacks by a leaf-mining insect, in which case control measures should primarily be directed against the latter.

Brown Spot (*Ceratophorum setosum* Kirchn.)

This not infrequently causes serious damage to laburnum in nurseries. Small brown spots appear on the leaves and enlarge into irregular brown blotches. They may occur also on the petioles and young shoots. The leaves fall, growth of the plant is arrested and in the worst cases it may be killed. A single Bordeaux spray has not controlled the disease; apparently it is necessary to spray repeatedly to keep the new leaves covered with the fungicide.

Canker (*Curcurbitaria laburni* (Pers. ex Fr.) de Not.)

Dead branches of laburnum may be covered by a black crust-like layer of perithecia of this fungus, breaking through the bark (Fig. 285).

The crust may be an inch or two long. The fungus enters through wounds and causes dark longitudinal stains in the wood. Eventually the wood turns brown and the bark yellow.

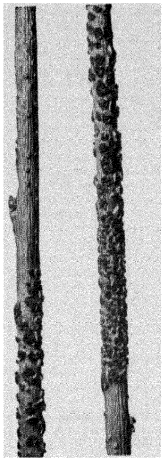


Fig. 285. *Cucurbitaria* on laburnum twig.

On the still-living infected branches and twigs the fungus forms two kinds of pycnidia, one with small pale, 1-septate spores, formerly called *Diplodia cytisi* Auersw. = D. *rudis* Desm. & Kickx., the other larger with brown multiseptate spores, formerly called *Camarosporium laburni* (Westd.) Sacc. & Roum.

A separate pycnidial fungus, *Phomopsis rudis* (Sacc.) Von Höhnel, is also sometimes found on dead twigs of laburnum.

Inoculation experiments have shown that *Cucurbitaria laburni* is unable to attack vigorously growing trees even through wounds. It can, however, establish itself on dead snags and spread thence into living branches of trees already weakened by some other cause.

Dead wood should be pruned away and the larger wounds coated with white lead paint or grafting wax (see silver leaf, p. 192, to which laburnum is also subject) (Green, F. M., 1932).

Downy Mildew (*Peronospora cytisi* Rostr.)

The leaves become spotted with brown on the upper surface, while the corresponding parts of the lower surface are covered with a layer of light grey mould. This downy mildew is specially damaging to nursery stock. Infected plants should be sprayed several times with Bordeaux mixture or other copper fungicide.

Rust (*Uromyces genistae-tinctoriae* Wint.)

The uredospores form cinnamon-coloured pustules on pale leaf spots; the teleutospores occur in similar but darker brown pustules at the end of the year. This rust was found on laburnum in Britain in 1946 but occurs also on many species of *Cytisus*, *Genista* and *Ulex*, including the common wild broom and gorse.

LACHENALIA

See *Iris*, Ink disease, p. 478.

LARKSPUR

See *Delphinium*, p. 446.

LARIX (Larch)

Canker (*Trichoscyphella willkommii* (Hart.) Nannfeldt = *Dasyscypha willkommii* (Hart.) Rehm.)

Where larch is grown under conditions not completely favourable to its growth it is often attacked by a little cup fungus which causes canker. Trees in bad situations, as in low wet soil or in the bottom of a valley, may be killed by it; others, if they survive, are marred by large open cankers on trunk and branches. These are flat and surrounded by a well-developed ridge of callus. The start of new cankers may be recognized by cracking of the bark and an outflow of resin. In spring the needles on diseased branches may be yellow. On the margin of the cankers the fungus forms its fruit bodies. They are from $\frac{1}{8}$–$\frac{1}{4}$ inch

across with a white and hairy outside, enclosing an apricot or orange-coloured disc which is fully exposed in wet weather, somewhat concealed by the inrolled margins in dry weather (Hahn, G. G., and Ayers, T. T., 1934) (Fig. 286). On dead and fallen twigs of healthy rows of white dots along the under-side of the needle. Microscopic examination is necessary for a definite diagnosis, however.

Peace and Holmes (1933) found that 10,000–360,000 spores might be produced on each infected needle.

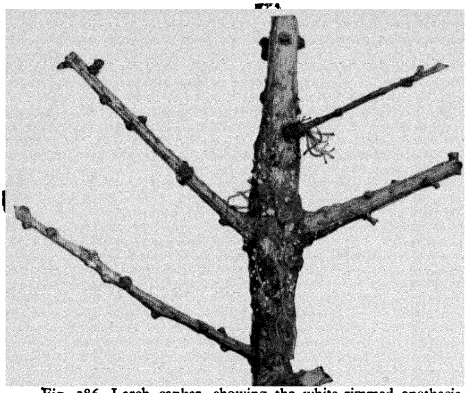


Fig. 286. Larch canker, showing the white-rimmed apothecia of *Trichoscyphella willkommii* (*Photo Dept. Agric. Scotland*).

larch a very similar but rather smaller orange and white cup fungus occurs very commonly, but this is harmless and does not cause cankers.

Hahn and Ayers have shown (1943) that though *T. willkommii* is particularly destructive to larches damaged by frost it is a wound parasite able to attack non-frosted trees. Infections occur especially during the summer months.

Larix leptolepis is comparatively resistant.

Leaf Cast (*Meria laricis* Vuillemin)

Leaf cast is the worst disease of young larch trees, especially of nursery stocks. The first sign of the trouble is a discolouration and browning of the needles, starting usually about the beginning of May. Infection may occur on any part of the needle, but soon afterwards all that part above the point of infection dies, so that the disease appears to be one in which the needles die from the tip. Fructifications of the fungus emerge through the stomata and can be seen under a strong lens as two

Infected needles are largely shed prematurely, but some remain attached to the plant over winter and do not fall till the following spring. The fungus overwinters in these and to a lesser extent in the fallen needles on the ground.

After trees have attained a height of 8–10 feet they suffer much less from the disease, which is therefore chiefly important in the nursery. It is greatly favoured by moisture and checked by dry weather.

Peace and Holmes found leaf cast was fairly easily controlled by sulphur sprays, of which the first should be at the end of February, the second at the end of March, followed by others every 2–3 weeks. They recommend for the first spray Amberene 3–5 gallons, Sulsol 2–3 gallons or liver of sulphur 14 lb. per 100 gallons, for subsequent sprays Amberene 1–1½ gallons., Sulsol 1 gallon or liver of sulphur 7 lb. per 100 gallons.

Damage by *Meria* can be distinguished from browning due to frost in the following ways:

Meria kills leaflets from the tip downwards; frost turns them brown all over at once. *Meria* seldom attacks needles at the tip of the shoot, which is the part most affected by frost. Needles killed by *Meria* fall soon after they have become completely brown; those killed by frost, though more shrivelled, tend to remain on the twigs throughout the season.

Rusts

A large number of rusts which have their uredo and teleuto stages on deciduous trees and shrubs, form their aecidial stage on larch needles. Those known to occur in Britain include:

Melampsora larici-caprearum Kleb. with uredo and teleutospores on goat willow.

M. larici-epitea Kleb. with uredo and teleutospores on osiers and other willows.

M. larici-pentandrae Kleb. with uredo and teleutospores on *Salix pentandra*.

M. larici-populina Kleb. with uredo and teleutospores on several species of poplar.

M. larici-tremulae with uredo and teleutospores on aspen and white poplar.

All these produce indistinguishable symptoms on larch needles, viz. bright yellowish-orange spots about $\frac{1}{25}$ inch across, which are very seldom noticed and apparently cause little damage to the trees.

Two other rusts on larch form larger cluster cups on the needles, up to about $\frac{1}{16}$ inch across, with torn white margins and reddish-orange contents. They are *Melampsoridium betulinum* Kleb. and *M. alni* Diet. with uredo and teleutospore stages on birch and alder respectively. Borthwick and Wilson (1913) found that these fungi infect larch needles early in the season, that the cluster cups, which form a row on one or both sides of the midrib on the under-side of the leaf, are ripe on needles 3 weeks old and that the discoloured leaves are shed by the end of July. Diseased needles may be much curved and distorted.

None of these rusts appears to be of any economic importance on larch.

LATHYRUS ODORATUS (Sweet Pea)

Downy Mildew (*Peronospora viciae* (Berk.) de Bary)

Pale spots occur on the leaves on the underside of which is a light-grey or pale-mauve layer of mould. The leaves slowly turn yellow and wither (see also p. 309).

The disease may be controlled by spraying or dusting with a copper fungicide.

White Mould (*Ramularia deusta* (Fuck.) Baker *et al.* = *Cladosporium album* Dows.)

This disease is common in the South and West of England and is known also in Holland and North America but not as yet in Denmark. It somewhat resembles ordinary powdery mildew, for on both sides of the leaves and on the wings along the stem there appears a mealy white powder, often covering a large part of the plant. The parts attacked become slightly shrunken; in severe attacks they develop a pale yellowish-brown colour and the leaves are shed.

White mould is worst under damp, warm conditions, which should therefore be avoided for sweet peas as far as possible. Dowson (1924), who described the fungus under the name *Cladosporium album*, controlled it by dusting with sulphur. *Erostrotheca multiformis* (Martin and Charles) has been claimed to be the perfect state of this mould but Baker *et al.* (1950) consider the evidence insufficient.

Root Rot (*Aphanomyces euteiches* Drechsl., *Fusarium culmorum* (W. G. Sm.) Sacc., *F. sambucinum* Fuck., *Thielaviopsis basicola* (Berk. & Br.) Ferraris)

This disease complex, already described under culinary pea (p. 308), also occurs on sweet pea.

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

Plants rot at the foot and some way up the stem. On the decayed part a white mycelium appears, followed by large black sclerotia (see p. 77).

Mildew (*Erysiphe polygoni* DC.)

This fairly common disease causes a thin, white floury coating over larger or smaller parts of the leaves and stems (see mildew, p. 63).

Leafy Gall (*Corynebacterium fascians* (Tilford) Dowson)

Many short, thick, fleshy and aborted stems with misshapen leaves develop at or below soil level. They originate either at the cotyledonary node, or at the node from which the first true leaves should spring. On old plants the mass of fasciated branches resembles a small witches' broom, about 1–2 inches tall (Fig. 287). This is quite different from the

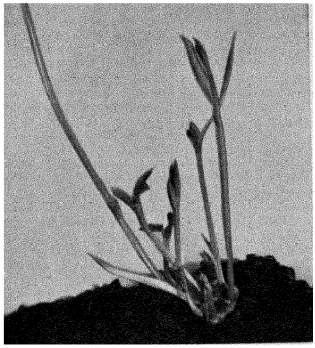


Fig. 287. Leafy gall of sweet pea (*Photo Dept. Agric. Scotland*).

little irregular nodular galls caused by the crown gall bacterium in sweet peas and is due to a different organism, first described under the name *Phytomonas fascians* Tilford (1936) but subsequently transferred by Dowson to the genus *Corynebacterium*. Tilford found that the leafy gall organism could cause similar symptoms on garden peas, petunia, pelargonium, tobacco plants, gypsophila and *Chrysanthemum indicum*. Lindfors (1938) found it on *Chrysanthemum maximum* in Sweden. It has also been shown to attack *Asparagus sprengeri*, dianthus, gladiolus, *Heuchera sanguinea* and strawberry (see p. 364).

Mosaic (*Pisum* Virus 2)

In Denmark the first serious attack of sweet pea mosaic was seen in greenhouse plants in 1924, but it has since been seen just as severe out of doors. The leaves turn pale along the veins, or less often between them, and may be a little curled or twisted. The paler parts of the leaves are thinner than normal and finally they become yellowish all over. The flower stalks are short and the petals spotted or feebly coloured. Varieties differ greatly in reaction to mosaic; some seem seldom to be infected, others grow away from it, while yet others are seriously deformed.

Infection is spread by aphides, with an incubation period of about ten days. The same virus infects sweet pea, culinary pea, red clover, beans, broad beans and lupins. Seed-borne infection has not been proved but there is strong circumstantial evidence that it does take place.

Streak (*Lactuca* Virus 1, *Lycopersicum* Virus 3, etc.)

Reddish-brown or chocolate-brown streaks appear on stems, leaf stalks and leaves. The attack usually starts near the base of the plant and spreads upwards. It was first described in England, then in America and has been known in Denmark since 1921.

Streak was originally ascribed to a bacterium, *Bacillus lathyri* Manns & Taubenhaus (1913), but this view is now discredited. Ainsworth and Ogilvie (1939) have shown that in England sweet pea streak is commonly due to infection with the virus of lettuce mosaic, which is spread by aphides. There is an incubation period of 3–4 weeks after infection, then severe streaks appear near the base of the stems (see p. 290).

Some shoots are killed but in those that survive the apical leaves, though full of the virus, show little or no symptoms except perhaps for a very mild mottle.

Snyder and Thomas (1936) have shown that a very similar disease is caused in sweet pea by the virus of tomato spotted wilt and other American authors have produced streak in sweet pea with viruses from clover.

Shedding Buds

In forced sweet peas the buds are apt to be shed instead of blooming during the short days of winter. In Denmark outdoor plants have also been observed to behave similarly after heavy rain.

In forcing, the grower should try to avoid the trouble by providing artificial light during the dark time of year and by avoiding excessive moisture and warmth. Out of doors the soil should not be allowed to get very dry. Regular watering should be adopted to prevent a sudden change from drought to plenty of water after rain.

Poor growth of sweet peas is sometimes due to lack of the usual nodules containing nitrogen-fixing bacteria on the roots. Where experience shows these organisms are likely to be absent from the soil the seed should be inoculated with a pure culture of the bacteria before being sown. Pure cultures are supplied by the State Laboratories at Lyngby.

LAVANDULA (Lavender)

Shab (*Phoma lavandulae* Gabotto)

The first symptom of the disease is a yellowing, wilting and death of some young shoots during the summer months from May to August. The disease spreads downwards from there, lower branches become infected at their bases and from them the fungus spreads upwards into others until all are involved. Finally the roots are killed.

The disease is caused by the fungus *Phoma lavandulae* Gabotto, which forms tiny black pycnidia in large numbers on the young dead twigs, less often on decorticated branches and on dead leaves, where they are hidden by the hairs. Infection takes place either through wounds, such as those made when the crop is harvested for the perfume industry, or through uninjured leaves or leaf axils. In the latter case the leaf base and adjacent part of the young stem turns brown while the leaf becomes at first bright yellow, then brown and curled. A long period, varying from two months to a year or more, elapses between infection and the outbreak of the characteristic symptoms of shab.

The disease is spread by cuttings from infected plants, by infected leaves and twigs being distributed about the field during cultural operations or by wind and, locally, by splashing of spores exuded from the pycnidia during rain storms. The fungus can exist as a saprophyte in the field on dead stems of the common weed fat hens (*Chenopodium album*).

Control means include rotation of crops, avoiding heavy ill-drained soils, eradication of weeds which weaken the bushes and help the fungus to survive, taking cuttings from healthy plants, using only green wood, and destroying infected plants. In an infected field some control may be obtained by clipping the plants drastically with shears at the end of February or beginning of March and collecting and burning the clippings. This practice tends to delay flowering and renders the plants more liable to damage by late frosts (Metcalfe, C. R., 1931).

LAVATERA

Leaf Spot and Dieback (*Colletotrichum malvarum* (Braun) Southw.)

This fungus may be very destructive to *Lavatera* and Malope. Brown oblong spots develop on the stems, on which the conidial pustules appear as small dark dots. Larger or smaller angular spots of similar appearance are found on the leaves (Fig. 288). The disease may kill both leaves and shoots.

An attack may perhaps be checked at the outset by spraying or dusting with a fungicide.

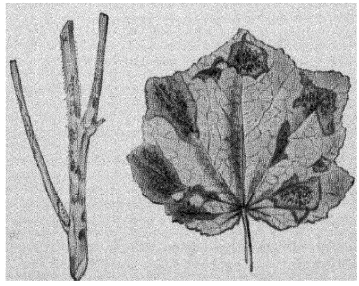


Fig. 288. *Colletotrichum malvarum* on *Lavatera* stem and leaf.

LIGUSTRUM (Privet)

Twig Blight (*Gnomonia cingulata* Beck= *Myxosporium cingulatum* Beck.)

In Germany in 1925 Beck found young privet plants on which the leaves were brown and limp and the twigs dying. At the junction of the current and past season's wood were dark conidial pustules.

Old plants may be similarly attacked, the twigs dying back to a constriction which marks the part invaded by the fungus.

Beck (1926) called the conidial state *Myxosporium cingulatum* and showed that it has a perithecial state *Gnomonia cingulata*.

In America a very similar disease of privet has been attributed to the fungus *Glomerella cingulata* (Stonem.) Spauld. & Schrenk, the cause of bitter rot of apple and other fruit and of various minor diebacks in shrubs and trees.

No control measures have been prescribed but probably an outbreak could be checked by clipping off the diseased twigs or removing affected plants from a seed bed and treating the remainder with a fungicide.

Leaf Fall and Dieback

It is a common occurrence, especially in allotments at Copenhagen where many privet hedges occur, that over part of a hedge the leaves become small and limp during the summer and then fall, leaving the branches almost bare. Affected bushes often die back severely or sometimes completely.

The symptoms suggest *Verticillium* wilt but we have never been able to demonstrate *Verticillium* or any other fungus or bacterium as the cause of this dieback.

It appears that aphis infestation may be connected with it. Leaves attacked by aphides roll up and are shed early. The aphides disappear during the summer and their presence earlier is often overlooked. By the time the leaves begin to fall the aphides have gone and the grower sees no reason for their dropping. The result is lack of normal ripening in the shoots, which then easily suffer from frost and are found to be dead in the following year.

It is nearly always *Ligustrum vulgare* and *L. v. atrovirens* which die back, and Consul Laurids Nielsen has informed us (1939) that in his many visits to allotments he has often observed these species infected by aphides while *L. ovalifolium* was never attacked. If the aphides are killed with nicotine or similar insecticide before the leaves are damaged the branches do not die in the following year. If leaf fall has commenced before the insecticide is applied, dieback will usually follow in spite of the treatment.

In England death of privet hedges has been ascribed to *Armillaria mellea* (Fr.) Quél. (see p. 456), *Rosellinia necatrix* Prill., *Verticillium dahliae* Kleb. and to a species of *Phytophthora* (Moore, W. C., 1943).

LILAC

See *Syringa*, p. 534.

LILIUM (Lily)

Lily Disease (*Botrytis elliptica* (Berk.) Cooke)

Symptoms. The lily disease may attack many species of *Lilium* but *L. auratum* is perhaps the most sensitive. Symptoms vary greatly with the weather. In dry weather only small red-edged spots appear on the leaves and flowers. Often they have orange centres. In this form the disease does very little damage.

If the summer is damp the fungus develops much more vigorously. The flower buds are destroyed and become covered with a grey layer of mould. Developing leaves wither at the tips and numerous large yellowish spots appear on leaves, stems and flowers (Fig. 289). If a dry period supervenes the fungus stops fructifying and may be difficult to find.

On the leaves the spots are often oval with their larger diameter towards tip and base of the leaf. Their appearance differs greatly on the various species of lily, they may be more or less grey, orange or reddish-brown, with or without a distinct marginal zone. On *L. candidum* they are usually grey and translucent.

When the plants are 6–8 inches high their tips may be attacked and growth of the stem arrested. Large, sunken spots may develop on the stem at any point between the tip and



Fig. 289. Lily disease.

soil level. Irregular, often brown, spots appear on the flowers which may be deformed. Under moist conditions small black sclerotia may develop on the rotting tissues.

Bulbs are very rarely attacked, but Moore (1939b) has observed rotting of the inner bulb scales and sclerotium production there in a few cases.

Cause. The disease was first described by Berkeley in England in 1881, who called the fungus *Ovularia elliptica*. In 1888 Smith called it *Peronospora elliptica* and in 1889 he referred to it as *Polyactis cana*. In 1888 Marshall Ward described the lily disease in detail and showed that it was due to a species of *Botrytis*. In 1901 Cooke recognized that this *Botrytis* was the same as Berkeley's fungus of 1881 and therefore named it *B. elliptica* (Berk.) Cooke. In 1931 Van Beyma and Van Hell found that two races of the fungus exist; one does not form sclerotia and is only a feeble parasite, the other produces them and is highly parasitic.

Control. Collect and burn the dead above-ground parts of lilies in winter and plant the bulbs in an airy place that is not too damp; but lilies cannot stand exposure to the wind either.

Bordeaux mixture 1 : 1 : 100 gives good control but the number of sprays required depends on the wetness of the season. A copper lime dust may also be effective.

Leaf Spot (*Cercosporella inconspicua* (Wint.) von Höhnel)

More or less circular chestnut-brown spots up to $\frac{1}{2}$ inch across may appear on the leaves of *Lilium martagon*, *L. thunbergianum* and *L. speciosum*. They have black borders and their centres become covered with a fine grey mould composed of the spores of *Cercosporella inconspicua*. Under moist conditions the disease may become serious enough to cause defoliation. Leaf spot caused by this fungus occurs in Italy, Hungary, Austria, Switzerland and France. A very similar disease of *L.*

candidum has been ascribed to *Cylindrosporium inconspicuum* Wint. var. *candidum* Sacc. & Fautr.

Leaf spot may be controlled by spraying with a copper fungicide.

Bacteriosis (*Bacillus lilii* Uyeda)

This disease was described in Japan in 1919 and has not been definitely identified elsewhere, but as Japanese lilies are distributed far and wide there is considerable opportunity for its spread.

The disease appears as dark-brown, oval or irregular spots on leaves and bulb scales. When the bulbs are infected the young plants grow slowly. Pale-brown streaks appear on the stalks which may be killed, and the infected leaves fall prematurely. Infection may be carried in the bulb or be contracted from the soil. It is important to scrutinize imported lily bulbs and to avoid planting any that appear diseased.

Rust (*Uromyces lilii* Fuck.)

Small powdery brown pustules appear on leaves and stalks and may join up to form larger continuous patches. Lily rust has been found occasionally in England on *L. candidum*.

Several early sprayings with Bordeaux mixture may protect against the disease. Infected leaves should be picked off and burnt.

Storage Rots of Bulbs (*Rhizopus* spp. & *Penicillium* spp.)

Lily bulbs are very prone to rot in storage and in transit. Often this is due merely to unfavourable conditions of storage or packing, but two fungi, *Rhizopus necans* Massee and *Penicillium corymbiferum* Westling, may play an active part in the decay.

Symptoms and causes. Bulbs attacked by *Rhizopus* develop watersoaked spots in the scales. These are darker than the normal tissue. From the scales the fungus grows down into the base and from that up again into other scales. In a few days a bulb may become completely rotten and soft with a sour smell. The fungus apparently penetrates through wounds, especially those made by removing the roots before packing.

The *Penicillium* causes a dryer rot, which starts as small sunken brown spots on the scales. As they increase in size a fine white mould appears on them, followed by the little blue-green tufts of spores. Gradually the rot spreads through the whole scale and down to the basal plate but it may take 2–3 months to involve the whole bulb. If only the outer scales are affected little harm is done, but if the fungus spreads to the scales round the bud no growth will take place after planting.

Rhizopus attacks bulbs under warm conditions, as in transit from Bermuda or Japan. *Penicillium* is active at lower temperatures in stored bulbs.

According to O'Leary and Guterman (1937) *Lilium auratum*, *L. rubellum* and *L. japonicum* are very susceptible to *Penicillium* bulb rot, *L. speciosum* less so and *L. longiflorum* is resistant.

American and European lily bulbs seem to suffer less than those from Japan, perhaps because of different treatment. In Japan the bulbs are packed in soil and sent in tightly closed wooden boxes. In America and Europe they are packed in more open boxes in peat moss or sawdust.

Control. Great care should be taken not to wound the bulbs. If possible the roots should not be cut off. If the bulbs are packed individually in waxed paper the spread of the fungus is checked. Packing material used should be such as does not absorb heat and should not be damp.

Dusting the bulbs with sulphur or copper dusts has not been effective. Mercury preparations control the fungi but damage the bulbs, staining the scales and stunting growth.

O'Leary and Guterman obtained excellent control of *Penicillium* rot by mixing bleaching powder (calcium hypochlorite) with the packing subsoil, at the rate of 5–6 oz. of powder to 50 lb. of soil. This caused no damage to the bulbs and also controlled the common bulb mite (*Rhizoglyphus echinopus*).

Stump Rot (*Phytophthora parasitica* Doster, etc.)

In North America and Bermuda *Phytophthora parasitica* has been identified as the cause of a rot which attacks the centre of the rosette or base of the leaves of *Lilium longiflorum* var. *eximium* soon after they appear above ground. The fungus occurs in the soil and is splashed on to the leaves especially during watering or by heavy rain.

The disease is controllable by draining the soil, avoiding wetting the leaves in indoor plants, and spraying with Bordeaux mixture. Pot plants may be watered with Cheshunt compound.

Both *P. cactorum* and *P. parasitica* may also attack the base of the stem in older plants of other lilies such as *L. candidum*, *L. pyrenaicum*, *L. regale*, *L. speciosum* and *L. washingtonianum*. The part of the stem below ground decays and the plant falls over. Control measures are similar to those for damping off (p. 68).

Root Rot and Bulb Rot (*Cylindrocarpon radicicola* Wollenw.)

Symptoms. Diseased plants develop more slowly than normal in autumn and have smaller, paler, sometimes curled leaves. When they are pulled up the bulb roots are found to be decayed and there are very few stem roots. Gradually the stems begin to dry up from the base and the lower leaves are shed. In warm, dry weather the stalks die completely but usually remain standing, though they easily break if pulled. The bulb is almost completely decayed, its basal plate is rotten and the scales fall apart. In mild attacks the bulb may survive all the season but the top fades early in autumn and when the bulb is lifted few or no roots are found.

In early stages of the attack a few or many oblong brown spots occur on the root, which spread until it is girdled by them. From the root the rot spreads into the base of the bulb and thence into the scales. If infected bulbs are planted the following year they are unable to form roots. The top develops but can only grow as long as the reserve of food in the bulb lasts. The infection may persist for several years before completely destroying the bulb.

Cause. Van Hell (1931) isolated *Cylindrocarpon radicicola* from diseased roots and thought it capable of reproducing the disease when he grew lilies in soil contaminated with it.

C. radicicola is also credited with causing root rot of *narcissus* but the general opinion is that it is a feeble parasite, at most able to attack roots injured by other causes.

Ogilvie (1937) was unable to reproduce the disease by inoculation with *C. radicicola* and concluded that the trouble is primarily due to waterlogging and other adverse soil conditions.

Rosette (*Lilium* Virus 1)

Rosette is a disease of *Lilium longiflorum* and its varieties, sometimes known as *L. formosum* and *L. giganteum*. It is prevalent in Bermuda, where it is known as " yellow flat," and in parts of North America. It seems to have been introduced into England, Holland, Sweden and North Germany in bulbs imported from Bermuda and Japan. Infected plants have yellow leaves which remain in a basal rosette. No flowering stem is produced. Plants infected during the current season appear normal below but have small yellow down-curled leaves above and their flower buds fail to open or give distorted blooms. The virus is spread by aphides, especially *Aphis gossypii*. In England and North Europe this is a disease of glasshouse plants, but in Bermuda, where *L. longiflorum* is cultivated out of doors on a grand scale, it occurs in patches in the fields. It is controlled there by regular inspection and roguing of the crop and by nicotine sprays and dusts. Export of lily bulbs is permitted only from fields certified free from rosette.

Mosaic (*Cucumis* Virus 1)

This is a much more widespread disease than rosette and attacks many species of *Lilium*. It causes pale spots and streaks in the leaves, differing somewhat according to the kind of lily concerned. There may also be dwarfing

and distortion of leaves and in *L. auratum* the flower buds and upper leaves of recently infected plants may drop, leaving a bare, crooked stem. The virus is spread by aphides and also to some extent by contact. Spread may occur in stored bulbs if aphides are present on them (Smith, T. F., and Brierley, P., 1948).

Control measures include destruction of infected plants, fumigation or spraying to keep down aphides and avoiding propagation from diseased plants. The disease is not transmitted through the seed.

Limber Neck (non-parasitic)

This condition in lilies has been observed only in North America and occurs there both out of doors and under glass.

When the plants are 1–2 feet high the stem collapses and shrivels at a point a few inches below the growing tip. If the wilted part is cut off the rest of the stem continues to flourish but, of course, it does not flower. Affected plants often have a poor root system. Damp, shady surroundings and low temperatures seem to be contributory causes.

The actual cause of the condition is unknown and we can therefore suggest no remedy, except that it is well known that lilies like " their feet in the shade and their heads in the sun."

LILY OF THE VALLEY

See *Convallaria*, p. 438.

LIME

See *Tilia*, p. 537.

LOBELIA

Leaf Blotch (*Septoria lobeliae* Peck)

Leaves of *Lobelia syphilitica* infected by this fungus develop irregular pale spots, spreading inwards from the tips or margins and bordered by broad pink or purplish bands. The older leaves may be killed. Pycnidia of the fungus appear as black dots, in small groups scattered over the upper surface (Moore, W. C., 1940a). The disease occurs also on *L. erinus* and *L. inflata*.

Lobelia cardinalis may be affected by a mosaic disease caused by the virus of cucumber mosaic (see p. 278).

LONICERA (Honeysuckle)

Leaf Spots

Many leaf-spotting fungi have been described on honeysuckle. The following are common in Britain:

Phyllosticta lonicerae Westd. causes rounded or irregular, reddish-brown spots, becoming paler with a brown border. Dotted over the surface are the rather conspicuous black pycnidia. *Ascochyta vulgaris* Kab. & Bub. may be the same fungus in a more developed state.

Leptothyrium periclymeni Sacc. causes similar round spots, about $\frac{1}{4}$ inch across, bearing shield-shaped black dot-like pycnidia on the upper surface.

Mycosphaerella clymenia (Sacc.) Oudem. forms minute dot-like perithecia in small dark brown spots, sometimes with a yellow margin.

Rust (*Puccinia festucae* Plowr.)

Small round yellow or brownish spots are formed by this rust on honeysuckle leaves in June and July. Their lower surface is covered by the cluster cup (aecidium) stage of the rust, which has its uredo and teleutospore stages on species of Fescue (see p. 554).

Mildew (*Microsphaera alni* (Wallr.) Wint. var. *Lonicerae* DC.)

This forms the usual powdery white coating of a powdery mildew on honeysuckle leaves (see mildew, p. 63).

LUNARIA (Honesty)

This plant sometimes suffers from club root (*Plasmodiophora brassicae*) (see p. 246, white blister (*Cystopus candidus*) (see p. 253) and by a virus disease causing mottling of the leaves and breaking of the flower colour. Both cabbage ring spot virus (see p. 258) and cucumber mosaic virus (see p. 278) can infect honesty (Moore, W. C., 1943).

LUPINUS (Lupin)

Dropping of Flowers

In 1931 white lupins shed the middle flowers of the spike at many places in Lolland and Falster, and we have later seen the trouble elsewhere. Probably it was due to drought but it is remarkable that red and blue lupins under the same conditions were unaffected.

Brown Spot (*Ceratophorum setosum* Kirchn.)

Leaves, stems, pods and seeds may all be attacked by this fungus which has also been called *Pestalotia lupini* Sor. Others refer it to the genus *Mastigosporium*. The spots may be small and dark brown but those on the leaves may attain a diameter of ½ inch or more (Fig. 290) and those on the stalks, which are often

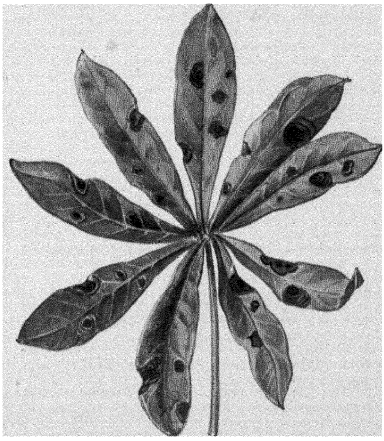


Fig. 290. Lupin leaf-spot caused by *Ceratophorum setosum*.

oblong, may be nearly an inch long. In the leaves they often have a yellow edge. If infected seed is sown the root or stem of the seedling may be attacked and the plant killed. Hence seed should only be saved from healthy plants. At the outset of an attack an attempt may be made to check it by spraying or dusting with a fungicide (Green, D. E., 1933).

Grey Mould (*Botrytis cinerea* Fr.)

The lower part of the stem is most attacked. Large brown patches appear which become covered with a grey mould (see p. 59).

Myrothecium roridum Tode ex Fr. may also attack the base of lupin stems and cause wilting (see p. 561).

Mildew (*Erysiphe polygoni* DC.)

This often forms a fine, white powdery coating on leaves of garden lupins (see mildew, p. 63).

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

A number of the leaves and stems wilt with watersoaked patches on them. On and in the stalks and leaves are found the white mycelium and large white or black sclerotia of the fungus. Pods and seed may also be attacked (see p. 77).

Browning (*Cucumis* Virus 1)

Infected plants develop dead brown spots on the leaves and brown streaks on the stems. The leaves are often distorted and the plant may fail to flower. When flowers are produced the spikes are thin with long spaces between the individual blooms. The plants are greatly weakened and may fall a prey to root rots or other diseases. This virus is spread by greenfly from a large number of ornamental plants (see p. 278).

Sore Shin

Is another virus disease caused by the common pea mosaic, *Pisum* Virus 2. A brown streak appears along one side of the stem, the young leaves wilt, turn black and fall, the older ones go yellow and fall also. Finally the stem turns black, the roots decay and the whole plant dies (Chamberlain, 1935) (see p. 310).

LING

See *Erica*, p. 455.

MAHONIA

Rust (*Cumminsiella sanguinea* (Peck) Arth. = *Puccinia mirabilissima* Peck)

This is an American rust which has become widespread in Northern Europe since its discovery in Scotland in 1922. It was first observed in Denmark on *M. aquifolia* in 1925, in a collection from Falster on 6-year-old plants which until then had been perfectly healthy.

Now it is common throughout the country and there are indications that in some places in Denmark it was present before 1925.

Symptoms. The rust pustules form small brown dots with a reddish margin on the under-side of the leaves. The red colour forms a conspicuous red spot on the upper surface as well (Plate 10, Fig. E.). Severe attacks not only spoil the appearance of the leaves but may cause them to wither and fall. These pustules contain uredospores mixed with a few teleutospores. Less often one finds small groups of yellow cluster cups seated on thickened blackish-green spots on the under-side of the leaves. The upper surface of these spots bears spermogonia (Fig. 291).

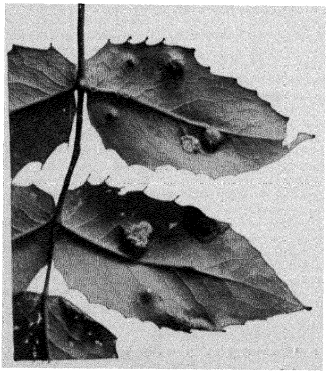


Fig. 291. Mahonia rust on under-side of leaves.

Mahonia bushes show considerable individual differences in susceptibility to this rust. Those with the darkest-green glossiest leaves seem least susceptible.

Control. Where there has been a severe outbreak the bushes should be cut level with the soil early in spring, the ground cleared of litter and dug between the stumps, and when the new shoots spring up they should be repeatedly sprayed or dusted with a copper fungicide.

Careful selection of *Mahonia* seedlings has led to the propagation of strains said to be resistant.

Black Rust (*Puccinia graminis* Pers.)

The cluster cups of the common barberry rust may also be found on the green berries of *Mahonia* and, exceptionally, on its leaves. They form groups up to ¼ inch across (see p. 74).

MALOPE

See *Lavatera*, p. 485.

MAPLE

See *Acer*, p. 408.

MARGUERITE

See *Chrysanthemum*, p. 431.

MARIGOLD

See *Calendula*, p. 427; *Tagetes*, p. 536.

MATTHIOLA (Stock)

Sclerotinia Rot (*Sclerotinia matthiolae* (Gay) Boiss.)

Sclerotinia rot of stocks is, at least usually, due to a particular species, *S. matthiolae*. The flower heads may be attacked and overgrown with downy white mycelium and black sclerotia, so that they wither and die. The stems may also be attacked and the whole plant killed. Probably the control measures recommended against *S. sclerotiorum* are applicable to this disease (see p. 77). The former has been reported in stocks in England.

Grey Mould (*Botrytis cinerea* Fr.)

Grey mould attacks especially indoor stocks about blossoming time. The flower stalks or the more or less developed flower heads are attacked by the fungus, which

causes a soft brown rotten area above which everything dies. The usual grey mould breaks out on the dead tissues (Fig. 59). There may also be large spots on the leaves.

Control. An outbreak of grey mould is an indication that the plants have been kept too moist. When it occurs in a house, therefore, the ventilation should be increased and the temperature raised. The diseased parts should be removed as soon as possible, preferably before the mould has broken out on them. It is best to place them at once in a container of milk of lime or other disinfectant to render the conidia harmless and prevent them spreading infection as one moves through the house.

Dusting with sulphur may have some effect, and Pomarsol may also be tried (see p. 598).

Club Root (*Plasmodiophora brassicae* Woron.)

Infected plants develop nodular swellings on their roots (see cabbage, p. 246). Some varieties of stock, however, are resistant.

Downy Mildew (*Peronospora parasitica* (Pers.) Tul. = *P. matthiolae* Gaum.)

This is caused by a specialized strain of the common downy mildew of crucifers, sometimes regarded as a distinct species *P. matthiolae*. It is found especially on young plants before they are planted out but may also appear later, especially on an indoor crop. On the upper surface of the leaves are pale spots, while on the corresponding parts of the undersurface is a whitish layer of mould. Stalks and flower heads may also be attacked. The diseased parts may be crippled but usually the plants grow away from the disease if conditions are not particularly favourable to it.

Control. See downy mildew of cabbages (p. 251).

Damping Off, Black-leg and Canker (*Corticium solani* (Prill. & Delacr.) Bourd. & Galz. and *Phoma lingam* (Fr.) Desm.)

Seedlings turn brown and soft just at soil level and fall over (see p. 68).

On older plants *P. lingam* may cause cankered stems (see p. 249).

Alternaria Leaf Spot (*Alternaria matthiolae* Neergaard)

Numerous grey or grey-brown spots up to $\frac{5}{8}$ inch across occur, especially on the lower leaves, but also on stems and pods. Seed from diseased plants is likely to be infected and may give seedlings that perish from damping off caused by the *Alternaria*. The same fungus attacks radishes (Ware, 1935). It would be advisable to treat stock seed with an organo-mercury seed disinfectant.

Black Rot (*Xanthomonas campestris* (Pamm.) Dowson)

This bacterium, best known on cabbage and other *Brassicae* (see p. 256) also attacks summer and winter stocks and wallflowers. The lower leaves turn yellow and fade. Their veins are darkened and in some cases this discolouration can be traced into the stems and through them to the vascular bundles of the roots.

A similar disease but with spots on the leaves, which may become curled and rolled, has been attributed in Italy to *Bacterium matthiolae* Bri. According to Burkholder (1938) this is only another name for *Pseudomonas syringae* Van Hall, the cause of lilac blight. Flowers of infected plants are delayed and malformed.

In either of these diseases we recommend that infected plants should be burned and that no stocks or wallflowers should be grown on the same soil until a year has elapsed. Nor should cabbage or any brassica be grown there. It would probably be well to disinfect stock seed with an organo-mercury seed disinfectant and to disinfect the soil used in seed boxes. Dung from cattle fed on turnips may contain *Xanthomonas campestris*, hence artificial manures are to be preferred for stocks.

Virus Diseases

Cabbage ring spot virus (*Brassica* Virus 1) produces white lines and spots on the petals of self-coloured varieties of stock (Fig. 292).

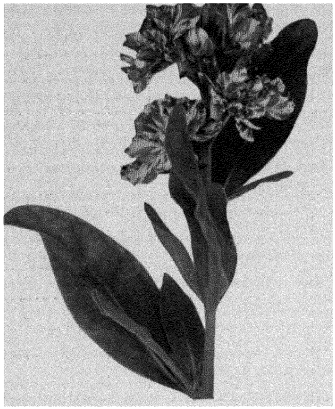


Fig. 292. " Broken colour " in stock flowers, caused by infection with Brassica Virus I (*Photo Dept. Agric. Scotland*).

The petals may also be twisted and deformed. Infection is not seed-borne but is spread by aphides. Similar breaking of flower colour may be caused by another virus, *Matthiola* Virus 1. Here too, the flowers are rather small and set little seed. The leaves have dark-green spots on a lighter ground. The disease is spread by aphides but not by infected seed. This virus differs from the preceding in not infecting cabbage or cauliflower.

Cauliflower mosaic virus (*Brassica* Virus 3) may also infect stocks, on which it has a rosetting effect owing to shortening of the internodes. Paling of the veins and a slight upward curling of the leaf edges may occur but there is no breaking of the flower colour.

When these troubles occur the grower should seek the source of infection, which is likely to be either old stock plants or an over-wintered brassica crop in the vicinity. These should be destroyed if possible and attempts should be made to keep down aphides.

MECONOPSIS

See *Papaver*, p. 504.

MEZEREUM

See *Daphne*, p. 446.

MICHAELMAS DAISY

See *Aster*, p. 416.

MIGNONETTE

See *Reseda*, p. 517.

MONTBRETIA

See *Gladiolus*, p. 463.

MUSCARI (Grape Hyacinth)

Smut (*Ustilago vaillantii* Tul.)

This smut occupies the anthers from which its spores are shed as a purplish-black dust. The mycelium is perennial in infected bulbs. Specialized races of the fungus apparently occur on different species of *Muscari* and *Scilla* (see p. 531).

Rust (*Uromyces scillarum* Wint.)

This rust, common on species of *Scilla*, is occasionally found on *Muscari* leaves, probably as a specialized race (see p. 531).

Black Slime (*Sclerotinia bulborum* (Wakk.) Rehm)

Sometimes attacks *Muscari*, as at one place in Denmark in 1923, causing many plants to rot (see p. 470).

MYOSOTIS (Forget-Me-Not)

Leaf Spot (*Entyloma fergussoni* (Berk. & Br.) Plowr.)

This leaf smut causes greyish-white spots on the leaves, up to about $\frac{1}{8}$ inch across. It is fairly common on wild forget-me-nots which should therefore be eradicated in the neighbourhood of cultivated ones.

Downy Mildew (*Peronospora myosotidis* de Bary)

Yellowish patches appear on the upper surface of the leaves, corresponding to a greyish-white layer of mould on the underside. The plants may be sprayed with a fungicide, preferably one containing copper.

When forget-me-nots are grown indoors the house should be well ventilated and the temperature kept at about 50–54° F. Wild forget-me-nots may harbour the fungus and should be destroyed.

Mildew (*Erysiphe cichoracearum* DC. = *E. horridula* (Wallr.) Lév.)

Leaves and stalks are covered with a white powdery coating (see p. 63).

Mosaic

In 1933 and once or twice since we have observed forget-me-nots which were backward in growth in autumn and had mottled leaves. Presumably they were infected with a virus, but which was not determined.

NARCISSUS

Fire (*Sclerotinia polyblastis* Gregory = *Botrytis polyblastis* Dowson)

The disease is called " fire " because of the rapidity with which the yellowish or dark reddish-brown spots appear and spread over the leaves causing them to wither. Usually it develops first on the flowers as small, watery, light-brown spots on the perianth and corona, at or about picking time. These may spread in transit and ruin the blooms before they arrive at market. During or after flowering the spots begin on the leaves, and in warm, moist weather the whole foliage may be killed in a few days. The fungus does not penetrate into the bulbs.

Botrytis conidia are formed on the leaf spots and in the dead leaves the fungus forms sclerotia up to $\frac{1}{5}$ inch long. In early spring these bear brown-stalked apothecia about $\frac{1}{8}$ inch across.

Burning all diseased foliage and spraying early in spring with Bordeaux mixture as against white mould appears to give reasonable control.

Smoulder (*Botrytis narcissicola* Kleb.)

This grey mould causes rotting of the shoots which may start as soon as they emerge above ground. The leaf tips are then crumpled and under damp conditions they become covered with the grey mould (Fig. 293). The shoot may also grow straight but a wet rot occurs at the base so that it becomes easily detached from the bulb. The decaying tissue is very slimy.

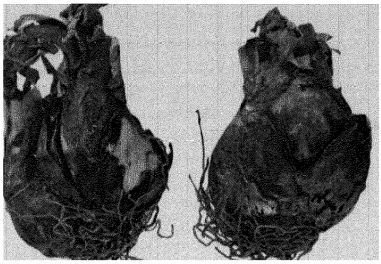


Fig. 293. Narcissus smoulder (*Photo Dept. Agric. Scotland*).

On the dead shoots and on dry stored bulbs the fungus forms black sclerotia about $\frac{1}{5}$ inch long but flattened and only $\frac{1}{25}$–$\frac{1}{16}$ inch thick. They occur specially near the tip of the outer scales (Westerdyk, J., and Van Beyma, F. H., 1928). Shoots apparently usually become infected from the bulbs but probably the sclerotia can also lie dormant in the soil for a time. The disease usually occurs scattered rather sparsely over the bulb fields. *B. narcissicola* does not attack mature foliage like *S. polyblastis*.

If the infected bulbs are detected at planting time they should be discarded. If they are planted they will decay, with or without producing a malformed, diseased shoot. All young plants that look obviously unhealthy or malformed should be dug out and burnt.

Leaf Scorch (*Stagonospora curtisii* (Berk.) Sacc.)

The first symptom is a reddish-brown scorching of the leaf tips soon after the young shoots emerge above ground. This is easily mistaken for frost damage. Under glasshouse conditions and in dry weather the disease may develop no further, but if the weather is wet the brown discolouration spreads several inches down from the tips and secondary brown, oval leaf spots appear lower down as the leaves grow up. When they are numerous the whole leaf soon turns yellow but the spots remain recognizable by their deeper red-brown colour.

The pycnidia of the fungus occur in large numbers as tiny dark-brown dots scattered over the dead areas. Flower stalks and spathes may also be attacked, and brown spots on the petals, inconspicuous at picking time, may spread in transit and spoil the consignment before it arrives at market. The fungus does not injure the bulbs but apparently overwinters in the dead leaf bases and round the neck of the bulb, ready to infect the young shoot when it emerges.

The best control, if labour is available, is to pick off the scorched tips as soon as the young shoots appear (Gregory, P. H., 1937). Its progress on the leaves may be checked by the Bordeaux spray used against white mould. According to Creager (1933) dipping the bulbs for 2 hours in ½% formalin solution just before planting kills the fungus on the old leaf bases.

White Mould (*Ramularia vallisumbrosae* Cav.)

This is very prevalent in South-west England. Small, sunken, greyish or yellowish spots appear on the leaves. They gradually become larger and darker, covered in damp weather with a conspicuous white powdery layer of conidia. Under warm, damp conditions the leaves are killed prematurely, and large numbers of very minute black sclerotia just visible to the naked eye develop on them. These germinate in the spring and bear clusters of spores which serve to infect the young leaves of narcissus. The leaves may be killed a month earlier than normal ripening and this weakens the bulb considerably (Gregory, P. H., 1939).

Control. The old overwintering narcissus leaves should be raked up and burned before the shoots appear in spring. The bulbs should also be lifted and replanted in fresh soil every few years, but as the sclerotia seem to survive only one year, the ground may safely be used for narcissus again after that interval, unless it is infested with the bulb eelworm (*Anguillulina dipsaci* Kühn).

When the shoots are 3–6 inches high they should be sprayed with 5 : 4 : 50 Bordeaux mixture plus a suitable wetting agent. In districts favourable to the disease the spray should be repeated at monthly intervals. Varieties differ greatly in susceptibility.

Basal Bulb Rot (*Fusarium bulbigenum* Cooke & Massee)

Symptoms. When the dry outer bulb scales are removed a chocolate-brown, or blackish-brown, or sometimes reddish-brown, discolouration is seen near the base. The discoloured part is often sharply divided by an irregular line from the white healthy part of the bulb (Fig. 294). Between the outer fleshy scales there is a white mycelium with little powdery, pink masses of spores. The diseased parts are soft and give a little when squeezed but are not waterlogged.

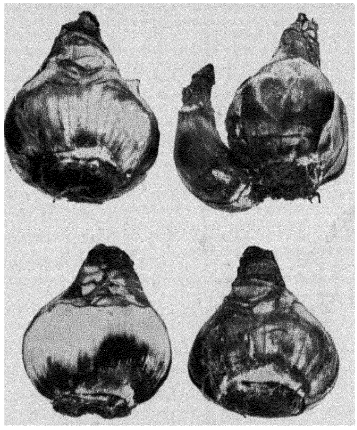


Fig. 294. Basal bulb rot of narcissus (*Photo v. Slogteren*).

In cross-section the bulbs usually show a continuous brown area with irregular white areas enclosed. In the bulb scales that are still white there is often found in the neighbourhood of the rotten area a discolouration of the vascular bundles which appear like black lines.

Severely infected bulbs are easily detected by their softness but slightly infected ones may easily be overlooked and planted. They either rot or give a poorly developed shoot with

a few yellowish narrow leaves and a flower bud which seldom opens (Gregory, P. H., 1932; Hawker, L. E., 1935).

Cause. True basal bulb rot is caused by *Fusarium bulbigenum* Cooke & Massee, though in Holland a bulb rot has been ascribed to *F. orthoceras* Appel & Wollenw. *F. bulbigenum* probably occurs naturally in some soils and it is introduced into others by planting infected bulbs. Under British conditions the bulbs usually appear sound when lifted but mycelium of the fungus is probably often present in dead adhering tissue or in dead and dying roots. From there it spreads into the bulb during storage, entering especially through a wound like that made by breaking off an offset or at the junction with the remnants of the mother bulb. In susceptible varieties entry often occurs through the roots (Hawker).

In regions with a higher soil temperature basal rot may be well advanced in the field before the bulbs are lifted.

Fusarium moniliforme Sheld., sometimes found in decaying bulbs and on the dead leaves and outer papery scales, seems to be a secondary invader following eelworm and is unable to attack sound bulbs.

According to van Slogteren the following are particularly susceptible: Bicolor horsfieldi, Mad. de Graaf, Sulphur Beauty, Henry Irving; quite susceptible are Bicolor Victoria and Glory of Sassenheim; less so, Sir Watkin, King Alfred and van Sion.

Control. Imported bulbs should be unpacked as soon as possible and stored in a cool, well-ventilated place, for infection depends on temperatures over 70° F. and does best at 80–90° F.

Shelving used to store narcissus bulbs the previous year should be washed with 4% formalin solution before the new consignment is received.

When the grower has raised his own bulbs it is an advantage to replant them as soon as possible after lifting, before the disease makes its appearance. New soil, new boxes and new covering soil should be used for forced bulbs each year.

Van Slogteren (1931) has investigated the effect on the incidence of *Fusarium* bulb rot of the warm-water treatment against bulb eelworm. He found it tends to increase the amount of decay, especially if the same water container is used several times. If, however, a fungicide such as Germisan is added to the warm water considerable control is achieved. A 0.5% solution of commercial formalin (1 quart per 50 gallons water in the tank) for the warm-water treatment—3 hours at 110° F. —is also effective.

Root Rot (*Cylindrocarpon radicicola* Wollenw.)

Symptoms. The plants languish and when they are pulled up the roots are found to be more or less rotten and partly remain in the ground. Closer examination shows that the root tips are brown and the roots greyish, often with oblong brown spots which gradually cover the whole root (Fig. 295). The small

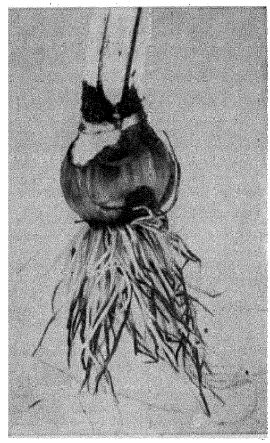


Fig. 295. Narcissus root rot (*Photo v. Slogteren*).

spots are at first yellow or yellowish-brown, then dark brown, and may begin near the middle of the root or nearer the bulb.

Occasionally the rot spreads a little into the basal plate, forming a layer of dead tissue

easily peeled off, but otherwise the bulb itself is not attacked.

Affected plants occur in patches in the field and are easily recognized by their pale colour and dwarfed growth.

Cause. Feekes (1931) found the fungus *Cylindrocarpon radicicola* always associated with narcissus root rot. Gregory (1932) had the same experience but was unable to infect the basal plate with *Cylindrocarpon*. Most workers regard this as a very weak parasite at most and the primary cause of narcissus root rot is probably not yet understood.

Control. Steam sterilization of the affected patches of soil has been shown to be effective but is not practicable on a commercial scale. Good results were also obtained by treating the beds with formalin (1–2 parts in 25 parts of water) at the rate of 2 gallons per square yard.

Rusts

Puccinia schroeteri Pass. is a rare rust which forms dark brown, rather large, pustules of teleutospores, mainly on the upper surface of narcissus leaves. They are surrounded by the torn skin of the leaf and by a brownish-purple leaf spot.

Coleosporium narcissi Grove is an even rarer rust, once found forming small oval yellow pustules on both sides of *Narcissus poeticus* leaves in Lincolnshire (Grove, 1922).

Smut

Urocystis colchici (Schl.) Rabh. (see p. 438) has occurred on leaves and scapes of narcissus in Spain.

Stripe

This is a virus disease, apparently due to a single virus which causes rather different symptoms in different narcissus varieties. These vary from silvery, brown or yellow stripes on the leaves to stunting of growth, which eventually leads to death of the plant. In many yellow-flowered varieties flecking of the flowers with white or lighter yellow occurs. Some, such as *Narcissus jonquilla* and its hybrids and *N. poeticus* varieties, show very little symptoms of infection. *N. triandrus* seem very susceptible. The wild daffodil *N. pseudonarcissus* appears to be resistant or perhaps immune. The symptoms tend to be suppressed in forced plants though they are still infected.

Pale spots on the leaves may also be due to eelworm, but these are thickened whereas those of stripe are thinner than the normal part of the leaf.

The virus is sap-transmissible but infection during cutting of the flowers is very unlikely to occur.

Infection can be spread by several species of aphides (*Myzus pseudosolani*, *Aphis rumicis* and *Macrosiphum euphorbiae* but not by *Myzus persicae*). But these seem to be seldom found on narcissus out of doors and the vector under field conditions has perhaps not yet been found. Symptoms of infection do not show until the following year (Caldwell, J., 1946).

The only effective control is periodic roguing of the stocks.

Malformed Flowers

If the temperature rises too high during the warm-water treatment against bulb eelworm the flowers may be injured. In the worst cases the buds dry up and are blind, in others the flowers open but are more or less misshapen.

In 1922 we saw a consignment of 30,000 Emperor bulbs many of which gave malformed flowers, the corona was partly or wholly closed, the leaves were wrinkled and there were dark patches on the bulbs. The cause was never ascertained but the bulbs were planted out of doors and have subsequently given quite normal blooms. It appears that drying bulbs in strong sun may cause adherence of the floral parts.

Outdoor daffodils not infrequently have large dry buds that do not open. As the bulbs have often been undisturbed in the soil for years obviously the condition is not then due to any mishandling of the bulb. Some unsatisfactory growth condition is to be suspected and one should try digging them up and replanting.

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NASTURTIUM

See *Tropaeolum*, p. 538.

NYMPHAEA (Water Lily)

Leaf Spot (*Gloeosporium nymphaearum* Allescher)

Rostrup observed cultivated water lilies in Denmark attacked by this fungus. It causes numerous round brown spots with dark edges, which bear small yellow wart-like conidial pustules on the upper surface of the leaf. The disease is disfiguring and an attempt should be made to check it by removing the first leaves found to be attacked.

On leaves carried a little above water level spraying with a fungicide may be tried.

OAK

See *Quercus*, p. 516.

ORCHIDS

Leaf Spots

Over 35 species of *Gloeosporium* and *Colletotrichum* have been described associated with spots on orchids, as well as other fungi belonging to the genera *Cercospora*, *Macrophoma*, *Phyllosticta*, etc. Many of these are almost certainly synonymous but no one has yet found time or opportunity to study them in detail.

Spots occur on leaves and bulbs. As a rule they are brown, round or irregular, more or

less sunken and sharply defined. Their size varies between about $\frac{1}{8}$ and $\frac{3}{4}$ inch, or several may unite to form larger irregular patches. In severe attacks both leaves and bulbs may be killed.

Newly bought plants should be kept away from the rest for a couple of months until one can be sure they are healthy. Severely affected leaves and bulbs should be removed and one may try to check spread of the disease by spraying or dusting with a fungicide. The plants should be well ventilated and not kept too warm. A general account of orchid leaf spots was given by Brierley (1919).

W. C. Moore (1941) has described a spot on *Cypripedium* leaves caused by the mould *Penicillium thomii* Maire. The blotches are uniformly deep brown, up to $1\frac{1}{2}$ inches across, sometimes with a paler margin $\frac{1}{8}$ inch wide and a narrow watersoaked halo outside that. The *Penicillium* fructifies rather sparsely on the spots which tend to become overgrown with the common but harmless black mould *Cladosporium herbarum*. Infection takes place only through wounds, which easily occur on orchid leaves because of their brittleness. Young leaves are less susceptible than older ones.

Propagation Fungus (*Moniliopsis aderholdi* Ruhl)

According to Van Poeteren (1928) serious attacks on young *Cypripedium* and *Odontoglossum grande* by a brown *Rhizoctonia*-like mycelium have occurred in Holland.

The plants of *Odontoglossum* had been imported and grown well for four years. Suddenly they became diseased, the new shoots did not grow, no new roots were formed and the old ones became brown and rotten. The plants flowered only a little. A brown mycelium like that of *Corticium solani* or *Moniliopsis aderholdi* (which seems to be a strain of the former fungus adapted to high temperatures, see p. 69) was found on the above-ground parts, on the roots and in the soil. About 100 plants were destroyed to check spread of the disease.

In such outbreaks we would recommend trying to check the disease by watering with dilute corrosive sublimate solution, about 1 : 3000 by weight, or with a proprietary organo-mercury fungicide. Corrosive sublimate is particularly effective against *Corticium solani* and its allies.

Repeated dusting with sulphur has checked the propagation fungus on above-ground parts of *Ficus pumila* and might well do the same on orchids if the already-damaged parts are removed. Dusting with Brassicol could also be tried.

Leaf and Heart Rot (*Phytophthora cactorum* (Leb. & Cohn) Schroet.)

Both on imported and locally grown orchids in Java a dark rot of the leaves has been observed. The dark, sharply defined spots spread rapidly, and in a few days the whole leaf was discoloured. In serious cases the disease spread to the new-formed leaves; in others, especially if the plants were kept dry, it could be restricted to a single old leaf. In houses it spreads rapidly both on *Vanda* and *Cattleya*.

The fungus usually responsible is *Phytophthora cactorum* (Leb. & Cohn) Schroet., which was constantly isolated from the diseased orchids and reproduced typical symptoms when inoculated into wounded leaves (Schwarz, M. B., 1928).

No control measures have been described but the species of *Phytophthora* are very sensitive to copper, and a spray with a copper fungicide would probably prevent spread of the fungus to the parts not already attacked. It would not check spread of the disease through an already-infected leaf.

Orchids should be planted well out of the soil so that water runs off them and should be kept as dry as they can bear.

Corticium rolfsii Curzi = *Sclerotium rolfsii* Sacc.

In 1934 this fungus caused 10,000 Reichsmarks' worth of damage to orchids at the Munich Botanic Garden.

Symptoms. The plants drooped as though suffering from lack of water and finally withered completely. Often they shrivelled at the base or fell over. The disease developed very rapidly; a plant which appeared almost completely healthy might be dead in 24 hours. Usually the decay was at or just above soil level. Here the tissues became discoloured and began to rot, and by degrees a white cobwebby mycelium appeared on them, which later became yellowish and more compact. Round hard sclerotia about $\frac{1}{24}$ inch across were formed in it. At first they were white, then yellowish, reddish and finally dark brown after which they became loose and fell off.

Cause. From the diseased orchids Flachs (1935) isolated the fungus *Corticium rolfsii*, which, like *C. solani*, is usually found only in its sterile state, called *Sclerotium rolfsii*. This is a very common parasite of crops in the warmer parts of Asia and America and is known to attack at least 150 plant species. It requires high atmospheric humidity, preferably 80–90%, and warmth. It begins to grow at 43° F. but makes only sparse growth at temperatures below 65° F.; the optimum temperature is 79–86° F. and its growth ceases at 104° F.

Control. The disease was completely checked by sprinkling the plants with a 0.5% solution of Danish Tillantin, taking care to see that the liquid penetrated well into the leaf sheaths. The treatment was repeated after 24 hours and after another 24 hours the plants were sprinkled with 0.25% Tillantin solution and again a week later. Simon, E., (1938) found this treatment might seriously damage the plants and it does not kill sclerotia within the tissues. He suggested soaking the roots 15 minutes in 0.1% Danish Tillantin solution.

PAEONIA (Paeony)

Blotch (*Septoria paeoniae* Westend.)

Brown spots with red margins sometimes occur on paeony leaves. Dotted over them are the small dark pycnidia of the fungus *Septoria paeoniae*. Spraying or dusting with a fungicide, preferably one containing copper, should be tried as a control measure.

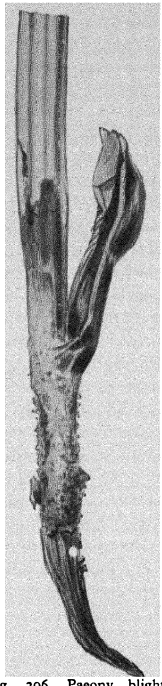


Fig. 296. Paeony blight attacking the base of the stem.

Blight (*Botrytis paeoniae* Oudem.)

Symptoms. At the base of the leaves and flower shoots a soft brown area appears which causes the shoots to wither (Fig. 296). Attacks may occur from the time the shoots have emerged a little way from the soil right on through the summer. They are most serious from some time before to a little after flowering. Under moist conditions the affected part becomes covered with a layer of grey mould.

Brown spots may also appear on the leaves, usually starting at the tip and spreading inwards; they are often round or triangular and may be zoned. Sometimes the buds are attacked when about the size of peas, cease to grow and turn brown. In wet years paeony blight may be very general and serious,

especially the attack on the stalks. All common paeony varieties may be attacked and also the tree paeony.

Cause. True paeony blight is caused by *Botrytis paeoniae*, but strains of the ubiquitous grey mould *B. cinerea* (see p. 59) may also attack paeony, especially fading parts or plants grown under unfavourable conditions.

Control. Like all diseases caused by species of *Botrytis* paeony blight is always difficult to check in very damp conditions and may do great damage in wet years.

As soon as shoots are seen to begin to droop they should be cut out right down to soil level. Infected buds should be cut off a little below the brown part. Spraying with Pomarsol or Shirlan should be tried.

In the autumn the plants should be cut right down to the root and the top layer of soil may be removed as well, as much as can be scraped away without injuring the shoots for next year. Or the dead stumps may be pulled away in spring, when they can be removed farther down without damaging the new shoots than could be done in autumn. Again the top soil layer should be scraped away and replaced with fresh soil. From early spring onwards one may try dusting with Brassicol or spraying with Pomarsol $\frac{3}{4}$: 100, especially at the base of the plants.

In severe attacks the plants should be dug up, soil and all above-ground parts carefully removed, and perhaps they may be dipped in a disinfectant solution, e.g. 1 : 100 Pomarsol, replanted in clean soil and covered over with the same.

If the soil is at all acid, lime should be supplied. At the end of the winter potassium and superphosphate should be applied at the rate of $1\frac{1}{2}$–3 oz. of each per square yard. In April about half that amount of chile saltpetre or other nitrogenous fertilizer should be given; or a complete fertilizer may be given in April.

Covering the plants with stable manure in autumn, or scattering dung around them in spring, favours the fungus and should be avoided, especially in places where blight is apt to occur. Winter coverings of leaves or other debris should also be avoided. Paeonies can stand our winters quite well without any such treatment.

Most of the varieties worked with by Winters (1930), who investigated varietal susceptibility of American paeonies to blight, are not grown in Europe. Amongst those he found resistant were Avalanche and Sarah Bernhard; susceptible were: Duc de Cazes, Duke of Wellington, Mme. de Verneville, Marie Lemoine and Strassburg.

Phytophthora Blight (*Phytophthora paeoniae* Coop. & Port.)

This fungus may attack stems, leaves or buds but the symptoms vary with age of the plant and part attacked. If a shoot is attacked at the tip the leaves and top of the stem turn dark brown and leathery. If the attack occurs lower down a large part of the stem becomes dark and everything above it withers. Stems may also be attacked at soil level, causing a foot rot.

This disease occurs in America where it has been described by Cooper and Porter (1928). No control measures were indicated but in all *Phytophthora* diseases spraying or dusting with a copper fungicide should be tried.

Mosaic (*Paeonia* Virus 1)

This is a conspicuous yellow mosaic in which large yellow or light-green patches, rings or arcs appear on the leaves (Fig. 297). The disease seems to be widespread but of little importance and not much is known about it. Green has observed paeony mosaic in England (1935).

Paeonies may also be infected with the viruses of tomato spotted wilt and cucumber mosaic.

PALMACEAE (Palms)

Leaf Spots

Exosporium palmivorum Sacc. causes irregular round or elliptical spots, at first yellowish and translucent, later larger and brown with a yellow translucent margin. On the spots appear small black conidial pustules. The disease is fairly common on species of *Phoenix*, especially on freshly imported plants.



Fig. 297. Paeony mosaic.

Various species of other fungus genera, *Colletotrichum*, *Cylindrocladium*, *Gloeosporium* and *Pestalotia*, cause spotting of leaves in various kinds of palm. The worst-affected leaves or parts of leaves should be cut out and the plants sprayed or dusted with a fungicide.

False Smut (*Graphiola phoenicis* (Moug.) Poit.)

Scab-like fungus bodies up to about $\frac{1}{10}$ inch across appear on the leaves of *Phoenix*, *Chamaerops* and other palms. They look like black cups filled with yellow powder. Around the fructification the leaf tissue turns yellow and the whole leaf may die prematurely.

The worst-affected leaves should be removed. In Holland Van Poeteren suggested spraying with Bordeaux mixture, at first every week, then at fortnightly intervals. Possibly other copper sprays, less likely to cause spotting, would also be effective.

Bud Rot

Both coconut palms in the tropics and various ornamental palms in greenhouses are apt to develop a wet rot at the base of the terminal bud, which becomes loose and eventually withers. This has been attributed to infection with the bacterium *Bacillus coli* (Esch.) Mig., common in contaminated water supplies, but it is doubtful if this is the primary cause of rot. More thorough investigations of coconut palms in the tropics have shown that the primary cause of bud rot there is *Phytophthora palmivora* and that bacteria come in afterwards to complete the destruction.

Diseased palms in greenhouses should be removed and burnt and the rest thoroughly sprayed with Bordeaux mixture.

Sun Scald

Palms under glass are apt to suffer from large leaf spots with dry yellow centres and brown margins. These are due to sun scald, often caused by bubbles in the glass concentrating the sun's rays like a burning glass (see Fig. 14).

Withered Leaf Tips

It often happens that a freshly purchased palm with healthy green leaves develops

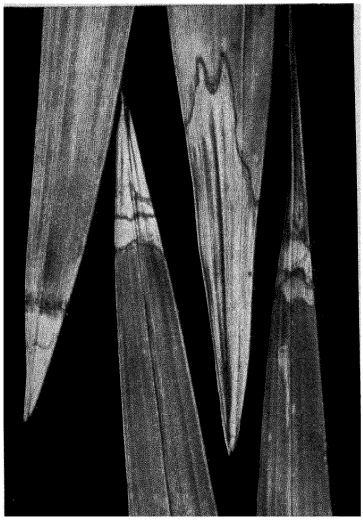


Fig. 298. Palm leaflets with withered tips.

withered leaf tips or even whole dead leaves soon after being brought into a living-room (Fig. 298).

Palms are apt to suffer greatly from a sudden change from a warm damp greenhouse to the dry air of a room. Buy palms with tough dark-green leaves. Do not repot them until absolutely necessary but give them a little very dilute manure water at intervals during the summer. When repotting becomes unavoidable use a pot only one size larger than that to be replaced and pot in leaf mould mixed with a little old manure and sand. Palms are said to need a fair amount of potash.

Be careful not to overwater or the roots will rot. If the soil has become very dry it is a good plan to stand the pot for a short time in luke-warm water. The leaves should also be sprayed occasionally with luke-warm water. Be careful not to be always knocking the leaves, and see that the plant does not stand in a draught.

PANSY

See *Viola*, p. 559.

PAPAVER (Poppy)

Downy Mildew (*Peronospora arborescens* (Berk.) de Bary)

This fungus forms a layer of close white mould, turning greyish mauve with age, on the under-side of poppy leaves. Affected leaves are usually yellowish and may be malformed. Most damage is done to seedlings. The disease is common on wild poppies in Denmark and England but also attacks ornamental poppies, opium poppy and *Meconopsis*.

Spraying or dusting with a copper fungicide will keep the disease in check.

PARTHENOCISSUS (Virginia Creeper)

Leaf Spots

In North America *Guignardia bidwellii*, cause of black rot and leaf spot in grapes, sometimes attacks the lower leaves of Virginia creeper, especially in damp, warm weather. The spots are grey-brown and angular with darker edges.

Phyllosticta viticola Sacc. & Speg. causes circular brown spots $\frac{1}{2}$ inch across. Neither of these is known in Denmark where, however, spotting has been caused by *Diplodina vitis* Brun. and *Phoma pallens* Berk. & Curt.

Spraying with Bordeaux mixture or other fungicide is advised against any of these troubles.

Downy Mildew (*Plasmopara viticola* (Berk. & Curt.) Berl. & de Toni)

Causes yellowish leaf spots which later turn brown. A close white mould occurs on the lower surface (see vine, p. 218).

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

This fungus may cause branches of Virginia creeper to wilt and die (see p. 60). It must be added, however, that the branches are apt to die suddenly without any apparent cause.

PELARGONIUM ("Geranium")

Black-leg (*Pythium splendens* Braun and other species)

This is a disease of the cuttings in which the stem turns black from the base upwards, the leaves wilt and the cutting dies. Infection comes from the soil or from a contaminated water supply. The fungi usually concerned are *Pythium splendens* Braun, *P. complectens* Braun, *P. de baryanum* Hesse and its variety *pelargonii* Braun. Less often they attack the stems of older plants, causing stem rot, leaf drop and ultimately death.

Botrytis cinerea may also cause stem rot of cuttings but the decaying tissue is usually browner and soon becomes covered with the usual grey mould.

To control grey mould the house should be kept dry and well ventilated. Attack by *Pythium* spp. may be prevented by using sterilized soil and keeping the water tanks clean and free from decaying plant remains. When black-leg does break out it may perhaps be checked by watering with Cheshunt compound. Brassicol may check the growth of grey mould.

Grey Mould (*Botrytis cinerea* Fr.)

Pelargoniums are very susceptible to grey mould. If the air of the house is at all moist, leaf spots, dead leaves and dying branches covered with this fungus will almost certainly be found. Grey mould may also attack and rot pelargonium cuttings (see p. 504).

Bacterial Leaf Spot

Two different kinds of bacteria, *Pseudomonas erodii* Lewis and *Ps. pelargonii* (Brown) Stapp, have been described from leaf spots of pelargonium in North America. Similar spots occur on pelargoniums in Europe.

The two organisms apparently cause very similar diseases. Spots appear first on the lower leaves. At first they are pale, then reddish or blackish brown with colourless edges. At the outset they are confined to the lower surface of the leaf but as they enlarge and join together they may penetrate to the upper surface. When they are numerous the leaves may turn yellow and drop off.

Control measures include picking off infected leaves, avoiding wetting the leaves, spacing the plants far apart and keeping the atmospheric humidity low. In Denmark Beauté Poitevine has been severely attacked, Meteor (Paul Crampel) much less so.

Leafy Gall (*Corynebacterium fascians* (Tilf.) Dowson)

This production of small clusters of malformed shoots sometimes occurs on pelargonium cuttings (see p. 484 and Moore, W. C., 1943).

Crown Gall (*Bacterium tumefaciens* E. F. Sm. & Towns.)

This causes cauliflower-like swellings on the stems and the plant's growth is checked (see p. 81).

Leaf Curl (*Pelargonium* Virus 1)

This virus disease is widespread on *Pelargonium zonale* in Europe and America. It has been known in Denmark for the last 30 years.

The symptoms usually appear in February or March, especially in cuttings. Light-yellowish spots $\frac{1}{24}$–$\frac{1}{16}$ inch across arise on the young leaves, occasionally accompanied by a clearing of the leaf veins. Later the spots develop into star-shaped or irregular blotches often with a brown dead spot at the centre. The leaves become crinkled and puckered and sometimes the whole plant is malformed and stunted, though this may be associated with aphis infestation. As the season progresses the new leaves appear without any symptoms and when the old scarred leaves drop off the plant appears quite healthy. It is still infected, however, and if cuttings are taken from it next spring they will show the same symptoms to the full.

The disease is transmitted by grafting and possibly by aphides.

Paul Crampel and King of Denmark are particularly susceptible varieties but, according to Pape (1927a), varieties with salmon-coloured flowers and *Pelargonium peltatum* are resistant.

The only possible control is to take cuttings from healthy plants, not from those which appear to have recovered from leaf curl, and to keep down aphides.

What appear to be two different virus diseases of pelargonium have been briefly described in Czechoslovakia (Blattny, C., 1933).

Oedema and Intumescences

Pelargoniums occasionally develop a number of raised scab-like corky patches, especially on the upper surface of the main vein in leaves as well as on stems and petioles.

The damage occurs when plants are kept in a very moist atmosphere, especially if they are suddenly brought in from a comparatively dry place. Night temperature in the glasshouse should not be allowed to fall much below the day temperature or the relative humidity will become excessive at night (see further, p. 392).

PERIWINKLE

See *Vinca*, p. 559.

PETUNIA

Leaf Spot (*Ramularia petuniae* Cooke)

This causes light-brown spots and blotches on the leaves. It has been reported at long

intervals on petunias in Devonshire. Spraying with a copper fungicide may be suggested as a control measure.

Foot Rot (*Phytophthora cryptogaea* Pethybr. & Laff.)

This soil fungus may cause rotting of the stem at the base of the plant. It has been observed killing petunias in England and Sweden. Watering with Cheshunt compound may be tried at the beginning of an outbreak.

Leafy Gall (*Corynebacterium fascians* (Tilf.) Dowson)

Occurs not infrequently on petunias (see *Lathyrus*, p. 484).

Virus Diseases

Several viruses are known to infect petunias. Most likely to be met with in nature are those with an abundant insect vector, especially common cucumber mosaic, *Cucumis* Virus 1. This causes mottling of the leaves, sometimes with dead spots, shortening of the internodes and sometimes brown streaks on the stems. The leaf blades may be distorted and flowering may be reduced or suppressed. Those blooms which do develop may fail to open normally or may be mottled and streaked.

Mottling and distortion of the leaves may also be caused by the virus of tomato and tobacco mosaic (*Nicotiana* Virus 1, see p. 383). In North America the plant is often infected with the aster yellows virus (*Callistephus* Virus 1, p. 428) which causes dwarfing, yellowing and abnormal branching.

PHLOX

Leaf Spot (*Septoria* spp.)

Several species of *Septoria* have been described causing leaf spots on phlox leaves. In England the disease is usually ascribed to *S. drummondii* Ell. & Ev., in Denmark to *S. phlogis* Sacc. & Speg.

The spots are round or elliptical and in their later stages have a white centre with a red-brown margin (Fig. 299). Besides the *Septoria* pycnidia, perithecia of *Leptosphaeria phlogis* Ritz. Bos may also be found on the spots.

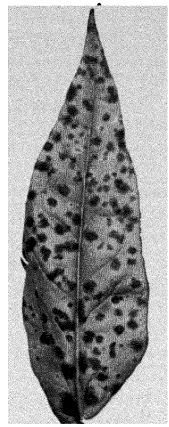


Fig. 299. Phlox leaf spot.

The mutual relationships of all these fungi still require investigation.

Infected leaves dry up and die prematurely, especially towards the base of the plant. Varieties differ in susceptibility and the most susceptible should be rejected. According to Pape (1939), Mia Ruys, Europa, Famos, Graf Schwerin and Antonio are particularly resistant. On other varieties one may try to keep the disease in check with a fungicidal spray or dust.

Mildew (*Erysiphe cichoracearum* DC.)

This may sometimes cover phlox leaves with its powdery white layer of conidia and even cause defoliation (see mildew, p. 63).

Leafy Gall (*Corynebacterium fascians* (Tilf.) Dowson)

May also occur on phlox (see p. 484), so may crown gall *Bacterium tumefaciens* (see p. 81).

PICEA (Spruce)

Grey Mould (*Botrytis cinerea* Fr.)

Seedlings and young plants of Douglas fir, pine and spruce may be attacked by grey

mould in wet seasons. Either individual twigs or the whole top may be killed but the fungus does not penetrate down to the roots. It attacks especially branches damaged in some way, as by being bent down under snow so that they touch the ground.

In the nursery attacks may be prevented by spreading gravel over the beds in autumn and by strewing cut branches over the seedlings to protect them from the weight of snow (Hartley *et al.*, 1919; Korstian, C. F., 1923). When snow has been lying for a long time it may be well to disperse it with a rake or a light harrow (see further, grey mould, p. 59).

Needle Rust (*Chrysomyxa abietis* (Wallr.) Unger)

On young spruce one sometimes sees during the summer pale cross-bands on the needles. These later turn yellow, and the following spring orange-coloured pustules of rust teleutospores break out from them. Basidiospores shed from these infect new needles and there is no alternate host and no other stage in the life history. Only very young needles can be infected, hence spruce that has made its new growth exceptionally early or trees that have not done so when the spores are shed escape the disease. The diseased needles are shed after the pustules have erupted, so that their span of life is reduced to one year. Trees over twenty years old seldom become infected.

Needle rust of *Picea excelsa* is fairly widespread in Scotland (Borthwick and Wilson, 1915a) where it has been found also on *Picea sitchensis* (Wilson, 1934).

Rhododendron Rust (*Chrysomyxa rhododendri* de Bary)

This differs from *C. abietis* in producing aecidia, not teleutospores, on the needles of *Picea excelsa*. The aecidia have a cylindrical white sheath which stands out like a column from the lower surface of the needle. Infected needles are shed the same year, and in severe outbreaks trees may be almost defoliated. As the fungus is not perennial in the spruce, however, the disease can only occur in the vicinity of infected rhododendrons. Of these the common *R. ponticum*, *R. catawbiense* and their hybrids are not susceptible (see p. 518) (Borthwick, A. W., and Wilson, M., 1915b).

Cone Rust (*Thecopsora areolata* Magn.)

This rust has its uredo- and teleutospore stages on leaves of the bird cherry (*Prunus padus*). The aecidia occur in swarms on the scales of cones of *Picea excelsa*. The mycelium does not penetrate the spruce branches and does no damage to the tree apart from the infection of the cones which takes place from the bird cherry every year. Infected cones are easily recognized by their scales standing stiffly open even in wet weather. The aecidia are brown and spherical and situated mainly on the inner side towards the base of each scale. When ripe they split open to expose the yellow spores. This rust is rare in Britain.

Leaf Cast (*Rhizosphaera Kalkhoffii* Bubák)

The first symptoms of the disease appear in May when the current season's needles turn pale with a purplish-brown tint. At this stage there is no sign of the fungus on the outside of the needle but later in the season knots of mycelium form in the cavities beneath the stomata. They push through the latter, ejecting the small mass of white wax which partially blocks the aperture, and form small black pycnidia on the surface. The dead leaves thus come to bear long rows of black dots on their underside, corresponding to the position of the stomata. Infected needles are shed prematurely leaving the branches very bare. Trees of the glaucous varieties of *Picea pungens* 14–20 years old may apparently be killed by it (Wilson, M., and Waldie, J., 1926).

The fungus causes defoliation of *Picea excelsa*, *P. alba*, *P. nigra*, *P. orientalis*, *P. schrenkiana*, *P. sitchensis*, *P. pungens* var. *argentea*, and is also found on *Pseudotsuga*, *Abies* and *Pinus* needles. Another species, *R. abietis*, causes reddening and defoliation of *Abies pectinata* in France (Mangin, L., and Hariot, P., 1907).

Leaf Scorch (*Lophodermium macrosporum* (Hartig) Rehm)

Spruce needles may be infected by this fungus at any time of the year. They turn brown and may be shed the same year or remain over winter, in which case the fructifications of the fungus mature on them in the second year. These are long black shining pustular swellings which open by a longitudinal split to shoot out their spores. Often almost the whole foliage of a spruce may be reddened by this fungus and the needles fall in showers when the tree is shaken.

Bud Blight (*Cucurbitaria piceae* Borthwick)

The buds at the tips of the shoots become covered with a dense black sheath the surface of which is crowded with little black spherical granules, the fructifications of the fungus *Cucurbitaria piceae*. The buds are permeated by the fungus mycelium and killed. Diseased buds may be found all the year round but the ascospores mature in autumn (Borthwick and Wilson, 1909). Bud blight has occurred in Scotland on *Picea excelsa*, *P. pungens* and *P. sitchensis*.

Ascochyta piniperda Lindau (= *Septoria parasitica* Hartig)

This disease attacks the current year's shoots and causes them to curve downwards and cast all or most of their needles. Pycnidia of the fungus *Ascochyta piniperda* occur as small black dots on the bark of the dead shoots and on the leaf scars. The mycelium penetrates also the previous year's growth and causes browning of the pith. This fungus attacks mainly seedlings of *Picea excelsa* and *P. sitchensis* (Laing, E. V., 1929).

Butt Rot (*Fomes annosus* (Fr.) Cooke = *Trametes radiciperda* Hartig)

Symptoms. Infection of spruce and pine trees 5–15 years old is usually discovered only when they suddenly die. At the base of the trunk there is an exudation of resin and often some small, flat, tough fruit bodies about ½ inch across, pressed flat against the bark or perhaps slightly projecting at their upper edge and covered with a thin whitish layer of tiny pores.

Older coniferous trees are not killed so suddenly. The forester soon learns to recognize the infected ones fairly easily; the needles are scanty and the twigs short, the butt end of the trunk is abnormally thickened. When the tree is felled and sawn up it is usually found in the case of common spruce that the fungus has caused a heart rot spreading 6–8 yards up the centre of the trunk. The uppermost part of the rotten wood is greyish; further down it is reddish-brown, often cracking into rings. Towards the base the trunk may be hollow with only the knots remaining. Round the butt, and on stumps and roots of felled trees, the fungus forms its fruit bodies, hard, tough, irregular crusts and brackets, blackish brown and rough above with a pale margin, covered on the under-side with a whitish layer of fine pores. The flesh is whitish. Often the fruit bodies have grown round and enclosed fallen needles, blades of grass, etc. (Fig. 300).

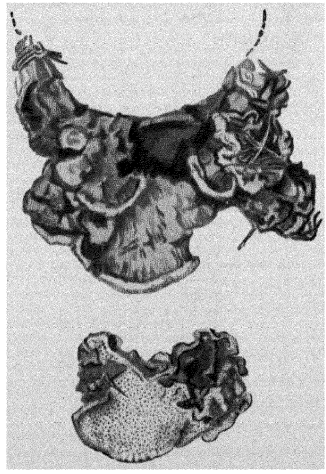


Fig. 300. Fructifications of *Fomes annosus*. Above: upper surface. Below: lower surface showing the pores.

Essentially *Fomes annosus* is a parasite of conifers, but deciduous trees may also be attacked, usually when there is an infected coniferous tree, stump or post in their vicinity. Then in a few years their foliage becomes scanty, their branches die back and eventually the tree is killed. Progress of the fungus from coniferous fencing posts to hawthorn hedge and thence to rowan, myrobalan or other fruit trees not infrequently occurs in gardens, especially in shelter belts. Fruit bodies of the fungus are often hard to find as they may develop only on the dead stump and seldom attain a large size.

Millions of basidiospores are shed from the pores of the fructifications, and on dead wood the fungus also produces conidia. Both can germinate in the soil. By mycelium arising in this way or growing out of diseased trees the fungus spreads through the soil. It seems to find difficulty in attacking young, vigorously growing roots; dead or dying roots are its typical invasion points, especially dead tap roots in the case of spruce (Jørgensen, Lund and Treschow, 1939).

Effect of environment. Losses caused by this fungus on shallow soils are only too well known. More surprising are the early and virulent attacks that sometimes occur when plantations are made on good deep arable land. One of the explanations put forward is that spruce is a shallow rooter, spreading its roots just below soil level, and that the wood of rapidly growing trees on rich soil is comparatively soft. Soil reaction seems to have little effect.

Relative susceptibility of trees. A considerable amount of information has been collected about *Fomes annosus* attacks in Denmark.

Of coniferous trees common spruce is most attacked, white spruce much less so. Sitka spruce suffers severely on arable soils but does well on deep sandy soils. Douglas fir and larch are susceptible when young but resistant later. Silver fir, Norwegian fir and the blue *Abies concolor* are very resistant. Scotch pine is most susceptible when young, mountain pine in old stands. Yew, *Thuja* and other conifers are also attacked.

Of deciduous trees beech may be attacked when young but old trees are resistant. Ash and oak are seldom infected but attack has been recorded on birch and hazel; in hedges on hawthorn and rowan; and in gardens on apple, pear and myrobalan. Even heather may be attacked by this fungus.

Control. First to be stressed is the importance of planting resistant kinds of tree. After an outbreak it is useful to clear away old stumps and main roots. Attempts made to check spread of an attack by digging trenches or applying copper sulphate, lime, etc., have given no satisfactory result. For the possibility of controlling it with carbon disulphide, see p. 593.

When there is any choice it is well to use the more resistant kinds of timber such as larch or oak (round or split) for fence posts in shelter belts and hedges and as stakes to young trees. In experiments in Sjaelland fencing poles of common spruce that had stood 1–2 days in 4% copper sulphate solution were practically unrotted after four years' use. Presumably any rotting fungi present in the wood had been killed. The posts must be cut from fresh sound wood as the solution does not penetrate dry or half dry posts.

Physiological Disorders

Common spruce often becomes redder than is desirable because the needles have been scorched by drying winds, generally in early spring. Many of the scorched needles are shed, and during the summer one may see comparatively well-developed current season's shoots with green needles while the branches with one- and two-year-old needles are more or less bare. Often it is obviously the most exposed branches that have been reddened. Common spruce near the sea nearly always has red shoot tips.

Yellowish-green needles in sitka spruce and common spruce may be due to lack of nitrogen; lack of potassium leads to a light reddish-purple tint with weak leading shoot and needles that die prematurely. White spruce apparently does not need much potassium.

Spruce over-manured with nitrogen, such

as " starling trees " regularly visited by the birds, becomes dark and very soft in the branches and eventually tufted in growth. Sitka spruce seems to stand nitrogenous manuring better than common spruce. White spruce, common spruce and sitka spruce all do best with a soil reaction under pH 6 and mountain spruce with pH below 7, but silver fir seems able to endure a certain amount of lime.

PINK

See *Dianthus*, p. 448.

PINUS (Pine)

Blister Rust (*Cronartium ribicola* J. C. Fischer)

This attacks only the Weymouth pine and other five-needled pines on which, in May and June, large yellow blisters appear, scattered over spindle-shaped swellings on branches and trunks. The blisters soon burst, shed a mass of aecidiospores and then dry up, leaving only a few shallow scars and exudations of resin. The blisters break out year after year until the infected branches die. Even whole trees may be killed. In the year of infection of a twig small yellow spots may appear on the needles which become deformed or are shed. In the early stages of an attack spermogonia can also be detected in the bark, but the blister rust phase is by far the easiest to see.

Pinus strobus, *P. cembra*, *P. monticola*, *P. excelsa*, *P. parviflora*, *P. ayacahuite*, *P. lambertiana* and *P. flexilis* have been found infected with blister rust in Scotland. Uredo- and teleutospores occur on species of currant (see p. 162).

Needle Rust (*Coleosporium senecionis* Fr. and related species)

This fungus occurs in many physiological races which have their uredo- and teleutospore stages on different dicotyledonous plants. Separate names have commonly been used for these, e.g. *Coleosporium senecionis* Fr. on groundsel and other species of *Senecio*, *C. tussilaginis* Kleb. on coltsfoot, *C. sonchi* Lév. on sowthistle, *C. petasites* Lév. on butterbur, *C. euphrasiae* Wint. on eyebright, yellow rattle and bartsia, *C. melampyri* Karst. on cow-wheat, *C. campanulae* Lév. on harebell and other campanulas, and so on. All these are common in Britain and all form indistinguishable aecidia or cluster cups on the needles of various species of pine, especially Scotch pine (*P. sylvestris*). The needles are infected in autumn, but not till the following spring do the small yellow-brown pustule-like spermogonia develop on them in rows. Later the aecidia break out in rows on the underside of the year-old needles. They are somewhat compressed, with a white membrane about $\frac{1}{8}$ inch long, enclosing chains of orange spores.

The uredo- and teleutospore stages appear on the leaves or stems of the appropriate alternate host as yellowish-orange powdery pustules and reddish-brown crusts respectively.

Older pines suffer little from this rust but it may do serious harm to young stock. Hence one should try to eradicate the alternate hosts, most of which are weeds, from the vicinity of forest nurseries and, if necessary, try to protect the pines by a summer spray with Bordeaux mixture or similar fungicide.

Peridermium pini (Willd. ex Pers.) Lév.

This is a rather similar rust which forms bladder-like pustules up to $\frac{1}{4}$ inch across, rather like those of *Cronartium ribicola*, on branches of Scotch pine (*Pinus sylvestris*). In the past it has been confused with *Coleosporium senecionis* and its allies, which only attacks the needles, but it is quite a different fungus, peculiar in that aecidiospores from pine are able to reinfect pine instead of a dicotyledonous host (Klebahn, 1918b). *P. pini* seems to be very rare in Britain, but occurs in the Scottish highlands.

Leaf Cast (*Lophodermium pinastri* (Schrad.) Chev.)

On dead fallen pine needles one often finds rows of black ridges about $\frac{1}{25}$–$\frac{1}{12}$ inch long, each with a single longitudinal split which opens wide in wet weather, exposing a greyish interior. This consists of a mass of asci containing thread-like ascospores which are

shot into the air. If they fall on one-to-three-year-old pine needles infection takes place, mostly in August and September. The following year the infected needles die and many are shed. In severe attacks the plants may be much weakened. Northern races of Scotch pine are more resistant than Southern races.

Young pines in nurseries may be protected by spraying with Bordeaux mixture plus a sticking agent in June or July and again in August.

Pine branches should not be used to protect the seedlings in winter because of the risk of infection.

On larger pine trees the leaf cast fungus is usually confined to the older needles. If the tree is weakened, especially if the needles have been dried by winds in spring, the fungus may spread actively in the damaged needles and may hasten their death.

Related fungi attack the needles of other conifers, e.g. *Lophodermium macrosporum* (Hart.) Rehm on *Picea excelsa* (see p. 508), *L. piceae* (Fuck.) Von Höhnel on many species of *Abies* and *Picea*, *L. laricinum* Duby on larch, *Hypodermella nervisequa* (Chev.) Lagerberg on silver fir and *L. juniperinum* (Fr.) de Not. on many species of juniper.

PLATANUS (Plane)

Leaf Disease (*Gnomonia veneta* (Sacc. & Speg.) Kleb. = *Gloeosporium nervisequum* (Fuck.) Sacc.)

Leaves of plane trees are often marked by striking brown patterns with jagged edges which follow the main veins of the leaf. After a time tiny light-brown conidial pustules of the fungus formerly called *Gloeosporium nervisequum* appear on the dead areas. In years favourable to it the disease may cause some defoliation, the young shoots may also be attacked and killed, the young leaves may be destroyed as they open and are then shed.

Slightly different conidial fructifications are produced on the dead wood, and during the winter perithecia of the perfect, *Gnomonia*, state of the fungus mature on the fallen infected leaves. These are tiny black bodies with slender black beaks projecting from the leaf tissue. Ascospores ejected from them are one source of infection in spring. No doubt infection largely comes, however, from conidial pustules on the twigs as in the case of apple scab, which has a very similar life history.

The fallen leaves should be collected and burnt or composted in autumn. If the disease remains prevalent spraying with Bordeaux mixture may be tried, but several trials may be required to discover the best timing of the sprays.

POINSETTIA

See *Euphorbia*, p. 456.

POLYGONUM

Leaf Spot (*Septoria polygonicola* Sacc.)

Round brown spots appear on the leaves and may be so numerous as to cause them to wither. A severe attack on *Polygonum orientale* was noted in Denmark in September 1936. Spraying or dusting with a fungicide may be tried.

POPULUS (Poplar)

Leaf Spots (*Marssonina* spp.)

Several species of *Marssonina* attack the leaves of different kinds of poplar, causing blackish-brown spots with irregular outlines. Sometimes they are so numerous that the leaves are more black than green and are shed early.

Lombardy poplars may suffer severely from such attacks; after extensive and premature leaf fall the shoots ripen badly, then frost damage and various feebly parasitic fungi kill the trees in a few years. Rainy summers seem to encourage leaf spot.

Klebahn (1918a) investigated these diseases and found several species of *Marssonina* involved which were very similar in microscopic characters but differed in their parasitic capacity.

Susceptible poplars may be protected by spraying 4–5 times with Bordeaux mixture if the summer is wet. Cutting back each year checks the disease. *Populus certinensis* is said to be resistant.

Didymosphaeria populina Vuill = *Napicladium tremulae* (Frank) Sacc.

Also causes blackish leaf spots on poplar leaves, especially on aspen. The fungus lives below the cuticle and produces an olive-coloured layer of conidia over the surface of the spots.

Canker and Dieback (*Dothichiza populea* Sacc. & Br.)

In some years Lombardy poplars, and others to a lesser extent, suffer severely from dieback of their branches or are even killed by it. In Belgium many young trees of *P. canadensis* have been killed by this disease after their resistance had been weakened by their being lifted too soon and the roots allowed to dry in transit (Quairière, C. J., 1931). Much loss of Lombardy poplars in nurseries has been reported from North America, and in Denmark there was great mortality among these trees, both old and nursery stock, in 1880–3. E. Rostrup considered *Dothichiza populea* played a considerable part in the loss but found the trees had been rendered susceptible by early spring frosts, with the ground free from snow, followed by icing and hard night frosts in May. Weakening of the trees by premature defoliation has been described above. Too dry or too wet a soil also renders them liable to attack.

From the time the buds burst brown discolouration of the bark may be found, roughened with small pimples in which the pycnidia develop. Cankers develop at the base of twigs, which then die back to the canker.

Extensive losses have occurred also in Holland and there too they seem to be bound up with environmental conditions. Attempts to select resistant varieties have been begun there (van Vloten, H., 1938).*

Rusts

Several closely related rusts occur on different species of poplar. All of them produce bright orange powdery pustules of uredospores on the under-side of living leaves, and blackish-brown crusts of teleutospores which mature within the tissues of the fallen leaves in autumn and winter.

Melampsora populina Lév. occurs on *Populus candicans, P. generosa, P. laurifolia, P. nigra, P. robusta, P. trichocarpa* and is divided into two sub-species one of which, *M. Allii-populina* Kleb., has its aecidia on the common broad-leaved garlic (*Allium ursinum*) while the other, *M. larici-populina* Kleb., has them on larch needles (see p. 483).

M. tremulae Tul. occurs on *Populus alba, P. balsamifera* and *P. tremula* (aspen) and is divided into several sub-species of which at least two occur commonly in Britain, viz. *M. larici-tremulae* Kleb. with aecidia on larch needles and *M. rostrupii* Wagner which forms very conspicuous clusters of orange, rather ill-defined, aecidia on leaves and stems of dog's mercury (*Mercurialis perennis*).

The poplar rusts probably cause little damage; they may be controlled if necessary by eradicating the alternate hosts.

Leaf Blister (*Taphrina aurea* (Pers.) Fr.)

Irregular blister-like swellings appear on the poplar leaves. The upper convex surface is smooth and green, the under concave surface is covered with a bright golden layer of the asci of *Taphrina aurea*. This disease is common on *Populus nigra*, also on *P. pyramidalis, P. canadensis, P. tremula* and *P. deltoides*. The fungus overwinters as mycelium in the buds.

A related species, *T. johnsonii* Sadeb., occurs on the carpels of female aspen catkins, causing them to become greatly enlarged.

Neither of these diseases causes any appreciable harm to the tree and no control measures have been worked out. A copper spray as used against peach leaf curl (see p. 179) would probably control *T. aurea*.

PRIMULA (Primrose, Polyanthus, Hothouse Primulas)

Leaf Spots

Various fungi, species of *Cercosporella, Phyllosticta, Ramularia, Ovularia* and *Septoria*, can cause leaf spots on primrose and polyanthus. The most abundant in Denmark is a round

* Poplar canker may also be caused by *Pseudomonas syringae* (see p. 535).

or angular leaf spot, first light yellowish-brown, then dark brown with a yellow margin and a fine white mould on the under-side, caused by *Ramularia primulae* Thüm (Figs. 301, 302).

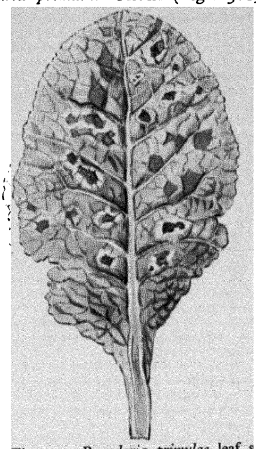


Fig. 301. *Ramularia primulae* leaf spots on primrose.

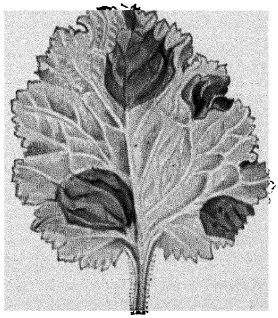


Fig. 302. *Ramularia* leaf spots on *Primula malachoides*.

Moore (1941) has quoted circumstantial evidence that the fungus known as *Cercosporella primulae* Allesch. may be the same organism. *Ovularia primulana* Karst. is common on leaves of wild primrose where it is easily recognized by the conspicuous white crust of conidia on the underside of the leaf spots.

Ascochyta primulae Trail and *Phyllosticta primulicola* Desm. cause very similar spots to those of *Ramularia* but instead of a fine mould on the under-surface there are small dark-brown dot-like pycnidia in the dead leaf tissue.

Spraying or dusting with a fungicide may check an attack if tried early enough.

Brown Root Rot (*Thielaviopsis basicola* (Berk. & Br.) Ferraris)

The leaves become pale green then quite yellow, and the plants cease to grow. The roots are brown, rotten and contain the fungus *Thielaviopsis basicola*.

Tiddens (1933) has studied this disease in Holland where it does much damage to *Primula obconica*. For control see *Begonia*, p. 422.

Primulas also often suffer from root rot and foot rot caused by various species of *Phytophthora* (see p. 68). Wilting and death of *Primula sinensis* seedlings caused by *Pythium spinosum* Sawada has been described by Cook and Collins (1938).

Grey Mould (*Botrytis cinerea* Fr.)

Out of doors, and especially under glass, primula may be attacked by grey mould. It may cause large rotten spots on the flower heads, which become covered with the usual grey layer of mycelium and conidia. *Primula sinensis* and *P. stellata* are prone to a foot rot caused by this fungus if they are overwatered or if the atmosphere becomes too damp and cold (see grey mould, p. 59).

Mildew (*Oidium primulae* Hammarlund)

In 1942 Hammarlund found a powdery mildew on *Primula malacoides* at Stockholm. Leaves, stems, flower stalks and flowers were covered with a floury white coating. The leaves withered often in the course of 5 or 6 days. This was the first observation of a powdery mildew on this host but owing to its natural mealy white surface slight attacks may no doubt have been overlooked.

By inoculation Hammarlund was able to infect flowers of *Primula sinensis* but not the leaves or flower stalks (see mildew, p. 63).

Downy Mildew (*Peronospora oerteliana* Kühn)

This downy mildew occurs occasionally on primrose and polyanthus leaves on which it causes rather vague yellowish spots with a whitish mould on the under-side. Control as for leaf spot.

Rust (*Puccinia primulae* Duby)

Primula rust occurs only on leaves of primrose, cowslip and their close allies. All stages occur on the same plant, the cluster cup (aecidia) in small groups on yellow spots on the under-side of leaves in May, uredo- and teleutospores in brown and blackish-brown pustules respectively in summer and autumn.

Bacterial Leaf Spot

This causes circular or angular dark-brown leaf spots with conspicuous yellow margins. In the early stages they are watersoaked and yellowish. Individual spots do not exceed ¼ inch in diameter but they may unite to kill large parts of the leaves. Infection occurs mainly on the older leaves. Many species of hardy primula are susceptible, including polyanthus, cowslip, *P. auricula* and *P. denticulata* (Ark, P. A., and Gardner, M. W., 1936). The disease was first described in California but occurs also in Britain. No control measures have been suggested.

Mosaic (*Cucumis* Virus 1)

In *Primula obconica* this virus causes slight mottling of the younger leaves, which may become distorted. The plants become stunted and cease to grow or may even be killed. *P. sinensis* and other species show similar symptoms, including breaking of the flower colour (Fig. 303). Polyanthus may also be infected and suffer from yellowing of the leaves and stunting of growth (see also p. 278).

Primulas are also susceptible to infection by the virus of tomato spotted wilt (*Lycopersicum* Virus 3). This causes yellowing, stunting and death of *P. malacoides*, stunting, yellowing of the veins and large dead marginal areas on leaves of *P. sinensis* (see p. 384).

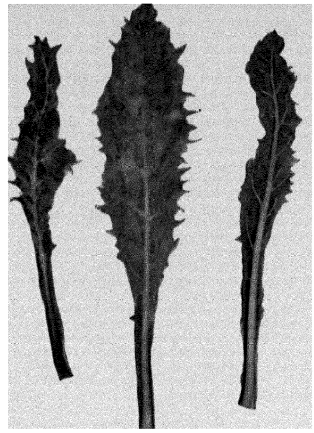


Fig. 303. Mosaic of *Primula bulliana* caused by cucumber mosaic virus.

Chlorosis

We have observed yellowing of the above-ground parts of primulas, like that associated with *Thielaviopsis* infection described above, which we have attributed to excess lime in the soil (see iron deficiency, p. 36).

PRIVET

See *Ligustrum*, p. 486.

PRUNUS (Ornamental Cherries and Almonds)

Bacterial Canker

Prunus cerasifera var. *pissartii* and *Prunus nana* have been attacked by this disease in England. The leaves become rolled or yellow and branches or even the whole tree dies suddenly (see p. 200).

Blossom Wilt (*Sclerotinia laxa* Aderh. & Ruhl.)

Prunus nana, *P. triloba* and other flowering cherries may have their blossom trusses attacked by this fungus and killed, or whole branches may be invaded and wilt (see cherry, p. 152).

PRUNUS LAUROCERASUS (Cherry Laurel)

Leaf Spots

Coryneum laurocerasi Prill. & Delacr. causes yellowish-brown spots with darker margins on cherry laurel leaves. Small brown pustules of conidia break out on the surface of the spots. In old spots the dead tissue dries up and falls out leaving a hole like hail damage. There was a severe outbreak at a nursery on Fyn, Denmark, in 1931.

An almost identical disease is caused by *Trochila laurocerasi* (Desm.) Fr. This fungus produces pustules of colourless sticky conidia (*Gloeosporium phacidiellum* Grove) which are soon followed by apothecia. The latter are about $\frac{1}{25}$ inch across, with a blackish upper surface which splits into 3 or 4 tooth-like flaps. In wet weather these bend back exposing the grey disc inside from which the ascospores are shot into the air.

Control measures include clipping the hedge only in autumn, supplemented if necessary by spraying with a fungicide (Gregor, M. J. F., 1936).

PSEUDOTSUGA (Douglas Fir)

Needle Cast (*Rhabdocline pseudotsugae* Syd.)

This fungus, like the Douglas fir which it attacks, is a native of the west coast of North America and has followed its host to Europe. It had appeared in Scotland by 1921, possibly earlier, and was seen in Germany and Holland in 1930. It was certainly present in Denmark in 1930 and may have been there by 1927 (Ferdinandsen, C., 1930).

On the needles of the current season's shoots infection by *Rhabdocline* causes the appearance of pale transverse bands from July to August. These later turn reddish brown. The following year in May-June the perithecia mature on the diseased needles. They are brown, elliptical and open by a slit in damp weather, exposing the orange-coloured layer of asci inside. After the spores have been shed the infected needles drop, so that diseased trees have their older twigs more or less naked (Day, W. R., 1927).

The green Douglas firs are more resistant than the grey and blue forms.

Importation of Douglas firs into Denmark is prohibited because of this disease.

Canker (*Phomopsis pseudotsugae* Wilson)

Infection of Douglas fir by this fungus occurs through wounds, such as those caused by pruning the side branches of trees 15–20 years old, by hail on young trees or by branches rubbing together in the wind. Damage by the fungus may occur in three ways: the young branches or shoots may die back, small branches may become completely girdled by a canker so that the whole part above the canker dies, or large cankers may develop on trunks and main branches during the winter and spring following infection and stop developing after the tree resumes growth in the spring. *Phomopsis pseudotsugae* is an active parasite and does not only attack trees weakened by frost or other unfavourable conditions. In addition to Douglas fir, especially the green form, the fungus causes dieback of cedars, and canker of larch, silver fir and *Sequoia*. *P. pseudotsugae* is unknown in North America, the home of the Douglas fir, and is apparently a European fungus native perhaps to larch, fir or cedar, which has found the Douglas fir an acceptable host after its introduction to Europe (Wilson, W., and Hahn, G. G., 1929).

PYRACANTHA (Pyracanth)

Scab (*Fusicladium pyracanthae* Thüm)

The berries of pyracanth may be completely covered by a dark olive-brown coating of this fungus. Attacks are sometimes so severe as completely to destroy the decorative value of the shrub. Scab also occurs on the leaves, especially on the petioles, and causes them to be shed.

The fungus responsible has been regarded by some authors as a physiologic race of the pear scab organism, but its conidia are distinctly shorter (Gante, T., 1935), so there is some justification for treating it as a distinct species. McKay was unable to transfer pyracanth scab to apple or pear leaves. According to him the fungus overwinters largely on

infected bud scales which give rise to whorls of infected leaves when the buds break in spring. A spray programme similar to that for apple and pear scabs should keep it in check. McKay (1944) found a lime sulphur spray satisfactory. He used a 1 : 40 spray three times in March and April and a 1 : 80 spray twice in June and if necessary in July. Copper sprays proved injurious to the foliage.

QUERCUS (Oak)

Mildew (*Microsphaera alphitoides* Griff. & Maubl.)

This disease spread over Europe with extraordinary rapidity in 1907–8.

Symptoms and development. Leaves and shoot tips become covered with a white floury coating of mycelium and conidia. The leaves wither and fall prematurely. New leaves may unfold but they are often crowded in a curious rosette-like fashion. Infected shoot tips become deformed, their wood does not ripen properly and they are easily killed by freezing in the winter. The fungus overwinters in the buds, the leaves that emerge from them are white with conidia and act as foci of infection for the rest of the foliage. Often the disease is worst on summer shoots, whose young leaves are easily attacked in early August.

Though the oak mildew is everywhere abundant in its conidial state, perithecia are very seldom seen in Britain. They appear to require a hot, dry summer for their development as a few were found in 1945 and more in 1946 (Robertson and Macfarlane, 1947).

Importance. Oak mildew is worst on sappy young shoots arising from the stumps of felled trees. It may also be severe on oak hedges and nursery stock. Infected seedlings may develop so slowly that they take an abnormally long time to attain a saleable size and infected hedges are far from ornamental. Hence oak is not desirable as a hedge plant.

Species of oak differ considerably in susceptibility. *Quercus cerris*, *Q. lanuginosa*, *Q. pedunculata*, *Q. robur* and *Q. sessiliflora* are highly susceptible while *Q. ilicifolia*, *Q. ilex*, *Q. macrocarpa*, *Q. phellos* and *Q. rubra* are much less so (see also mildew, p. 63).

Leaf Spot (*Sclerotinia candolleana* (Lév.) Fuck.)

Yellow spots appear on the oak leaves about the end of June. At first they are small and circular but soon increase in size and turn brown with a darker marginal zone. Later in the season the central part of the spot may fall out like a shot-hole. The fungus does not fructify on the living leaf, but after it has fallen in autumn a small, hard, black, seed-like sclerotium develops within the decaying tissue. It may be $\frac{1}{8}$ inch long, and in the following spring it germinates to form a small yellowish-brown cup fungus with a disc about $\frac{1}{8}$ inch across on a long slender stalk. Ascospores shot from this, usually in May, infect the young oak leaves (Wilson, M., and Waldie, J. S. L., 1927).

Heart Rots

Many bracket fungi cause decay of the heart wood in standing oak trees but this is a problem for the forester and timber merchant rather than the gardener or nurseryman. *Polyporus sulphureus* Fr. forms large clusters of bright yellow fleshy overlapping brackets usually fairly high up the main trunk. When old they dry up and become white and chalk-like. *P. dryadeus* Fr. forms single, much thicker and tougher, brown brackets, usually near the base of the trunk or on the main roots. The margin is pale and exudes drops of clear liquid in damp weather. *Fistulina hepatica* Fr., the vegetable beefsteak, is a large red bracket fungus with flesh that looks much like raw meat. It is a common cause of dieback in old oaks, causing the "stag-headed" condition.

These fungi usually gain access to the wood through wounds. Damaged branches should be cut back flush with the main branch or trunk. Large old wounds should be cleaned of all rotting wood and bark and the resulting surface should be coated with a soft bituminous preparation. Metal sheets should not be nailed over wounds. Cavity wounds should be cleaned out, disinfected and filled, if necessary, with concrete or "Synthaprufe" (Le Sueur, 1936).

RESEDA (Mignonette)

Leaf Spot (*Cercospora resedae* Fuck.)

Numerous small straw-coloured circular spots appear on the leaves, upon which the needle-shaped conidia of *Cercospora resedae* are produced in small clusters. Stalks and seed pods may also be attacked. In severe attacks, especially on the lower part of the plant, the spots may run together and the whole leaf turns reddish and dies.

The disease is prevalent in North America, where experience has shown that it can be checked by a Bordeaux spray 2: 2: 50. Seed should not be saved from diseased plants or, if that is unavoidable, it should be treated with a disinfectant (see p. 576).

RHODODENDRON

Gall (*Exobasidium vaccinii* (Fuck.) Woron.)

See *Azalea*, p. 420.

Leaf Scorch and Dead Buds (*Pycnostysanus azaleae* (Peck) Mason = *Sporocybe azaleae* (Peck) Sacc.)

This fungus attacks the terminal flower buds in July and August, killing them so that they turn brown but do not decay. Instead they remain on the twigs for two or three years and become covered with the short, black, bristle-like fructifications of the fungus, each bearing a head of spores. Leaf buds and twigs are also attacked and in severe cases there may be a certain amount of dieback. On the leaves the fungus causes large brown blotches.

Control measures include cutting out the dead twigs and buds and spraying the bushes with Bordeaux mixture after they have bloomed.

Rhododendron flower buds may also be found covered with grey mould. In this case it is probable that they had been previously damaged by some other agency such as late frosts.

Yellow Leaves and Withered Leaf Edges

When rhododendrons develop yellow leaves the most likely cause is an excess of lime in the soil. If the soil reaction much exceeds pH 6 peat should be added, and if a nitrogenous manure is given it should be sulphate of ammonia. Rhododendrons seem able to endure a higher soil reaction on heavy clays than on light soils.

If the soil is not naturally peaty it is advisable to mix a quantity of peat with it before planting rhododendrons, for subsequent applications only affect the top layers and are therefore likely to be inadequate.

If the soil is stiff or too dry or the plants are exposed to wind the edges of rhododendron leaves are apt to wither and dry up.

Root Rot (*Armillaria mellea* (Vahl ex Fr.) Quél.)

We have observed one very serious instance of damage by the honey agaric to old rhododendrons in a park full of oak trees.

The bushes died and examination showed that the wood for a short distance upwards from soil level was brown, while just beneath the bark were the white mycelial fans and brown rhizomorphs of *Armillaria mellea* (see *Fagus*, p. 456).

In Scotland *Rhododendron sanguineum*, *R. lapponicum* and *R. saluenense* have been attacked by rhizomorphs from old stumps ten yards away.

Leaf Spots

Angular brown spots caused by *Septoria rhododendri* Cooke were found by us in 1938 on the lower leaves of rhododendrons in North Zeeland.

Lophodermium rhododendri Ces. causes whitish leaf spots with a brown margin. The flat, shiny, elliptical, black fructifications, which open by a longitudinal slit, occur scattered over the dead areas.

Other leaf spots have been attributed to *Pestalotia macrotricha*, *Phyllosticta rhododendri* Westend., *Macrophoma cylindrospora* Berl. & Vogl., etc., but most of these are probably secondary invaders attacking leaves damaged by other causes including frost and sun scald.

Where an outbreak of leaf spot has occurred, dead fallen leaves should be raked up and

removed before March in case parasitic fungi are overwintering on them. Several protective sprays with a fungicide such as Bordeaux mixture may be given from springtime onwards.

Wilt (*Phytophthora cambivora* (Petri) Buism.)

The first sign of the disease is a pale yellow-green colouring of the leaves which then wither completely. The roots and bases of the stems are more or less brown and decayed. If the bark is removed reddish-brown streaks are seen in the cambium, and when these extend upwards to the shoots brown sunken cankers appear.

Cause. This root rot and wilt is caused by *Phytophthora cambivora* (Petri) Buism., which is closely allied to *P. cinnamomi* Rands (see erica, p. 455) or perhaps even identical with it. The fungus causes root rot and death of many kinds of tree in fairly warm climates, e.g. sweet chestnut in Italy.

Importance. Rhododendron wilt was first found in New Jersey in 1927 (White, R. P., 1930, 1933). It occurs on one-, two- or three-year-old seedlings of *Rhododendron ponticum* and on many *R. catawbiense* hybrids, as well as on one- or two-year-old grafted plants of most commercial varieties. The following have been shown to be susceptible by artificial inoculation: *R. carolinianum*, *R. californicum*, *R. catawbiense*, *R. caucasicum* and *R. maximum*.

R. ponticum, and especially the Hoogendijk type, has shown itself to be the most susceptible of all those examined. No natural infection has been seen on *R. maximum*, and artificial inoculations do not always succeed on it. In New Jersey the disease is worst on seedlings and spreads from individual plants to many more. According to White it could not be found in 1930 in any nursery examined in France, Holland or England. It seems unlikely to be introduced into Denmark.

Control. The optimum temperature for this fungus lies between 77 and 82° F. and it does not thrive in soils with pH below 4.25. Hence when an attack seems probable the soil should be kept as acid as possible and its temperature below 77° F. The fungus does not survive severe frost, so soil intended for rhododendrons should be exposed to frost as much as possible before being used.

Rust (*Chrysomyxa rhododendri* (DC.) de Bary)

This disease is known to occur on *Rhododendron dauricum*, *R. ferrugineum*, *R. hirsutum*, *R. intermedium*, *R. kaempferi*, *R. kotschyi*, *R. myrtifolium* and *R. roylei*. Irregular brownish-yellow patches appear on the upper surface of the leaves and powdery yellow pustules of uredospores break out on the corresponding parts of the under-surface. The aecidiospore stage occurs on the common spruce (see p. 507), so that rhododendron rust is common only where spruce and susceptible species of rhododendron grow side by side, as in the Alps and Pyrenees. The disease has, however, been seen occasionally on rhododendrons in Britain (Ashworth, D., 1938).

In North America another rust, *Pucciniastrum myrtilli*, occurs on *Rhododendron ponticum*.

RICHARDIA

See *Zantedeschia*, p. 562.

ROBINIA (False Acacia)

Leaf Spot (*Septoria curvata* Sacc.)

This causes large irregular spots on the leaves, on the under-side of which the small black pycnidia of the fungus appear like dots. Rostrup has observed serious defoliation by this disease in Denmark.

Mildew (*Erysiphe polygoni* DC. = *E. martii* Lév.)

This powdery mildew occurs occasionally on *Robinia* (see mildew, p. 63).

Heart Rot (*Polyporus sulphureus* Fr.)

This bright yellow bracket fungus is not uncommon on old *Robinia* trees. It probably gains access to the heart wood through scars left by removal of branches (see p. 516).

ROCK ROSE

See *Helianthemum*, p. 468.

ROSA (Rose)

Rust (*Phragmidium mucronatum* (Pers.) Schlecht.)

Symptoms. Like many other rust fungi, rose rust has several stages in its life history but they all occur on the same plant; there is no alternate host. Most familiar are the pale-orange powdery pustules of uredospores and the almost black pustules of teleutospores that occur dotted over the under-side of leaves. The latter survive the winter.

This rust may also form large, often elliptical, orange-yellow cushions of spores over leaves, young shoots, leaf stalks and canes, particularly when it has overwintered as mycelium in twigs or buds (Plate 10, Fig. D). This is the aecidial stage corresponding to the cluster cup of other rusts, but instead of individual cups each with its own membrane there is a spreading mass of spores called a caeoma.

Attacks on the leaves cause them to fall too early. The caeoma stage deforms the affected parts and makes the stems liable to snap easily, but is mainly important as a source of infection for the leaves. Rust is seen mainly on outdoor roses, perhaps because bushes under glass are seldom exposed to frost. Williams' (1938) experiments at Cheshunt showed that teleutospores germinated when they had passed the winter on rose leaves out of doors, but none did so that had overwintered in glasshouses or in the laboratory. Roses under glass are cut back so hard that branches and buds which may contain an over-wintering mycelium are almost certain to be removed.

Cause. Rose rust is mainly caused by *Phragmidium mucronatum* but other allied species occur on roses. They can only be distinguished by microscopic examination.

In Norway Jørstad found *P. mucronatum* and *P. tuberculatum* Müll equally common out of doors but it was the latter that occurred under glass. Rust on the stems was due to *P. mucronatum* but that on the leaves was often *P. tuberculatum*.

Williams undertook cross-inoculation experiments in England with strains of *Phragmidium* from nine different rose sources, *R. canina*, *R. laxa*, *R. rugosa* and garden hybrids. At least one strain from *R. laxa* and one from *R. rugosa* was able to infect the cultivated roses while strains from *R. canina* could not do so. The strains of rust from garden roses could infect the briar Schmidts Special.

Control. The disease is difficult to control by the usual sprays and dusts. As with most rusts they are not fully effective. If, however, a grower has reason to fear a severe outbreak he should undertake several sprayings with Bordeaux mixture, lime sulphur or similar sprays, or dust repeatedly with sulphur before the disease gets a hold, for only by early treatment can it be kept in check. Copper sprays seem more effective than sulphur compounds. At Cheshunt in 1933 the best results were obtained by spraying with a copper fungicide plus spraying oil.

It is important to cope with the over-wintering stage of the fungus. Infection of the branches is usually first seen after the caeoma stage is fully developed and ready to shed its spores. They should be cut out but to prevent the spores being shaken out in doing so the caeomata should be smeared with spirit or carbolineum before the branches are touched. The affected parts should then be cut out and burnt.

In Germany and Switzerland spraying late in autumn and again early in spring with a tar oil 8: 100 or lime sulphur 1: 10 is recommended.

As the fungus overwinters also as teleutospores on fallen leaves they should be raked up in autumn and destroyed.

Varieties differ greatly in susceptibility, and those found to be most attacked should be eradicated or they will serve as sources of infection for the others. In general the Remontant, Moss and Centifolia roses are most susceptible. Tea, Polyantha and climbing roses as well as many hybrid teas are resistant. Noisette and monthly roses are fairly resistant, Bourbon roses some more some less so.

Black Spot (*Diplocarpon rosae* Wolf. = *Actinonema rosae* (Lib.) Fr.)

Symptoms. This disease is often injurious to many varieties, especially out of doors. It first appears as small dark leaf spots, at the edges of which is a radiating mycelium growing out over the leaf surface (Fig. 304). Later the

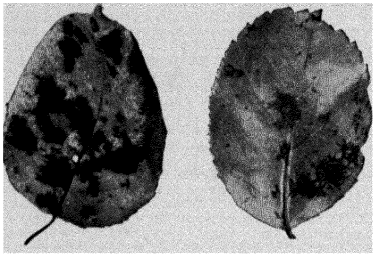


Fig. 304. Black spot on rose leaves.

spots grow bigger and may join together so as to cover much of the leaf surface. Infected leaves may turn yellow and easily fall off so that the bushes may become leafless by early summer. This checks ripening of the buds and makes them more susceptible to winter injury. The leafless condition of the bushes also renders them unsightly. Dark irregular spots may appear also on the leaf stalks and on the stems, especially of varieties with soft growth (Fig. 305).

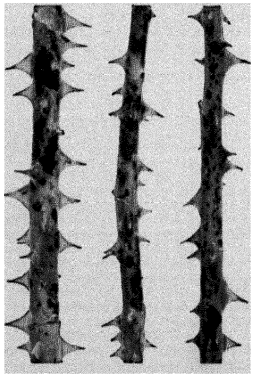


Fig. 305. Black spot on rose stems (*Photo Massey & Westcott*).

Cause. The imperfect state described above was first called *Actinonema rosae*. The mycelium on the spots bears tiny conidial pustules which are freely spread from diseased to healthy leaves all through the summer, especially in damp weather, which gives them every opportunity to germinate.

During the winter pycnidia and apothecia develop on the fallen leaves, but the latter, though found in North America, have not yet been seen in Europe.

Control. The main overwintering place of the fungus is on the fallen leaves which should therefore be carefully raked up and destroyed in autumn. It may also overwinter on infected shoots and these should be cut out before the buds break.

The disease is difficult to control by spraying or dusting.

In 1939 Massey reported that experiments at Ithaca, New York, had shown that a weak Bordeaux mixture containing about $\frac{1}{4}\%$ copper sulphate had given excellent results. But it had to be applied 15–25 times in the course of the summer. This would appear prohibitive but it seems from American publications that such repeated sprayings or dustings are undertaken there. Special emphasis was laid on the importance of frequent sprayings in rainy weather, even 2–3 times a week, whereas in dry weather once every 8–10 days was enough.

Spraying with stronger Bordeaux mixture causes too much spotting, and lime sulphur is not very effective. The efficacy of sprays is increased by adding a sticking agent. Very frequent dusting with sulphur before rain may also be beneficial.

In North America black spot does much damage to roses under glass, evidently more so than in Denmark. Detailed investigations (Lyle, E. W., 1938) have shown that rose leaves must be damp for over 6 hours—to enable the conidia to germinate on them and cause a spot. If, then, one does not spray the plants with water the conditions are not likely to become favourable for infection. Practical

experience on a large scale has shown that roses can do quite well under glass without being sprayed with water if they are otherwise well grown. To avoid red spider flourishing on the dry leaves they must be sprayed with a suitable acaricide, that mostly used is a proprietary American preparation Selocide which gives excellent control in Denmark. Where that is not available oil or petroleum sprays may be used against red spider if the prevalence of black spot makes it advisable not to spray the roses with water.

In 1938 and 1939 at Lyngby it was observed that roses under a pear tree that had received 3–4 summer sprays with Bordeaux mixture for several years had less Black spot and better foliage in the autumn than others in the vicinity which had not been affected by the sprays.

Mildew (*Sphaerotheca pannosa* (Wallr.) Lév. var. *rosae* Woronich.)

Symptoms. Mildew is undoubtedly the most prevalent disease of roses, both out of doors and under glass. It may appear on all the green parts and even on the petals, forming a white mealy coating, that later turns grey,

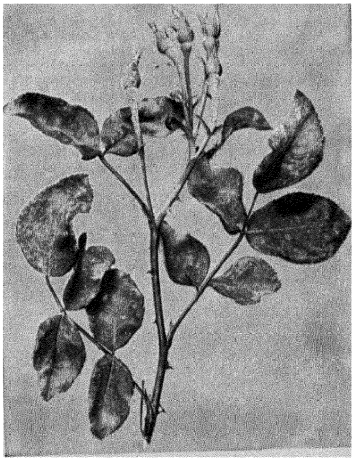


Fig. 306. Rose mildew.

made up of mycelium and chains of conidia (Fig. 306). In old infections tiny dark spherical perithecia occur embedded in the felted coating of mycelium.

The coating on young shoots and buds is thicker and more felted than that on leaves. If buds are attacked when young they do not open or yield only malformed blooms. On the leaves mildew is not only unsightly but by interfering with assimilation it may weaken the plant and upset flowering.

Cause. The rose mildew is closely allied to that which attacks peach and almond, but Woronichin has shown that the mildews on these plants are distinct varieties, viz. *S. pannosa* var. *rosae* which attacks roses and *S. pannosa* var. *persicae* on peach and almond. Hence there is no risk of mildew spreading from rose to peach, though if conditions are favourable for mildew the two varieties are likely to occur side by side, each on its own host plant.

Mildew may become very serious on roses lacking water and nourishment.

Out of doors mildew is always worst in warm sheltered places, particularly on climbers on south walls unshaded by trees.

Varieties differ greatly in susceptibility to mildew. When the crimsom rambler raised by Turner in 1894 first appeared on the market it became very popular, but this soon waned when it was found to be extremely susceptible to mildew. Only crosses between it and more resistant varieties have proved to be of permanent value.

The fungus overwinters partly as perithecia formed out of doors on infected shoots and fallen leaves but largely by mycelium in infected buds. In spring these give small shoots that are white with mildew.

Control. The fallen leaves should be collected and destroyed in autumn and the bushes should be pruned hard before the buds burst. Spraying with lime sulphur 1:10 just after leaf fall and again just before the buds break may have a beneficial effect. One should aim at growth that is both vigorous and hard; manuring with nitrogen increases susceptibility to mildew, potassium checks it.

Susceptible varieties should not be planted against a wall, particularly not against a south wall, or, at least in sheltered localities, they will give little pleasure to the grower. No rose varieties are immune to mildew, but in general smooth-leaved varieties are fairly resistant.

Though mildew is most prevalent in climbing roses it may also attack free-standing bush roses out of doors. The extreme susceptibility of Else Poulsen is well known and one should not grow such a variety unless one is prepared to spray or dust it as often as necessary.

According to Dutch experience, roses on their own roots and those worked on stocks of *Rose rugosa* and *R. rubiginosa* are less susceptible than those worked on *R. multiflora*.

Sulphur and sulphur compounds are the usual fungicides employed. Under glass vaporization of sulphur may be tried, either from vaporizers or by putting it on the central steam heating pipe if there is one. The heat of ordinary warm water pipes is not enough to vaporize it. Sulphur dusts and sprays with lime sulphur, liver of sulphur or colloidal sulphur may be used under glass and out of doors (see p. 600). For dusting the finest possible grade is required.

All sulphur treatments tend to cause spotting and are hence objectionable. At Cheshunt spraying with oil 1:100 plus Bouisol has worked well and causes little spotting. Others that may be used are salicyl-spirit-soap, formalin, shirlan and white oils. The last-named do not kill the mildew but the mycelium and conidia become stuck together so that new conidia are not formed for some time after spraying. C. J. Hansen has successfully used 1 gallon Albolineum + 1½ gallons petroleum to 100 gallons water. Proprietary preparations like Agrosol, Cosan, Pomarsol and Sulsol may also be used. They cause less spotting than lime sulphur.

Sulphur and copper sprays protect the plants for some time after; some of the other sprays, especially formalin, only kill the mildew and leave no protective film to prevent fresh infections. It is therefore necessary to repeat the spraying with them at very short intervals if conditions are favourable to mildew and the varieties are susceptible. It is important to start control measures before the attack has spread far and obtained a hold on the plants.

Downy Mildew (*Peronospora sparsa* Berk.)

Symptoms. Downy mildew is mostly found in Denmark on roses under glass but it also occurs out of doors.

On the upper surface of the leaves occur reddish patches which slowly turn brown while the fungus breaks out as a fine greyish mould on the corresponding parts of the lower surface. The mould is made up of innumerable, much-branched conidiophores bearing nearly spherical conidia. In the tissues of the older spots oospores are formed which can survive the winter.

Affected leaflets are shed early; even the quite young ones on the flowering shoot. The calyx lobes may also be attacked.

Peronospora sparsa, the downy mildew which causes these spots, is believed to have come from India to Europe and was first found in England in 1862. Rostrup found it in Denmark in 1884 on 2,000 roses at Odense and subsequently at many other places.

Control. In rose houses one should try to avoid the disease by firing well and ventilating thoroughly night and day. Condensation must not occur in the house at night. If the disease still appears, its spread may be checked by spraying at once with a copper fungicide. The fungus may overwinter in fallen leaves, and these should be carefully removed during the resting period of the roses.

Leaf Spot (*Sphaceloma rosarum* (Pass.) Jenk.)

This fungus causes small, roundish, brown to dark-purple or black spots on the leaves, the centre of the spots gradually turns grey or whitish (Fig. 307). Severely attacked leaflets may turn red, yellow or brown and the leaves may be shed, but leaf fall from this cause is much less prevalent than from black spot.

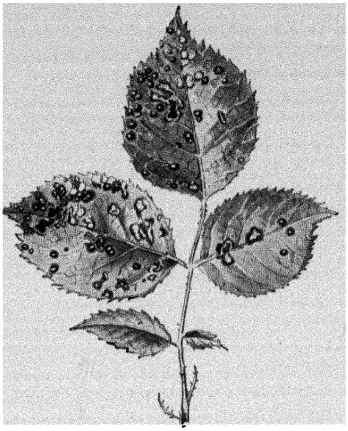


Fig. 307. Roseleaf spot, *Sphaceloma rosarum*.

Round or elliptical brown or purple raised spots like small scabs with sunken paler centres may appear on the stems. The variety Dr. W. Van Fleet is particularly prone to these.

Other minor leaf-spotting fungi on roses are *Phyllosticta rosarum* Pass. and *Septoria rosae* Desm., which cause small, round, pale-brown or grey spots with reddish or purple margins. A few pycnidia appear like black dots in the centre of the spots.

Roseleaf spots may be controlled by spraying or dusting with a fungicide when the disease spoils the appearance of the bushes. Fallen leaves should be raked up and destroyed in autumn.

Stem Canker (*Coniothyrium wernsdorffiae* Laur.)

Symptoms. Discoloured spots occur on the overwintering branches, varying in size and appearance according to age. The first symptom is a small red spot that increases in size and becomes darker. As the cells die the centre of the spot turns light brown while the margin remains reddish-brown or purple, in striking contrast to the green bark (Fig. 308). On the spots pycnidia develop which break

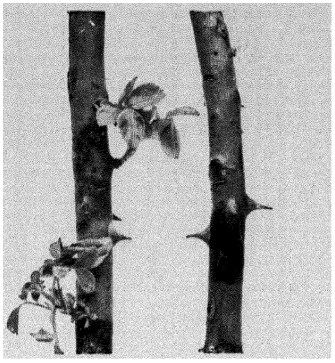


Fig. 308. Rose canker, early stage (*Photo Westcott*).

out through small cracks in the bark (Fig. 309). These small cracks are very characteristic of the disease.

If the spots developed on stems beneath covering material during winter they first appear as black spots but after being exposed to the light for a week or two they take on the appearance described above. In spring the spots are more visible than later in the summer when the bark of the branches gradually becomes darker all over.

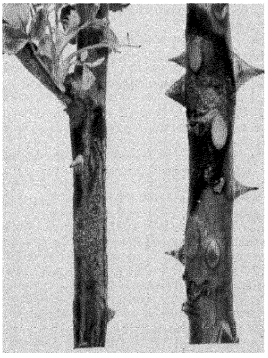


Fig. 309. Rose canker, later stage with pycnidia (*Photo Westcott*).

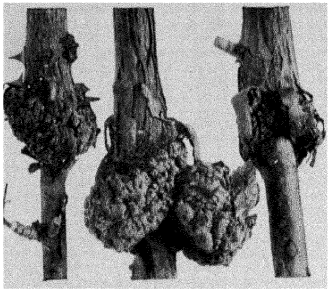


Fig. 310. Callus growth over *Coniothyrium wernsdorffiae* (*Photo Westcott*).

The spots may become an inch or more long or several may join up so as to affect a long piece of stem.

The stems may become girdled by spots and if this happens in the spring the part above dies. If girdling only occurs later in the summer a gall-like outgrowth may grow out over the spots, which may become 2 inches across in the course of the summer (Fig. 310). The branches above the gall do not die but become thickened.

Cause. The spots are caused by the fungus *Coniothyrium wernsdorffiae*. Infection occurs in winter or early spring from the old stems at least 1½ years old, for shoots are not infected in their first summer (Westcott, C., 1934). Infection only occurs through wounds but these are always plentiful on roses, either from insect attack or by thorns tearing branches that strike against one another in the wind or get torn off themselves.

Control. As the disease occurs almost exclusively on roses covered during the winter there is an obvious advantage in growing varieties that can withstand frost. The fungus seems worst in districts with severe winters, more so than in Denmark where it is not the practice to cover roses very heavily in winter. Many varieties are susceptible so that there seems little likelihood of controlling the disease by selection of varieties.

New roses should be carefully examined before planting and branches obviously wounded should be cut off. It is best to plant roses that have not had an attack of stem canker. On older bushes all damaged branches should be removed as soon as seen.

Westcott found it best to cover the plants with coarse sacking or with evergreen branches for protection against the winter.

It is important that the shoots be well ripened in autumn. Soil conditions should therefore be good and care should be taken not to give the plants too much nitrogen.

Other Canker and Dieback Fungi

Leptosphaeria coniothyrium (Fuck.) Sacc., which has a conidial state called *Coniothyrium fuckelii* Sacc. or *C. rosarum* Cooke and Harkn., causes stem cankers rather like *C. wernsdorffiae*. It has caused great losses in Holland both out of doors and under glass. The plant's growth is checked and it develops yellow leaves after it has flowered for the first time. Such plants often die altogether. In one nursery many roses which had shown symptoms of the disease during the summer died while being overwintered in a shed. Newly budded roses either did not take or died after budding. The scion could easily be broken from the stock. On the dead plants there was more or less brown discolouration extending both upwards and downwards from the place they budded (Van Poeteren, 1932). The fungus is known to attack grafts in other countries also. Wood for grafting or budding should not be taken from infected beds or houses.

L. coniothyrium is also well known as a parasite of raspberries (see p. 211).

Brown Canker caused by *Diaporthe umbrina* Jenk. starts as a small round dark-edged spot with whitish centre on young shoots. Later the spots grow larger and finally girdle the stem which then dies. The large lesions are cinnamon-brown with purple margins. Brown spots may appear also on the leaves and flowers (Jenkins, 1918, 1931; Ogilvie, L., 1932). The surfaces of the cankers become dotted with the tiny protruding beaks of the perithecia of *D. umbrina*.

Cylindrocladium scoparium Morgan causes brown areas to develop on the bark at soil

level which later become blackened and sunken. The lesions may spread upwards along the stem and down into the roots.

Botryosphaeria dothidea (Fr.) Ces. & de Not. may attack the branches, causing somewhat raised black patches from which the bark falls off with more or less concentric cracks.

Griphosphaeria corticola (Fuck.) Von Höhnel, which has the conidial state *Coryneum microstictum* Berk. & Br., has been described causing canker and dieback of roses in England by Brooks and Alaily (1939). Cankers start as smooth brown depressions with purplish edges on the green bark, usually round a dead bud, thorn scar, rust pustule or other injury. Usually they cease to extend before the stem is girdled and become sunken, but do not split to expose the wood as with those caused by *L. coniothyrium*. Black pycnidia and perithecia of the fungus appear on the dead bark. Less often the canker girdles the stem and causes it to die back or the fungus infects a pruning wound and the stem dies back from that. Infection can occur at any time except the summer months, when the plant is able to isolate the fungus by barriers of gum in the wood. The varieties Duchess of Atholl, Lady Inchiquin and Golden Emblem were found to be particularly susceptible.

Cryptosporium minimum Laub. is a conidial fungus which makes conspicuous rounded blackish spots with purple edges on branches of roses, especially ramblers. It seems to occur mainly on plants weakened by other factors such as frost. Very minute colourless pustules of spores break out on the dead bark.

Control. As a general rule one should avoid giving roses too much nitrogen and should plant them on a good soil not too damp or acid, so that the shoots may ripen well. The bushes should not be covered in winter any more than is absolutely necessary. Spotted leaves and cankered shoots should be removed at least before the winter covering is put on. In severe outbreaks of any of these diseases frequent sprayings or dustings with a good fungicide should be made throughout the whole growing period.

Roses should not be planted again where a cankered rose has been taken out. Those under glass should be kept no damper than is absolutely necessary.

Grey Mould and Dieback (*Botrytis cinerea* Fr.)

Deacon (1933) has shown that the common grey mould *Botrytis cinerea* may attack rose stems through snags left where blooms have been cut, also by infecting thorns and spreading from them into the stem beneath. It can also infect stems directly through the bark of rapidly growing shoots. Once the wood has been killed the fungus bursts out through the bark and forms the familiar layer of grey mould on the surface. Varieties differ considerably in their liability to dieback from this cause. Mrs. A. R. Barraclough was found to be particularly susceptible.

Under damp conditions grey mould may attack the flower buds either through the outer petals or by invading the flower stalk, which turns dark brown and the bud dies without opening. On buds that have opened the fungus may cause brown spotting of the petals (see grey mould, p. 59).

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

Verticillium wilt of roses has been described by Martin (1926) and two cases were verified in Denmark in 1933 (see *Verticillium* wilt, p. 60).

Bacterial Blight (*Pseudomonas syringae* Van Hall)

In 1938 roses in Arkansas developed sunken black spots and streaks, sometimes with narrow red edges, on calyxes, flower stalks and leaf stalks. These were due to a bacterium identified as *Pseudomonas syringae*, the cause of lilac blight (see p. 535).

Rose Mosaic (*Rosa* Virus 1)

A mosaic disease of roses is known in North America and in England. The leaves become distinctly mottled and in some varieties they

may be distorted. Flower buds may appear bleached and the flowers may lose their colour and be imperfect. The whole plant may be dwarfed. Hybrid tea roses and Wichuriana hybrids are most susceptible. The disease is spread by grafting and budding but the insect vector if any is unknown.

Several other virus diseases known as streak, wilt and infectious chlorosis have been described on roses in North America and Australia but are as yet unknown in Europe.

Bronzing of Foliage

Bronzing of the leaves occurs occasionally on young weak greenhouse roses which have been cut back hard and where buds have been destroyed, or in roses that have been forced hard.

It may result in the leaves turning quite yellow. The damage has been observed on Claude Pernet and Wilhelm Kordes.

Frost

Roses may be damaged by winter frosts and still more by hard night frosts after warm, sunny spring days. The branches die back from the tip and bushes may even be killed altogether.

Rose varieties differ greatly in susceptibility to frost. The tea roses should always be covered in winter but many hybrid teas may also be damaged by frost.

Varieties liable to be damaged in this way should be covered by heaping up the soil about them and then adding a loose covering of pine twigs, dry seaweed or horse dung. Coarse sacking may also be used, especially over the crowns of standard roses. The covering material should not be too thick or tightly packed and should not be pressed closely round the plants or it may encourage canker fungi (see p. 524).

Spring frosts are the most dangerous, so one should not remove the coverings too early.

Chlorosis

If the soil is very rich in lime, as in gardens where old mortar may have been mixed with it, rose leaves often turn yellow (see Fig. 15). The same condition is sometimes seen in greenhouse roses.

In such cases it is often helpful to water the soil with ferrous sulphate solution, 1: 100 by weight (see iron deficiency, p. 36, and manganese deficiency, p. 39).

Unopened or Falling Buds, Blackened Flower Stalks and Shoot Tips

The cause of these phenomena has not been explained but there is considerable evidence

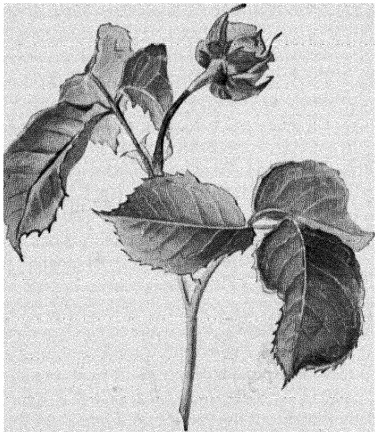


Fig. 311. Blackening of rose flower stalk.

to show that they are connected with unsatisfactory nutrition, for example excess nitrogen or great fluctuations in water supply (Fig. 311). The affected parts should be removed as they easily fall a prey to grey mould (see above).

Summary of Rose Diseases

		page
I. Roots and Crown		
Irregular nodules	Crown Gall	81
II. Branches and Shoots		
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E. Mealy white coating on young shoots	Mildew	521
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(2) Blackish irregular spots, leaves similarly marked	Black Spot	520
(3) White or grey spots with a red margin	Leaf Spot	522
(4) Red spots turning brown at the centre, sometimes girdling stem and overgrown with gall-like callus	Stem Canker	523
(5) Brown cankers cracked open to expose the wood	*Leptosphaeria coniothyrium*	524
(6) Brown cankers with purple edges, not cracking open to the wood	*Griphosphaeria*	525
(7) Large blackish blotches, not cracked or split	*Cryptosporium minimum*	525
(8) Raised black blotches marked with concentric cracks	*Botryosphaeria*	525
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(1) Whole leaf yellow or yellowish green	Iron Deficiency	36
(2) Leaves yellow-spotted	Manganese Deficiency	39
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C. Small orange or black spore pustules on under-side	Rust	519
D. White coating, especially on upper surface, also on shoots	Mildew	521
E. Spots on leaves:		
(1) Vague reddish-brown patches with fine grey mould on under-side	Downy Mildew	522
(2) Dark-brown or black spots with fringed edges	Black Spot	520
(3) Grey or white spots with red edges	Leaf Spot	522
	Septoria	523
	Phyllosticta	523
(4) Brown spots	*Diaporthe*	524
F. Sunken black streaks on petioles	Bacterial Disease	525

		page
G. Leaf fall:		
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IV. Flower Buds and Blooms		
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B. Brown decaying buds or buds dying with blackish lesion below them, grey mould breaking out	Grey Mould	525
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D. Blackened flower stalks and sometimes blackened calyces. No fungus present	Cultural Faults Bacterial Disease	526 525

RUSSIAN VINE

See *Cissus*, p. 436.

SAINT JOHN'S WORT

See *Hypericum*, p. 477.

SAINTPAULIA (African Violet)

Saintpaulia plants are very apt to develop pale spots on their leaves. These form rings about $\frac{1}{16}$ inch wide and barely $\frac{1}{4}$ inch in diameter (Fig. 312).

There is much difference of opinion about the cause of these spots. No fungus or bacterium has been found associated with them, and some growers believe they form where drops of water have lain on the leaves. The ring type of leaf spot is associated with many virus diseases but we have seen affected *Saintpaulia* plants lose the symptoms entirely when moved to another nursery. This strengthens us in the opinion that some cultural factor is responsible; the spots are not found on quick-growing plants shielded from direct sunlight.

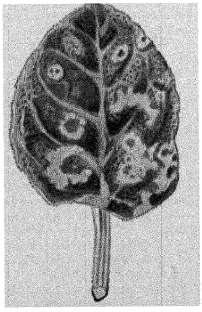


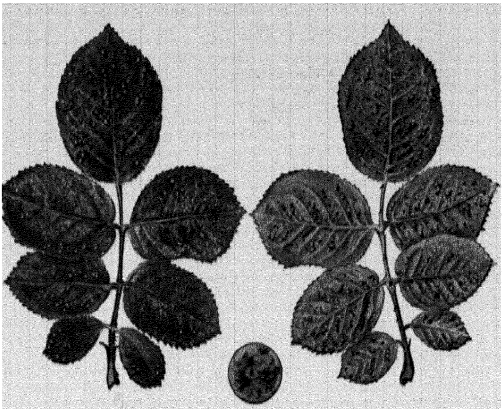
Fig. 312. Spotting of *Saintpaulia* leaves.

SALIX (Willow)

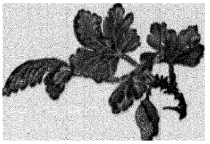
Rusts (*Melampsora* spp.)

The rust *Melampsora amygdalinae* Kleb., of basket willow (*Salix triandra*) differs from all the other willow rusts in having all its stages on the willow; there is no alternate host. Moreover it occurs only on *S. triandra*, *S. amygdalina* and *S. pentandra* and cannot infect other species of willow.

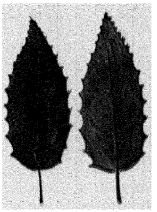
The aecidia appear as elongated bright orange cankers at the base of the young shoots soon after they appear on the stumps in April. Spores from these aecidia alight on the leaves and give rise to the uredospore stage, which occurs in swarms of bright orange pustules on the under-side of the leaves, sometimes causing them to be distorted and fall prematurely. Infections also occur on the green stems in July, usually near an infected leaf stalk, and set up small cankers which spoil the



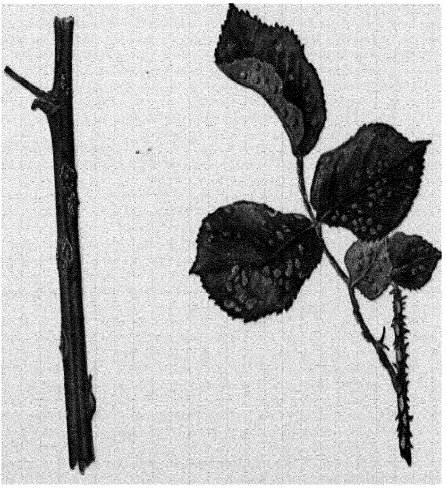
A. Rose rust. Left: upper side of leaf. Right: lower side. Inset: pustules of uredo-and teleutospores as seen under a lens.



B. Gooseberry rust.

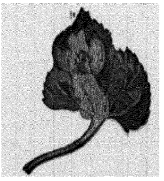


E. Mahonia rust. Left: upper side of leaf. Right: lower side.



C. Asparagus rust, aecidia on the stem.

D. Rose rust in the caeoma stage.



F. Viola rust, aecidial stage.

rods for basket work. The teleutospores appear on the lower leaves in July in the form of dark-brown crusts beneath the skin of the under-side of the leaf. They mature on the fallen leaves in winter and germinate in spring, each forming four basidiospores which are shot into the air to infect the new shoots in April. The uredospore stem cankers also survive the winter and produce a fresh crop of uredospores in spring.

Control measures include cutting the rods close to the stump in winter, to remove all uredo-cankers, drainage of hollows into which the infected leaves tend to be washed after leaf fall and dusting the stumps with copper lime dust or ground sulphur as soon as they begin to show green, repeating the dusting about a week later (Ogilvie, L., 1932). An alternative method of control suggested by later experiments at Long Ashton is to destroy the first shoots in spring, i.e. those made at the critical time when the teleuto-spores are germinating. For this purpose spraying with 10% tar-oil emulsion when the shoots are about 2 inches long was suggested.

Many other closely related species of the rust genus *Melampsora* occur on the leaves of various species of willow. During the summer the under-side of willow leaves may become completely covered with bright orange powdery pustules of uredospores. The teleuto-spores form beneath the skin of infected leaves after they have fallen in autumn, they form small scab-like dark-brown or black crusts just beneath the skin of the leaf.

These willow rusts cannot always be recognized with certainty until their alternate host has been found. Three of them have their aecidia on larch needles, viz:

M. larici-caprearum Kleb. with uredospores on goat willow (*S. caprea*, *S. cinerea*, *S. aurita*).

M. larici-epitea Kleb. with uredospores on the above species and also on *S. nigricans*, *S. purpurea*, *S. viminalis* and a few others.

M. larici-pentandrae Kleb. with uredospores on *S. pentandra*.

M. euonymi-caprearum Kleb. has aecidia on spindle tree and uredospores on *S. cinerea*.

M. ribesii-viminalis Kleb. has aecidia on currant or gooseberry and uredospores on the osier (*S. viminalis*).

M. orchidi-repentis Kleb. has aecidia on wild orchids and uredospores on the creeping willow (*S. repens*).

Two species have their aecidia on the broad-leaved garlic (*Allium ursinum*) and uredospores on the different tree willows, viz.

M. Allii-fragilis Kleb. with uredospores on *S. fragilis* and *S. pentandra*, and

M. Allii-salicis-albae Kleb. with uredospores on *S. alba* and *S. vitellina*.

Several other species with aecidia on Alpine saxifrages attack only the little Alpine willows.

Usually when a willow is found to be infected with rust it is necessary to see which of the possible alternate hosts is growing in the vicinity and, if possible, to search for the aecidia on it in spring.

On cultivated willows these rusts may be controlled by spraying or dusting with a copper fungicide. The first treatment should be made when the buds are green at the tip, a second treatment given 8–15 days later and repeated at some weeks' interval.

Scab (*Venturia chlorospora* Ces. = *Fusicladium saliciperdum* (All. & Tub.) Lindau)

Leaves and sometimes the young shoots become covered by a close olive-green mould. The leaves wither and the shoots may die back (Alcock, N. L., 1926b). A copper spray as suggested for rust will control scab.

Black Canker (*Physalospora miyabeana* Fukushi)

This disease attacks willow leaves and rods especially in *Salix alba* and its varieties and *S. americana*. Leaves are usually infected at the tip which blackens in *S. alba* and turns reddish-brown in *S. americana*. The diseased area spreads down the leaf in a wedge shape and the dead tip bends sharply downwards. Infection may be confined to one side of the midrib. Under favourable conditions the whole leaf is killed and the fungus spreads

down the petiole into the stem, where a brown patch develops. The cambium is killed so that as the rest of the stem increases in thickness the infected part becomes sunken and forms a canker, over which the bark shrivels and cracks open. As infection occurs by way of the leaves the cankers are situated at the nodes. Young succulent rods may be infected at the very tip, which blackens, dies back and curls over very much like a plum twig with wither tip. Minute pink pustules of conidia break out on the young cankers in summer, while later in the season small dark perithecia develop embedded in the dead bark. No fructifications appear on the leaves, which usually become overgrown by the harmless olive-coloured mould *Cladosporium herbarum.*

Control measures are based on preventing infection of the leaves. For this purpose they should be sprayed with Bordeaux mixture at intervals of about 3 weeks through the summer (Nattrass, R. M., 1928; Dennis, R. W. G., 1931).

Black Blister (*Cryptomyces maximus* (Fr.) Rehm)

This forms elongated black blisters along willow branches. They may be 6 or 8 inches long by about $\frac{1}{4}$–$\frac{3}{4}$ inch broad, burst through the bark and then split open, especially in wet weather, and expose the yellowish-brown ascus layer inside. If the branch is girdled it may die back, otherwise the fungus seems to cause little damage. The only control measure one can suggest is to cut out infected branches.

Black Spot (*Rhytisma salicinum* (Pers.) Fr. ex Fr.)

Black spot of willows is very like tar spot of sycamore (see p. 408). It forms black, shining, slightly raised patches up to about $\frac{1}{4}$ inch across on living leaves of various species of *Salix*. No control measures are known.

Mildew (*Uncinula salicis* (DC.) Wint.)

A thin white powdery layer of mycelium and conidia grows over the infected leaves. Scattered amongst the conidia are tiny dark spherical perithecia covered with fine hooked bristles only visible under a microscope. This mildew attacks all species of willow and also occurs on aspen and many species of poplar (see mildew, p. 63).

Wood-rotting Fungi

The following fungi commonly cause decay of trunk and branches in old willow trees: *Fomes igniarius*, with black hoof-shaped fructifications with a hard, cracked upper surface; *Trametes rubescens*, with rather thin, bracket-like fructifications, the under-side of which is covered with deep irregular slots, whitish when fresh but staining reddish-brown when handled; and *Polyporus sulphureus* (see oak, p. 516).

Watermark Disease (*Bacterium salicis* Day)

Watermark disease of the cricket bat willow (*Salix coerulea*) has been studied in England by Day (1924) who ascribed it to *Bacterium salicis*. The first symptom, generally in May, is a wilting and withering of the leaves on a number of branches. They turn brown and hang for some time but the disease progresses no further that year. Thin, sticky, colourless exudations of slime, crowded with bacteria, ooze out of cracks on the dying branches. As the slime dries up it turns brown. In subsequent years the disease extends its hold and in two or more seasons the whole tree may be killed. When the affected branch is cut through, light or dark brown or even black stains are seen in the wood. *B. salicis* occurs in swarms but only in the larger wood vessels.

Infection of the crown takes place through wounds made by insects, which may themselves carry the bacteria about on their bodies.

Death of the bark of infected branches is hastened by infection with the otherwise harmless pycnidial fungus *Cytospora chrysosperma* Fr. The orange-coloured spores ooze out in tendrils from the pycnidia in damp weather, making it rather conspicuous on the dead branches.

Watermark disease also affects *Salix alba* and its hybrids (Dowson, W. J., 1937). A very

similar disease of *S. alba*, *S. amygdalina* and *S. purpurea* in Holland has been attributed to a different organism, *Pseudomonas saliciperda* Lindeier.

Day recommends spacing trees farther apart than is usually practised, a distance of 25 ft. is suggested, avoiding localities that are marshy or have a wet subsoil. Infected trees should be felled and their tops burned by autumn. Essex County Council has made felling of infected trees compulsory.

SALPIGLOSSIS

Grey Mould (*Botrytis cinerea* Fr.)

In Denmark *Salpiglossis* only does well in really fine summers, largely because it so easily falls a prey to grey mould. Large brown areas appear especially on the stems and become overgrown by that fungus (see p. 59).

Salpiglossis is also liable to be infected by the virus of tomato spotted wilt (see p. 384).

SANSEVIERIA

Leaf Spots (*Fusarium moniliforme* Sheldon)

On *Sansevieria* leaves the mould *Fusarium moniliforme* causes circular somewhat sunken spots $\frac{1}{4}$–$\frac{1}{2}$ inch across. They are reddish brown with a yellow edge and may completely penetrate the leaf; eventually the centre of the spot may dry up and fall out.

The disease has been studied in detail by Kotthoff in Germany (1937a) and L. K. Jones in North America (1940). What was apparently this disease has also been observed in Denmark.

Infected leaves should be cut out and the plants sprayed with Bordeaux mixture or other fungicide, preferably with a sticking agent added.

SCABIOUS

Mildew (*Erysiphe polygoni* DC.)

A race of this mildew causes a powdery white coating on scabious leaves (see mildew, p. 63).

Leaf Spot (*Ramularia* sp.)

Moore (1943) has found a species of *Ramularia* on scabious leaves, causing leaf spots which dropped out like shot-holes. This was perhaps *R. knautiae* (Massal.) Bubak, which causes round blackish-purple spots on leaves of wild scabious.

SCHIZANTHUS (Poor Man's Orchid)

Schizanthus often becomes infected with the virus of tomato spotted wilt (see p. 384), which causes a temporary stoppage of growth followed by production of a bushy, stunted plant. The plant may also become infected with *Solanum* Virus 2, the cause of leaf drop streak in potato (see p. 339), but the virus seems to do no harm to *Schizanthus*. Other diseases known to attack this plant are mildew (*Oidium* sp., see p. 63), *Sclerotinia* rot (see p. 77), foot rot (*Corticium solani*, see p. 68), and leafy gall (see p. 484).

SCILLA

Rust (*Uromyces scillarum* Wint.)

This rust attacks the wild bluebell (*Scilla nutans*) and cultivated *S. campanulata* and *S. bifolia*. It forms only teleutospores, in small brown pustules which occur crowded together in elliptical patches about $\frac{1}{4}$–$\frac{1}{2}$ inch across. If they are very numerous the leaves may turn yellow and die.

Anther Smut (*Ustilago vaillantii* Tul.)

This affects flowers of *S. bifolia*, *S. taurica* and the wild vernal squill, *S. verna*. Plants only become infected in the seedling stage but then remain infected for life. The fungus forms dark-purple masses of spores in the anthers, spoiling the appearance of the flowers, but otherwise does little harm to the plant (Massee, I., 1914).

Septoria scillae Westd. causes vague pale-brown spots, dotted with the tiny brown pycnidia, on living leaves of bluebell, other species of *Scilla* and *Muscari*. They occur mainly at the tips of the leaves.

Scillas may also be attacked by *Sclerotinia bulborum* (see p. 470), *Sclerotium tuliparum* (see p. 542), *S. delphinii* (see p. 479), and *Penicillium gladioli* (see p. 465).

SEA PINK

See *Armeria*, p. 415.

SEMPERVIVUM (House Leek)

Rust (*Endophyllum sempervivi* de Bary)

Affected plants have unusually narrow and pointed upright heart leaves, on which small orange or yellow pustules of rust break out (see p. 455).

SENECIO (Cineraria, etc.)

Mildew (*Oidium* sp.)

Whitish, floury spots occur on all the green parts of the plant and may join up to form extensive patches. The parts affected may be somewhat deformed. As perithecia have never been found in this mildew it cannot yet be fully identified (see mildew, p. 63).

Rust (*Coleosporium senecionis* Fr.)

Uredospores of this rust occur in orange-coloured pustules on the under-side of the leaves on cinerarias and occasionally on *S. smithii*. The rust is common everywhere on groundsel. The aecidial stage occurs on the needles of pine trees (see p. 510) but it seems clear that the rust can persist indefinitely in the uredo stage on species of *Senecio* without returning to the pine. Thus it occurs on the Isle of Coll from which pines are completely absent (Wilson, 1934).

Where rust is prevalent on cinerarias the plants should be sprayed with a copper fungicide like Burgundy mixture while still small. A trial spraying on a few plants should be made to make sure the spray is not too strong for them.

Foot Rot (*Phytophthora cryptogea* Pethybr. & Laff., *Pythium* spp. *Corticium solani*, etc.)

Symptoms. A soft rot develops on the stem just above soil level and the plant wilts. It often occurs just before the plant is due to flower. Usually a few plants out of a batch develop the disease first and are then followed by many more. Any of the usual damping-off and foot-rotting fungi can attack cinerarias, *Phytophthora* spp., *Pythium* spp., *Corticium solani* and even species of *Fusarium*. Probably *Phythopthora cryptogea* and its allies are the most destructive. Under moist conditions the stems may also be attacked by the grey mould *Botrytis cinerea* (see p. 59).

Control. The plants should not be grown under very damp conditions. As soon as an infected plant is seen the grower must realize that the soil is infected with one of the above organisms and that many plants will probably be killed unless steps are taken to check the disease at once. The whole batch of plants should be watered with a disinfecting solution such as 2 oz. corrosive sublimate and 1 lb. chile saltpetre in 22 gallons water or with one of the proprietary organo-mercury soil fungicides. The soil should be damp when the watering with disinfectant is done, and if any of it falls on the leaves it should be washed off with clean water.

Soil for cinerarias should be disinfected by either steam or formalin (see p. 577).

Leaf Spot (*Ascochyta cinerariae* Tassi)

This causes circular brown or black spots on the leaves which in the end make them shrivel and rot. The disease is known in Italy and Germany, and Moore (1943) has recorded this or a related species causing rotting of cineraria stems in England.

A similar leaf spot of Cineraria caused by *Alternaria senecionis* was described by Neergaard in Denmark and probably occurs also in England (Green and Hewlett, 1949a).

Virus Disease

Cineraria plants often become infected with the virus of tomato spotted wilt (see p. 384). Pale-yellow spots develop on the leaves, often followed by brown streaks on the under-side of the veins. The plants are usually severely stunted and may be killed.

SINNINGIA (Gloxinia)

Foot Rot and Corm Rot (*Phytophthora parasitica* Dastur., *P. cryptogea* Pethybr. & Laff. and other species)

In Britain, Denmark, Germany and Holland species of *Phytophthora* cause rotting of gloxinias. The rot starts at the base of the

plant, spreading up the leaf stalks and flower stalks and down into the corm.

Blue varieties seem more susceptible than white ones, and the varieties Defiance, Kaiser Friedrich and Kaiser Wilhelm are fairly resistant.

Affected plants should be removed and burnt. One may try spraying with a copper fungicide to prevent infection of those plants not already attacked. Watering with Cheshunt compound may help to kill the fungus in the soil. Packing sand round the corms may also be beneficial.

Grey Mould (*Botrytis cinerea* Fr.)

Under damp conditions a brown rot appears on the flowers and afterwards on the leaves, and becomes covered with the common grey mould (see p. 59).

Virus Disease (*Lycopersicum* Virus 3)

Leaves of infected gloxinias show very conspicuous brown rings with green tissue inside. The rings may be quite wide and may be so numerous as to cover the leaves. In the latter case the leaves may be killed and infected plants are usually more or less stunted and disappointing (see tomato, p. 384) (Green, D. E., 1934a).

SNAPDRAGON

See *Antirrhinum*, p. 412.

SNOWDROP

See *Galanthus*, p. 461.

SPRUCE

See *Picea*, p. 506.

STATICE

Leaf Spot (*Cercospora* sp.)

In September 1936 we found numerous brown-ringed spots on leaves of *Statice rosea superba* (Fig. 313). They were associated with a species of *Cercospora*. Spraying or dusting with a fungicide may be tried as a control.

What may have been the same disease has been ascribed to *Cercosporella* sp. in England.

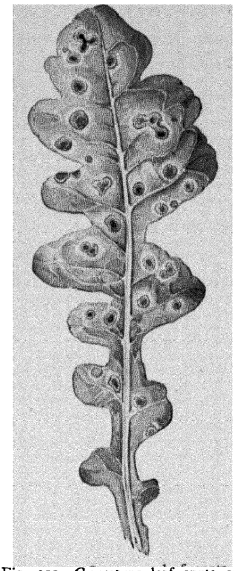


Fig. 313. *Cercospora* leaf spots on statice.

Other diseases recorded on statice in England are mildew (*Oidium* sp., see p. 63), rust (*Uromyces limonii* Lév., which forms aecidia, uredo and teleutospores all on statice leaves), grey mould (*Botrytis cinerea* Fr., on flowers and shoots, see p. 59), and the virus of tomato spotted wilt (Moore, W. C., 1943) (see p. 384).

STOCK

See *Matthiola*, p. 492.

SUNFLOWER

See *Helianthus*, p. 468.

SWEET PEA

See *Lathyrus*, p. 483.

SWEET WILLIAM

See *Dianthus*, p. 448.

SYCAMORE

See *Acer*, p. 408.

SYRINGA (Lilac)

Leaf Spot (*Heterosporium syringae* Oudem., etc.)

Various fungi cause leaf spots on lilac, especially *Septoria syringae* Sacc. & Speg., *Gloeosporium syringae* All., *Phyllosticta syringae* West. and *Heterosporium syringae* Oudem. The last-named, which occurs in Denmark and England, causes large, irregular, grey-brown, dry patches on the leaves. It fructifies as minute tufts of blackish mould dotted over the surface. *Phyllosticta syringae* also causes large brown leaf spots but they have a darker edge and are dotted with pycnidia on the upper surface.

The obvious control measure to recommend is spraying with a copper fungicide, but Dutch experience has shown that lilacs are sensitive to Bordeaux mixture. Hence any spraying done should be preceded by a small-scale test to find out the effect on the foliage.

Leaf Roll

Leaves of lilac not infrequently roll up and turn yellowish (Fig. 314) and the development of the plant is checked. Unfortunately the cause of this tiresome phenomenon is still unknown. Leaf rolling may occur after frost and also if a ring of bark has been cut or damaged, as by the wire holding an old name label. Apparently excessive pruning and damage to the roots or unsatisfactory nutrition may all cause rolling of the leaves. C. H. Cadman (1940) has shown that rolled, brittle and yellowish leaves are produced by lilac scions grafted on to privet stocks and that the trouble is due to incompatibility between scion and stock. Finally, very similar symptoms have been attributed to virus infection (Smith, N. M., 1940).

Verticillium Wilt (*Verticillium albo-atrum* Reinke & Berth.)

In 1922 Van Der Meer proved that serious attacks of wilt on lilac at Aalsmeer, the centre of the Dutch lilac industry, were due to *Verticillium*. The disease has occurred several times in Denmark.



Fig. 314. Leaf rolling of lilac.

The whole bush or only one side of it may be affected. The leaves take on a milky hue, starting at the bottom of the bush and spreading upwards. They are then shed except for those at the tips of the branches, which may remain hanging a long time, with shrivelled margins overgrown by various dark moulds.

Slightly infected shoots become short and thick and may carry only small clusters of flowers. Severely affected twigs and branches die but their wood is only slightly discoloured. Diseased plants should be grubbed and burned, roots and all (see *Verticillium* wilt, p. 60).

Lilac Fungus (*Phytophthora syringae* Kleb.)

Symptoms. The disease very seldom occurs on lilac bushes in gardens but almost exclusively on forced plants of grafted varieties. Dead brown patches appear on the stems and branches and may girdle them so that all above dies. In Denmark we have found the plants attacked at the graft so that the whole bush is killed. The buds may be partially or wholly dark brown with a shrivelled surface.

Any of the buds may be affected, both terminals and laterals, but the number may vary from one to many. In Holland De Bruyn (1924, 1928) found the leaves also infected, but we have not seen this in Denmark (Fig. 315). Irregular brown spots appear scattered over the leaves; their shape is irregular but they gradually involve much of the blade. Their colour differs somewhat in different varieties; on dark types they are almost sepia while on Marie Legraye they are paler; often there is a pale-green edge to them. The usual symptoms in Denmark are the brown patches on the bark.

Cause. All these symptoms are caused by the water mould *Phytophthora syringae* which attacks *Syringa vulgaris*, *S. persica*, *Ligustrum vulgare* (privet), *Jasminum nudiflorum*, *Forsythia viridissima*, and hawthorn, also apple and pear fruit (see p. 125). It lives in the soil and is splashed on to the plants by heavy rain or during watering, thus reaching and infecting both leaves and branches. From a single infected leaf, on which conidia develop during a dewy night, the disease may spread further. It may grow into the branch through the stalk of an infected leaf but more often the leaf is shed before this can happen. Infection occurs mainly in a damp August or September.

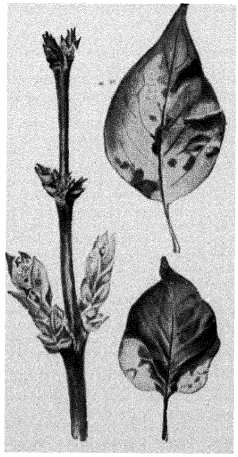


Fig. 315. *Phytophthora syringae* lesions on lilac buds and leaves (after de Bruyn).

Control. In Holland the expedient was tried of picking the leaves off early so that infection of the branches could not occur via the leaf stalks, but it was found that this checked development of the flowers and was therefore inadvisable.

Bordeaux mixture and lime sulphur sprays had a tendency to scorch the leaves.

In Denmark we have observed great varietal differences in susceptibility. Charles X is very susceptible and may be replaced by Jacques Callot and Hugo Koster.

To avoid the disease the grower should set his plants out of doors on raised beds with channels between, especially if the soil be damp. When they are planted they should be kept upright so that leaves and shoots do not touch the soil and so become exposed to infection. If grafting is done fairly high up it lessens the risk of infection at the graft by soil getting into the cut.

Spraying stems and branches with Bordeaux mixture just after leaf fall may to some extent protect against infection. Boxes and pails are to be preferred to pots as the latter have to be covered with soil to protect them from frost.

Silver Leaf (*Stereum purpureum* (Fr.) Fr.)

The leaves take on a milky hue or silvery sheen (see plum, p. 191).

The bushes may also be killed by *Armillaria* root rot (see p. 456).

Bacterial Blight (*Pseudomonas syringae* Van Hall)

The bacterium which blights lilacs, described by the Dutchman Van Hall in 1902, is now known to have a wide host range; not only is it found on lilac in Europe and North America, but the bacteria causing citrus diseases in the Mediterranean region and the Western States of America, formerly called *Ps. citriputeale* (C. O. Smith) Stapp. and *Ps. citrarefaciens* (Lee) Stapp., are now believed

to be the same as *Ps. syringae*. It is now known to attack french beans, roses and chrysanthemums, and in inoculation experiments it has infected ash, lettuce, oak, pear, poplar, *Prunus* spp., tomato, chrysanthemum, etc. It is a close ally of *Ps. prunicola* Wormald (see p. 199) and *Ps. cerasi* Griffin, so widespread in western North America.

In May or June, or earlier under glass, watersoaked brown spots, which soon spread and turn black, appear on the lilac bushes. They are first seen on the green skin of the young shoots, but spread up through the leaf stalks on to the leaves. The shoots may be girdled and die or they may merely become distorted, when the dead blackened patches are easily invaded by fungi such as *Botrytis*, *Phoma* sp., etc. The bacteria may travel along the vascular bundles and so reach other leaves on which spots appear along the veins or in the blade, sometimes so many that the leaf withers. The general appearance is suggestive of frost damage and it appears that spring frosts, cold, or damp, predispose the bushes to an attack. Infection easily occurs through wounds but can occur without them. *Syringa vulgaris* seems less susceptible than *S. persica* or *S. sinensis*.

Control consists essentially in cutting out the diseased branches and, under glass, in watering sparingly. Apparently heavy nitrogenous manuring favours the disease while lime and phosphoric acid make the plant more resistant. The bacteria may survive some time in the soil so that a rotation of crops is advisable in nurseries and care should be taken over the source of potting soil. Lilac cuttings should be kept out of reach of frost.

TAGETES (African Marigold)

Bacterial Leaf Spot

In July 1937 we first saw almost black round or angular spots in *Tagetes* leaves (Fig. 316). The plants were of the variety Yellow Supreme, from a nursery near Copenhagen. In June and July 1938 similar infected leaves were received from Sjaelland, Fyn and Jylland, and we were told by a seed firm that the variety concerned had been attacked at various places where other varieties were unaffected.

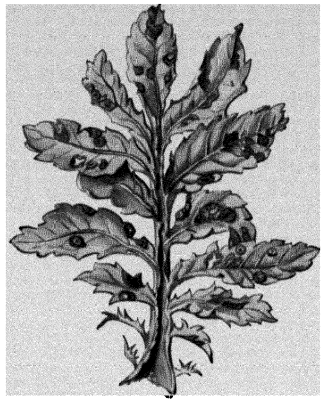


Fig. 316. Bacterial leaf spot of *Tagetes*.

Bacteria were invariably found in the leaf spots but the disease was not further investigated. Circumstantial evidence points to transmission with the seed so it is worth while to try seed disinfection (see p. 576).

Grey Mould (*Botrytis cinerea* Fr.)

Tagetes is often attacked by grey mould, especially the woody type grown under glass. Large rotten patches appear on the plants, and under damp conditions they become covered with the hairy grey mould, sometimes accompanied by black sclerotia.

When the decay girdles the stem everything above the point of attack wilts and dies.

The brittleness of *Tagetes* shoots makes them easily broken and multiplies wounds through which *Botrytis* can gain access.

Control should be directed to avoiding wounding, keeping the plants dry and the atmosphere as dry as they can stand. Growth should not be too fast. Plants grown in pots are less prone to attack than those bedded out. As a last resort spraying with Pomarsol (p. 598) or dusting with Brassicol (p. 592) may be tried.

Foot Rot (*Phytophthora cryptogaea* Pethybr. & Laff.)

Tagetes may rot at the foot, which turns black and shrivels so that the plant wilts and dies. The cause is usually *Phytophthora cryptogaea*, which causes similar trouble in so many plants, e.g. tomato (see p. 375).

Tagetes varieties differ somewhat in susceptibility. As the fungus usually comes from the soil it should either be disinfected with steam or formalin or fresh soil obtained.

THALICTRUM (Meadow Rue)

Many species of *Thalictrum* can act as aecidial host to a group of related rust fungi which have their uredo and teleutospore stages on cereals or grasses, notably *Puccinia triticina* Erikss., which attacks wheat, and *P. persistens* Plowr., which attacks couch grass (see rust, p. 74).

Thalictrum may also be attacked by a powdery mildew *Sphaerotheca humuli* (DC.) Burrill var. *fuliginea* (Schlecht.) Salin (see mildew, p. 63).

THRIFT

See *Armeria*, p. 415.

THUJA

Leaf Scorch (*Didymascella thujina* (Dur.) Maire = *Keithia thujina* Durand)

This fungus was first found in North America in 1913 and is thought to have come from there with *Thuja plicata* and *T. occidentalis* on which it is found. It was found in Denmark in 1935 and is quite common in Britain. The living leaves are attacked and turn brown. Infected leaves may be scattered amongst healthy ones or most of those on a twig may be killed, making the whole twig look brown. This happens especially towards the base of the tree. The fructifications of the fungus appear on the upper side of the dead leaves, like tiny dark-brown circular cushions $\frac{1}{50}$–$\frac{1}{25}$ inch across. They become exposed by the falling away of the overlaying skin like a lid, and after the ascospores have been shot out the whole fructification drops out, leaving a little hole in the leaf tissue.

The disease may become serious under damp conditions, but may be prevented by spraying with Bordeaux mixture (Buchwald, 1936a).

Thuja on soil poor in lime may develop somewhat similar brown leaves and twigs, so one should look for the fungus fructifications before making a diagnosis.

TILIA (Lime)

Leaf Spots

Various fungi may cause leaf spots on lime. In Denmark the common lime (*T. vulgaris*) often has its leaves marked with brown spots $\frac{1}{16}$–$\frac{1}{8}$ inch across, sometimes with pale centres, caused by the fungus *Cercospora microsora* Sacc.

On common lime and small-leaved lime (*T. cordata*) considerable leaf fall occurs in early summer in some years; on the leaves are large pale spots with blackish-brown margins and there may be similar spots on leaf stalks and twigs. The cause is the fungus *Gloeosporium tiliae* Oud.

Lime leaves may also have numerous brown or black angular spots $\frac{1}{16}$–$\frac{1}{8}$ inch across. For eight years we have seen them on a tree of *T. platyphyllos* var. *pyramidalis* from Riga, planted among nine other kinds of lime which were unaffected. The cause is unknown.

Canker (*Pyrenochaeta pubescens* Rostr.)

In the nursery state *Tilia platyphyllos* and *T. vulgaris* growing on rather acid soils may be attacked by a fungus which produces deeply sunken spots on the bark, up to $\frac{1}{2}$ inch across. At first the spots are dark red or blackish brown, then greyish white dotted with small, black, pimple-like, hairy pycnidia.

Spraying during the winter with 2–3% copper sulphate solution or brushing with the same solution has had a beneficial effect (Lind J., 1909).

Butt Rot (*Ustulina vulgaris* Tul.)

This fungus is a Pyrenomycete whose fructifications form hard black shining crusts 2 or 3 inches across on dead stumps. It is a

wound parasite of deciduous trees, notably beech, elm and lime. The trunk is invaded through a wound, such as a fire scar, at the butt, and decay spreads through the heart wood both upwards and down into the larger roots. The decayed wood, which is paler and lighter than the normal, is bounded and permeated by narrow dark zone lines (Wilkins, W. H., 1936, 1938, 1939).

TROPAEOLUM (Garden Nasturtium)

Leaf Spot (*Lycopersicum* Virus 3)

Garden nasturtiums infected with the virus of tomato spotted wilt (see p. 384) are stunted, with yellowish, distorted leaves. Usually numerous pale dead spots appear almost covering the leaf surface.

The plant may also be infected by leafy gall (*Corynebacterium fascians*, see p. 484).

TSUGA

Leaf Scorch (*Keithia tsugae* Farlow)

Infected leaves occur scattered amongst the healthy ones or sometimes all the leaves on a twig become diseased. They turn brown and die and in severe cases the whole twig may be killed. Fructifications of *Keithia tsugae* appear on the under-side of the dead leaves. They are dark brown, cushion-like, circular or elliptical and about $\frac{1}{50}$ inch across. The epidermis covering them is turned back like a lid and after the spores have been shed the whole fructification drops out, leaving a minute pit in the dead leaf. This disease occurs on *Tsuga canadensis* in North America and was found on the same host in Scotland in 1937 (Wilson M., 1937).

TULIPA (Tulip)

Fire (*Botrytis tulipae* (Lib.) Lind)

Symptoms and development. Fire attacks both the bulb and the above-ground parts, though most of the damage is done to the latter. It is very prevalent in Britain, Denmark and Holland and probably occurs wherever tulips are grown. On the brown outer scales, or on the outermost white bulb scale, occur small

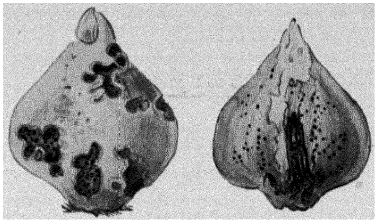


Fig. 317. Tulip bulbs bearing sclerotia of *Botrytis tulipae*.

round sclerotia, $\frac{1}{25}$–$\frac{1}{12}$ inch across. At first they are white, but later become black and shiny, and it is in this state that they are found on the dry bulb (Figs. 317, 318). In very severe attacks they may be so numerous as to form a continuous scab over large parts of the bulb. Under damp conditions the outer fleshy scale may be decayed by the fungus.

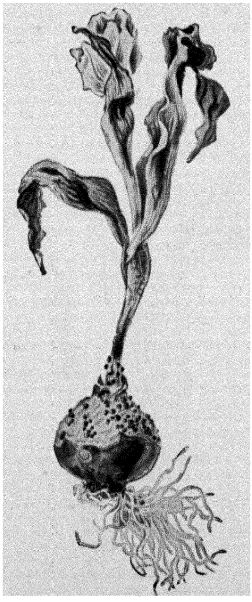


Fig. 318. Tulip fire on Murillo.

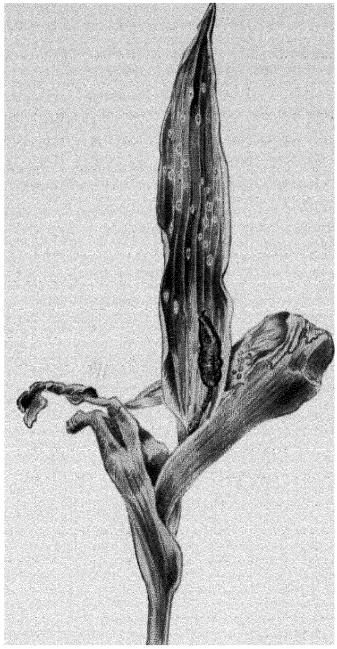


Fig. 319. Tulip fire on the leaves.

On the leaves there appear small greyish-yellow spots with reddish-brown margins, outside which a translucent yellow line is usually discernible by transmitted light (Fig. 319). Gradually the spots become larger and develop on their centres a grey coating of conidiophores and conidia. In severe attacks the whole leaf is malformed. On the stalks there first appear small spots like those on the leaves, but later sclerotia may be developed and the stalk may bend over, somewhat as in topple. If the flower buds are severely attacked when young they do not open at all, but if the flowers are attacked after they have partially or entirely opened, small pale spots with dark edges arise, giving the blooms a spotted appearance, especially in red varieties

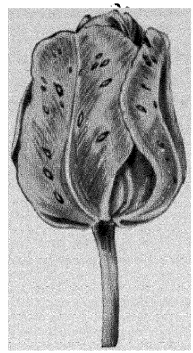


Fig. 320. Tulip fire on petals of Darwin tulip.

(Fig. 320). If tulips are standing in a moist atmosphere the buds decay and the flowers are wholly overgrown by the grey mould. Conidia are not formed on the bulbs themselves. Infection of leaves, buds and flowers is very general in wet years on Darwin tulips out of doors, especially when the bulbs have been in the same ground for several years.

On early varieties, such as the Murillo group, the attack may be even more severe. The whole shoot shrivels up and becomes covered with the grey mould of conidiophores and conidia as soon as it emerges above ground. Sclerotia may be abundant on these dead shoots and on the bulbs beneath. In the most severe attacks the shoot does not even emerge from the soil. Tulip is the only plant attacked by this species of *Botrytis*.

Cause. Tulip fire is caused by the fungus *Botrytis tulipae* (Lib.) Lind, first described by Mlle Libert in Belgium in 1830. *B. parasitica* Cavara is a later name for the same fungus.

Infection may come (1) from infected bulbs, (2) from infected soil, and (3) by secondary spread of the disease through the air by conidia from plants infected in one of the first two ways. This secondary spread is greatly favoured by a damp atmosphere. Temperature is less important, for the conidia

can germinate over a wide range of temperatures up to 77° F.

Experience has shown that the attack on bedding tulips is worst in a moist spring. Frequent light showers are more favourable to the fungus than the same amount of heavy rain. It is the atmospheric humidity rather than the amount of rain which is important.

Conidia are easily spread with the rain and by rain splashes from plant to plant, but Beaumont, Weston and Wallace (1936) do not consider secondary infection serious at a greater distance than 10 yards from a primarily infected bulb.

At 65–68° F. the conidia begin to germinate in 2 hours. Those that have been kept dry retain their germinative capacity for 50 days or more.

Tulips planted in infected soil had their shoots attacked within a week and when they were dug up a fortnight later sclerotia had formed on the tip of the bulb and on the lowest part of the stalk.

The sclerotia are very resistant to chemicals. According to Beaumont *et al.* (1936) they can endure 2% copper sulphate solution for several days, and several germinated after 15 minutes' soaking in undiluted formalin. They did not germinate after 2 hours in 0.05% corrosive sublimate solution. Methylmercuric-chloride at 1: 100,000 killed the sclerotia in 2 hours and at double that strength (1: 50,000) it did not kill 500 William Pitt bulbs dipped in it.

Varietal susceptibility. All tulip varieties may be attacked, but Baronne de la Tonnaye is said to show considerable resistance.

The Murillo types are very susceptible and also Darwins, especially Bartigon, William Copland and William Pitt.

Control. Whether one is a bulb grower or a tulip forcer one should avoid planting bulbs which bear sclerotia. The bulb grower should sort the bulbs thoroughly after lifting and rather discard those that are only suspected of harbouring the disease than keep any that are diseased, for these will act as primary foci of infection.

When the disease is noticed on sprouting bulbs they should be removed at once, to avoid spread of the leaf and flower spot phase of fire by conidia from the rotted shoot.

Wounds favour infection but if enough moisture is present the conidia germinate on and attack quite uninjured tissues.

Spacing the bulbs well, both in boxes and in beds, checks spread of the disease. In houses watering should be restricted and the air kept dry, hence overhead pipes are an advantage.

Spraying of outdoor tulips has not proved very successful and is not generally practised. Bordeaux mixture may cause much scorching and by producing damaged spots on the plants may aid penetration by the fungus rather than check it. Sulphur sprays and dusts are ineffective.

Of late years the salicylanilide spray Shirlan (20–40 oz. per 20 gallons water) has been extensively used in Denmark and Holland with good results. The effect of a spray can only be assessed when an unsprayed control area has been left, as otherwise an apparently beneficial effect may have been due to the fungus being checked by dry weather. In Holland 0.8% Pomarsol and 1.2% Tulisan sprays have also been recommended (Plantenziektenkundige Dienst, 1939).

As infection may be contracted from the soil and the sclerotia remain alive there for 2 years and more, tulips should not be replanted on soil that has carried tulips within the past four years.

Steam sterilization is the only reliable way of destroying the sclerotia in the soil.

Bulb fields should be inspected at least once a week and all diseased tissue removed. As soon as it is picked off the plant it should be dropped in a can of disinfectant, e.g. weak formalin solution or even lime water. If it is laid in an open basket and carried round dry it will be shedding conidia all the way. All diseased parts should be removed daily from tulips under glass.

Disinfection of the bulbs by soaking ½–1 hour in Aretan solution or similar organo-mercury dip is to be recommended.

Shanking (*Phytophthora cryptogaea* Pethybr. & Laff. and *P. erythroseptica* Pethybr.)

These two water moulds have caused enormous losses to British tulip growers. In 1913 one grower lost 12,000 out of 30,000 William Coplands by shanking. Infection takes place from the surrounding soil by way of the roots and starts as soon as the first roots begin to grow out. The fungus penetrates through the base of the bulb into the bottom of the flower stalk, leaving the old bulb scales almost untouched. When the attack progresses rapidly the bulb does not shoot (Fig. 321) but more often it starts to develop

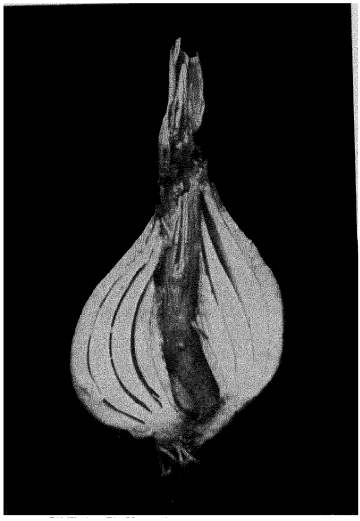


Fig. 321. Shanking of Wm. Pitt tulip (*Courtesy Annals of Applied Biology*).

normally; a flower bud emerges which remains blind because the base of the stalk has decayed. Its leaves, too, are poorly developed. This form of the disease is the typical " shanking." Sometimes if only a few roots are attacked the flower opens but is under-developed. The fungus may occur on the bulbs but serious outbreaks of shanking arise from soil infection. Infected soil may be disinfected by steam or by watering it with 2% formalin solution and leaving covered for 2 days with sacking soaked in formalin. Cheshunt compound is not effective against shanking (Foister, C. E., 1930).

Root Rot (*Pythium ultimum* Trow)

Symptoms. When the bulbs are brought in from the pits to be forced there is usually nothing to show they are diseased, but in about a week symptoms become apparent. The growth of the shoots is arrested and often they can easily be pulled out and are grey and soft at the base, usually with a narrow brown zone between this and the healthy tissue above. Similar large or small greyish rotting areas may be seen in the bulb scales, delimited by a darker grey or brownish line (Fig. 322). The attack often starts at the base of the bulb and spreads upwards on one side, but it may involve the whole bulb and reduce it to a soft putrid mass.

Development of the bulb may be stopped by the disease at various stages, sometimes when the shoots are only 2–4 inches long, sometimes after the leaves have unrolled and the buds are about to bloom. As a rule the roots develop well at first but stop growing and rot if the fungus attacks the base of the bulb.

Cause. The water mould *Pythium ultimum* Trow is found in the rotting bulb scales and shoots. It can infect tulip bulbs both directly and by way of infected soil, but it apparently needs either specially favourable environmental conditions or specially susceptible bulbs (Lund, A. A., and Weber, A., 1935).

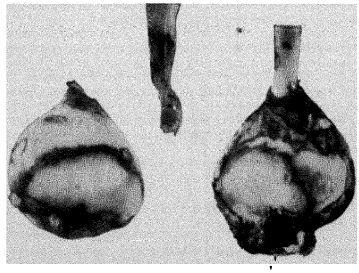


Fig. 322. *Pythium* rot of tulips.

The same fungus can attack seedlings of cucumber, bean, cabbage, cress, tomato, red beet and peas, causing root rot and damping off, and may rot cucumber fruit (see p. 276) and potato tubers (see p. 333).

The disease was particularly prevalent round Copenhagen in 1933–4, and the same fungus has been isolated from diseased tulips in Holland. In England tulip root rot and basal rot may be caused by it or by related species of *Pythium* (Moore, W. C., and Buddin, W., 1937). If the roots only are attacked the bulb may flower more or less normally.

Control. As the disease only occurs occasionally little attempt has been made to devise control measures. Bulbs for forcing should be planted in sterilized soil. Consignments should be examined when received and any rotting ones discarded at once.

Grey Bulb Rot (*Sclerotium tuliparum* Kleb. = *Rhizoctonia tuliparum* (Kleb.) Whetz. & Arth.)

Symptoms. On tulips for forcing the grey bulb rot fungus shows up when the boxes are brought in from the pits or a little later. Out of doors the attack becomes visible when the bulbs have reached a corresponding stage in development. In severe early attacks the shoot often does not come up, or if it does it is rotten from the outset. Or the sprout may then be brown and decayed at the tip only and the rot spreads down it later into the bulb. Sometimes there is a small brown spot on the sprout just above the bulb, which gradually spreads and involves sprout and bulb in a common decay. Inside the diseased bulbs the fleshy scales take on a rather characteristic light-grey or pinkish-grey colour, often darker in the upper than in the lower half of the bulb (Fig. 323). Between the scales one may find the whitish mycelium of the fungus but often the picture is complicated by the appearance there of other secondary fungi. The roots are almost always well developed and healthy and the basal plate is seldom attacked. Sclerotia develop on the surface of the bulb, especially towards the tip and in the surrounding soil, occasionally also between the roots (Fig. 324).

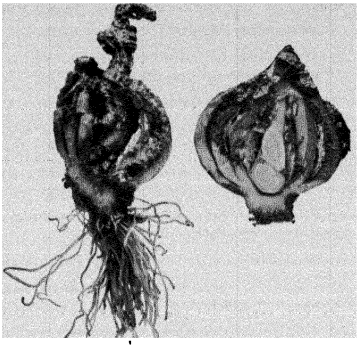


Fig. 323. Grey bulb rot of tulip (*Photo Whetzel & Arthur*).

They only loosely adhere to the bulb and are easily left in the soil. At first they are soft and white and embedded in mycelium, later they turn light yellowish brown, then dark brown and finally become almost black and hard. They are pale yellowish-brown inside, unlike the sclerotia of *Sclerotinia sclerotiorum*, which are white inside with a black rind. The sclerotia of *S. tuliparum* vary greatly in shape from spherical to nodular and in size from that of a cabbage seed to that of a small maize

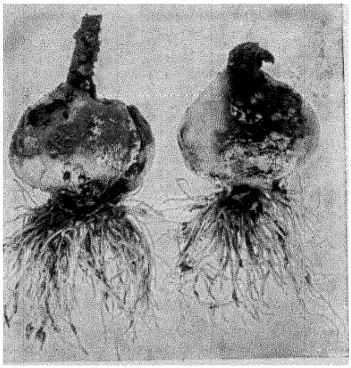


Fig. 324. Mycelium and sclerotia of grey bulb rot fungus on tulip (*Photo Whetzel & Arthur*).

grain. Infection almost invariably leads to the complete decay of the bulb.

Cause and development. Grey bulb rot is caused by the fungus *Sclerotium tuliparum*. It was noticed in Holland by Wakker as early as 1884 but he did not give it a name. Ritzema Bos in 1903 confused this disease with fire. In a series of papers from 1903 to 1907 Klebahn studied the fungus in Germany. At first he, too, confused it with fire but later he realized its distinctness and called it *Sclerotium tuliparum* Kleb. In 1925 Whetzel and Arthur in North America re-examined the fungus and changed the name to *Rhizoctonia tuliparum* because they considered the structure of the sclerotium more like that of *Corticium* (*Rhizoctonia*) *solani* than that of *Sclerotinia sclerotiorum*. This change of name has not been generally adapted, however.

S. tuliparum forms sclerotia more quickly than does *Botrytis tulipae*, and so far no spore form belonging to it has been described. Presence of ripe sclerotia on the decayed bulbs does not mean they were infected before planting. If sclerotia are present in the soil the mycelium grows out of them and attacks bulbs in the vicinity, at least to a distance of 2 inches. If the bulbs are closely planted the disease may spread from bulb to bulb. Then infection from a single sclerotium in the soil may spread to several bulbs; in Klebahn's experiments it spread 7 inches from the sclerotium. Sclerotia may remain alive for several years; Klebahn found those 3 years old could still infect bulbs but not so vigorously as fresh sclerotia under the same conditions.

Cross-inoculation experiments have shown that *Iris hispanica* is attacked in the same way as tulip, and Brooks (1926) found it in England attacking *Iris reticulata*. Hyacinth, yellow narcissus and *Scilla sibirica* are only attacked when plentiful inoculum is present on the shoot, and usually infection of these species does not kill them. The rotted part dries up leaving a malformation of the organ attacked. Hyacinth and narcissus bulbs are usually unattacked, but those of *Scilla* and *Fritillaria imperialis* may be completely destroyed. Sclerotia develop only sparsely on these four hosts. *Muscari*, *Narcissus poeticus*, snowdrop and crocus were almost immune from attack in Klebahn's experiments, but in England crocus foliage has been destroyed though the corms were untouched. Forced irises, especially Wedgewood and Imperator, are often severely attacked. Buddin (1937–8) found *Colchicum* and *Ixia* to be susceptible. A map of the known distribution of *S. tuliparum* in England and Wales will be found in *Min. Agric. Bull.* 139, 1948.

The fungus does a great deal of damage to tulips both under glass and in the field.

It is exceptional for infected bulbs to be sold, but circumstantial evidence points to the disease having been introduced to several countries with bulbs from Holland. As the fungus is likely to do only a little damage for a few years after a casual introduction it is very difficult to estimate when the introduction actually occurred in a particular instance. In trying to estimate this one has to consider the following facts:

(1) Sclerotia can retain their power of germination for 3 years.

(2) On a single bulb cut in half 20–30 sclerotia may form in the course of 3 weeks; we found this at Lyngby in the spring of 1931. Thus the soil may become heavily contaminated by sclerotia from a few diseased bulbs when they decay and become broken up.

(3) The disease arising from a single sclerotium can spread from bulb to bulb for at least 7 inches.

(4) Various other bulbous plants and corms may be attacked and the soil may have become infected by them at an earlier period. As daffodil and hyacinth may be attacked by the fungus without suffering much damage, bulbs of these plants, raised in infected soil, may act as carriers of the fungus without it being noticed.

Hence when grey bulb rot is found in a consignment it is not possible to say whether the infection came in with those bulbs or was derived from tulip or other diseased bulbs or corms that disintegrated in the soil a year or so before.

When the disease is present but the bulbs and perhaps the tips of the shoots are healthy

it is an indication that the bulbs and the soil in which they were planted were free from the disease, but that the soil covering the boxes in the pits was contaminated and the shoots became infected from that. Sometimes they are infected not at the tip but a little lower down.

Control. The bulbs should be planted in fresh soil in new or disinfected boxes (see p. 580); these should be covered with fresh or disinfected soil (see p. 577). Hence in practice one must change the pits every year or find new covering soil. Out of doors tulips should be planted in fresh ground or, if a grower wants to use one bed for tulips every year, he should change the soil periodically or disinfect it. Change of soil is, of course, imperative once the disease has appeared.

Both in boxes and outdoor beds diseased bulbs should be removed at once, with the surrounding soil. This applies also to blanks where bulbs have decayed before coming up.

According to Whetzel and Arthur (1925) infected soil can be disinfected by watering with 2% formalin solution at the rate of a gallon per square yard. It is best to cover the soil with sacks for 2 days after the treatment and we think a thorough soaking will give best results. Buddin did not get satisfactory results by disinfecting soil with 2% formalin. He did obtain 80–90% good blooms when bulbs planted in infected soil were set so high that from $\frac{1}{3}$ to $\frac{1}{2}$ of their surface was above the soil and covered with straw instead of soil. Bulbs planted to the usual depth in the same soil were all attacked by *S. tuliparum*. Of late years Brassicol has been extensively used for soil disinfection.

Smoulder (*Sclerotium perniciosum* van Slogt. & Thomas)

So far this disease has only been found in Holland but elsewhere it may perhaps have been confused with grey bulb rot or fire. It was first fully described by Van Slogteren and Thomas (1930) who thought it had probably been present in Holland for at least 12 years before that.

Smoulder occurs in tulips planted out of doors. There are gaps in the rows where bulbs have failed to grow and the leaves of others hang limp. The stalk of an infected leaf is decayed at soil level and covered by a web of white fungus mycelium which grows out also into the soil. Small black sclerotia, very like those of *Botrytis tulipae*, occur on the decayed tissues. In the bulb fields the disease is found mostly in the plants from the smallest bulbs, that have been planted unpeeled. When large bulbs are attacked it is usually at the base of the stem by infection from the soil. The bulbs themselves are not usually attacked.

Infection comes largely on the bulbs, apparently on the outer brown scale, as peeled bulbs largely escape the disease. Soil that has carried an infected crop, however, becomes contaminated with the fungus. Control measures include digging up and burning affected plants together with the surrounding soil, peeling all the bulbs and storing them in well-ventilated conditions. Disinfection by soaking the bulbs in Aretan or similar preparation for 30 minutes or more may also be tried.

Spotting of Leaves, Stalks and Flowers

(1) On Darwin tulips 8–12 inches high we have seen numerous small, brown, oval spots that often united to form broad bands running the length of the leaves. Similar small spots may occur on the stalks and on the white bulb scales. Their cause is unknown. We noticed the condition in March 1929 on tulips from Jylland. On enquiry from Van Slogteren we were told the condition had not been seen until that winter, when affected bulbs were sent to Holland from all over the world. No parasite was found in the affected tissues and no other cause was demonstrated, but it was suspected that damage by frost was involved.

In March 1940 we saw another instance of this condition in tulips. The winters 1928–9 and 1939–40 were both unusually severe, so it is likely enough that some form of frost damage had occurred.

(2) We found a somewhat similar condition in 1932 in out-door tulips. They developed small pale spots on the leaves. These, too, have occurred in Holland. They are not due to fungi or bacteria and their cause is quite unknown.

(3) Sudden spotting of Darwin tulip leaves occurred at several places in the United States in 1919. The spots were small, sometimes circular but more often elliptical with the long axis parallel to the veins. They were mostly from $\frac{1}{8} \times \frac{1}{25}$ to $\frac{1}{5} \times \frac{1}{16}$ inches, a few larger. There were also numerous, irregular, larger, dead areas which may in some cases have arisen by the fusion of small spots. The smallest spots had a brown centre with a yellowish-green watersoaked margin, larger ones had pale-yellow or brown dead centres surrounded by a brown zone with similar margin. The whole picture was suggestive of a bacterial leaf spot, but no bacteria were found and there was considerable evidence that the spotting was due to frost a week earlier (Jones and Miller, 1919).

(4) On the variety Hobbema rows of small spots along the edges of the perianth leaves have been ascribed by Van Slogteren to some unexplained physiological condition.

(5) Beaumont and Buddin (1938) have described small spots about $\frac{1}{4}$ inch across on forced tulip leaves caused by infection with *Fusarium avenaceum* (Fr.) Sacc. The fungus is common on old straw and the disease seems most prevalent where straw has been used to cover the boxes during the rooting period. The flowers are unaffected and the disease is evidently of minor importance.

Chalking

Symptoms. There is a discolouration of the outer bulb scales and in the more severe attacks it penetrates the inner white scales causing them to become grey or brown, or eventually the whole bulb is converted into a hard, white, chalky mass.

Spierenburg has reproduced the condition artificially by infecting bulbs with a species of *Penicillium*, keeping them damp for a time and then drying. In severe cases or when slightly affected bulbs are left damp, a grey-green fungus appears either on the surface of the bulbs or between the scales where, in severe attacks, it may form a layer as thick as one of the bulb scales (Plate 9, Fig. C).

Cause and development. A species of *Penicillium* is always associated with chalking. Many species of these moulds are commonly found on decaying plant remains, on " mouldy" bread and other foods, on damp leather, in the soil, etc. They are also often found on living parts of plants, especially resting or storage organs like bulbs, corms and tubers. Most of them, however, seem able to cause decay only when the plant organ has been weakened in some way beforehand.

Among factors conducive to chalking and to *Penicillium* invasion are mechanical wounding of the bulbs, lifting before they are mature and storing under badly ventilated and damp conditions. Besides tulip the condition may arise in hyacinth, narcissus and lily bulbs and in crocus corms. It is usually very difficult to tell exactly when the trouble started in a consignment.

Penicillium corymbiferum Westling is more actively parasitic than most of the blue-green moulds and can actually cause decay on its own account.

Calcification of the bulb scales may perhaps be caused by the bulbs having been grown on too damp or too heavy a soil, and the *Penicillium* is then only secondary. Moore (1939) states that chalking has been produced experimentally by stripping the outer brown scales from tulip bulbs and exposing them for several hours to hot sunshine. After storing 2–3 weeks in a dry airy place the bulbs were chalky throughout and useless.

Though *Penicillium* may completely permeate the bulbs between the scales the attack usually looks worse than it is. We have often planted infected tulip and hyacinth bulbs in soil and obtained excellent flowers from those lightly attacked while even severely affected tulip bulbs gave good but small blooms. Only the very severely affected bulbs failed to grow. On hyacinth bulbs grown in glass the *Penicillium* spreads rapidly if the water reaches up to the base of the bulb.

Control. Examine the bulbs when received, and throw away those severely affected. The bulbs should be handled carefully to avoid wounding them and should be kept dry until setting time.

Breaking (*Tulipa* Virus 1)

Breaking in tulips has been known at least since 1576, when it was considered to be an inherited malformation, and mutations of a similar appearance may indeed occasionally arise. The diseased plants are as a rule a little later and weaker in growth and their leaves may be mottled or striped. The conspicuous feature of the disease, from which it is named, however, is the breaking of the flower colour (Fig. 325), by which Darwin tulips are converted into Rembrandt tulips. The symptoms are milder in forced bulbs than out of doors. Infection can be spread artificially by sap



Fig. 325. Tulip breaking, normal flower on right.

inoculation or by grafting bulbs and naturally by aphides. McWhorter (1938) has shown that two viruses are involved in tulip breaking. *Tulipa* Virus 1 is responsible for the mottling of leaves, stunting of growth and causes pale streaks of a different colour on the flowers (full-break). *Tulipa* Virus 2 has no effect on leaves or growth but intensifies the red colour of the perianth leaves (self-break). The commercial varieties of broken-flowered tulips contain a balanced mixture of the two viruses. As a rule there is no sign of disease in the year when infection takes place (Brierly, P., and McKay, M. B., 1938).

If a grower wishes to plant Rembrandts or other " broken " tulips he should keep them well away from self-coloured varieties.

To control the disease in the latter, infected plants should be removed as soon as possible and aphides in houses and bulb stores should be controlled by fumigation. Outdoor tulip beds should not be very sheltered as this may encourage aphides.

McWhorter found *Tulipa* Virus 1 causing a mosaic disease of *Lilium speciosum* and found it to be carried without visible symptoms by *L. tigrinum*, *L. longiflorum* and *L. candidum*. This virus is also said to cause mosaic disease of hyacinths.

Blind Buds

Symptoms. As a rule the blind buds are first noticed when the normal ones begin to show colour or burst. Then the flower stalk from the topmost leaf to the bud is seen to be only an inch or so long and is quite dried up. The bud itself is $\frac{1}{2}$–$\frac{3}{4}$ inch long, yellowish-white and shrivelled (Fig. 326), hence the descriptive Dutch term *Stroobloemen* = "strawflowers."

Cause. As a rule it is very difficult to discover why tulips have formed blind buds, for often it is due to a tendency on the part of the bulb itself, complicated by unsuitable conditions of forcing. A consignment of tulips with this tendency will have many blind buds at early forcing and perhaps also at late forcing; the higher the temperature of forcing, the more blind buds.

The time the bulbs were lifted the previous summer has some influence on the matter. The after-treatment of bulbs treated to cold storage in order to prepare them for early forcing may increase the tendency to produce blind buds.

Blindness is often due directly to heating in transit or during storage before planting. Van Slogteren was able to produce the condition by exposing bulbs to fixed temperatures for particular periods between August and November. At such periods a temperature of 78° F. for a week or two may cause much damage. A warm autumn is therefore unfavourable to tulips for forcing.

Even at planting time the grower can judge whether consignments of tulip bulbs are suitable for forcing or not. The size of the bulb gives no guidance, but one should cut through a small number from each consignment and examine the shoot inside.

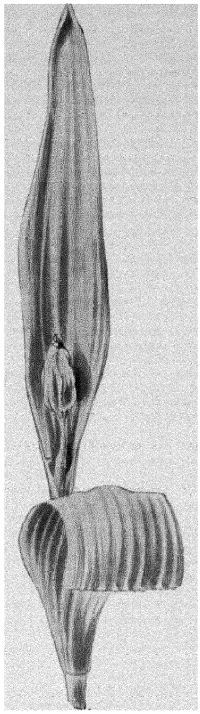


Fig. 326. Blind bud of tulip.

Samples that have a long thick shoot are better suited to early forcing than those with a slender shoot. The consignments that appear most suitable for forcing first should then be stored in such a position that one takes them first when planting. When one undertakes early forcing it is well to get several consignments of bulbs from different places so as to be able to choose the most suitable for the early forcing. When bulbs are planted for forcing not only should they have a long shoot but the flower bud should be already outside the bulb for then only can it be forced. It is also a good plan to put up for forcing a box from every consignment one hopes to force early. Then in a short time one can see which will force most easily.

High temperatures are tolerated less at early than at late forcing. Strong underheat is specially to be avoided as it may cause drying and failure of the roots. Dry soil encourages the production of blind buds, especially in forced bulbs.

Topple

Symptoms. A glassy, watersoaked region appears on the flower stalk, usually when the bloom is nearly fully developed. It may arise at any level except just above the bulb, where there is always a healthy piece of stem. In Holland Pinkhof (1929) found the affected part usually in the upper, slender half of the stem. In Denmark we have often found it to occur just above the lowest leaf on the stalk, and Moore (1939) has made a similar observation in England. After a time the affected area extends to an inch or so and drops of sugary liquid exude from small cracks in the skin, or raise it to form small sap-blisters. Then the watersoaked part shrivels and the flower falls over at that point (Fig. 327). This wet form of the disease is prevalent in early double varieties like Murillo and Therose.

In Darwin tulips (Le Notre, Sanders, Pride of Haarlem, etc.) only a short piece of stem is usually affected and it shrivels and falls over without any preliminary glassy watersoaked stage. Other varieties particularly prone to topple are Rose Grisdelin, La Précieuse, L'Immaculée and Tournesol.

Cause. Topple is almost exclusively a disease of tulips forced under glass. Varieties may show great differences in their tendency to topple, for under almost identical growing conditions one will have none and another almost 100% affected. There may also be great differences between consignments of the same variety grown under the same conditions and between parts of the same consignment grown under different conditions.

We have, therefore, to differentiate between a predisposition on the part of the bulb to develop topple and the cultural conditions during forcing which allow this predisposition

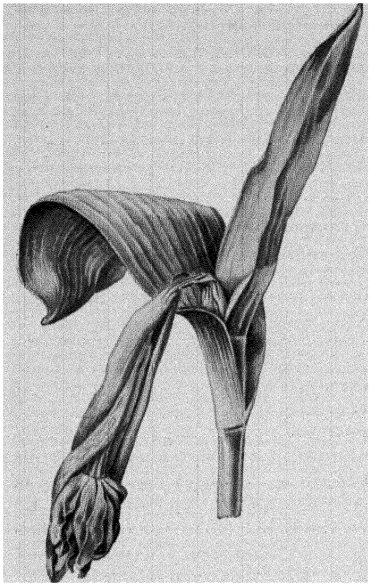


Fig. 327. Tulip topple.

to express itself and actually produce the disease.

Pinkhof concluded that topple was due to the living cells at a particular place on a tulip stalk becoming charged with sugar solution and that in forced bulbs this happens prematurely when the tissues are unable to cope with it. Uyldert (1933-4) found that cut tulip stems bearing flower buds developed topple if they were placed in sugar solution but not if calcium salts were added to the solution.

According to Pinkhof any factor which disturbs normal development of the young flower bud predisposes the plant to topple. She found no difference between tulips grown on sand and clay soils. Damage to the root system, time of planting and temperature before forcing all have an effect. In some varieties low temperature storage to " prepare " the bulbs for forcing markedly increases the amount of topple (Beijer and van Slogteren 1930).

If environmental conditions are the same more plants topple in January than in March. In practice many more cases are seen in March than in January but that is because of the usually higher temperature in March. By the middle of April topple ceases altogether, even in bulbs grown under conditions which earlier in the year would have given rise to it. Thus, when tulips are allowed to develop at the normal rate they do not develop topple, which is evidently due to a disturbance in the normal rate of development.

In North America a similar falling over of the blooms occurs in outdoor tulips but is due to rotting of the stems by *Phytophthora cactorum* (Leb. & Cohn) Schroet., especially in wet weather.

From the grower's standpoint it is important to know what are the conditions during forcing which bring out the tendency to topple. Hammarlund (1915) in Sweden concluded the disease was due to excessive atmospheric humidity in the forcing houses. Pinkhof, however, found that high temperature has a greater effect than high humidity.

High atmospheric humidity and high temperature often occur together in the forcing houses because much watering is often done in warm weather and because then the night temperature is often much lower than the day temperature, leading to high relative humidity at night.

Pinkhof's conclusions were (1) that variations in temperature were only important when they raised the mean temperature, (2) that ventilation was useful because it reduced temperature and humidity and (3) that light had no direct influence on topple but that sunshine was injurious by raising the temperature.

Control. The temperature in the forcing houses for varieties prone to topple should be kept as low as possible. Atmospheric humidity should be no higher than is absolutely necessary and shade should be provided when required.

Experiments have shown that topple can be effectively controlled by adding 2 oz.

calcium nitrate per gallon to the water used for watering the forcing boxes (*Gartnertidende* liii, p. 287, 1937; liv, p. 180, 1938) but the solution may damage the leaves and flowers of some varieties (Wenzl, H., 1938).

Glassy Buds

Symptoms. The buds remain down among the leaves which are about 6 inches high and sometimes fail to open. They have a peculiar glassy or watery look which, however, may disappear, leaving the buds apparently uninjured.

Cause. The plants look frozen but as the condition appears in tulips that have been in the warm ever since they were brought indoors this cannot be the case.

We consulted Van Slogteren about the matter who replied as follows in March 1929: " Your tulip, Therose, shows a condition well known to us in forced and outdoor bulbs. They give the impression of being glassy and full of water. The plants are backward in development but in the field they usually recover. The root system is often good and we have found no explanation of the phenomenon which, in my opinion, is of a physiological nature. As a rule the plants are already affected when taken into the houses."

We have seen houses in which the tulips suddenly become glassy and in a couple of days returned to normal again.

As we do not know the cause of the trouble we can suggest no means of preventing it.

Green-tipped Flowers

Symptoms. The perianth leaves have green tips.

Cause and development. This tiresome phenomenon may be due to various cultural faults. Usually the flowers are green-tipped because the bulbs for forcing were planted too early. The time to plant depends on the time they are to be forced; bulbs to be forced early should be planted early. The condition is particularly widespread in years in which the bulbs did not mature well so that when planted at the normal time they are naturally not ready for forcing. It has also been stated that tulips forced too late may also become green-tipped. This can be prevented by setting the bulbs that are to be forced late later than those destined for early forcing.

Too much warmth in late summer may cause it. If the boxes stand too long on the cold soil after they have been brought in the top of the plant becomes active but not the roots, and this may cause the flowers to become green-tipped. It is therefore useful to raise the boxes above the soil, as on boards, so that air can circulate beneath them, which will help root development.

Dryness during forcing may cause green-tipped flowers.

Some Danish bulb growers have told us that Couleur de Cardinal should have heat as soon as it is brought into the house or it will be green-tipped.

Sometimes just below the tulip flower there is a leaf which is partly green and partly coloured like the flower. In form it somewhat resembles a perianth leaf and the flower is often malformed. This is the uppermost stem leaf which has happened to develop too high up. The fault appears to be inherited, but variation in the direction of malformation in the top stem leaf is particularly frequent in Bartigon and also in other Darwin tulips such as William Copland. It is particularly prevalent when big bulbs are used.

Irregular Perianth Leaves

Tulip bulbs subjected to cool storage from the time of lifting until setting, to make them suitable for early forcing, may produce flowers in which the floral leaves have a more or less indented margin, indicating that the flowers were incompletely developed.

Suffocated Bulbs

The outer bulb scales become black and the inner ones a soft putrid mass. This complete destruction of the bulbs occurs if they are stored in very damp surroundings, for instance if there is standing water in the store. It has also been observed when they are stored where a load of manure has lain for a time or if soil has been tramped over the bulb boxes. The

decayed bulbs will then be found under the trampled places. A thick layer of stiff covering soil may also be injurious, especially if the bulbs are set in a closely packed heavy soil.

If necessary the bulb stores should be drained and the boxes may eventually be stood on slats.

Heating of Bulbs

Symptoms. The bulbs appear quite normal outside but when forcing starts the shoots are found to be only an inch long when they should be much more forward. When the bulbs are cut through the base of the shoot within the bulb is found to be yellow-brown, one or two of the scales next to it are discoloured and as a rule the shoot itself is also discoloured on the outside. The shoot is often quite healthy, however, except for the flower bud itself, which is shrivelled; in other cases the whole shoot is shrivelled and the bulb scales may be so dried up that there are air spaces between them (Fig. 328).



Fig. 328. Tulip bulb damaged by heating (*Photo v. Slogteren*).

At a later stage of forcing the leaves usually develop small dry edges or streaks and there are often brown-edged flaws in the margin or on the surface of the blade. The flower stalks are abnormally short in relation to the leaves and the flower buds are completely dried up and very small, scarcely $\frac{1}{2}$ inch long (Fig. 329). In some cases the flowers may be fairly big but then the perianth leaves are misshapen (Fig. 330). No fungus, bacterium or insect that might cause the damage can be found in the affected bulbs.

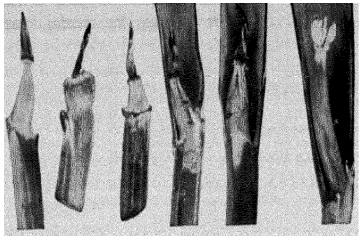


Fig. 329. Tulip buds killed by heating (*Photo v. Slogteren*).

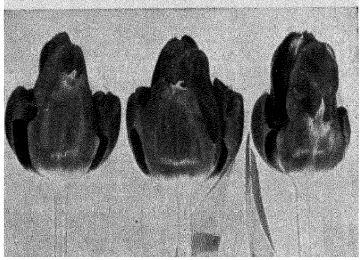


Fig. 330. Malformed tulip flowers, variety Fred Moore, from bulbs damaged by heating (*Photo v. Slogteren*).

Cause. In 1928 we received many tulips with these symptoms. Van Slogteren thought they were to be ascribed to heating. This most frequently occurs during transit but it may also arise when the bulbs are too warm in the pits. It cannot be too strongly emphasized that all bulbs should be kept cool from the time they are received until they are planted. Preferably they should be planted at once.

Control. As the damage to the bulb is done before it is planted the grower can do nothing to mitigate the loss. Comparison with the behaviour of bulbs from the same consignment at other nurseries will help to form an opinion whether the heating took place in transit or after delivery at his own premises.

Torn and Perforated Leaves

Leaves and leaf stalks are completely perforated by longitudinal slits, particularly in the lower part. The condition is seen only

in outdoor tulips and seems to be due to frost during winter or night frosts later. The affected leaves are easily attacked by grey mould and should therefore be removed. Tulip beds should be protected against severe frost.

Doubled Shoots

In tulips one often sees a condition in which the tip of the shoot remains bent down and caught in the bulb. The shoot is therefore bent double. Such bulbs often sprout late, and if they are cut through before shooting the shoot may be found doubled up, with its tip curved round to lie beside its base (Fig. 331). The reason for this is not known but

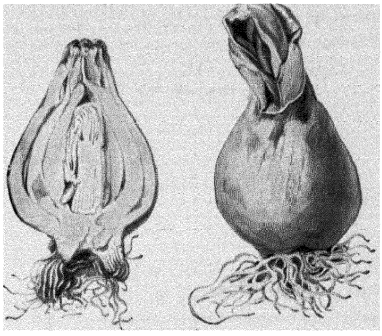


Fig. 331. Tulip bulbs with abnormal shoots.

possibly dry bulb scales may be a contributory cause. This may be the result of dryness during storage or earlier. It seems to be worst in naked or large bulbs. One should liberate the tip of the shoot from the bulb by hand, for if it remains caught back too long leaves and flowers will both be malformed.

Hard Base

This is a malformation of the bulb in which the base is smooth and hemispherical instead of having the normal ring-like ridge from which the roots arise. When bulbs of this type are planted the roots grow upwards through the space inside the outermost fleshy scale and usually fail to escape into the soil

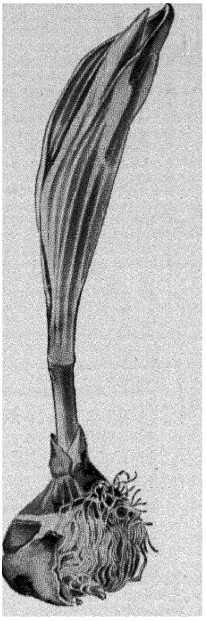


Fig. 332. Tulip hard base.

until they reach the nose of the bulb (Fig. 332). Moore (1939) believes that, at least in some varieties, the hard base is due to the mother bulb having been planted upside down.

Another factor contributing to failure of the roots to grow out normally may be lifting the bulbs before they are ripe, so that the outer brown scale remains unbroken. It may also be due to damaging the base of the bulb or to lack of air, as when the bulbs are pressed too hard into firm earth or when the storage pits are dug in soil that is too wet and heavy.

Bulbs with the hemispherical hard base can be recognized at planting and in them the outermost fleshy scale should be cut with the point of a knife just above the rooting base. Storage pits should be well drained and the boxes placed on slats. If cement benches are used the boxes should stand on boards so that air may circulate beneath them.

Summary of Tulip Diseases

I. Bulb and Shoots *page*

A. Abnormal sprouting without decay:

(1) Tip of shoot adheres to bulb ..... Doubled Shoot 551

(2) Shoot short, flower bud dried up, inner bulb scales yellow-brown ..... Heating 550

(3) Roots growing upwards below the outer scale .. Hard Base 551

B. Decay of bulb or shoot:

(1) Outer bulb scales black, inner grey and putrid .. Suffocated Bulbs 549

(2) Bulb scales grey-brown-chalky white, sometimes a little grey-green mould, shoot generally normal ..... Chalking 545

(3) Bulb scales normal, base of shoot rotten ..... Shanking 541

C. Bulb scales and shoots attacked:

(1) Black sclerotia present. Shoot decays early .... Fire 538

(2) White, yellowish-brown or black sclerotia, bulb scales pinkish grey, later decayed, shoots spotted brown or rotten ..... Grey Bulb Rot 542

(3) No sclerotia, bulb scales greyish with dark-grey or brown zone against healthy tissue, shoot small, rotten at base, roots decayed ..... Root Rot 541

II. Stalks and Bulbs

A. Weak or altogether lacking:

(1) Nothing appears above ground ..... See Grey Bulb Rot ... 542

(2) Small leaves, stem rotten at the base ..... Shanking 541

(3) Tip of shoot attached to bulb ..... Doubled Shoot 551

B. Yellowish-brown spots or streaks:

(1) Oblong small spots or streaks ..... Small Spots 544

(2) Larger streaks and brown leaf edges ..... Heating 550

(3) Larger irregular dead spots or small yellowish-grey leaf spots with red edges and often grey mould at the centre. Stalks often spotted, perhaps cracked or decayed with small black sclerotia ..... Fire 538

C. No dead spots, leaf stalks fall over at a glassy shrivelled place ..... Topple 547

D. Leaves wilt, leaf stalk decayed with small black sclerotia ..... Smoulder 544

E. Small pale spots on leaf ..... Small Spots 544

F. Leaves mottled with lighter and darker green ..... Breaking 546

G. Longitudinal slits in leaves ..... Torn Leaves 550

H. Leaves glassy, dark green, frosted looking ..... Glassy Shoots 549

III. Flowers

		page
A. Flowers missing:		
(1) No shoot appears above ground	See Grey Bulb Rot	542
(2) Whitish, shrivelled bud on a short stalk	Blindness	546
(3) Glassy watersoaked buds hidden among leaves	Glassy Bud	549
(4) Flower bud dried up, very small	Heating	550
(5) Bud weak or wanting, stalk decayed at base	Shanking	541
B. Flowers spotted or speckled:		
(1) Small pale spots with darker edges or whole flower decays	Fire	538
(2) Small spots on edge of perianth leaves	Small Spots	544
(3) Light or dark streaks and blotches in flower colour	Breaking	546
C. Flowers with perianth leaves green-tipped or one wholly green	Green-tipped Flowers	549
D. Perianth leaves indented or lobed	Irregular Perianth Leaves	549 550
E. Flowers fall over:		
(1) Glassy or shrivelled place on stalk, flower normal	Topple	547
(2) Bud or flower rotten, plant spotted all over, stalk possibly cracked	Fire	538

TURF

Brown Patch

In mown turf, especially on golf courses and bowling greens, dead patches quite often appear. They have been studied particularly in England, Holland and North America. Two main types may be distinguished: large irregular brown patches that may be a yard or more across, and small circular spots an inch or so in diameter, popularly known in America as " dollar spots."

The large patches are caused by one of the two related fungi *Corticium solani* (see p. 68) or *Corticium fuciforme* (Berk.) Wakef., which forms tiny red hair-like bodies projecting from the dead leaves. The small spots are caused by *Fusarium nivale* or, less often, by *Sclerotinia homoeocarpa* F. T. Bennet.

In countries with a heavy snowfall *F. nivale* is particularly destructive, not only to turf but to autumn-sown cereals, on which it causes the disease known as snow mould. Large areas of grass or cereal are killed and become covered with the white mycelium of the fungus and its pink spore pustules (Fig. 333). The condition seldom arises in Britain, but if snow lies long on fine turf it may be as well to clear it artificially.

In Holland Van Luijk (1934) has isolated from dead patches in turf species of *Pythium*, *Fusarium*, *Helminthosporium* and *Sclerotium*. By inoculation experiments with pure cultures he has shown that *Pythium* species attack especially grass seedlings or perhaps prevent

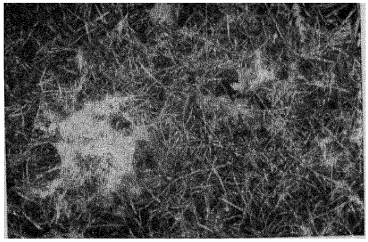


Fig. 333. Snow mould mycelium on turf (*S. p. F.*).

the seed germinating; the other fungi attack established turf.

His experiments were not carried out under natural conditions of growth.

We have obtained excellent control of the large brown patch by watering with a solution of 2 oz. corrosive sublimate and 18 oz. Chile saltpetre in 22 gallons of water, applied at the rate of about 1 gallon to 2 square yards. A similar solution should be effective against dollar spot. There are also a number of proprietary fungicides sold for this purpose, most of which contain an organo-mercury fungicide. These are usually effective if used according to the makers' directions.

Covering the patches with a thin layer of good sterilized compost encourages new growth and helps new sown grass to become established. It may be well to apply the compost before watering with the fungicide.

Watering of greens should be done in the morning so that the excess water may dry during the day.

Fairy Rings

On greens, lawns and old turf of all kinds more or less regular rings of darker grass may appear, that grow larger year by year. Fungi, especially toadstools or puffballs, may appear in the rings in autumn. Next season, usually about May, a ring of dead grass appears on the outer edge of the old ring. Then during the summer the site of the old ring becomes covered with darker more vigorous grass, growing more strongly because the fungus has liberated nutrients from organic matter in the soil and made them available to the grass roots. On the outer side of the ring of dead or dying grass the grass may also be unusually green. Hence a typical fairy ring is thus a compound structure of three adjacent concentric zones. Very large rings are usually not continuous but have gaps where the grass looks normal.

The soil beneath the ring is permeated by fungus mycelium though there is none in the soil within the ring.

Various fungi may cause rings in turf. The best known, often called the fairy ring mushroom, is the small, rather tough, cream-coloured *Marasmius oreades* Fr. Common in old pasture are the St. George's mushroom (*Tricholoma gambosum* Fr.) and the Blewit (*T. personatum*). Less common but often more destructive to the turf is the very large, short-stalked, white toadstool *Clitocybe gigantea* Fr. Kolderup Rosenvinge found a ring of this fungus was about 21 years old and increased in diameter about 1½ yards each year. We have found annual increases in diameter of 5 feet in a ring of *M. oreades*, nearly 8 feet in one of *T. gambosum*, and 4 feet in one of *T. personatum*.

Fairy rings show up most on poor, exhausted soils so that liberal manuring may make them less noticeable. Watering with one of the fungicides used against brown patch may be tried, care being taken especially to soak the soil on the outer edge of the ring. A great quantity of liquid would be required, for we have found the topmost foot or more of soil may be densely packed with mycelium. Hence the soil must be well wetted with the fungicide to a depth of at least 18 inches.

Rusts

Turf grasses may be attacked by several species of rust fungus. The most important are crown rust (*Puccinia coronata* Corda), specialised races of which attack rye grass, bent, other common pasture grasses or oats; *Puccinia festucae* Plowr., found on the fine-leaved fescues; *P. poarum* Niels, which attacks the meadow grasses (*Poa*); *Uredo festucae* DC. on fescue; and many others. Crown rust forms elliptical orange-coloured pustules of uredospores, especially on the upper side of the grass leaves during the summer months. Later black pustules of teleutospores form on the under-side of the leaves, often in rings round the uredo pustules. The cluster cup stage (aecidium) of this rust is common on *Rhamnus cathartica* (buckthorn) especially in the South of England (Fig. 39).

It is doubtful if the buckthorn is necessary for the rust as the uredo stage probably survives the winter. In any case once the rust has passed from buckthorn to rye grass, or

its other grass hosts, uredospores are produced in such vast numbers that in favourable years the disease spreads far from the original focus of infection. In Denmark, however, by the law of 27th March, 1933, one can demand eradication of buckthorn bushes growing within 600 ft. of one's own ground. Dietz (1923) has shown that the cluster cup spores can cause infection a mile and a half away in the course of 7 days. The disease was severe near the source of infection, becoming less and less noticeable with distance from the buckthorn bush. But as soon as the first generation of uredospores matured it spread very rapidly. Thirteen days later it had spread over 50 miles from the source of infection.

Puccinia poarum on meadow grasses causes pustules like those of crown rust but rounder. Its cluster-cup stage is very common from June onwards on the leaves of coltsfoot, on which it forms very conspicuous orange spots. Coltsfoot should be eradicated from the vicinity of turf containing species of *Poa*.

The finer-leaved fescues are attacked by two rusts. *Uredo festucae* causes very conspicuous yellow bands on the leaf surrounding each pustule of uredospores. Its teleutospores and alternate host if any are unknown. *Puccinia festucae* has its cluster-cup stage on honeysuckle but is comparatively rare.

ULMUS (Elm)

Coral Spot (*Nectria cinnabarina* (Tode) Fr. = *Tubercularia vulgaris* Tode)

Symptoms. The branches die back and cinnabar or blood-red fructifications burst through the dead bark. The wood of diseased branches may first become dark green or black and afterwards brown.

Cause and development. The fungus colonizes dead twigs or wounds and spreads thence into the adjacent healthy branch which it kills. It advances so quickly that no callus forms round the point of infection as usually happens with the canker fungus *Nectria galligena*, a close ally of the coral spot fungus. The fungus can also penetrate the lenticels of dead or injured bark but this is probably exceptional.

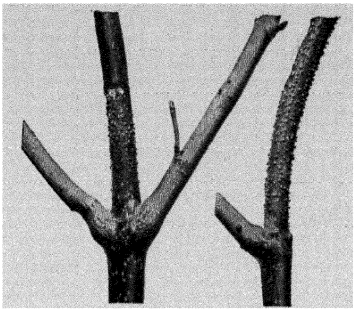


Fig. 334. Conidial pustules of coral spot fungus on twigs.

On the surface of branches it has killed, the fungus first produces swarms of light-red to brick-red pustules of conidia, each from $\frac{1}{25}$–$\frac{1}{10}$ inch across (Fig. 334). These are covered with elliptical one-celled colourless conidia and were formerly called *Tubercularia vulgaris*. Later on, small clusters of perithecia appear on and among the conidial pustules. They have the deep-red colour of the mercury ore cinnabar after which the fungus is named.

Importance. The fungus is ubiquitous on dead twigs and branches and is common as a wound parasite of many trees and bushes. In Denmark we have frequently observed it on elm, especially on trees that have been cut back or otherwise wounded in streets and beside roads, also on lime, sycamore, maple, horsechestnut, ash, apple, currant, etc.

Various physiological races exist with different degrees of virulence.

Its presence is an indication that something is wrong with the tree or bush attacked; either the branches have not matured properly, or the fungus has gained entrance through unprotected wounds, cankers or twigs that died from some other cause.

Prevention. Good normal growth with thorough ripening of the wood and protection of pruning wounds and others are the chief preventive measures. It may be necessary to keep an eye on large wounds for several years, renewing the covering material as it

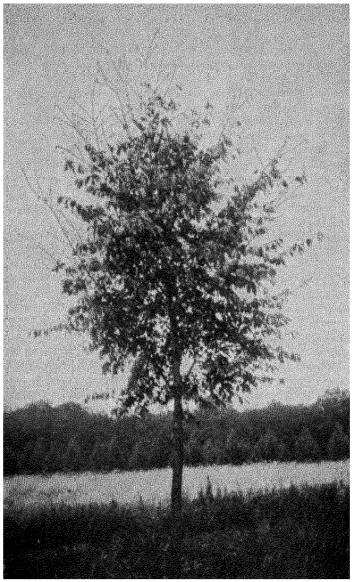


Fig. 335. Elm tree dying of Dutch elm disease (*Photo Westerdijk*).

becomes cracked or flakes off. When penetration by the fungus has occurred the affected branches should be cut right back to healthy wood and the cut trimmed and coated with a protective paint (see canker, p. 115).

Dutch Elm Disease (*Ophiostoma ulmi* (Buism.) Nannf. = *Ceratostomella ulmi* Buism. = *Graphium ulmi* Schwarz)

Symptoms. This disease was first studied in Holland. There symptoms appear at the beginning of June. The elm leaves on some branches suddenly wilt and in a few days dry up so that the trees look as though scorched by fire and the dead leaves fall off (Fig. 335). The branches die back from the tip; one or two leaves may hang on at the tip of each shoot while all the rest fall (Fig. 336).



Fig. 336. Elm tree inoculated with Dutch elm disease (*Photo Buisman*).

Later in the summer whole trees may die suddenly. If a tree or some of its branches lose its leaves from this disease early in the season the bare twigs may produce new leaves during the summer but they will be few and small. The dormant buds on trunks and main branches of diseased trees often break, covering them with a mass of small green shoots while all the rest of the tree is bare. In other cases these shoots wither soon after they appear.

As well as trees with dead and leafless crowns one may see others during the summer which have thin foliage, with some branches quite bare, and have a generally sickly appearance. In the following autumn some of these die from the elm disease. The bark is loose and the wood dead. However, all the trees whose leaves wilted in the summer do not die that year. They may survive for several years but an infected tree seldom recovers completely.

The wood of infected branches has a characteristic brown discolouration which appears as brown dots in a cross-section and as brown stripes in a longitudinal section (Fig. 337). In elm disease these streaks in the wood are brown, interrupted, not sharply

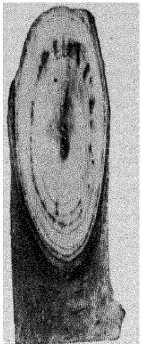


Fig. 337. Dutch elm disease, brown spots in the wood (*Photo Buisman*).

defined, and the surrounding wood develops a reddish-purple tint. It must be remembered that similar brown colouring occurs in the wood of trees attacked by *Verticillium alboatrum* and by a bacterium, *Pseudomonas lignicola* Buism. It is curious that this bacterium may be present in large numbers and cause long streaks in the branches apparently without harming the trees. Its presence may lead to an erroneous diagnosis as Dutch elm disease, and confusion of this kind is probably responsible for the opinion that the disease is comparatively harmless.

Cause and development. Schwarz, who first in 1922 discovered the fungus causing Dutch elm disease, found only the conidial stage and called it *Graphium ulmi*. Buisman in 1932 proved that there was an ascus stage with perithecia which she called *Ceratostomella ulmi*.

Fransen (1937, 1939) and others have found various insects spread the disease, especially the large and small elm bark beetles, *Scolytus scolytus* and *S. multistriatus*. The females lay their eggs in the bark of diseased elms and the larvae gnaw tunnels partly in the wood, partly in the bark. Pupation takes place in these tunnels. The mature beetles that leave the tunnels may easily be heavily contaminated with spores of the fungus, for it produces conidia and perithecia freely in them. The fungus has been isolated from intestines of the beetles. When the bark beetles have matured they fly to the top of healthy trees where they eat the bark of young shoots and twigs, infecting them with the fungus which grows into the wood. It is thus easy to understand why the disease begins in the top of the tree. Once introduced into a tree the fungus spreads very quickly.

Importance. Elm disease was first described in Holland by Spierenburg, who thought it was first seen in 1918; what was probably the same disease was seen in France also in 1918 according to Guyot. In Germany Tubeuf recorded dead elm branches in 1918 and 1920. Since then the disease has spread tremendously in West, South and Central Europe (Fig. 338)

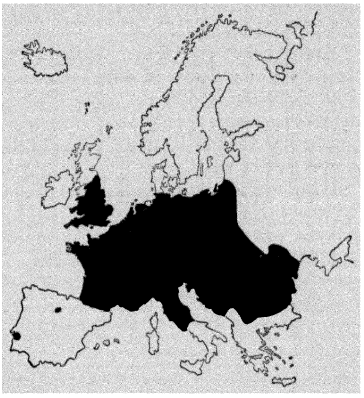


Fig. 338. Distribution of Dutch elm disease in Europe, 1936 (after Broekhuizen).

and has become well established in the North-eastern United States. Denmark has luckily been spared so far, but the disease occurs in Schleswig Holstein spreading gradually farther north, so that importation of elms into Denmark is forbidden. In England it is prevalent in the South, less so in the North and is unknown in Scotland.

It is mostly trees between 10 and 40 years old which are attacked.

The destructive character of the disease may be illustrated by data from Holland. In

Rotterdam there were 30,000 trees in 1920 of which 24,000 were elm; by 1932, 11,000-12,000 had been destroyed on account of the disease. Utrecht had 7,000 elms in 1920, but by 1932 half of them were gone and in the following winter a further 7% had to go.

Control. Attempts at control can follow two courses:

(1) Destruction of the bark bettles which spread the disease; and

(2) Attempts to find and propagate species, varieties or individual elms which are resistant to the disease.

Dead trees should be cut down as soon as possible, and if they are too big to burn, the bark beetles in them should be destroyed and prevented from laying eggs in the bark. Egg laying may be hindered by smearing the felled tree with Carbolineum.

Bark of felled diseased trees should at once be removed and burnt. Coating the outside with Carbolineum does not check development of the beetles already there. If the trunk can be immersed in water for 3 months the beetles will be killed.

It has been found that some species of elm, especially those from Eastern Asia, are not attacked by Dutch elm disease, and the horticultural value of these and resistance of other species is under trial in Holland. As infection occurs through the crown one can graft a resistant elm on to a susceptible one and thus escape the disease.

The following Eastern Asiatic elms are resistant or only slightly susceptible: *Ulmus pumila L.*, *U. pumila* var *pinnatoramosa* Henry, *U. Wilsoniana* Schneider, *U. macrocarpa* Hance, *U. parvifolia* Jacquin. The following varieties are less susceptible than those commonly planted: *U. foliacea* Gilibert var. *dampieri* Rehder (= *U. campestris dampieri* Späth) and *U. glabra* Hudson var *fastigiata* Rehder (= *U. plumosa pyramidalis* Hort.). *U. macrocarpa* is sensitive to frost.

Dieback

Various fungi may cause a dieback of elm branches which may sometimes be confused with Dutch elm disease. Amongst these are *Verticillium albo-atrum* (see below) and coral spot (p. 555). In Holland Buisman found a species of *Diaporthe* on young elm branches which did not leaf or bore only small leaves which soon died. The leaves might also wither later in the summer but usually only slowly after turning rose-red in colour; this withering occurred only on shaded branches. Conidial fructifications of the *Phomopsis* type appeared, and when they were grown in pure culture the perithecial stage, a species of *Diaporthe*, appeared. Infection experiments with this fungus gave inconclusive results. In general species of *Diaporthe* are only feeble wound parasites at most. A *Phomopsis* also occurs on elm branches in Denmark (Lind, 1913). In North America a virulent dieback of elms is caused by *Dothiorella ulmi* (Verrall and May, 1937). Death of elm branches may not necessarily be due to fungus invasion of the branches themselves but may be the result of unfavourable growing conditions, such as covering or compacting the soil surface by road operations, or of invasion of the trunk by bracket fungi. Wilkins (1939) has shown that *Ustulina vulgaris* may also attack elm (see Tilia, p. 537).

Sometimes after a very abundant flowering and fruiting an elm has appeared so much weakened that presence of the elm disease has been suspected.

Verticillium Wilt (*Verticillium albo-atrum* (Reinke & Berth.))

Symptoms of *Verticillium* wilt in elm may closely resemble those of Dutch elm disease. The latter occurs mainly in trees 10-40 years old, whereas *Verticillium* mostly attacks young elms.

The tip leaves of diseased trees begin to hang limply, then dry up and the damage gradually spreads to the lower leaves. In the wood is a brown discolouration which may be difficult to distinguish from that of Dutch elm disease but which is usually more evenly distributed. It may be found even in the wood of the smaller twigs. Young infected elms may be stunted in growth and gradually lose their leaves.

The disease was found in Denmark in 1928 on a 5-year-old elm and in 1936 on many others of the same age. Those worst affected were cut down and in 1937 the remainder had apparently recovered.

Verticillium usually enters the tree through the roots and may appear, for example, when elms are planted on soil which formerly grew potatoes (see p. 60).

Mycosphaerella ulmi Kleb. = *Phleospora ulmi* (Fr.) Wallr.

This fungus forms small white conidial pustules on the under-side of elm leaves which then wither early. No control has been attempted but presumably a fungicidal spray or dust would be beneficial (Klebahn, 1905).

VALERIANA (Valerian)

Mildew (*Erysiphe cichoracearum* DC.)

A powdery white coating appears on the upper surface of the leaves (see mildew, p. 63).

Rust (*Uromyces valerianae* Fuck.)

All stages, aecidia, uredo- and teleutospores, occur on the same plant, the first in spring, the others in summer and autumn. The aecidia occur as groups of " cluster cups " on pale thickened spots on leaves, petioles and stems. Uredospores form tiny brown pustules scattered over vague yellowish spots on both sides of the leaves; the teleutospores are similar but darker brown (see rust, p. 74).

VERBENA

Mildew (*Oidium verbenae* Thüm. & Boll.)

We have seen *Verbena* leaves and stems quite white with this mildew (see mildew, p. 63).

VERONICA

Mildew (*Sphaerotheca fuliginea* (Schlecht.) Lind)

This mildew occurs occasionally on wild speedwells such as *Veronica chamaedrys* and *V. spicata*, also on cultivated *V. andersonii* and *V. myrtifolia*. On the latter it may attack leaves and shoots so severely that the youngest shrivel (Buchwald, N. F., 1936b). Infection seems most prevalent in winter. The plants should be grown in the most favourable situation and should be sprayed with a fungicide, preferably one which will not spot the leaves. Mildew has been seen in England on *V. longiflora subsessilis*.

Downy Mildew (*Peronospora grisea* Unger)

Downy mildew is common on wild speedwell and the brooklime *V. beccabunga*, on which it causes a conspicuous yellowing of the infected leaves. Their lower surface is covered with a close grey mould of conidiophores and conidia. The disease has been observed on cultivated shrubby veronicas in the South of England.

Leaf Spot (*Septoria exotica* Speg.)

Round greyish or brown spots with purplish edges appear on both sides of the leaves in cultivated shrubby veronicas. Scattered over them are dark brown dots, the pycnidia of *Septoria exotica* Speg.

The disease is not uncommon in the South of England but there seems to be no known control.

VINCA (Periwinkle)

Rust (*Puccinia vincae* Berk.)

This is rather a rare rust but once a periwinkle bed has become infected the disease appears in it year after year, as the fungus apparently overwinters as mycelium within the plants. There is no alternate host. Uredo- and teleutospore pustules are very similar and occur in swarms on the under-side of the leaves. They are very often attacked by the hyperparasite *Tuberculina persicina*, which forms its own spore layer like a mauve bloom over the rust pustules. The only control is to root up and destroy all infected plants.

VIOLA (Pansy, Violet)

Smut (*Tuburcinia violae* (Sow.) Liro = *Urocystis violae* (Sow.) Fisch. v. Waldh.)

The fungus causes large, dark-purple, spindle-shaped swellings on the leaf stalks, often in a row. Occasionally they occur also

on leaf blades, stems, calyxes and capsules of sweet violet, pansy, *Viola cornuta* and other species. The swellings burst and a mass of black smut spores is shed from them.

The disease is not common in Denmark but it is reported periodically in Britain. Diseased plants should be destroyed as soon as noticed and cuttings should not be taken from infected plants or beds. Fresh healthy plants should be planted on clean soil well away from the infected beds.

Rust (*Puccinia violae* DC. & *P. aegra* Grove)

This disease has been abundant, at Lyngby and elsewhere, on sweet violet and *Viola cornuta* without attacking pansies in the vicinity. Some authors prefer to treat the pansy rust as a separate species, *P. aegra* Grove.

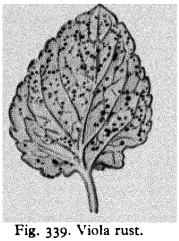


Fig. 339. Viola rust.

Pale-green spots appear on the leaves in spring, the under-side of which are covered with yellow cluster cups. They may also be found covering swollen parts of the veins, leaf stalks and stems. Cluster cups of *P. aegra* may be found as late as August, those of *P. violae* not usually after June. During the summer small light-brown pustules of uredospores appear on the under-side of leaves in *P. violae*, on both sides in *P. aegra*. In autumn come the darker-brown teleutospore pustules. Severely infected plants do not flower freely.

Control. In old-established plants remove as much as possible of the above-ground parts in autumn. Cuttings should not be taken from diseased plants. Fresh healthy stock should be planted in clean ground (see rust, p. 74).

Scab (*Sphaceloma violae* Mass. & Jenk.)

This disease is not yet known in Europe but it has been destructive to violets in the United States of America since 1925 and is now widespread there. It has been studied by Massey and Jenkins (1935).

Spots appear on the leaves between the veins. At first they are small and watersoaked and visible on the under-side only but soon become apparent on both sides of the leaf. They grow into grey or whitish scab-like circular spots about $\frac{1}{4}$ inch across, often with a dark-green margin. The adjacent normal green tissue often becomes much raised on one side and depressed on the other. A row of spots or a single elongated spot may spread along a vein for $\frac{1}{2}$ inch or so, causing it to become twisted and deformed. The spots may dry up and fall out, leaving a perforated leaf, and in severe attacks the tissue between the spots also becomes brown and withered.

Spots also occur on runners, leaf stalks and flower stalks, where they cause conspicuous swellings. On these organs they are elongated, about $\frac{1}{4}$ inch long and either raised or sunken. On calyxes and capsules the spots may be round or irregular and seldom attain more than $\frac{1}{18}$ inch diameter.

In America successful control has been obtained by spraying with Bordeaux mixture at intervals of 10–14 days.

Foot Rot

Both *Viola cornuta* and pansy may suffer from foot rot. The first sign of the disease is often a dull green or bluish hue about the leaves, sometimes with a reddish-purple or bronze tint. Soon afterwards a pale-coloured rot develops at the base of the stems, spreading both downwards to the underground parts and some distance up the stems. Diseased plants are easily pulled up by a slight tug.

The disease was specially prevalent in pansies in Denmark in the autumn of 1943, where pansies or other violas had been grown for some time on the same ground.

In Norway Solberg (1926) found *Pythium de baryanum*, *P. artotrogus* and a third *Pythium* sp. in rotting pansy roots. In Holland Van

Eek (1937) made a thorough study of pansy root rot. He found plants might suffer damping off in the seedling stage and might be infected with root rot before being planted out. The general symptom picture resembled that described above. From the infected plants he isolated 43 fungi and 6 from pansy seed. Of these the most virulent parasites were *Brevilegnia gracilis* Van Eek, *Pythium aphanidermatum* (Edson) Fitzpatrick, *P. de baryanum* Hesse, *P. perniciosum*, *Fusarium culmorum* and *Corticium solani*, i.e. the usual damping-off and foot-rot fungi which attack so many plants. As *F. culmorum* may be seed-borne he recommended seed disinfection by soaking it for 45 minutes in a $\frac{1}{5}$–$\frac{1}{3}$% solution of Danish Tillantin.

At Hamburg Pape (1942a) found the disease very prevalent and caused by *Pythium de baryanum*. Young pansy plants began to wither 2–3 weeks after being planted in soil infected with the *Pythium* and in another week or two they were dead. The soil was effectively disinfected by heating to 205–208° F. for 1 hour or by watering a layer 4 inches deep with 1% formalin solution at the rate of 2 gallons per square yard, covering for 2 days and then ventilating for 14 days before planting. Liming had no effect on the disease.

In England Chesters and Hickman (1944) found the commonest cause of viola foot rot was a new species of *Pythium* which they named *P. violae*. *P. oligandrum* Drechsler, *P. mamillatum* Meurs, *Corticium solani* (see p. 68) and *Myrothecium roridum* Tode ex Fr. (see p. 49) also sometimes caused the trouble.

Halo Blight (*Centrospora macrospora* (Osterw.) Neerg.)

The leaves develop small blackish-brown spots with light-green translucent edges. Plants may be much weakened by the disease. As the fungus forms a resting mycelium which may probably overwinter in the soil, pansy seed should only be sown in fresh or disinfected soil and the plants should be planted out on soil where there have been no pansies or violas for several years. The fungus is also apparently seed-borne, and seed disinfection with an organo-mercury preparation should therefore be tried. If the disease appears, spraying or dusting with a fungicide should be tried at once.

Leaf Spots (*Ramularia* spp.)

Several species of *Ramularia* have been described on leaf spots of *Viola* spp. Campbell (1938) has studied *R. deflectens* Bres. on the cultivated viola Kate Blyth. Dark-coloured spots spread inwards from the leaf edges and may eventually cover the whole blade. As they age a white mycelium appears on the surface and very tiny black dot-like microsclerotia arise in the dead leaf tissue. The fungus is a weak parasite which only attacks the viola in early spring. It is favoured by a cool, moist atmosphere.

More often reported is *R. agrestis* Sacc. on small, round, greenish, then yellowish, and ultimately white spots on pansy leaves and on the wild heartsease *V. tricolor*.

R. lactea is a species found on circular whitish or straw-coloured spots with a brown edge on violet leaves (*V. odorata* and the various wild " dog violets "). Under damp conditions the spots are covered with tiny white dots, formed of clusters of short conidiophores and conidia. The spots are seldom as much as $\frac{1}{4}$ inch across, and as they age the brown edge disappears and the spot becomes thin and dry. It may fall out leaving a hole in the leaf (Fig. 340). The presence of tufts of conidiophores helps to distinguish *Ramularia* spots from *Centrospora* blight (see above).

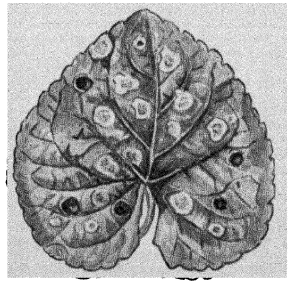


Fig. 340. Violet leaf with *Ramularia* leaf spot.

Control. One may try spraying or dusting with a fungicide or perhaps removing the diseased leaves. In old plants complete cutting back to ground level is advisable when the attack is severe, followed by a fungicidal spray on the young new shoots. As the fungi may be seed-borne it is well to shake up viola and pansy seed with a seed disinfectant before sowing.

Black Root Rot (*Thielaviopsis basicola* (Berk. & Br.) Ferraris)

Viola roots become brown and rotten and development of the plants is checked, they appear sickly and may die. The fungus *Thielaviopsis basicola* is to be found in the roots (see begonia, p. 422).

Soft Rot

In 1921 Lacey (1922) encountered a soft rot of violas in England caused by *Bacterium carotovorum* (L. R. Jones) Lehm (see p. 257). The whole interior of the stem was reduced to a soft mush and the rot was invading the petioles, causing the leaves to drop when their bases decayed. Inoculation experiments on healthy violas showed the rot to progress very rapidly once the organism gained entrance to the stems.

Colour Breaking (*Cucumis* Virus 1)

Violas and pansies infected with the virus of common cucumber mosaic (see p. 278) show a " clearing " or yellowing of the veins in the youngest leaves, best seen if they are held up to the light. Later a mild yellowish mottle develops on them and growth may be stunted. The most conspicuous symptom, however, is in the flowers. Pale streaks appear on the petals and gradually fade to white. Occasionally the whole flower may turn pale or white, and it is then often small and misshapen. The virus is spread by aphides. Control measures include destroying infected plants, careful inspection of freshly purchased plants or cuttings to detect the early leaf symptoms of infection, and regular nicotine sprays to keep down aphides (Severin, 1947).

VIRGINIA CREEPER

See *Parthenocissus*, p. 504.

WALLFLOWER

See *Cheiranthus*, p. 430.

WATER LILY

See *Nymphaea*, p. 499.

WILD VINE

See *Parthenocissus*, p. 504.

WILLOW

See *Salix*, p. 528.

WINTER ACONITE

See *Eranthus*, p. 455.

YUCCA

Leaf Spots (*Coniothyrium concentricum* (Desm.) Sacc.)

On the leaves are large round well-defined brown spots, greyish at the centre and with purple margins. Dark dot-like pycnidia gradually appear on them, often in concentric rings, whence the name of the fungus.

Remove the leaves most severely attacked and spray the plant with a copper fungicide.

ZANTEDESCHIA (Arum Lily)

Leaf Spots

Phyllosticta richardiae Brooks cause brown spots of various sizes on leaves, petioles and flower stalks and also on the white spathes. Spots on the green parts are often surrounded by a yellow zone (Fig. 341). Quite severe attacks were observed in Denmark in 1932 and 1935 but the fungus was probably there long before. It is quite common in Britain.

Cercospora richardiaecola Atk. causes round, dark-brown spots with white centres on arum lily leaves in North America, and *Gloeosporium callae* Oud. produces large yellow spots that ultimately turn brown. Neither is known in Denmark or Britain.

To control leaf spots one should remove the most severely affected leaves and then spray or dust the plant with a fungicide such as Bordeaux mixture. The house should be well heated and ventilated.

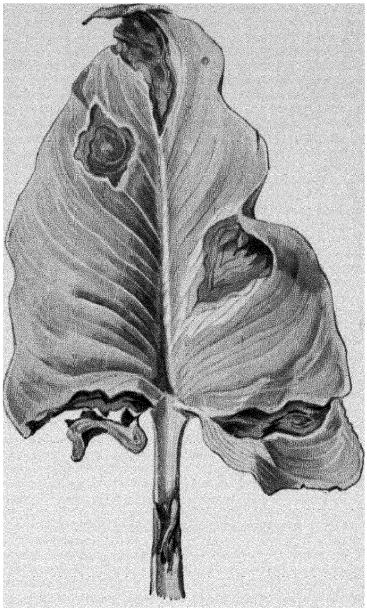


Fig. 341. Arum lily leaf attacked by *Phyllosticta richardiae*.

Root Rot (*Phytophthora cryptogea* Pethybr. & Laff. var. *richardiae* (Buism.) Ashby)

This disease was first described in Holland and is quite common in Britain but has yet to be found in Denmark.

Symptoms. When the plants begin to put out new leaves after their resting period the outermost turn yellow. The yellow discolouration spreads, the leaf stalk becomes limp, the leaf wilts, falls over and turns brown. After a time the next leaf follows suit. The plant makes no real growth but does not die outright, and at intervals new leaves are formed which turn yellow and die. The plant is dwarfed and the flowers small.

An affected arum lily is easily pulled up, for most of the roots are rotten, often reduced to hollow tubes of skin. The corms may also be attacked and partially decayed. Only a few roots remain on the pulled-up corm and these may be translucent, soft and glassy, sometimes with a reddish tint. The more completely decayed roots are left in the soil.

Control. Infected plants should preferably be destroyed. If it is necessary to try to save them the corms should be shaken free of soil after the resting period and the roots removed. Wash them in water with a stiff brush and scrape away all decayed parts. Then soak for 2 hours in a solution of formalin 1: 50 or corrosive sublimate 1: 1000. Repot in disinfected soil in new or disinfected pots.

It is also important to disinfect the soil surface or staging on which the plants are to stand. Glass, walls and staging of the house should be disinfected by burning sulphur or spraying with formalin.

Corm Rot (*Bacterium aroideae* (Towns.) Stapp)

This soft bacterial rot of arum lily corms is fairly common in Britain. The same symptoms may be caused by either of the two related bacteria *B. aroideae* or *B. carotovorum* (L. R. Jones) Lehm. The rot usually starts at the base of the stalks, near soil level, and spreads downwards into the corm. Finally the whole plant wilts, dies and is destroyed by a soft rot.

Control, as for root rot.

Virus Infection (*Lycopersicum* Virus 3)

Infection of arum lily with the virus of tomato spotted wilt was first demonstrated in England in March 1934 but it is now known to be widespread both there and in Denmark. Infected plants have twisted or curled leaves marked with whitish spots and streaks. There may also be streaks on leaf and flower stalks and spotting of the spathes, which may also be malformed (Ainsworth, G. C., 1935). As the season advances the plants usually appear to recover from the disease though the leaves may still bear whitish ring-like spots.

The virus is spread by thrips, which are fond of feeding on arum lily flowers.

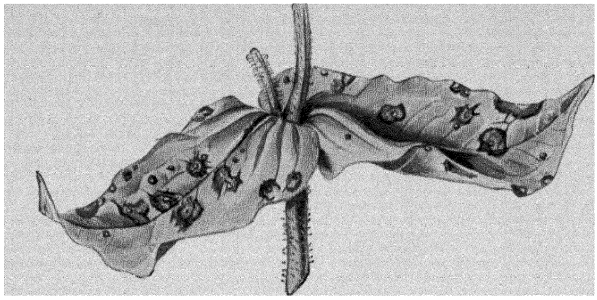


Fig. 342. Zinnia leaf spot.

ZINNIA

Leaf Spot (*Alternaria zinniae* Pape)

This disease was described by Pape (1942b) in Germany. Large, brown, somewhat angular spots appear on the leaves. Usually they are whitish at the centre and may join up to form big continuous brown blotches (Fig. 342). The attack tends to be most serious in autumn but may appear quite early on transplanted zinnias. There may also be brown spots on the flowers but these are less common.

The fungus is seed-borne and hence it is advisable to disinfect zinnia seed with an organo-mercury seed disinfectant even if the germinative capacity is thereby somewhat reduced. Soaking the seed for 3 hours in $\frac{1}{4}\%$ Germisan solution may be recommended.

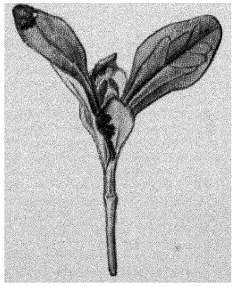


Fig. 343. Zinnia seedling attacked by *Alternaria*.

The plants should also be sprayed or dusted with a fungicide, preferably while still in boxes.

Damping off of zinnia seedlings associated with a species of *Alternaria*, apparently different from the above, has been recorded in England (Moore, W. C., 1943).

Grey Mould (*Botrytis cinerea* Fr.)

Especially under glass grey mould may cause rotting of zinnia flowers, which turn brown and gradually become covered by a coating of grey conidiophores and conidia. The houses should be well heated and ventilated (see further, p. 59).

Sclerotinia Rot (*Sclerotinia sclerotiorum* (Lib.) de Bary)

Zinnia is not infrequently attacked by *Sclerotinia*, the white mycelium of which destroys the stems and kills the plant. Large white sclerotia which gradually turn black occur in the mycelium (see p. 77).

Mosaic and Ring Spot

Zinnias are very commonly infected with the virus of tomato spotted wilt which causes the leaves to be mottled with numerous light and dark green spots or with small yellowish rings on a green background (see p. 384).

Infection with cucumber mosaic virus causes similar mottling and may also lead to dirty-grey or brown markings on the petals which are often twisted and distorted (see p. 278).

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V

CONTROL MEASURES

5 CONTROL MEASURES

I. ADMINISTRATIVE ACTION

In the realm of plant disease control administrative action takes two forms: quarantine regulations intended to exclude dangerous diseases not already in the country, and measures designed to improve the health of crops and to check the spread of diseases already present.

1. Quarantine Regulations

All civilized countries try to keep out infectious diseases that might become injurious to their people, stock or crops. Countries isolated by the ocean, mountain ranges or deserts find this comparatively easy. Neighbouring countries separated by no such natural boundaries cannot prevent the exchange of diseases spread by the wind or by insects. Thus in North America wheat rust spores are carried by wind from Texas far into Canada. In such cases the only limit to the spread of a disease is the drying up and death of spores after suspension for some time in the air. But, of course, a disease introduced into a new country by wind-blown spores will not become established there unless it finds conditions of temperature, moisture, etc., suitable for spore germination and growth of the fungus.

Parasites not spread by the wind may be carried long distances by trains, ships, motor cars and flying machines; at an inspection of a German zeppelin in U.S.A. seven insects and two parasitic fungi were found in three bunches of flowers. Importation of corn, fruit and animal foodstuffs offers obvious opportunities for the introduction of diseases affecting those crops.

Under reasonable supervision these dangers can be overcome; for example, many shiploads of potatoes have been sent from Europe to North America without introducing any dangerous parasites, but when workmen from Central Europe emigrating to Pennsylvania took their own seed potatoes with them they introduced potato wart disease into North America.

Not every "new disease" recorded in a country is due to importation from abroad. Often, in the case of a minor disease, it has been present from time immemorial but no one has bothered to record it before. Or a disease long present may suddenly attract attention because an unusually favourable combination of circumstances in a particular year has enabled it to spread. In such cases it may almost disappear again in the following year. Other new diseases are apparently due to a new race of some old fungus appearing which is either more virulent than the old or is able to attack a new plant. Again, when the acreage under a certain crop is suddenly increased, diseases always present on it have an opportunity to spread to an extent hitherto impossible and hence they attract notice for the first time. In the period 1928–37, 82 diseases due to bacteria, fungi or viruses were observed in Denmark which had not been seen there before. Only with 17 or 18 of these is there clear evidence that they were recent importations; some were even found on imported plants.

To frame reasonable quarantine regulations a knowledge of the biology and interrelationship of parasite and host is required. All civilized countries can contribute to the common fund of information about the behaviour of plant diseases though they do not all do so to the same degree. Even when the life history of a parasite and its apparent importance in

its native home are known, one cannot always foresee how it will behave when introduced into a new country. When the Weymouth pine was brought from North America to Europe it was attacked by the currant rust (see p. 162) and suffered from blister rust, a disease never before seen on Weymouth pine. When, later, young Weymouth pines were exported to America from European nurseries the fungus went with them, and its introduction into the United States has ruined many of the fine stands of 5-needled pines in that country.

Quarantine regulations have to be based on biological data. There is no point in enforcing quarantine against diseases already widespread in a country, and at the same time their economic consequences have to be considered. The primary reason for quarantine is to prevent loss within the country enforcing the regulations but it may also be employed to protect an export trade. Once an objectionable disease is introduced into a country, other countries in which the disease is not yet present are liable to exclude the first nation's exports of the crop concerned.

The disadvantages of quarantine are obvious; it restricts foreign trade, it is a nuisance to travellers bringing in plants from abroad and it is expensive to enforce. A country whose major exports are excluded may react by changing its agricultural production and may become a competitor in the sale of other products elsewhere.

Even if quarantine cannot keep out a dreaded parasite indefinitely its exclusion for even a few years may be worth while. In 1934, the first year the Colorado beetle entered Germany from France, 108,000 Rmk. were spent on attempts to control it and 100,000 Rmk. in the following year. Once a parasite has become established similar additional sums may have to be spent on an endeavour to breed crop varieties resistant to it.

Quarantine regulations may be of various degrees of severity:

(*a*) **Embargo,** usually accompanied by strict control of goods in transit; thus in Denmark potatoes in transit may not be unloaded on to the quay and reloaded but must be transferred directly from ship to ship without being landed at all.

(*b*) **Detention** of tubers, bulbs, nursery stock, etc. This is the original meaning of quarantine (= 40 days) and is widely practised with plants imported into U.S.A. They are grown at special quarantine stations until the authorities are satisfied they have not brought any dangerous parasites with them.

(*c*) **Disinfection** of the plant on arrival may be practised with imported seeds and various products such as cotton.

(*d*) **Inspection** on arrival is done in many civilized countries. Its value is greatly enhanced if the plant has already been inspected before it left the exporting country and has been issued with a health certificate. This may certify that a particular parasite is not known to occur in the country or district of origin, or that the crop from which the plant came, or the individual plant itself, has been inspected and been found to be free from injurious insects or diseases. Plants to be exported from Britain may be issued with a certificate of this kind by the Ministry of Agriculture or Department of Agriculture for Scotland after they have been inspected by an appropriate officer of the Ministry or Department.

Importation into England and Wales. The Importation of Plants Order 1947 is designed to prevent the introduction of pests and diseases into England and Wales from overseas. It applies to " all living plants and parts thereof (except seeds) for planting " as well as to potatoes, raw vegetables and certain raw apples. Its chief provision is that all plants, as defined, and all potatoes imported must be accompanied by official certificates of health. There are certain exceptions to the general rules. Thus the importation of sugar-beet and mangold plants is totally prohibited, to guard against the introduction of virus diseases, as also is the importation of potatoes from U.S.A., Canada and European France to guard against bacterial wilt and the viruses of spindle tuber and yellow dwarf.

Another order deals specifically with the importation of cherries, to prevent the

introduction of cherry fruit fly. The Importation of Elm Trees and Conifers Orders prohibit the importation of living plants of *Ulmus* and of eight named genera of the order *Pinaceae*.

After a new pest or disease has been introduced into England and Wales attempts to prevent its spread may be made under the provisions of the Destructive Insects and Pests Order 1933. Action under this order was taken against, for example, bacterial canker of tomato.

Control of spread of old-established diseases.

Regulations may be made to check the spread of serious diseases whose distribution in a country is still limited. They are most likely to be effective in the case of soil-borne diseases, which persist for long periods once soil has become infected, but which have few natural means of spread except on infected plants. A familiar example is potato wart disease (see p. 318). In Britain an attempt is made to check spread of certain other diseases by the Sale of Diseased Plants Orders 1927–43. These make it an offence to sell, offer or expose for sale, or, having sold, to deliver for planting any plant substantially affected with fruit tree cankers (caused by fungi or bacteria), American gooseberry mildew, silver leaf, powdery scab of potatoes, club root, or onion and leek smut, or infested with named insects, as well as any tubers, or narcissus or daffodil plants, or bulbs, that are visibly rendered unfit for planting by reason of their being or having been affected by *any disease or pest*.

The Silver Leaf Order 1923 requires the destruction by fire of all dead wood in plum or apple trees by 15th July in each year.

Compulsory notification of outbreaks is prescribed by the Wart Disease of Potatoes Order 1941 and the Progressive Verticillium Wilt of Hops Order 1947.

It is often impossible to tell by inspection of seeds or tubers on the market whether they are healthy or not. In the case of seeds the presence of disease fungi may often be detected during the routine test for germinative capacity. Thus, in England a report on the presence or absence of the celery leaf spot fungus on celery seed may be obtained for a small fee from the official Seed Testing Station, National Institute for Agricultural Botany, Cambridge (see p. 267). Provision of a similar service in the case of many other seed-borne diseases is hindered by difficulties of administration, staffing and housing rather than by lack of scientific knowledge.

Examination of the health of the growing crop from which the tubers, runners or other planting stock are to be taken offers the only practicable means of checking the spread of many virus diseases. It has also proved very effective in Holland in stamping out hyacinth yellow disease, now seldom found in Dutch bulbs (see p. 471). A similar measure against iris mosaic, now so widespread, would be of great value to importing countries.

Since 1944 the Jutland Garden Association has arranged a scheme of inspection of shallot crops to protect growers from the purchase of sets infected with mosaic and neck rot (see pp. 303 and 299).

The schemes under which the health of seed potato crops is certified in various parts of Britain have been outlined on p. 341.

Similar schemes are operated for the certification of hops, blackcurrant, raspberry and strawberry crops from which planting stock is to be taken (see pp. 214 and 366).

Some of these schemes are linked with compulsory orders which prohibit the selling of planting material except from certified stocks, e.g. Sale of Strawberry Plants and Black Currant Bushes Order 1946.

2. Advisory Services

Growers frequently encounter problems in plant disease which they cannot solve with the books and equipment available to them. In such cases they need the assistance of a specialist in plant pathology. Since 1946 a National Agricultural Advisory Service has been set up in England and Wales to provide the necessary information gratis to all growers. For this purpose the country has been divided into eight provinces, each with a centre at

which an advisory plant pathologist and one or more assistants are stationed. Two of the provinces have sub-centres with an additional advisor. The provinces are as follows:

South-west: Counties of Cornwall, Devon, Dorset, Somerset, Wilts and Gloucester. Centre at Bracken Hill, North Road, Leigh Woods, Bristol 8; sub-centre at Staplake Mount, Starcross, near Exeter.

South-east: Counties of Hants, Berks, Oxford, Buckingham, Middlesex, Surrey, London, Sussex and Kent. Centre at Chiltern Court, St. Peter's Avenue, Caversham, Reading; sub-centre at Olantigh Road, Wye, Ashford, Kent.

Eastern: Counties of Hertford, Essex, Bedford, Huntingdon, Cambridge, Suffolk, Norfolk, the Holland division of Lincolnshire and the Soke of Peterborough. Centre at Anstey Hall, Trumpington, near Cambridge.

East Midland: Counties of Northampton, Rutland, Leicester, Derby, Nottingham and the Kesteven and Lindsey divisions of Lincolnshire. Centre at Shardlow Hall, Shardlow, near Derby.

West Midland: Counties of Warwick, Worcester, Hereford, Salop, Stafford, Cheshire. Centre at Woodthorne, Wergs Road, Tettenhall, Staffs.

Lancashire and Yorkshire: Centre at Quarry Dene, Weetwood Lane, Leeds 6.

Northern: Counties of Westmorland, Cumberland, Durham and Northumberland. Centre at Elswick Hall, Elswick Park, Newcastle-upon-Tyne 4.

Wales and Monmouth: Centre at Trawscoed, Aberystwyth, Cardiganshire; sub-centres at Brynn Adda, Bangor, Caernarvonshire and University College, Cathays Park, Cardiff.

In Scotland a separate advisory service operates through the three Agricultural Colleges which divide the country between them as follows:

Counties of Wigtown, Kirkcudbright, Dumfries, Ayr, Renfrew, Lanark, Bute, Argyll, Dunbarton, Stirling and West Perth; West of Scotland Agricultural College, Auchincruive, Ayr.

Counties of Berwick, Roxburgh, Selkirk, Peebles, East, Mid and West Lothian, Fife, Kinross, Clacmannan, East Perth and Angus; East of Scotland Agricultural College, George Square, Edinburgh.

Counties of Kincardine, Aberdeen, Banff, Moray, Nairn, Inverness, Ross and Cromarty, Sutherland, Caithness, Orkney and Zetland; North of Scotland Agricultural College, 41½ Union Street, Aberdeen.

Growers in Northern Ireland may seek advice from the Department of Plant Pathology, Queen's University, Belfast, and those in Eire from the Department of Plant Pathology, Albert Agricultural College, Glasnevin, Dublin.

II. ACTION BY THE INDIVIDUAL GROWER

1. Seed Disinfection

Spores and other resting bodies of very many kinds of bacteria and fungi may be found on seeds. Some of these are able to attack and rot seed of low vitality; others belong to dangerous parasites which cause diseases of the established plant and are introduced to new localities on the seed. Seed disinfection may be aimed at preventing loss in either or both of these ways.

Seed disinfection may also protect the seedling from damping-off fungi during the first stages of germination. Protection of this kind is only transient but it may help the seedling to survive.

Disinfection of garden seeds might with advantage be practised much more extensively than at present, especially against damping-off fungi. Many diseases, of course, do not depend entirely or at all on seed-borne infection and against these seed disinfection can give no protection. Also different kinds of seed require different treatment; even different consignments of the same kind of seed may differ in sensitivity to fungicides. Some kinds of seed may advantageously be disinfected in bulk, others only just before they are to be sown.

A treatment which is normally both effective and harmless to the seed may damage a

particular sample which is perhaps unusually damp or has been injured during threshing. Hence with the more valuable consignments of seed it may be wise to treat a small sample first and check its germinative capacity before proceeding to treat the whole consignment.

The following four methods of seed disinfection are of general application:

(*a*) **Dry disinfection with red copper oxide.** The seed should be placed in a box or other tightly shut container that it less than half fills. The red copper oxide powder should be sprinkled over the seeds and the container closed and shaken up or churned over for 4–5 minutes until every seed is well coated with the dust. Treated seed may be kept or sown at once.

Many kinds of seed such as:

Bean	Melon
Beetroot	Pine
Carrot	Pumpkin
Cucumber	Spanish pepper
Egg plant	Spinach
Lettuce	Stock

can be protected against damping off by $\frac{1}{4}$–$\frac{1}{2}$ oz. of the red copper oxide per pound of seed. Peas need only $\frac{1}{10}$–$\frac{1}{8}$ oz. per pound. Cabbage, radish and other cruciferous seed and celery seed may be damaged by this treatment.

(*b*) **Dry disinfection with zinc oxide.** $\frac{1}{3}$–$\frac{1}{2}$ oz. per pound of seed may usually be tried against damping off of cabbage, radish, beetroot, lettuce, spinach and tomato seed.

(*c*) **Soaking in chloride of lime.** 3 oz. calcium hypochlorite are mixed with 1 quart of water and stirred for several minutes. It is then allowed to stand for a short time, after which the clear liquid is poured off or filtered and is ready for use. Seeds shaken up in this liquid for a few minutes are freed from bacteria and superficial moulds without any injurious effect on their germination.

At Cheshunt Experiment Station 4–8 hours soaking in this solution has been found satisfactory for tomato seed. For lettuce seed infected with *Marssonina* see p. 287.

(*d*) **Organo-mercury seed disinfectants.** These are of two kinds, those intended for use in solution and those to be shaken up as dusts with the dry seed. Of the former one of the first was Uspulun but there are now several proprietary preparations of the kind, any of which may be tried. The grower is advised to make a few preliminary trials with them, using weak solutions, to make certain they do not injure the seed. The following was the procedure with Uspulun: Pour the seed into the solution and stir several times. Skim off the refuse and empty seeds that float on top. Pour off the liquid and sow the seed at once or spread it to dry quickly. The liquid should be at about room temperature, say 65–68° F.; cold solutions are less active than warm. The recommended times to soak seed in Uspulun were:

5–60 *mins.*	30 *mins.*	1 *hour.*	6 *hours.*
Tomato	Cucumber	Bean	Celery
(risky)	Leek	Callistephus	
	Onion	Carrot	
	Spruce	Pea	
	Wallflower		

The dry organo-mercury seed disinfectants, of which several equally effective are on the market, will no doubt be widely used, but so far not much has been published about their effect on garden seeds. Peas should be treated at the rate of about 1 oz. to 13 lb.; small seeds may need slightly larger doses.

Special Methods

Turnip, swede and cabbage seed may be treated against ring spot, dry rot, canker and black rot by soaking in hot water just before it is sown. It should be steeped in water at 122° F. for 25 minutes (Millard, 1945). Care is necessary not to exceed this temperature or the seed will be killed. Samples of poor germinative capacity may be injured by this treatment so it is as well always to treat a small trial lot first and see if the germination has been affected before proceeding with hot-water treatment of a large consignment of seed. Drying the seed quickly after the soaking is one of the great difficulties in applying

this method of control to large quantities of seed.

To control ring spot, cabbage seed may also be shaken up with an organo-mercury dust. Soaking for 20–30 minutes in a 1: 1000 corrosive sublimate solution has proved effective against black rot.

Celery seed. For treatment against leaf spot, see p. 266.

Spinach seed may be treated dry with red copper oxide (p. 576) or by soaking 1 hour in ¼% Germisan solution or 1% copper sulphate solution.

Tomato seeds may be sensitive both to copper and mercury but there seem to be considerable differences in the behaviour of different lots. Chloride of lime is safe and effective.

Potato tubers may be dipped in an organo-mercury solution to control *Corticium solani*, skin spot, dry rot, etc. (see under the various diseases, pp. 328 and 331).

2. Soil Disinfection

In many diseases infection is mainly contracted from the soil. This is especially the case under glass where continuous cropping or at most a very short rotation is usual. In such circumstances the soil must either be renewed frequently or be disinfected. The latter practice is becoming an essential part of intensive cultivation.

Soil disinfection may be done by heat or by chemicals. The former method has the advantage that the treated soil can be used almost as soon as it is cool. Both methods often increase the soil fertility. This applies especially to soils rich in nutrients which are rendered more available by the treatment. Soil bacteria too, after an immediate temporary decline in numbers, increase rapidly; the colloid content of the soil is changed and bacteria, fungi and insects killed by the treatments are broken down to supplement the nitrogen supply in the soil (Elveden, 1921, Waksman and Starkey, 1923).

Baking. Soil for seed boxes and pots may be very thoroughly disinfected by dry heat. An oven for baking small amounts may be

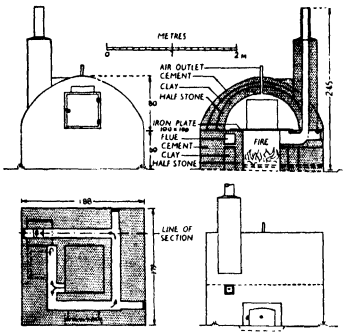


Fig. 344. Oven for disinfection of soil by heat.

built at comparatively small cost and be of service for many years. A practical design is given in Fig. 344 where sizes are expressed in metres (1 metre = 39.37 inches or 1.09 yards). In such an oven the soil is heated to about 212° F., kept at this temperature for an hour and then left in the oven overnight to cool. The soil near the bottom of the heap reaches a higher temperature than that at the top so the heap should be stirred several times with a shovel during the process (Burgess, P. S., 1922). The reaction of the heated soil may be slightly more acid.

When a large amount of soil is to be baked the oven should be built for continuous use so that soil can be filled in on top and taken out below. Flues are built in the wall and a chimney is carried up the middle. The fire is made up during the day and kept low at night and thus soil can be removed from the oven every day.

Electricity. Electrical heating of soil in special containers is practised where electricity is cheap. One may use ordinary heating elements or pass the current through the soil itself. As the capacity of the latter for conducting electricity varies with its water and salt content and with temperature the results are apt to be erratic.

Steam, which was first adapted for practical use by Rudd in 1893, has several advantages. Once installed the apparatus can be used

for greenhouse beds, benches and potting soil.

The soil should be well loosened and be fairly moist but not wet. Only high-pressure steam should be used, with a working pressure of about 70—100 lb. per square inch (Bewley, 1926, 1950). After disinfection by steam the soil may be planted or sown as soon as it has cooled. A stiff clay gives up the heat in 24 hours while a light soil rich in humus may retain it for 2–3 days. Bewley (1950) advocates flooding the soil with water after steaming. A considerable amount of nutrients is released in the soil by steaming. Hence, especially in old houses that have previously received much manure, one can afford to wait until the middle of the growing period before applying fertilizers. Potassium and phosphoric acid may then be required.

To kill the usual greenhouse fungi the soil should be warmed to 194–212° F. for 15–40 minutes. As a rough practical guide of its effectiveness potatoes should be dug into the soil at different depths; when they are tender the treatment has been completed. Weed seeds, insects, beetles, etc., are all killed but beetle larvae and others may escape if they have burrowed below the level at which the soil is being steamed. It is important to cover the soil during treatment so as to keep the warmth in, but in practice the soil is seldom kept covered after treatment. As mentioned above, light soils rich in humus retain the warmth longest; the top layer dries quickly and acts as an insulator, keeping the heat in the lower layers. Walls and supports, tools and footwear not affected by the steam should be disinfected by chemicals, e.g. formalin (see p. 595).

Various methods of soil disinfection by steam are in use. A fully illustrated account of them is readily accessible in Ministry of Agriculture Bulletin 22 which should be consulted by those requiring further information than can be given here. (See also Bewley, 1950.) Some common types of apparatus are:

(*a*) A harrow-like grid (Fig. 345) where the steam issues from small holes at the bottom of the upright tubes. There may also be smaller holes higher up the tubes. The grid is pressed well down into loose, recently dug soil. The soil surface may advantageously be covered during steaming with a flat metal tray turned upside down to keep the steam in.



Fig. 345. Oil disinfection by steam with the harrow-like grid. On left in operation; on right lifted up.

(*b*) One may also use a grid of horizontal perforated iron tubes connected at one end by a cross-tube through which the steam enters. This is a slower process than (*a*) but usually more efficient.

The first step is to remove the soil one spit deep from an area the size of the grid, and, if necessary, to loosen the subsoil with a fork to allow penetration by the steam. The grid is then laid out, buried with soil from the adjoining area and covered with a metal or wooden box or a tarpaulin. Steam is then introduced. After steaming is completed the grid is pulled sideways out of the soil and laid in the neighbouring space, buried again and so on.

(*c*) A permanent system of pipes may be laid at a depth of about 18 inches in the soil, e.g. drainpipes of 3–4 inch diameter. The pipes are about 18 inches apart and every group of 4 or 5 pipes is united by a cross-pipe of iron 2–3 inches in diameter, through which the steam enters. When the pipes are well laid so as not to be disturbed by cultivation the arrangement may remain serviceable for many years. Steam is turned on for 2–6 hours.

(*d*) Steaming in boxes. 2 or 3 heavy boxes are built with a perforated false bottom on which soil can be laid a short distance above

the base. Soil is piled on in a layer some 20 inches thick and steam at high pressure is let in beneath the false bottom. When the soil has been at 212° F. for 3–4 minutes the steam is shut off. The soil remains in the box another 20–25 minutes while the next box is being similarly treated. The method is said to be quick, cheap and reliable (Lawrence and Newell, 1935).

Chemical disinfection of soil is extensively practised against eelworm and other pests and sometimes against fungi. As for steam sterilization the soil should be loosened and kept sufficiently moist. It is almost impossible to kill dry sclerotia.

After disinfection by chemicals the grower must allow some time to elapse before sowing or planting in the treated soil.

Where large greenhouses are to be disinfected the area is divided into suitable plots, say about 50 sq. yards each, surrounded by little walls of earth. This is particularly important if the soil surface slopes. The liquid is prepared separately for each plot and poured over it (Fig. 346). The grower

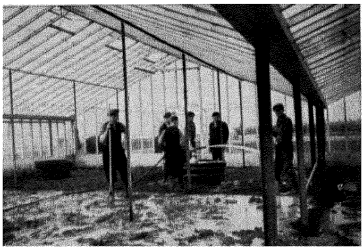


Fig. 346. Soil disinfection with liquid.

tries to ensure an even distribution and penetration by shovelling and raking; then, when almost all the liquid has percolated into the soil, the surrounding earthen walls are thrown down. The walls and supports of the house are sprayed with the same liquid. Four men can disinfect 1,000 square yards of soil in about 4 hours.

A large number of chemicals has been used for soil disinfection of greenhouses and only the cheapest and best known will be mentioned here. The following have proved efficient in practice: formalin (p. 595), kerol (p. 597), cresylic acid (p. 594) and corrosive sublimate (p. 594), also carbon disulphide for root eelworm. Organo-mercury disinfectants have also been used. So has chlorpicrin (trichlornitromethane) in America, and much may be hoped for from future researches in the use of more complex halogenated nitroderivatives of aromatic hydro-carbons.

Watering of growing plants with disinfectant solutions is sometimes effective against damping off and foot rot fungi and *Moniliopsis aderholdi*.

Cheshunt compound (see p. 593) is used particularly against *Pythium* and *Phytophthora* attacks on seedlings.

Formalin has been used on seed boxes that are watered after sowing with a solution of $\frac{1}{4}$–$\frac{1}{2}$ of 1% commercial formalin in water. This weak solution does well with cucumber, pea, sweet pea, and tomato but may damage seed of stock and other ornamental plants (Ogilvie *et al.*, 1939).

Corrosive sublimate $\frac{1}{4}$ oz. plus 2 oz. saltpetre dissolved in $2\frac{1}{2}$ gallons water is an excellent solution against *Corticium solani* and *Moniliopsis aderholdi*, as e.g. on stocks, but growth of many plants is checked by it, such as begonia, hydrangea, young tomato plants and seedlings of cauliflower and brussels sprouts.

3. Nursery Hygiene

Under glass. Every careful grower regularly picks off withered leaves and flowers from his plants because he knows they soon bear a crop of grey mould. If individual diseased plants appear in an otherwise healthy crop it is generally enough to remove them. This is especially desirable in propagating material. Debris should be collected in a pail containing lime water or some disinfectant fluid, otherwise as it is being carried through the house it will scatter spores everywhere and its removal will do more harm than good. It may sometimes be advisable to sear diseased plants with a blow lamp before they are dug up, to prevent

them from infecting adjacent plants when dragged across them during removal.

Plant pots, boxes, canes, string should be disinfected by soaking for 24 hours in formalin solution 1: 50 and then ventilated. Plant boxes are often disinfected by flaming for a moment, or by dipping in 4% copper sulphate solution, followed by a dip in lime water. Both surface disinfection by flame and dipping in copper sulphate and lime water are impracticable with boxes put together with galvanized nails.

Water tanks may be centres of infection by *Phytophthora* and *Pythium* spp. causing damping off and foot rot and should periodically be emptied and thoroughly cleaned. The tank and its surroundings should be sprayed with 1% formalin. Then the tank is filled with 1% formalin, high enough to enter the overflow tube and left covered with sacking for two days, after which it is emptied and washed with clean water, being then refilled for use.

For burning of sulphur in greenhouses see p. 600.

Store-rooms. Fruit stores are liable to become contaminated with infectious matter from rotting fruit, partly in the trays, partly on the shelves, floor, walls and ceiling. The sooner spotted and decaying fruit is removed from store and destroyed, the less is the chance of infection.

Of the three common apple-rotting fungi *Sclerotinia fructigena* is in our experience the most susceptible to formalin, spores on the apple surface being killed if placed in 1% formalin. *Penicillium* is similarly killed by ½% chloride of lime, while *Gloeosporium album* seems to be destroyed by 2% chloride of lime.

Many of the fungi that cause rot of stored fruit are brought in with the fruit. Nevertheless during the summer the trays should be freed from infectious matter by thorough washing and the store and packing rooms should also be cleaned. Three methods of disinfection may be recommended, the relative advantages of which have not been thoroughly compared:

(*a*) Burning of sulphur, 4 lb. per 100 cubic yards (see p. 600).

(*b*) Evaporation of formalin, 4 lb. per 100 cubic yards.

(*c*) Thorough spraying with 2% chloride of lime solution.

After treatment the room should be left locked for 24 hours and then thoroughly ventilated. The treatments should be done long before fruit is to be brought into the store. The fruit may be damaged if it comes in contact with boxes strongly impregnated with formalin (Fig. 347).

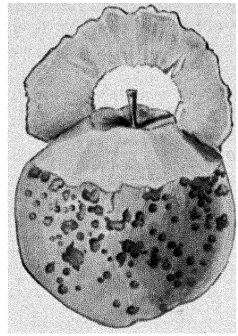


Fig. 347. Cox's orange pippin damaged by formalin from inadequately aired case.

Gardens. Diseased branches may be observed at any time during the summer and it is easiest to remove them at once; diseased fruits should also be removed as soon as possible. The longer they remain the more likely are they to produce infectious matter. Even if this is conscientiously done all summer there will still be dead branches and mummified fruit to be collected and burnt after leaf fall (Figs. 69, 124, 125).

Old leaves may carry much infectious matter (see the part they play in overwintering of apple scab, p. 104). Fallen leaves should, therefore, be well dug in or collected for composting; leaves drifted into hedges, etc., may form an important reservoir of infection. In kitchen gardens both fungi and insects are found on old cabbage stumps; rust occurs on asparagus tops. It is well to dry and burn material of this kind as soon as possible. Other



Fig. 348. Apples with brown rot, collected from beneath the trees.

plant debris is more suitable for the compost heap but one must remember that it will contain a large amount of infectious matter. During decay the rotting bacteria destroy or crowd out many parasitic fungi, hence the longer the material lies in the compost heap the better. Spores of the club root fungus must be expected to remain able to germinate for 5 years and those of wart disease even longer.

Boxes and canes should be disinfected as described above (p. 580).

4. Choice of Variety and Method of Cultivation

Varietal susceptibility. Varieties very susceptible to an important disease soon disappear from cultivation as soon as others of equal quality and greater resistance become available.

If the resistant varieties are not quite such high yielders or of such fine quality it may still be best to grow them.

Where direct control measures are ineffective or too expensive and immune varieties are not available one may have to abandon cultivation of a particular crop. For example, where severe attacks of club root occur it may be better to grow potatoes or carrots instead of brassicas.

Where it is practicable to treat seed against fungus diseases (see p. 576) this will be a help in getting a crop of healthy seedlings.

Density and depth of seeding are of great importance. Dense seeding encourages damping off and the propagation fungus (see p. 68). When seed is sown too deeply only the strongest seedlings will come up. In very early sowings out of doors the soil may be too cold; seed that then germinates very slowly may be overwhelmed by fungi and bacteria from which it would have escaped by rapid germination later in the year. This often happens with pea seedlings. Seed treatment may be a help in protecting early sown seed but the condition of the soil is also important.

Soil. On soggy ground the choice of crops is very limited until it has been drained. Lime deficiency and over-liming have been discussed on p. 29, where it has been seen that some crops are quite at home on lime-deficient soils. Some "over-liming injury" is really deficiency disease due to lime-induced deficiencies of available boron, manganese, iron, etc. (see p. 36).

For the action of the common artificial manures see Section 1: nitrogen, p. 40; phosphoric acid, p. 41; and potassium, p. 42.

Stable manure is less liable to misuse but runs the danger of introducing infectious material with it, especially club root (p. 246) or poisons like chlorine (see p. 34).

Rotation of crops. Because most diseases attack only one crop or a group of related crops and become more prevalent when these crops are grown continuously in the same place, rotation of crops is a valuable aid in disease control.

In general, when planning annual crops, the grower will arrange his rotation so as to use up the manure to the best advantage. When laying down a crop for several years, e.g. strawberries, he will consider what would be the best previous crop, in this case probably corn or peas. After a disease has broken out severely the grower will choose subsequent nonsusceptible crops for that plot of land.

When planning his cropping he should also consider as far as possible what are to be the adjacent crops. Because of blight early potatoes are bad neighbours for late potatoes. A new consignment of virus-free seed potatoes should

not be planted alongside an old stock heavily infected with mosaic or leaf roll. Seed crops of biennial plants should be kept at a distance from first-year crops of the same plant or they will probably infect the seedlings with some disease. It is, however, difficult always to keep susceptible crops apart; thus virus from cucumbers may infect spinach and cause it to be ruined by mosaic.

Biological control. Noxious insects have enemies of their own, hyperparasites that prey upon them, and so, to some extent, do eelworms and parasitic fungi. Much work has been done on the " antagonism " of the green mould *Trichoderma lignorum* to *Corticium solani* and other fungi. It is probably the mutual antagonism between fungi and the competition between them when living as saprophytes that prevents plant disease fungi from being more universally destructive than they are. The practical application of this hyperparasitism and antagonism as and when required, however, presents difficulties that have yet to be solved. Most of the successes in biological control have been with insects introduced into a new country without the parasites that kept them in check in the old. An instance of fairly successful biological control familiar to most growers in Britain is that of the greenhouse white-fly by its parasite.

Some form of antagonism is apparently involved in the control of potato scab by green manuring (see p. 322).

5. Spraying, Dusting and Fumigating

The chemicals employed to control plant diseases and insect pests are usually applied by spraying machines designed to cover the plant with a layer of solution of the chemical used. Some are quite effectively employed dry in the form of finely divided powders, applied by special dusting machines. Under glass various forms of fumigation and evaporation of fungicides are practised.

Winter spraying. Most winter sprays applied to fruit trees from December to February are designed primarily for the control of insect pests. Amongst these sprays are the various " water soluble " carbolineums, combinations of tar distillates and an emulsifying agent, and mineral oil emulsions. These have some effect also on the overwintering stages of some fungi, notably *Sclerotinia laxa*. Too strong a winter oil spray or too frequent application may lead to bark canker formation.

Copper sulphate solution 4–6% has been used for winter spraying against American gooseberry mildew. Formerly 2% copper sulphate was used for winter spraying against apple scab but the practice has been abandoned (see also spring spraying, below).

Lime sulphur 1 : 9 is used in winter or up to March as a spray against American gooseberry mildew, vine mildew, peach leaf curl and pocket plums.

Formalin 1 : 40 is used as a winter spray against American gooseberry mildew.

Spring sprays. In the short period while the buds are swelling and before they show a green tip various pests may be attacked more successfully than either before or after (Fig. 349). This is the case with the use of emulsified carbolineum against apple weevil, of lime



Fig. 349. Apple buds at green tip stage, the limit of development for use of carbolineum sprays on apple.

sulphur against pear gall mite, etc., but it also applies in some degree to fungi. Spring carbolineum has a distinct effect on *Sclerotinia laxa*, especially when mixed with Bordeaux mixture.

Spraying with Bordeaux mixture 48 : 24 : 100 when the buds are breaking has had definite

effect on apple and pear scab in Switzerland and Germany (Osterwalder, A., 1935). We cannot, however, fully endorse the statement that after such a spray only the post-blossom summer sprays are required.

Summer sprays, according to long-established usage, include all those applied to trees or bushes bearing green leaves. This necessarily restricts the strength of the sprays that can be used with safety (see spray damage, p. 45). As regards fungi summer sprays are mainly preventive but may be used to check further development of mildew or scab outbreaks that have already begun.

Apple trees. The scheme on p. 584 gives a summer spray schedule designed primarily to control apple scab but the usual insecticidal sprays are also included in it. Where mildew is prevalent one should use lime sulphur instead of Bordeaux mixture at the green cluster stage just as at the pink bud stage. In later summer sprayings lime sulphur 1: 100 may be used instead of Bordeaux mixture. The latter is useful for varieties which cannot stand lime sulphur (see p. 47).

The scheme set out on p. 584 is the standard spray in Denmark but it is subject to local variations according to the variety grown, method of spraying, etc. Bordeaux mixture is the most effective spray against scab but when used just before flowering and in post-blossom sprays it may cause much damage, especially on varieties like Cox's Orange, Belle de Boskoop and others. Therefore the standard scheme relies on lime sulphur for the pink bud and 80% petal fall sprays. In addition to its fungicidal properties lime sulphur is also active against red spider but it too may cause spray damage and leaf fall in some varieties. On these red spider may be controlled by a $\frac{3}{4}$ or 1% spraying oil spray. Damage may be caused by spraying in succession with lime sulphur and oil.

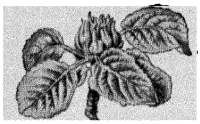
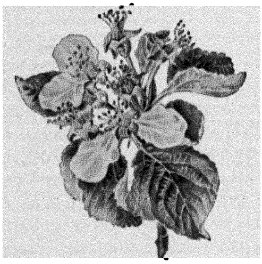
It is very important to spray thoroughly before flowering for it is then that the first scab infections take place by spores from last year's leaves and twigs. In damp years and on susceptible varieties scab at this early stage may get such a hold that it is very difficult to check it later. Where scab is particularly to be feared emphasis should be laid on spraying twice before flowering. Often 4–6 summer sprays may be needed to ensure a scab-free crop. On late varieties and those susceptible to scab it may even be necessary to spray in August.

When a spring carbolineum spray has been given at the green tip stage, with 3 gallons lime sulphur added per 100 gallons of spray to increase the effect on scab, the grower should find one spray against scab sufficient between leafing and flowering unless the danger of infection is abnormally great.

Various proprietary sprays may also be used.

Pear trees are sprayed, particularly against scab, at the same periods as shown for apple trees on p. 584, but as a rule a Bordeaux mixture 8: 8: 100 or white Bordeaux mixture 4: 8: 100 is used. Lime sulphur is useful against red spider but it may scorch some pear varieties. Against red spider a summer petroleum wash may be added to the Bordeaux mixture at the rate of $\frac{3}{4}$–1 gallon per 100 gallons. As insecticides, lead arsenate and nicotine may be added as with apple.

Standard Summer Spray Schedule for Apple Trees

Sprays	Effect
 *Green Cluster Stage* Bordeaux mixture 8: 8: 100 +20–30 oz. 80% nicotine per 100 gallons* +4 lb. lead arsenate per 100 gallons.	Bordeaux against scab and other fungi. Nicotine against apple sucker, aphides and capsids, also young caterpillars. Lead arsenate against winter moths and other leaf-eating caterpillars.
 *Pink Bud Stage* Lime sulphur 1: 60 +nicotine and lead arsenate as above.	Lime sulphur against scab, etc., and red spider. Nicotine against capsids, apple sucker and aphides. Lead arsenate against winter moth, leaf-eating caterpillars.
 80% *Petal Fall Stage* Lime sulphur 1: 100 + nicotine and lead arsenate as above.	Lime sulphur against scab, mildew, etc., and red spider. Lead arsenate and nicotine against codling moth, apple sawfly, etc.
Fruitlet Stage (fruit the size of marbles) White Bordeaux mixture 4: 8: 100	Bordeaux mixture against scab and *Sclerotinia.* Lead arsenate against codling moth.
In Wet Years Subsequent sprays with white Bordeaux mixture 4: 8: 100 at interval of 2–3 weeks.	Bordeaux against scab and *Sclerotinia.*

* A more usual nicotine spray in England is 5–8 fluid ounces 95–98% nicotine per 100 gallons.

Cherries and plums cannot, unfortunately, be included in the same schedule because, for one thing, plum leaves may be scorched by the Bordeaux mixture. In the schedule below particular attention has been paid to control of blossom wilt. Where brown rot of the fruit is prevalent it is tempting to continue with Bordeaux mixture or lime sulphur after blossoming. In such a case the expense the crop can stand decides how many sprays to apply. Cherry may be scorched by Bordeaux mixture during the summer. For control of Bacterial Dieback see p. 204.

Gooseberry. Burgundy mixture is very effective against American gooseberry mildew though it may scorch if used later than just after flowering. Formalin soap has a milder action but may be used on the growing berries as it is non-poisonous and does not cause spotting. Washing soda sprays are also quite effective (see pp. 171–2).

Gooseberries, red and black currants. Burgundy mixture is less effective against leaf spot than Bordeaux mixture 8: 8: 100, which is therefore preferred when American gooseberry mildew is not concerned and is always preferred for the late summer sprays. Spraying against leaf spot is done just before and just after flowering and once or more after the berries have been picked.

Standard Summer Spray Schedule for Cherries

Spray	Effect
Flower Buds in Close Cluster Stage (10–15 days prior to blooming)	Bordeaux mixture against blossom wilt.
Bordeaux mixture 8: 8: 100	Nicotine against aphides and small caterpillars.
80% nicotine 20–30 oz. per 100 gallons.	
Petals Visible (2–5 days before blooming)	Bordeaux against blossom wilt.
Bordeaux mixture 8: 8: 100	Lead arsenate against leaf-eating caterpillars.
Lead arsenate 4 lb. per 100 gallons.	

Standard Summer Spray Schedule for Plums

Spray	Effect
Flower Buds in Close Cluster Stage (10–15 days before blooming)	Lime sulphur against blossom wilt.
Lime sulphur 1: 50	Nicotine against aphides, fruit sawfly and small caterpillars.
80% nicotine 20–30 oz. per 100 gallons.	N.B. Czar may be sensitive to nicotine.
Petals Visible (2–5 days before blooming)	Lime sulphur against blossom wilt and red spider.
Lime sulphur 1: 50	

The **remunerativeness** of intensive summer spraying on a scale like that outlined on p. 584 is well illustrated by Danish experiments (Esbjerg, N., 1929; Dullum, N. 1931; Dullum and Esbjerg, 1937). The results show that rational disease control is necessary in economic fruit growing; small charges in the price of chemicals or fruit cannot destroy the remunerativeness of spraying. But the outlay remains fairly large and therefore any new method that may reduce the quantity of chemicals or amount of labour required is worth investigating. It will always pay a grower to estimate carefully the probable intensity of an attack in relation to weather conditions and to decide which sprayings will be most important in a given season and which can be omitted or postponed. The grading of the fruit may also give clear indications which sprayings were worth while.

For winter or spring spraying the following approximate quantities are needed: for small dwarf trees, 2–4 pints per tree; for 20-year-old free-standing trees, 14–21 pints according to the growth of the variety; for fruit bushes, 1–3 pints per bush. The whole surface of the bark must be wetted, and for old, neglected trees a definitely greater quantity of spray will be necessary.

In summer spraying a little more liquid is used in the post-blossom spray than in the first and last application; for five summer sprays one may need in all 14–21 pints to a 3–4-year-old tree, 70–100 pints for a 10–12-year-old tree and 130–175 pints for a 15–20-year-old tree.

In much fruit cultivation, however, remunerativeness is not the chief consideration. Much good fruit is grown in private gardens and gives pleasure to the grower, even if he knows he could have bought it more cheaply in the market. How few sprayings will suffice to give a satisfactory crop for home use is a long-standing problem. In 1939 about 3,000 gardens in Sjaelland were sprayed by spraying associations. A copper sulphate spray or a spring carbolineum and Bordeaux mixture (p. 582) may reduce the number of summer sprayings required but scarcely reduces the expense.

In the official publications of the English Ministry of Agriculture distinctions are drawn between the intensive cultivation of table fruit, fruit cultivation in small gardens and extensive cultivation of cider apples. In the latter the expense is small for it is only necessary to keep the fruit free from scab; but a heavy yield and high sugar percentage is required, hence the tree leaves must be well developed and sound (Turnbull, J., 1938; Taylor *et al.*, 1946; 1948; Barker, B. T. P., 1937; M. of A. Advisory Leaflet 114). English practice is illustrated by the following examples.

A. Intensive Apple Cultivation

1. 7.5% tar oil emulsion in January.
2. 5% mineral oil emulsion at bursting of buds.
3. Lime sulphur 1 : 33 at green bud stage.
4. Lime sulphur 1 : 40—lead arsenate at pink bud stage.
5. Lime sulphur 1 : 100—lead arsenate—nicotine at 80% petal fall stage.
6. As 5 a week later.
7. Possibly several subsequent applications according to season and variety. For post-blossom applications a copper lime dust or sulphur dust may replace the spray.

B. Apple Cultivation in Private Gardens

1. Tar oil emulsion in January.
2. Lime sulphur or Bordeaux spray—lead arsenate—nicotine at pink bud stage.
   Trees should be banded with sacking or corrugated cardboard to catch codling moth and grease banded to trap winter moths.

C. Cider Apple Cultivation

1. 7.5–10% tar oil emulsion, the first and most essential spray.
2. Lime sulphur 3 : 100—lead arsenate at pink bud stage.
   A well-managed cider orchard only needs one spray a year, a winter and summer spray alternatively. Grease bands are used against winter moth.

Turnbull drew attention to the importance of having enough sprayers; no fruit grower has regretted the purchase of too powerful a

sprayer. One should be able to deal with all the apple and pear plantations in 6 days. Slow work may be due to low pressure, too great loss of pressure between sprayer and nozzle or too small a sprayer; double nozzles are recommended. If one works with too narrow a jet the nearest branches become either too heavily sprayed or too little. Winter washes are a definite part of the spray programme in England, but there is a tendency towards a light winter wash with tar oil instead of the thorough soaking practised up to now. Experiments are also being made in increasing the quantity of liquid and decreasing the concentration of the summer sprays, especially where only scab control is involved.

Sprayers. The sprayer must be kept clean. Water should be sprayed through tube and nozzle after it has been used. All rubber tubing and connections should be inspected critically at the end of each season. Make sure the manometer is registering correctly.

After spraying with carbolineum the sprayer should be well washed through with warm water and the rubber should be particularly well looked after. Bordeaux mixture and Burgundy mixture do not affect the metal parts, but the sprayers should be cleaned after use so as to keep the air vents, etc., clear.

Dusting as a control method is about as old as spraying. In England Kyle used it against vine mildew in 1846, and in 1850–1 experiments in dusting with sulphur powder were carried out at Versailles and in the South of France. Immense quantities of sulphur have subsequently been used in vineyards. An advance in dusting fruit trees was made in North America when a finer preparation with a colloid base was found to be more effective (Reddick and Crosby, 1916).

Sulphur powder is still the principal remedy for the various powdery mildews, and it has been used against scab on fruit trees. In Danish experiments six dustings during the summer have been found to keep scab under control in normal seasons. Starting with 1–2 sprays in spring followed by 4–5 dustings one obtains excellent control of disease, little damage by chemicals and beautiful fruit.

Dusting with sulphur when the trees are in bloom may hinder germination of pollen on the stigmas; this is undesirable when pollination is scanty (MacDaniels and Furr, 1929).

Copper lime dusts have been much used for potatoes and vegetables. In some fruit-growing districts, e.g. Nova Scotia, much copper lime dust is used on apple trees. To prevent scorching a dust of low copper content may be used.

The advantages of dusting are: (1) The possibility of quick and prompt application; a motor duster can treat 25–34 acres of apple orchard in 8–9 hours as against a motor sprayer's $7\frac{1}{2}$ acres. (2) Weighing of chemicals and transport of water is avoided. (3) Dusting machines are comparatively cheaper than sprayers and on the whole more reliable. (4) The comparatively light weight of a dusting machine is an advantage on undulating ground or wet soil. (5) The dust has greater penetrating power. (6) Dusting tends to scorch less and give brighter-looking fruit. (7) Houses, etc., in the vicinity are not marked by the dust.

The disadvantages of dusting are: (1) Weaker and less lasting effect against fungi than the corresponding spray. (2) In an apple orchard the cost of chemicals is about three times as great for dusting as for spraying. (3) It needs more settled weather, as slight wind drifts the dust away and rising air currents may carry it too high. (4) The workers have to be protected against the fine dust.

Evaporation and fumigation. In combating insect pests under glass burning or evaporation of various chemicals is very largely employed. Against fungi sulphur vapour is used, particularly against mildew, but various kinds of plants are sensitive to sulphur vapour. Evaporation of sulphur is described on p. 600.

Any plant will be damaged if the sulphur catches fire, for then sulphur dioxide is formed which is highly poisonous (see p. 51). In fumigating empty houses, however, e.g. mushroom houses during the fermentation, sulphur dioxide may be used and large quantities of sulphur are burnt.

Formalin vapour may be used for disinfection of fruit stores, etc. (see pp. 580, 595).

III. CHEMICAL FUNGICIDES

Brief notes are given on the best-known fungicides, arranged alphabetically. It is not practicable to list all the proprietary preparations, marketed under trade names, and their omission must not be regarded as implying any doubt as to their efficiency when used according to the manufacturers' directions. Preparations are not listed if the name under which they are sold clearly indicates their composition and purpose.

The Ministry of Agriculture in England has laid down specifications for the well-known non-proprietary fungicides such as lime sulphur, copper sulphate, Bordeaux powder, Burgundy powder, Cheshunt compound and formaldehyde and for the common insecticides. Members of the Association of British Insecticide Manufacturers have agreed to conform to these standards which have been published as *Min. Agric. Bull.* 82, 1934.

Since 1943 the Ministry of Agriculture in England and the Department of Agriculture for Scotland have jointly operated a scheme for the official approval of proprietary fungicides and insecticides. These include those brands of substances covered by the specifications above mentioned which are guaranteed to conform to those specifications and also products for which no official specification exists, but whose composition has been declared in confidence to the Advisory Committee which operates the approval scheme and for which evidence of efficiency is available. Products whose composition is kept wholly secret from the public are not eligible for approval under the scheme (see *Agriculture* l, pp. 331–4, 1943 and liv, pp. 134–7, 1941). Lists of approved products have been published from time to time (see *Agriculture* l, pp. 478–80 and 570, li, p. 190, and pp. 383–4, 1944; li, p. 523, lii, p. 282, 1945; lii, p. 476, liii, pp. 94–5, 371–2, 1946; liv, pp. 46–7, 286, 1947; liv, p. 478, lv, p. 227 and 364, 1948).

Only fungicides and insecticides of the following types are as yet (1948) accepted for approval. Lead arsenate powders, lead arsenate pastes, lime sulphur washes, miscible tar oil winter washes, stock emulsion tar oil winter washes, organo-mercury dry seed dressings, derris and lonchocarpus insecticidal dusts, and sprays, copper fungicidal dusts (excluding seed dressings), copper fungicidal sprays, nicotine dusts, nicotine sprays, nicotine sulphate, miscible petroleum oil winter washes, stock emulsion petroleum oil summer washes for glasshouse use and orchard use, wetters and spreaders, derris petroleum oil washes for glasshouse and orchard use, thiocyanate petroleum winter washes, sulphur dusts and sprays, D.D.T. dusts and sprays, stock emulsion D.D.T. petroleum oil winter washes, derris pyrethrum insecticidal sprays, stock emulsion tar and petroleum oil winter washes, miscible tar and petroleum oil winter washes, benzen hexachloride insecticidal dusts, stock emulsion benzen hexachloride—petroleum oil winter washes, D.N.C. petroleum oil winter washes, 4% calomel dusts, banding greases and prepared grease bands and metaldehyde slug baits.

Approved products are entitled to bear a lozenge-shaped design surrounding a crown, with a statement that they conform to a standard approved by the Ministry and Department.

List of Therapeutants

Abavit B—an organo-mercury dry seed dressing.

Agral—a wetting agent and spreader (see p. 602).

Agrosan G—an organo-mercury dry seed dressing.

Alkaline copper soda mixture (see p. 593).

Aluminium sulphate may improve the growth of rhododendron in the same way as magnesium sulphate. The information about rate of application is very vague so growers should use it carefully, starting with about ½ lb. per 100 lb. of potting soil or about 1 oz. to 3 square yards out of doors.

Ammonium polysulphide is a dark-red, evil-smelling fluid formerly much advocated as a spray against hop mildew and American

gooseberry mildew because it does not cause spotting. It is used diluted with water at the rate of about 1: 200 plus a little soft soap.

In early English experiments Bordeaux mixture was found more effective than ammonium polysulphide against apple scab. Lime sulphur and ammonium polysulphide seemed about equally effective. Of late years there has been some revival of interest in it as a spray for apple trees. Ammonium polysulphide reacts with lead arsenate to yield dissolved arsenic so that a combined ammonium polysulphide and lead arsenate spray may be liable to scorch.

Aretan (organo-mercury preparation) is much used on the Continent for disinfection of flower bulbs, e.g. tulip against fire, narcissus and hyacinth against various mould fungi. The bulbs are soaked for 30 minutes in a ¼% solution.

Potatoes may be treated against black scurf and stem canker by soaking for 15 minutes in 0.15% solution. Treatment immediately after lifting may control dry rot and skin spot (see p. 331 and Hortosan p. 591).

Blue vitriol, see copper sulphate.

Borax is used as a fertilizer on boron-deficient soils (see p. 32). The rate of application is about 15–20 lb. per acre = 1 oz. to about 20 square yards. Larger quantities are liable to be injurious except in special circumstances. Such small quantities are best applied thoroughly mixed with dry sand, superphosphates or Chile saltpetre or may be dissolved in water and applied by spraying.

Bordeaux mixture is used as a fungicidal spray to protect growing plants against disease fungi. One of its advantages is that its composition can be greatly varied according to circumstances. The formula is usually quoted as three successive figures, meaning pounds of copper sulphate, pounds of quick lime and gallons of water. For fruit trees a usual formula is 8 : 8 : 100 or white Bordeaux mixture (see p. 584); for potatoes and other plants 16 : 8 : 100 is more usual. The solution is made as follows:

(1) In a wooden vessel add 4½ lb. powdered copper sulphate to 11 gallons water and stir vigorously until dissolved.

(2) In another vessel place 2¼ lb. quicklime and cover with water until the lime breaks down into a fine powder. Add as much water as is needed to give 11 gallons of milk of lime. If slaked lime is used 6½ lb. will be needed.

(3) The milk of lime should be stirred vigorously and the copper sulphate solution poured into it and the mixture stirred vigorously for two minutes.

If the lime is pure and free from flint, chalk or other adulterants, the Bordeaux mixture will be alkaline. To test it dip a red litmus paper in the final solution. The paper should turn blue; if it does not, more lime should be added. The mixture should then be stirred afresh before being poured through a strainer into the sprayer. Bordeaux mixture should be used the day it is made.

White Bordeaux mixture contains a large excess of lime and is used mainly for fruit trees in the proportion 4 : 8 : 100. It is made up as described above, but a litmus test is unnecessary.

Where Bordeaux mixture is to be prepared in bulk it is usual to have the casks of copper sulphate solution and milk of lime on a high staging which the two solutions flow down into a lower cask in which they mix. From the latter the prepared mixture is run into the sprayer.

Any deposit of Bordeaux mixture on vegetables, etc., for the table may be removed by rinsing in dilute acetic acid (1 : 50 by volume).

The French doctor and botanist Millardet studied downy mildew of the vine in the vineyards round Bordeaux. In 1882 he observed that the farmers had sprinkled vines beside the road with a mixture of copper sulphate and lime to discourage passers-by from stealing the grapes. These sprinkled vines were free from the mildew which was prevalent farther inside the plantation. By experimenting with these ingredients he obtained a solution which was tried successfully in 1883. In 1884 there was too little mildew for conclusive experiments. On 1st May 1885 Millardet published

an account of the good results obtained with the solution which has been known ever since as Bordeaux mixture (*Phytopathological Classics* No. 3, 1933). The same year Jouet tried it on tomato plants and in 1891 Prillieux used it on potatoes.

As soon as the principle of using a copper solution with an alkali had been discovered several other similar fungicides were devised. Bordeaux powder and a copper ammonia solution were used in 1885 and a copper soda solution (Burgundy mixture) in 1887. The first experiment with Bordeaux mixture in Denmark was made in 1886 but lack of suitable sprayers prevented its use on a commercial scale until much later.

The precise nature of Bordeaux mixture is still somewhat obscure. A copper sulphate solution in water is acid, and a solution of quicklime in water (limewater) is alkaline. If dilute solutions of the two at the rate of 10 of the former and 3 of the latter by weight are mixed a neutral solution is obtained. The mixture is a colloid which slowly becomes precipitated. If it is sprayed on to leaves and left to dry the mixture forms a coating that adheres well.

Bordeaux mixtures are complex in nature and not very stable. Their composition varies with the relative proportions of copper sulphate and lime and with dilution and temperature. As already mentioned, several different formulæ are in use for different purposes.

Evaluation of Bordeaux mixture. The simplest method of evaluating a Bordeaux mixture is to measure its stability, the suspensive capacity of its particles. One pours the mixture into a tall glass vessel, noting the level of the sediment from time to time. By simple experiments one can demonstrate that stability is greatest in cold solutions, in dilute solutions and in those where the copper sulphate was poured into the milk of lime, not vice versa. If one cools a solution from 64.4 to 55.4° F. or from 55.4 to 50° F. its stability is approximately doubled (Butler, O., 1915). If the limewater is poured into the copper sulphate solution a coarse sediment with very low stability is obtained, but a very good mixture results if the copper sulphate solution and limewater are allowed to run together, each from its own container, and mixed in a third. Vigorous stirring while mixing increased stability. In America "Express Bordeaux" (Instant Bordeaux) is much used. Here the copper sulphate is first dissolved in a large quantity of water in the tank of the sprayer, and the lime is placed on the strainer and washed down with more water. The mixture is, of course, used at once.

The persistence of Bordeaux mixture has been examined particularly on hop leaves and cabbage leaves. It has considerable adhesive capacity in itself, but both its dispersal and stability may be increased by the addition of various colloids.

Butler recommended adding sugar, but casein is better (Ramsay, A. A., 1921). In practice casein is often added in the form of skimmed milk and gives a mixture that spreads well but the dried film is apt to scale off the leaves. Addition of gelatine or carpenter's glue gives a fine spreading spray and a more lasting film. Addition of soap as a rule tends to make the mixture coagulate, but addition of a potash resin soap gives a Bordeaux mixture which can moisten leaves of cabbage and other wax-covered plants and give a film that persists well (Newton *et al.*, 1930). Addition of iron sulphate reduces the adhesive property (Butler and Smith, 1919). The lime used also affects the stability of the mixture. The best is a clear limewater but this will only make a very dilute Bordeaux mixture. The next best is a chemically precipitated lime, then milk of lime and thoroughly soaked hydrated lime (Holland *et al.*, 1926, 1927). Adhesiveness is reduced by either too little or too much lime; the ratio 8 : 4 : 100 seems to adhere best. Pure lime does less damage to cylinders and nozzles than lime contaminated with silicon or other hard particles. Rather less hydrated lime is needed if it is finely divided than if it is coarse (Cunningham, G. H., 1935).

The effect of Bordeaux mixture as a fungicide depends largely on the dried-up layer protecting the plant for a considerable period against reinfection. The direct action in killing

fungus spores already present is of less value. Some fungus spores, at least, excrete small quantities of organic acids (Cerasoli, E., 1921) which may act on the film of Bordeaux mixture, dissolving a small quantity of copper which within a few hours is absorbed by the spores and causes them to lose their capacity for germination. This is the case, for example, with conidia of *Sclerotinia* (Goldsworthy and Green, 1936). The copper in the film of Bordeaux mixture is also gradually liberated by the oxygen, ammonia and nitrate in the air, as well as to a very small degree by secretions of the plant. The liberated substances are dissolved in rainwater and washed into the soil. Naturally the activity of Bordeaux mixture depends upon its composition. For example, against apple scab or potato blight, Bordeaux mixture 8 : 4 : 100 is 4 times as toxic as 8 : 8 : 100 or mixtures still higher in lime (Doran, W. L., 1923); but, as is well known, Bordeaux mixtures low in lime may cause much scorching in apple trees.

The different reactions of plants to Bordeaux sprays have led to numerous modifications of the formula for particular crops. There seems to be a great difference in behaviour between plants with a very acid cell sap and those with less acid sap.

Bordeaux mixture affects the growth of the plants, not only if they are deficient in copper (see p. 36). Potatoes sprayed early develop more leaves and a heavier yield of tubers; later spraying gives fewer leaves and lower tuber weight. On both vine and potato Bordeaux mixture may stimulate chlorophyll formation and cause thickening of the leaves. It may lengthen the growing period and retard ripening, e.g. in tomato. It is an old experience in North America that Bordeaux mixture helps to protect potato against tip-burn of the leaves, due to heat and insects. It is therefore surprising that several research workers have found that a film of Bordeaux mixture increases the transpiration of leaves. In fact the effect on transpiration is not a simple one and it is difficult to compare results obtained with thin-leaved greenhouse plants with others on outdoor plants. Bordeaux mixture 8 : 12 : 100 did not increase transpiration of stock, increased it very little in celery but much in coleus, hollyhock, bean and tomato (Wilson and Runnels, 1934); potato (Duggar and Cooley, 1914); and cherry (Dutton and Wells, 1924). If warm weather occurs at the time of spraying, Bordeaux mixture increases transpiration more than when warm weather occurs after spraying; that seems to be the effect of the lime rather than of the copper. The shading effect of the Bordeaux mixture film has also to be reckoned with. It is usually beneficial (under certain conditions Bordeaux mixture may diminish transpiration by day and increase it at night). Finally it is an open question whether increased evaporation from the leaf has anything to do with the eventual scorching by Bordeaux mixture (Butler, O., 1923; Horsfall and Harrison, 1939).

Small tomato plants are easily damaged by drought if sprayed with Bordeaux mixture immediately after re-potting. The damage may be reduced by adding 1% white oil to the Bordeaux mixture and is prevented altogether by not spraying until 5–6 days after re-potting (Wilson and Runnels, 1937).

Spray damage was more fully described on p. 45; it may be due to copper penetrating into the base of hairs through pores or through insect wounds. The effect of copper on the leaf epidermis is uncertain. Lime does not in itself counteract spray damage, for example, vine is sensitive to excess of lime, but it hinders the liberation of appreciable quantities of free copper ions.

As already mentioned, Bordeaux mixture 8 : 2½ : 100 is neutral but when left standing it becomes feebly acid. Some plant species cannot endure mixtures containing excess lime; for instance tomato does not like Bordeaux mixture 8 : 24 : 100 but stands 24 : 8 : 100 quite well (Shutak and Christopher, 1939).

Bordeaux mixture 8 : 4 : 100 is permanently neutral provided it is made with pure quicklime. The reaction can be tested with red litmus powder which should turn distinctly blue. Bordeaux mixture 16 : 8 : 100 has usually given best results with potatoes and is easier to spray than 2 : 2 : 100. A very strong mixture

of the neutral type is Osterwalder's 48 : 24 : 100 (see p. 582).

Bordeaux mixture 4 : 8 : 100, "white Bordeaux mixture," is used particularly for spraying apple trees.

Massey (1939) found Bordeaux mixture 2 : 1 : 100 particularly suitable for use all the year round as a spray against black spot on roses.

Bordeaux mixture 4 : 1 : 100, which may be expected to contain a certain quantity of free copper ions, is tolerated by pecan trees (Cole and Large, 1936).

Ready mixed Bordeaux powders have been brought on the market several times but have not been found cheaper or better than the home-made mixtures. Bordeaux creams are, as a rule, expensive in regard to their very small copper contents.

Bouisol, a proprietary copper fungicidal spray, is used in the same way as Bordeaux mixture and is tolerated to about the same extent as white Bordeaux mixture. It has the advantage that it causes hardly any spotting and is thus very suitable for use on flowering plants such as rose.

Against *Botrytis* on tomato (p. 372) a bouisol and oil combination may be used. A bouisol and white oil emulsion is also recommended against rose mildew under glass (Orchard, O. B., 1937).

Brassicol (20% pentachlor-nitrobenzene + 80% inert filler) has been found in Denmark to control cabbage club root and brown rot of tomato when used at 1¾ oz. per sq. yard. Planting may be done soon after the dust has been mixed with the soil, as long as the sun does not shine on the soil for an hour after treatment. The more thorough the mixing the better; for example, the dust may be well mixed with superphosphate or sand to increase its bulk and make it easier to distribute evenly over the area to be treated.

Dusting with Brassicol has given good control of *Botrytis* but, especially in sunshine, it may scorch delicate plant tissues. Both Brassicol or a Brassicol and talc mixture (1 : 4 at a rate of 893 lb. per acre) gave good control of clover rot (*Sclerotinia trifoliorum*) (Gram, E., 1944). Its possible use against *Sclerotinia* rot of other plants (see p. 77) and *Fusarium* wilt of asparagus (p. 234) needs further investigation.

To control onion smut the seed may be slightly damped and mixed with Brassicol at the rate of ½ lb. per 1 lb. of seed.

Soil for seed boxes and transplanting boxes may be disinfected with 10 oz. Brassicol per cubic yard to prevent damping off. Seed beds out of doors may be treated with it at the rate of 1 oz. per sq. yard. To disinfect deeper layers as for flower bulbs, use 2–3 oz. per sq. yard. Brassicol mixed with sand at the rate of 1 oz. to 9–20 gallons (4½–10 pecks) may be used to cover soil in controlling the propagation fungus.

Brassisan (20% trichlor-dinitrobenzene + 80% filler) is active against club root; for seed beds and seedlings the German recommendation is 1½–2 oz. Brassisan per sq. yard, for cabbage fields 700–900 lb. oiled calcium cyanamide followed a week later by 180–210 lb. Brassisan per acre.

Burgundy mixture is easier to prepare and spray than Bordeaux mixture and has a similar effect on potato blight and downy mildew of vine. It is apt to scorch fruit trees. The recommended proportions are copper sulphate 4 oz., washing soda 5 oz., water 2½ gallons. The copper sulphate is dissolved in a little boiling water and the washing soda, which should be fresh and crystalline, in the rest of the water. While the latter is being vigorously stirred the copper sulphate solution is poured in. Burgundy mixture should preferably be used in 24 hours. There is no need to strain the mixture. It cannot be tested with litmus paper so care must be taken to weigh out the chemicals correctly.

Burgundy mixture may also be made by using ready mixed copper soda powders which are on the market.

Burgundy mixture tends to spot leaves less than Bordeaux mixture; its adhesive power is much the same or slightly superior. The same spray mixtures may be made as with Bordeaux mixture (see p. 589), but addition of lead arsenate or Paris green may be very injurious to plants.

Alkaline Burgundy mixture is recommended for summer sprays against American gooseberry mildew (see p. 168); about 6½ oz. copper sulphate, 1 lb. 3 oz. of washing soda and 2½ gallons of water.

Calcium cyanamide is used as an artificial fertilizer and has sometimes had some effect against club rot. In Jorgensen's experiments it had little effect against damping off. It is used to some extent as a weed killer.

Calcium hypochlorite is used as a seed disinfectant (see p. 576). In one experiment when it was dug into soil at the rate of 2½ oz. per sq. yard it gave some protection against violet root rot of carrot. See also packing soil for lilies (p. 488). For watering mushroom beds calcium hypochlorite is used at the rate of 1–4 lb. in 20 gallons water. For cleaning fruit stores a 2% solution of calcium hypochlorite may be used (see p. 580). Apples with fly speck (p. 127) may be washed in 5% calcium hypochlorite to bleach them. For its use on walnuts see p. 222.

Calomel (mercurous chloride). A 4% calomel dust is much used today in controlling cabbage and onion flies. Preston (1941) has shown that one ounce of calomel per 50 plants applied just before planting, either in the dibble hole or along a shallow drill, gave considerable control of club root in cabbage, cauliflower, etc.

Carbolic acid. See cresylic acid (p. 594).

Carbon disulphide is an evil-smelling substance, dangerously inflammable, used particularly against eelworm, cockchafer larvæ and other noxious soil insects. Its fungicidal activity seems to be small but like other soil disinfectants it has a fertilizing effect. Experiments have shown carbon disulphide to have some effect against soil sickness in tree nurseries (p. 53) and brown root rot (p. 376) but only in quantities which make the cost prohibitive.

Thomas and Lawyer (1939), after 3 years experiments in North American orchards, claimed that carbon disulphide killed *Armillaria mellea* without destroying apple roots. The most favourable conditions for the fungicide are a dry soil covered with a surface blanket of moistened earth 3–6 inches thick. Holes are bored 18 inches apart and 8–9 inches deep, 45 c.c. of carbon disulphide poured into each and the holes stamped down. It takes up to 2 months to kill the fungus.

Casein. See spreading agents (p. 602).

Cheshunt Compound is used to disinfect soil against damping off of the seedlings. When used in a thorough watering of the soil it does not damage the growing plants. The compound contains 2 oz. copper sulphate and 11oz. ammonium carbonate well powdered and thoroughly mixed. The dry mixture should be stored 24 hours in a tightly corked glass or stone jar before using. 1 oz. is then dissolved in a little hot water and the solution made up to 2 gallons with cold water. The dry mixture may be stored in an airtight receptacle but if left exposed to the air it gradually loses ammonia and becomes less potent. The solution should be used at once and must not be put in vessels of iron, tin or zinc as it will corrode them (Bewley, W. F., 1921). One pint of the solution is sufficient for an ordinary seed box, 14+ 9 + 2 inches.

Copper ammonia sprays do not produce spots like Bordeaux mixture and are therefore suitable for use on roses, chrysanthemums, etc., under glass. In our experience they have given some control of *Botrytis*. The deep-blue solution obtained by adding excess of ammonium hydroxide to copper sulphate solutions has been called "Eau Celeste" and "Azurin." 4 oz. copper sulphate are dissolved in 25 gallons water and ammonia is added until the sediment that first appears dissolves again, giving a deep-blue, clear liquid. Instead of ammonia, ammonium carbonate may be used, about 8 oz. of which will be needed. With this salt the change to the deep-blue colour is quite obvious.

Eau Celeste is rather unstable and may give off ammonia, which is apt to scald plants in a crowded greenhouse. Also when the ammonia is given off a deposit of copper may remain on the leaves and cause injury.

Copper oxide (red). See p. 576.

Copper oxychloride is a substitute for Bordeaux mixture which is on the market

under several proprietary names. It is recommended largely for spraying potatoes, vegetables, etc., as it is apt to cause spray damage to fruit trees.

Copper soda spray. See Burgundy mixture (p. 592).

Copper sulphate. For sprays a powdered copper sulphate easily dissolved in cold water is used. It should be dissolved in a wooden vessel as copper sulphate corrodes iron and zinc. If the powder is first covered with a gallon or so of boiling water it will dissolve more quickly.

As a winter spray against American gooseberry mildew a 4–6% solution is used (4–6 lb. in 10 gallons). For disinfection of celery seed, see p. 267; spinach, see p. 356; red pepper, see p. 352.

For preparation of Bordeaux mixture see above.

Copper deficiency in cereals (white tip, see p. 36) is controlled by applying 45–70 lb. copper sulphate per acre. It may be mixed with sulphate of ammonia, potassium sulphate or ordinary potassium fertilizer or mixed with enough sand to enable it to be broadcast evenly.

For disinfection of boxes see p. 580.

Coppesan, a proprietary copper fungicidal spray.

Corrosive sublimate (mercuric chloride) is a dangerous poison the sale of which is strictly controlled by pharmacists. It is sometimes sold in tablets of known strength, so prepared that 1 tablet makes 1 pint of a 1 : 1000 solution. It reacts with metals and should be dissolved for use in stone jars or wooden pails, preferably with boiling water. The concentrated solution can then be poured into the required quantity of cold water in a wooden barrel or other vessel. Watering cans for use with sublimate should be painted inside, or one may use an enamelled can.

For use against *Corticium solani* in seed beds, etc., see p. 73. 1¾ oz. corrosive sublimate and 1 lb. saltpetre are dissolved in 22 gallons water and used at the rate of 1 gallon to ¾–1 square yard. Fine turf may be watered with a similar solution against brown patch and Dollar spot, but less is used, say 1 gallon to 2 square yards (see p. 554). The effect of sublimate on *Pythium* is uncertain but it gives some control of club root on cabbage, etc. For this purpose ½ pint of a 1 : 2000 solution is poured into each dibble hole at planting time (Preston, N. C., 1931, 1934).

Against tomato canker (*Didymella*) the plants should be watered several times with 1 : 1000 corrosive sublimate, 1 pint per plant.

Begonia and hydrangea are sensitive to corrosive sublimate. Seedlings of cauliflower and brussels sprout endured only solutions not exceeding 1½ oz. in 22 gallons water. Tomato seedlings are also sensitive. After watering with corrosive sublimate under glass poisoning by mercury vapour has been observed (see p. 40).

According to Ferdinandsen and Jørgensen (1939) conifer seed beds may be disinfected with one of the following solutions at the rate of 1 gallon to 1 square yard:

(1) 1¾ oz. corrosive sublimate in 22 gallons water.

(2) 1 oz. corrosive sublimate—1 pint hydrochloric acid in 22 gallons water.

Because of the difficulty in obtaining corrosive sublimate the organo-mercury preparations are generally to be preferred as seed disinfectants. Corrosive sublimate is, however, still used against a few diseases:

Disease	*1 oz. corrosive sublimate in the undermentioned volume of water*	*Soak and stir for*
	galls.	*mins.*
Cucumber: bacterial leaf spot	6	5–10
Cabbage: black rot	6	20–30
Onion: *Fusarium*	3–6	15
Tomato: *Bacterium vesicatorium*	18	5
Egg plant: *Phomopsis*	12	10
Pea: *Ascochyta*	6	15
Narcissus: root rot	6	60
Pepper seedlings: rot	6	15

Cresylic acid (liquid carbolic acid, 97–99% purity). This is used for soil disinfection in greenhouses at the rate of 1 gallon in 39 of

water per 9 square yards. The soil is first dug, watered with cresylic acid, left for a fortnight, dug again and watered with the same quantity of cresylic acid. It cannot be used for planting until 5–6 weeks later. If all the cresylic acid is to be applied at once 3 pints cresylic acid in 16 gallons water are enough for 3 square yards of light soil; for a heavy soil use 2½ pints in 12 gallons water (see also Russell and Petherbridge, 1913).

Cresylic acid is also active against *Pythium* and club root. As a simultaneous control of damping off and *Corticium solani* one may try watering with 1 oz. corrosive sublimate + 2½ pints cresylic acid in 13 gallons water per 6 square yards. After watering the soil should be ventilated for 3 weeks (C. A. Jørgensen, 1933).

Cuprous oxide. See red copper oxide p. 599).

Dichlornaphthoquinone is a comparatively new spray which has shown some promise against Leaf Spot of black currant (Marsh, 1947).

Dry seed dressings. Several fungicidal dusts containing small quantities of complex organomercury compounds are on sale under proprietary names for use as dry seed dressings. A small quantity of the dust shaken up with seed in a closed container kills fungus spores and mycelium adhering to the surface. Among the preparations of this type on the British market are Agrosan, Ceresan, Harvesan, Leytosan, and Lunasan Seed Dressing (see also red copper oxide, p. 599).

Eau Celeste. See copper ammonia spray (p. 593).

Ferric citrate is sometimes used to control iron deficiency (see p. 36). Because of its chemical composition it is much less readily available to plants than ferrous sulphate. It has been used in Holland against chlorosis and other forms of iron deficiency. It may be applied as a spray at the strength of 3½ oz. in 22 gallons water or, for azalea, 2¼ lb. in 22 gallons water.

It may also be added to Bordeaux mixture at the rate of 10 oz. per 22 gallons but it tends to reduce the adhesiveness of the Bordeaux mixture so that a sticker should then be added as well.

For use with lime sulphur see p. 597.

Folosan is a proprietary pentachlor-nitrobenzene dust (see Brassicol) recommended by its makers for the control of *Botrytis* on lettuce. It is applied to the plants at the rate of ¼ oz. per square yard, preferably when they are wet with dew.

Formalin is a clear, transparent, strongly smelling liquid that should contain 36–40% of the gas formaldehyde. It irritates the skin and mucous membranes and is highly toxic to many fungi and bacteria.

For soil disinfection against club root, *Pythium* and *Verticillium*, etc., 1 pint of formalin in 5 gallons water is used per 2 square yards. It is less effective against *Corticium solani*. After watering the soil is covered with a sack for 24 hours, after which one should attempt to accelerate evaporation of the formalin by turning the soil with a spade. The soil should be quite free from the smell of formalin before it is used, and if the smell lingers it may be helpful to water with dilute ammonia. One of the special uses of formalin is as a disinfectant against *Sclerotinia* rot at the rate of ¾ pint in 4½–9 gallons water per square yard (see p. 79) and against onion smut (see p. 299).

Soil for boxes and pots should be turned over in a heap whilst it is being sprayed with formalin solution (for 1 ton soil use 2¼ lb. formalin in 1½–2¼ gallons water). After thorough turning over with a spade the soil should be covered and then left to air thoroughly like that in the beds.

In America a more convenient method employed is to mix with the soil a formaldehyde dust. For 100 square yards 4½ pints of formalin are mixed with 88 lb. of a carrier made up of 1 part kaolin and 2 parts diatomaceous earth. This is thoroughly incorporated with the soil to a depth of 2½–3 inches. Next day the seed is sown and the soil thoroughly watered (Alexander, L. I., *et al.*, 1931). The method has been used successfully with tomato, celery and various ornamental plants, but we have seen growth of

celery somewhat retarded by formalin treatment. Seeds of campanula, petunia, lettuce, and most crucifers should not be sown until 24 hours after the soil has been treated (Wilson, J.D., and Tilford, P. E., 1933).

Plant boxes, pots, cans and string should be disinfected by soaking in formalin solution 1: 50 and afterwards airing them until needed. Flower pots may be strung along a wire so that they can easily be dipped and pulled out again.

As a summer spray against American gooseberry mildew and other powdery mildews a formalin solution 1: 200 may be used, to which soft soap has been added at the rate of 1 lb. in 10 gallons. Such a solution has the advantage that it does not cause spotting but it may scorch the plants if applied in strong sunshine.

As a winter spray against American gooseberry mildew, formalin has been used at the rate of 3½–4 pints in 22 gallons water. It is useful in destroying moss and larvæ but has no effect on insect eggs. As a disinfectant against fungus spread in transit on tomatoes, beans and other vegetables formalin has the advantage that it quickly disappears. The American Department of Agriculture recommends dipping tomatoes for 2 minutes in a solution of 1¾ pints formalin in 66 gallons water and allowing them to stand 4–5 days before being packed. In a single experiment we have seen good results from such treatment. It is universally agreed that to protect the outdoor tomato crops a Bordeaux spray is to be preferred.

After seed has been sown the seed boxes may be watered with a formalin solution from 1: 500–1: 200 by volume.

Formalin may also be used to treat celery seed against leaf spot (see p. 267) and narcissus bulbs against bulb rot (see p. 497).

Stores may be disinfected by evaporating formalin at the rate of 2½ lb. per 100 cubic yards of space. Evaporation is best done in a saucepan over a lamp or over a special petroleum burner filled up with enough petroleum for the flame to last until the formalin is evaporated and then go out. Mixtures of formalin and potassium permanganate or chloride of lime may be almost explosive and are to be discouraged.

Formalin is also useful for disinfecting water tanks (see p. 580).

Fungex, a proprietary copper fungicidal spray recommended by the makers for use on tomatoes.

Gelatine. See spreaders (p. 602).

Germisan, an organo-mercury disinfectant used in solution, has been used on celery seed to control leaf spot (see p. 267) and on narcissus bulbs (see p. 497).

It was a German proprietary preparation containing cresyl mercuric cyanide.

Green vitriol. See ferrous sulphate (p. 36).

Heptadecylglyoxalidine is a comparatively new fungicide which may in some instances replace lime sulphur. Marsh (1948) reported that in a season favourable to apple scab spraying Worcester Pearmain trees 4 times with a preparation of glyoxalidine reduced the amount of leaf infection by 90% and of fruit infection by 80% as compared with that on unsprayed trees. On the leaves the glyoxalidine treatment provided significantly better control than that given by the standard lime sulphur spray programme. The glyoxalidine spraying caused no damage to foliage or fruit of Worcester Pearmain and only negligible foliage damage to Cox's Orange Pippin. It was also effective against *Pseudopeziza ribis* on black currant.

Hortosan, a proprietary fungicide used for steeping bulbs, tubers, etc., is used at the rate of 1 oz. per 2½ gallons of water to water seed beds for the prevention of damping off.

Hydrated lime can be used to replace quicklime in making Bordeaux mixture, provided it is free from impurities and has not been converted into carbonate of lime. 1½ lb. hydrated lime should be used to replace 1 lb. of quicklime.

Against calcium deficiency (see p. 33) hydrated lime is quite effective.

Ialine Colloidal copper compound, a proprietary copper fungicidal spray is recommended by the makers for use on apple, pear and hop.

Iron sulphate. See ferrous sulphate (p. 36).

Kerol is a proprietary tar-oil preparation found by C. A. Jørgensen (1933) to control *Pythium de Baryanum* and have effect against club root but none against *Corticium solani*. He used it in 1 : 400 of water, at the rate of 9 gallons per square yard. The soil was thoroughly watered 3–4 days later and planted after 3 weeks.

Lime sulphur is supplied by the trade as a reddish-brown liquid. According to the Ministry of Agriculture's specifications it should be clear and free from sludge and have a specific gravity of approximately 1.3. It should contain not less than 18.5% weight in weight of polysulphide sulphur, equivalent to about 24% weight in volume.

Lime sulphur should not be exposed to frost and should be kept in a tightly closed container.

As a winter spray it is used at a dilution of 1 : 9 (10 gallons lime sulphur to 90 gallons water) against peach leaf curl, American gooseberry mildew and vine mildew. It also cleans the trees of moss and lichen.

A lime sulphur spray 5 : 95 (1 : 49 on sulphur-shy varieties) when the young leaves are the size of a florin and the flowers have appeared but not yet opened controls the big bud mite on blackcurrants.

It is used as a summer spray 3 : 100 against American gooseberry mildew and at 1 : 50 to 1 : 100 against apple and pear scab, apple mildew, red spider, etc. For the post-blossom sprays on apples weaker sprays are recommended. For the usual English recommendation see p. 586. The Dutch recommendation is:

(1) Before flowering a lime sulphur spray 4 : 100.

(2) After flowering a lime sulphur spray 1–1¼ : 100.

(3) Three weeks after (2) a lime sulphur spray ¾–1 : 100.

(4) Later a lime sulphur spray ½–1 : 100.

In America a 1⅛ : 100 lime sulphur spray has been found best when the apple scab control and spray damage had both to be considered (Thurston, H. W., and Miller, H. J., 1938).

Sticking agents are often useful but soap should not be used as it curdles the spray. Gelatine, casein and various proprietary preparations may be used with advantage. Lead arsenate reacts with lime sulphur, turning the liquid grey-black, and increasing its fungicidal activity. The same is true of ferrous sulphate which is used especially in Switzerland and Canada in winter sprays, e.g. 10 gallons lime sulphur and 1–2¼ lb. ferrous sulphate in 22 gallons water, and summer sprays, e.g. 2 gallons lime sulphur and 3½ oz. ferrous sulphate in 22 gallons water. In Canada sometimes as much as 4½ lb. ferrous sulphate may be used with 2½ gallons lime sulphur in 22 gallons water. See chlorosis (p. 37).

Spray damage with lime sulphur occurs especially with (1) too strong a jet of spray, (2) at temperatures over 79° F. and (3) on particularly sensitive varieties of apple, etc. Scorching of the leaves may be partly avoided by adding ferrous sulphate to the spray but this does not prevent subsequent shedding of leaves and fruit. Spraying with a petroleum oil 3–4 weeks before or after lime sulphur is very risky.

Lime sulphur as a fungicide has had a curious career. In 1852 Grison wrote in the *Gardener's Chronicle* of lime sulphur for use in mildew control. After that it seems to have been forgotten until it was used as a sheep wash in California (" California mixture "). Then it was tried successfully against scale insects on various fruit trees and in 1902 Lowe and Parrott in New York discovered its effect on apple scab. Its fungicidal activity is somewhat dependent on temperature so that its popularity has suffered some fluctuations. Its effect on apple mildew and red spider is, however, appreciated by all.

The action of the dried film of lime sulphur is a complicated one and seems to depend partly on its power of absorbing oxygen, partly on the alkaline properties of the film which affects the wax coating on scale insects and partly on the slow liberation of free sulphur. As far as apple scab is concerned

lime sulphur both kills the colonies already present and protects the leaf against reinfection (Gloyer, W. O., 1933).

The polysulphide content is considered to be the active agent.

A dry lime sulphur has been produced in America by evaporating ordinary lime sulphur and adding sugar. It may contain for example 70% calcium polysulphide and over 5% pure sulphur but its composition is apt to be variable.

Liver of sulphur is the general name applied to the product obtained by fusing an alkali carbonate with sulphur. That usually employed is crude potassium carbonate, but both potash and soda livers of sulphur are on the market. The predominant constituent is potassium polysulphide or sodium polysulphide respectively. Liver of sulphur was formerly used against powdery mildew, *Corynespora melonis* (see p. 274), carnation rust (see p. 451) and red spider. A spray made up of 14 oz. liver of sulphur and flour paste made from 1 lb. of flour in 22 gallons water scarcely removes the waxy coating of carnations. For plants without a waxy coating 11 oz. liver of sulphur and 1 lb. soft soap in 22 gallons may be used. The danger of spray damage is greater than with lime sulphur which is therefore to be preferred in most cases.

Magnesium sulphate is used against magnesium deficiency (see p. 38). Soil for rhododendrons may have about 3 lb. magnesium sulphate mixed with every 2 cwt. In North America magnesium deficiency is countered by using dolomitic lime (containing magnesium) in preparing Bordeaux mixture.

Malachite Green is a dyestuff recommended by Bennett (1933) for control of brown patch of turf.

Manganese sulphate is used to control manganese deficiency (see pp. 39 and 158). It is usually applied broadcast at the rate of 150 lb. per acre. To apply this small quantity evenly it should first be thoroughly diluted with dry sand. Manganese sulphate may also be applied to growing crops as a spray, at the rate of at most 10 lb. in 20 gallons water. After spraying with this strong solution the plants should be rinsed with clean water. Manganese sulphate may also be dissolved in water and added to ready-mixed Bordeaux mixture. In our experience various apple varieties have been unharmed by 7 oz. manganese sulphate per 22 gallons of Bordeaux mixture. In practice 3½–7 oz. manganese sulphate has often been added per 22 gallons lime sulphur for the pink bud and post-blossom sprays in Denmark.

Milk. See spreaders (p. 603).

Mercuric chloride. See corrosive sublimate (p. 594).

Mercurous chloride. See calomel (p. 593).

Perelan. A proprietary copper fungicidal dust.

Perenox. A proprietary copper fungicidal spray recommended by the makers for use on celery, hops, potatoes and tomatoes.

Petroleum emulsion for control of powdery mildew, aphides, red spider and woolly aphis may be made up as follows: Mix 1¾ pints petroleum with 3½ oz. train oil and cresol soap, and stir carefully into 2¼ lb. soft soap dissolved in 1¾ pints water. Then stir vigorously into 22 gallons water. If the plants can stand it the mixture may be used at double strength.

Phenyl mercury chloride has shown some promise at reducing brown rot of plums when added to an oil emulsion winter spray (Marsh, 1946).

Pomarsol (formerly 0672) is a copper-free organic compound of sulphur much used as a fungicide on the Continent. A solution of 27 oz. in 22 gallons water controls apple and pear scab, grey mould on vines, tulip fire, etc. It may be especially valuable on apple varieties which cannot stand copper sprays.

Potassium permanganate has sometimes been used as a very weak fungicide, at the rate of 1–5 oz. in 4½ gallons water per square yard. It has only a slight disinfectant capacity but its potassium content may be of some value and it may stimulate root growth of seedlings.

Potassium polysulphide. See liver of sulphur (above).

Lime is used to improve the physical constitution of soil and reduce its acidity

and also has an indirect effect on club root of brassicas. Its value as a direct fungicide is otherwise very small (see p. 29).

Quicklime is an essential ingredient of Bordeaux mixture. Whitewashing of tree trunks destroys moss and lichens but is of little value compared with a regular winter spray.

Red copper oxide (Cuprous oxide) in a finely divided condition (300 mesh) is used as a seed disinfectant (see p. 576). The powder should be at least 95% CuO.

Salicylanilide. See shirlan (below).

Salicylic acid has been used as a spray against powdery mildew. 3½ oz. salicylic acid may be dissolved in 1¾ pints methylated spirit and poured into a solution of 4½ lb. soft soap in 22 gallons water. It may scorch vines and peaches as well as roses in full bloom. On account of the soap content this spray kills aphides and leaf-hoppers.

Salt (common salt, sodium chloride). A 2–4% solution of common salt, equivalent in strength to sea water, has sometimes been used against American gooseberry mildew. It may scorch the leaves.

Saponin. See spreaders (p. 603).

Seed disinfectants. See dry seed dressings (p. 595), red copper oxide (p. 576).

Seo. A proprietary copper fungicidal spray.

Shirlan AG (salicylanilide emulsified with Agral). At a strength of 10–21 oz. per 22 gallons water this makes a good spray against tomato leaf mould. According to Dutch experience a stronger solution (22–43 oz. per 22 gallons water) prevents tulip fire. It has also been recommended against powdery mildews, but sulphur fungicides are generally preferred for this purpose.

Skimmed milk. See spreaders (p. 603).

Slaked lime is made by slaking quicklime with half its weight of water (e.g. 20 lb. quick lime—1 gallon water), when the lime forms a very fine powder. Added to soil slaked lime acts much like ordinary agricultural carbonate of lime. If not preserved in tightly shut containers slaked lime is gradually converted into carbonate of lime. Slaked lime will burn the eyes.

Soap. See spreaders (p. 603).

Soap-soda solution. 5½ lb. soft soap + 5½ lb. washing soda to 22 gallons water has been used as a spray against American gooseberry mildew but may cause scorching of leaves and fruit, especially if the solution is not carefully made up or if the spraying is done in bright sunshine.

Sodium chlorate is very poisonous to flowering plants and therefore valuable as a weed-killer. A 1% solution is also used to kill potato haulm in autumn, thus checking tuber infection and making the crop easier to lift. It should be used with care as clothing soaked in sodium chlorate and afterwards dried is highly inflammable. Also if used too freely there is a risk that the chlorate may not be washed out completely during the winter and enough may remain in the soil to poison next year's crop. Potted plants may also be poisoned if the pots are stood on paths recently watered with sodium chlorate as a weed-killer (p. 34).

Sodium hypochlorite may be used to disinfect fruit boxes, stores, etc., instead of chloride of lime (see p. 593). The commercial product has a somewhat variable chlorine content. Baker and Heald (1934) found spraying picking boxes and packing equipment with a sodium hypochlorite solution containing 0.4% available chlorine helped in control of blue mould of apples.

Spersul. A proprietary sulphur fungicidal spray.

Soltosan. A proprietary copper fungicidal spray.

Spraying oil. See white oil (p. 601).

Sulphate of ammonia is an "acid" nitrogenous fertilizer that neutralizes the lime content of a soil and is therefore especially suitable on alkaline or overlimed soils. In manurial experiments on a light sandy soil over a period of years every 100 lb. sulphate of ammonia neutralized 150–200 lb. calcium carbonate.

Sulphocloud, a proprietary sulphur fungicidal dust.

Sulphur. The reason for the great fungicidal activity of sulphur is not clear. Conversion to hydrogen sulphide or sulphur dioxide has

been found of no significance in this connection. The important reaction seems to be a formation of polysulphides. The activity of sulphur depends largely on temperature. It is too feeble at low temperatures and may be injurious to the plants in bright sunshine and warmth (cf. p. 45). The more finely divided the sulphur deposit the greater is its fungicidal activity.

For dusting in mountain vineyards crushed sulphur is used, a loose powder made up of crystalline fragments. For use in a blower a fine-grained sulphur is preferred. Flowers of sulphur, produced by sublimation, and sulphur flour produced by grinding are objectionable because the fine powder tends to cohere in small balls difficult to break down. This tendency may be overcome by mixing it with a small percentage of inert matter such as kaolin, lead arsenate, bentonite, etc. (see sulphur dusting, p. 587).

Green sulphur is an impure by-product of the coal-gas industry.

Ferrox sulphur is a similar substance prepared by a slightly different process which may leave 2–6% ferric oxide in the final product.

Grey flotation sulphur is produced in the liquid purification of water gas.

Ordinary finely divided sulphur requires addition of a wetter if it is to be used in a spray. Soap is often used, especially when the sulphur is added to a nicotine wash. In America a mixture of sulphur, casein and hydrated lime has been used as a substitute for lime sulphur. These " wettable " sulphurs are apt to have the disadvantage that their particles do not remain in suspension in the spray tank unless continually agitated. By special processes, however, it is possible to obtain very minute particles that easily remain in suspension and are termed " colloidal " sulphurs. The earliest reference to the use of sulphur as a plant protectant seems to be that by Robertson to the London Horticultural Society, 1821, and dealt with a spray of sulphur mixed with soapy water against peach mildew.

Sulphur vapour is also used, especially in the control of powdery mildew under glass and to some extent against gummosis and blotch in cucumber. The practice of painting the hot-water pipes of vine houses with sulphur mixed with linseed oil is also a form of slow vaporization (Barker, B. T. P., Gimingham, C. T., and Wiltshire, S. P., 1920). The temperature of the pipes should be between 140° and 176° F.

Sulphur is usually evaporated in generators at the rate of $1\frac{3}{4}$ oz. per 100 cubic yards of glasshouse space. Generators in which air is pumped through the molten sulphur are to be preferred. When steam is used hydrogen sulphide may be formed (Barker and Wallace, 1922). The sulphur vapour settles on the plants in a fine film which adheres well.

The melting point of sulphur is about 115° C., its ignition point in air about 260° C. and its boiling point about 444° C. It is important that the sulphur is not allowed to catch fire as then sulphur dioxide is formed. When sulphur is evaporated in bright sunshine flowers should be shaded. Those of tomato and antirrhinum, for example, may be scorched by sulphur vapour.

Burning of sulphur is practised in greenhouses that have been emptied of plants at the end of a season. The purpose is to kill fungus spores on walls, staging and soil surface. The sulphur dioxide formed is very poisonous to living plants (see p. 50). In the case of a row of glasshouses the fumes may penetrate from the house being fumigated into those adjacent, for glass walls are not usually airtight enough to keep them back. For the reaction of sulphur dioxide with zinc oxide and galvanised pipes see p. 50.

$2\frac{1}{2}$–7 lb. of sulphur are used per 100 cubic yards of house. $2\frac{1}{2}$ lb. is a small dose, insufficient to kill tomato leaf mould in leaky houses. In mushroom houses 17 lb. sulphur per 100 cubic yards are used during fermentation. To disinfect empty fruit stores, etc., $4\frac{1}{2}$ lb. per 100 cubic yards is enough. A small fire of charcoal or wood chips moistened with petroleum is made on some fire-resistant surface. On this is placed the sulphur, the fire is lit and the house kept closed overnight.

In a Danish chrysanthemum house we found

that burning 5 lb. sulphur per 100 cubic yards after forcing was over greatly checked *Botrytis* on the flower stalks but did not affect the sclerotia of *Sclerotinia* which germinated freely after the treatment. Sulphur dioxide has no effect on fungi protected by even a thin coating of soil, hence soil disinfection is necessary as well (see p. 577).

Sulphur may be used to change the reaction of a soil. Powdered sulphur mixed with soil gradually becomes converted into sulphuric acid and if the soil has been over-limed the sulphur will correct the excess alkalinity. The quantity needed can be determined by laboratory analysis; it may amount to 450–900 lb. per acre. When a quick and marked change in reaction is not imperative the use of sulphate of ammonia is more economical (see p. 31). Some American workers have advocated adding sulphur to the potato drill to control common scab (see also lime sulphur, p. 597; liver of sulphur, p. 598; pomarsol, p. 598; sulsol, below).

Sulsol is a sulphur fungicide used, amongst other things, instead of lime sulphur on sulphur-sensitive apple varieties. 1 lb. per 20 gallons water is used. Against rose rust 21 oz. sulsol + 10 oz. soft soap in 22 gallons water has been quite effective and caused only insignificant spotting of the plants.

Thiocarbamates are complex organic compounds of iron or zinc which show some promise as fungicidal sprays for fruit (Marsh, 1947).

Tillantin (Danish) an organo-mercury fungicide used against *Sclerotium rolfsii* on orchids (see p. 501) and against crown gall in seed beds (p. 83) at the rate of 1 lb. in 20 gallons water. In making a clay gruel to dip roots against crown gall a solution containing 2 lb. per 20 gallons is used.

Danish Tillantin and other proprietary organo-mercury fungicides may be used for spraying the soil of seed boxes and of boxes to which seedlings are to be transplanted about 8 days before use. For this purpose 1¼ oz. should be dissolved in enough water to wet 1 cubic yard of soil thoroughly. Some use 9 oz. per cubic yard but if so the soil should be left 3 months before being used, e.g. for cucumber, melon or tomato. For bench soil for the same plants 1¼ oz. in 2¼ gallons is used per square yard.

Tillantin 1875 is a dry seed dressing.

Train oil cresol soap used in emulsifying petroleum and other spraying oils is made as follows: 11 lb. veterinary liver train oil and 11 lb. cresol (raw carbolic acid) are mixed and heated almost to boiling point. Then carefully add 2¼ lb. commercial caustic potash and stir well. The product may be used immediately but is improved by keeping.

Uspulun, an organo-mercury fungicide containing chlorphenol mercury, was designed primarily as a steep for seed disinfection but used also against soil fungi. For the former purpose it has been replaced by the organo-mercury dry seed dressings and is no longer on the market.

White lead paint is used to cover pruning wounds on trees, especially to prevent infection with *Stereum purpureum* (see p. 192).

To improve its appearance a little black paint or lamp-black may be added.

White oil is a highly refined emulsified petroleum oil sold under various trade names. White oils are used particularly against insect pests in glasshouses but they also have some effect on powdery mildews and other fungi.

It is generally known that a white oil used 3–4 weeks before or after lime sulphur, sulphur dust or other sulphur fungicide, may cause severe scorching. A similar effect has sometimes been observed with white oil following Burgundy mixture and it may be unsafe to use it after Bordeaux mixture or other copper sprays.

White oil may also cause scorching if applied in bright sunshine.

Winter spray oils. Numerous proprietary preparations are on the market and have been approved by the Ministry of Agriculture (see p. 588).

Zinc oxide. As a fungicide see p. 576.

LIST OF SPREADING AGENTS

Spreaders (spreading agents and sticking agents). The behaviour of a spray on a leaf

Spreading Agents

depends on several chemical and physical factors. Even the force with which the spray strikes the plant may play some part. It is well known that a misty spray may be very satisfactory while a jet directed straight on to the foliage may cause a great deal of damage.

The electrical conditions are important but difficult to control. Most damp leaves carry a negative charge and the same is true of the spray particles in most of the common preparations. Actually one has to distinguish between sticking agents, spreading agents and stabilizing agents, but as most substances used act in more than one way we are considering them all under one heading, namely as additions made to a spray to increase or extend its effect.

Spreading power refers to the area the mixture can cover and spread out over and may therefore be different on bark, a mildewed surface, fruit and various kinds of leaf. In evaluating it one examines the foaming capacity of the spray, its ability to wet a waxed carton as well as hop and cabbage leaves and perhaps its capacity to wet certain textiles.

Sticking power is an expression of the length of time the dried liquid can remain on the sprayed plant. It is increased when the particles of the fungicide or insecticide are small; hence, for example, Bordeaux mixture should be made in cold and dilute solution (see p. 589). Sticking power is increased by the addition of various substances like starch (flour paste) glue, molasses, dextrin. The kind of clay called bentonite has a corresponding effect on dusts. Sticking power may sometimes be too great, thus lead arsenate should not stick to fruit so thoroughly that rain cannot wash it off before the fruit is picked.

Stability is a term to express how well the particles of the spray can keep in suspension when the mixture is left to stand. Stability increases with fineness of particles. Addition of glue, gelatine, sulphite lye and other "protective colloids" may greatly reduce the rate of precipitation of the particles.

As a rule a spreading agent or sticking agent is desired to be chemically inert but in practice this means that it is desired to increase the activity of the other ingredients. In practice a spreader may increase the spreading capacity of the spray but may have other less desirable effects such as checking the liberation of copper, sulphur or arsenic. Its collective action has to be found out by experiment.

Agral is a sulphonized oil used as a spreader or wetting agent, e.g. in shirlan (see p. 599).

Alcohols. Lauryl and other higher alcohols are used, usually as sulphates, in some proprietary preparations. So are secondary alcohol sulphates, by-products of the petroleum industry. They may be used in most spray mixtures, are harmless to plants and ultimately evaporate.

Sulphonation products of terpene alcohols have been used as spreaders in France.

Carpenter's glue, 3½ oz. per 22 gallons of spray, may be used instead of gelatine and is much cheaper. It should be set to soak some time before and be dissolved in boiling water.

Casein is the albuminoid separated from skim milk by addition of rennets or acids. At 3½ oz. per 22 gallons of spray it increases the spreading power of Bordeaux mixture or lime sulphur but not the sticking power. Casein is insoluble in water and should therefore be mixed with an equal part of washing soda the day before it is to be used, stirred into water and left there to dissove. Lime casein or calcium casenate is an intimate mixture of hydrated lime and casein and can be used direct.

Cresol soap. See p. 601.

Flour. Addition of 18 oz. wheat flour to 22 gallons of liquid increases the sticking power. The flour is carefully mixed into a fairly smooth gruel, is then slowly diluted to 2 gallons water and stirred into 20 gallons of the spray. The flour may be cooked into a paste in advance.

Gelatine. 2½ oz. gelatine per 22 gallons of liquid may be added to all the common sprays to increase their spreading and sticking capacity. It may slightly increase the quantity of soluble arsenic, which can be counteracted by mixing lead arsenate with lime. Gelatine dissolves in warm water and is then added to the prepared spray.

Glue. See carpenter's glue (above).

Lime does not increase either spreading or fungicidal power. When lime and lead arsenate are mixed together (e.g. 28 oz. slaked lime + 14 oz. lead arsenate) it is to prevent gelatine or casein releasing soluble arsenic and causing spray damage. Lime slightly reduces the fungicidal capacity of lime sulphur but this is increased by the lead arsenate (Goodwin, W., and Martin, H., 1925).

Molasses, 2–4 lb. per 20 gallons Bordeaux mixture, increases the sticking power. Use of large quantities of molasses is not advised as they may attract bees which will be poisoned if the spray contains, for example, lead arsenate. When added to lime sulphur the smell of the latter masks that of the molasses and bees are not attracted.

Oils. By sulphonation of oils (treatment with sulphuric acid) various spreading agents have been produced (e.g. Agral, Penetrol). White oils are also useful for spreading and sticking but may be objectionable if they cause lead arsenate to adhere to fruit too long. In English experiments cottonseed oil and other plant oils have reduced spray damage by Bordeaux mixture on apple trees. The oil is emulsified by stirring into ready-made Bordeaux mixture and is then a good carrier of nicotine (Austin, M. D., Jary, S. G., and Martin, H., 1932).

Resinates are obtained by treating resin with alkali. They have been employed to adulterate soaps, and hence in England the percentage of resin acids in soft soaps for spraying purposes has to be declared. Resinates can be used in Bordeaux mixture and lime sulphur for use on cabbage and other wax-coated plants but must not be added to sprays containing Paris green. The resinate consists of $4\frac{1}{2}$ lb. resin, $2\frac{1}{4}$ lb. potassium carbonate and $1\frac{1}{2}$ gallons of water. Potassium carbonate is added to boiling water and then the resin which has previously been dissolved by boiling for some time. 7–16 oz. of resinate go to 22 gallons of spray.

Saponins are a group of glucosides, obtained commercially largely from the inner layers of the bark of *Quillaja saponaria*. There are a number of preparations on the market but ordinary soft soap is usually preferable. Saponin does not form chemical combinations with any of the usual sprays.

Skim milk. Addition of 1 gallon skim milk or $3\frac{1}{2}$–7 oz. skimmed milk powder to 22 gallons Bordeaux mixture has proved useful on potato. For smooth-leaved plants it may be inadequate as a spreader.

Soap may be used with nicotine ($2\frac{1}{4}$ lb. soap to 22 gallons water) and with formalin and salicylic acid. Soft soap (potassium soap) is generally used. When added to Paris green, lead arsenate and other arsenic compounds, soaps may render substances soluble that will cause spray damage. Soap makes lime sulphur curdle and cannot be used with it. It is probably the oldest spreading agent. Soap is easily combined with other chemicals; even in "hard" water a little is precipitated in a compound with lime. Much more is precipitated in Bordeaux mixture thus reducing its spreading power.

Sugar behaves like molasses and is used at the rate of 9 oz. per 22 gallons of liquid.

Train oil cresol soap. See p. 601.

LITERATURE

Throughout the text references have been given to the more important sources of information. In editing the English edition an attempt has been made to include references to most of the more recent British papers on the diseases dealt with. These should be fairly easily accessible to many readers but they are likely to find difficulty in consulting the Continental and American papers cited. Excellent summaries of most of those published since 1922 will, however, be found in the *Review of Applied Mycology*, either in the volume for the year in which the paper was published or in that for the following year. The original papers are often available to serious students in the library of the Commonwealth Mycological Institute, Kew, Surrey, which publishes the *Review*.

Periodical summaries of plant diseases reported in England and Wales are also published as Bulletins of the Ministry of Agriculture and Fisheries. These have been

freely consulted and quoted in the text of Parts II–IV, especially Bulletins. 126 and 139, covering the period 1933–46. Informative accounts of many of the more important British plant diseases are available in Advisory Leaflets of the Ministry of Agriculture and Fisheries. A list of these is appended but not all may be in print. There are also a few leaflets on diseases and pests of forest trees issued by the Forestry Commission. Much valuable and topical information can be gleaned from the Annual Reports of Horticultural Research Stations, notably those at Long Ashton, East Malling and Cheshunt.

Following the list of Advisory Leaflets will be found a list of authoritative handbooks likely to be useful to persons wishing to undertake further studies in plant diseases.

In each country of Western Europe and North America there is usually one or more journals devoted largely to plant pathological papers. Work on the more academic aspects of plant-disease fungi is often published in the transactions of the various mycological societies. A list of such journals, arranged geographically, appears below.

Advisory Leaflets, Ministry of Agriculture and Fisheries

No. 38 Mosaic Disease and Streak of Tomato.
No. 53 Tomato Wilt or " Sleepy " Disease.
No. 62 White Rot Disease of Onion Bulbs.
No. 85 The Downy Mildew of the Onion.
No. 99 Powdery Scab of Potatoes.
No. 100 Apple and Pear Canker.
No. 107 Black-leg of Potatoes.
No. 139 Potato Virus Diseases.
No. 198 Gooseberry Cluster Cup Rust.
No. 204 Dieback Disease of Gooseberries.
No. 205 Apple Mildew.
No. 207 Powdery Mildew of the Vine.
No. 218 Dry Rot of Potatoes.
No. 241 Leaf Spot of Celery.
No. 245 Apple and Pear Scab.
No. 246 Silver Leaf Disease of Fruit Trees.
No. 248 Brown Rot and Allied Diseases of Plum.
No. 253 Crown Gall.
No. 261 Onion and Leek Smut.

No. 263 Tomato Leaf Mould.
No. 265 Sclerotinia Disease of Potatoes.
No. 273 American Gooseberry Mildew.
No. 274 Wart Disease of Potatoes.
No. 276 Club Root.
No. 277 Reversion in Blackcurrants.
No. 278 Potato Leaf Roll.
No. 279 Skin Spot and Silver Scurf of Potatoes.
No. 290 Spraing, Internal Rust Spot and Net Necrosis of the Potato Tuber.
No. 291 Black Scurf and Stem Canker of the Potato.
No. 292 Pink Rot and Watery Wound Rot of Potatoes.
No. 296 *Verticillium* Wilt and the Black Dot Disease of the Potato.
No. 319 Soil Sterilization.

Department of Agriculture for Scotland

No. 77. Raspberry Diseases in Scotland.

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INDEX

USE OF THE INDEX

Because the crop plants are alphabetically arranged in three categories, tree and bush fruit (pp. 101–224), vegetables and herbaceous fruit (pp. 231–398) and ornamental plants and trees (pp. 407–564) their names are not entered again in the index. The reader should note that strawberry diseases are dealt with in Chapter 3, pp. 358–68. In the first two categories the plants are arranged under their English names, in the third the botanical names are used throughout but adequate cross references are inserted to enable the reader familiar with the common English names to find the disease of the plant he seeks. The index therefore contains the scientific names of the pathogens and such common names of diseases as are distinctive. Vague general names such as leaf spot, rust, etc. are not indexed. Thus to find antirrhinum rust the reader will turn to antirrhinum in Chapter 4 or look up *Puccinia antirrhini* in the index. The therapeutant chemicals and spreading agents discussed alphabetically on pp. 588–603 are not entered again in the index.

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