PAGES MISSING WITHIN THE BOOK ONLY

(67 TO 112)

UNIVERSAL
LIBRARY

OU_156580

UNIVERSAL
LIBRARY

OSMANIA UNIVERSITY LIBRARY

Call No. 581.2 / M51E Accession No. G 11755

Author Mathews, Irving E., and
Icent, George C.

Title Elements of plant pathology. 194?

This book should be returned on or before the date last marked below.

Elements of

PLANT PATHOLOGY

THE MACMILLAN COMPANY
NEW YORK · BOSTON · CHICAGO · DALLAS
ATLANTA · SAN FRANCISCO
MACMILLAN AND CO., LIMITED
LONDON · BOMBAY · CALCUTTA · MADRAS
MELBOURNE
THE MACMILLAN COMPANY
OF CANADA, LIMITED
TORONTO

Elements of Plant Pathology

BY IRVING E. MELHUS
Professor of Plant Pathology
and Head of the Department of Botany

AND GEORGE C. KENT
Instructor in Plant Pathology

IOWA STATE COLLEGE

NEW YORK

The Macmillan Company

1948

COPYRIGHT, 1939,
BY THE MACMILLAN COMPANY

All rights reserved—no part of this book may be reproduced in any form without permission in writing from the publisher, except by a reviewer who wishes to quote brief passages in connection with a review written for inclusion in magazine or newspaper.

Published May, 1939

Reprinted June, 1948

— PRINTED IN THE UNITED STATES OF AMERICA —

PREFACE

The *Elements of Plant Pathology* is an attempted solution of a practical teaching situation from the standpoint of subject matter. The chief contribution of the book lies in the emphasis placed on parasitism in disease processes and the principles relating to control measures, coupled with the condensation and omission of unnecessary morphological and mycological data. In its development three obstacles had to be overcome. First, many of the students had no intention of becoming professional plant pathologists and took the course merely that they might become better farmers, teachers or agriculturists. Obviously the course content had to be different from what one might offer a student specializing in one of the main divisions of botany.

Second, the time allotted to the subject was not more than five hours for one quarter or four hours for one semester. It was our task, therefore, to teach the student as much about the elements of plant pathology and their application as such limited time would permit. Much material that an enthusiastic teacher would like to give, of necessity, had to be omitted. The real task has been to choose appropriate material, without omitting important subject matter illustrating the principles and practices of the science. In doing this, the following four objectives have been developed as the minimum need of the student in plant pathology: (1) To appreciate the influence of plant pathology on human affairs, (2) to acquire an understanding of health and disease in plants, (3) to understand the phenomena of parasitism and (4) to acquire as much information as possible about the characteristics of diseases, their symptoms, cause and control.

The third condition concerned the availability of appropriate subject matter in sufficiently concise form. The beginning student invariably is lost if assigned reading in monographic treatises and seldom grasps the principles involved if referred to popular papers and bulletins. Material of this kind is not prepared for the beginning student. Such publications are intended either for the more mature student and investigator or, on the other hand, for the layman interested primarily in the practice of control measures.

This book is not intended to contain all of the subject matter that may be taught in a beginning course in all parts of the country. To include subject matter suited to all sections of the country would make the book too large, too involved and too costly to serve the best interest of the beginning student. The local importance of certain diseases may call for additions to or substitutions for the material presented. Such additions may illustrate the principles equally well and confront the students with a local problem having clearer meaning and sometimes greater significance to them than the material used in this text. Furthermore, the organization of subject matter of local interest by the instructor adds a creative touch that insures better teaching. There are several different methods, well known to the instructor, of making such material available to the student. It is true, however, that the material in the text is of significance to most agricultural regions and illustrates the principles fundamental to the subject.

In the treatment of most of the subject matter, reference to investigators has been omitted, as is commonly practiced in many of our text books of general botany, except where the name of the authority is of classic importance. In the chapter on virus diseases, however, it was not possible to follow this rule because of the newness and the uncertain state of much of the knowledge in this field.

Different methods of subject matter presentation have been tried, the most satisfactory of which consists of placing the subject matter in the student's hands and utilizing the class period for discussion and thought. In the task of thinking

with the student in plant pathology, many new ideas have developed as to selection and organization of the subject matter. This book constitutes the subject matter that is used at present in teaching a course involving two discussion periods and two three-hour group conference periods per week.

The instructor will observe that the emphasis placed on the mycological aspect of pathology has been minimized, while the main emphasis has been placed on the phenomena of parasitism in disease processes. The diseases chosen to illustrate the elements of plant pathology have been grouped into chapters according to the larger systematic divisions, as phycomycetes, bacteria, ascomycetes, etc. These chapters have been arranged, however, not according to their systematic positions, but in a manner that has proved to facilitate a more rapid understanding of parasitism. Within the chapters the arrangement is based on similarity of pathological changes that facilitates the student's understanding of disease processes. The diseases chosen as types have been selected because of their illustration of certain principles or practices, because of their relation to the development of the science of plant pathology or because of their importance in agriculture.

The authors have taken the material presented from many books, bulletins and journals. To the contributors of all these we freely and gladly extend our acknowledgments. Parts of the manuscript have been read by Dr. O. H. Elmer, Professor J. M. Raeder, Dr. H. C. Murphy, Dr. J. C. Gilman, Dr. C. S. Reddy, Miss Marie Corkle, Dr. R. E. Vaughan, William Kreutzer, Dr. L. D. Leach, Dean R. E. Buchanan, Dr. K. S. Chester, Dr. C. J. Eide, Dr. G. L. McNew, Dr. H. W. Thurston, Jr., and Mr. Edward Williamson, to all of whom the authors are happy to extend acknowledgments. The authors stand ready, however, to assume responsibility for the errors that may have been overlooked.

I. E. M.
G. C. K.

AMES, IOWA
April, 1939

CONTENTS

CHAPTER		PAGE
I.	INTRODUCTION	1
II.	PLANT PATHOLOGY AND HUMAN AFFAIRS . . . . Ancient Records of Disease, Modern Records of Disease	3
III.	DEVELOPMENT OF PLANT PATHOLOGY	18
IV.	DISEASE IN PLANTS . . . . . Signs and Symptoms of Disease in Plants, Classification of Disease in Plants	22
V.	PARASITISM . . . . . Infection Phenomena, Resistance and Susceptibility, Immunity, Specialization in Parasites, Polymorphism and Heteroecism, Perennial Mycelium, Mycorrhiza, Epiphytotic Diseases	31
VI.	THE INFLUENCE OF ENVIRONMENT ON PLANT DISEASES . . . . . Environment and the Parasite, Environment and the Host, Effect of Environment on Disease	51
VII.	PRINCIPLES OF CONTROL MEASURES . . . . . Temporary Control Practices: Modification of Cultural Practice, Disease-free Seed and Propagating Stock, Spraying and Dusting, Seed Treatment, Wood preservation Permanent Control Practices: Quarantine, Eradication, Disease Resistance	68
VIII.	DISEASES CAUSED BY PHYCOMYCETES . . . . . Soft Rot of Sweet Potatoes, Damping-off of Seedlings, Late Blight of Potatoes, White Rust of Crucifers, Downy Mildew of Grapes, Brown Spot of Corn, Club Root of Cabbage, Powdery Scab of Potatoes	114
IX.	DISEASES CAUSED BY BACTERIA . . . . . Plant Pathogenic Species: Fire Blight of Apple and Pear. Jones' Soft Rot of Vegetables, Black Leg of Potatoes, Bac-	159

CHAPTER PAGE

terial Blight of Beans, Angular Leaf Spot of Cotton, Wilt of Cucurbits, Stewart's Disease of Sweet Corn, Black Rot of Cruciferous Crops, Crown Gall of Apples, Potato Scab

X. VIRUS-DISEASES OF PLANTS 206

Discovery of Virus-Diseases, Economic Losses, Symptoms, Nature of Causal Agent, Properties of Viruses, Influence of Environment, Possible Size of the Virus Entity, Influence of Physical Agents, Influence of Chemicals, Movement and Multiplication of Viruses, Dissemination, Insect Vectors, Control, Common Tobacco Mosaic, Sugar Cane Mosaic, Curly Top of Sugar Beets, Aster Yellows

XI. DISEASES CAUSED BY ASCOMYCETES 250

Chestnut Blight, Brown Rot of Stone Fruits, Lettuce Drop, Thielavia Root Rot of Tobacco, Ergot of Rye, Alfalfa Leaf Spot, Apple Scab, Powdery Mildew of the Grape, Powdery Mildew of Barley, Peach Leaf Curl, Graphium Wilt of Elm

XII. DISEASES CAUSED BY FUNGI IMPERFECTI 309

Diplodia Dry Rot of Corn, Phymatotrichum Root Rot, Cercospora Leaf Spot of the Beet, Watermelon Wilt, Cotton Wilt

XIII. DISEASES CAUSED BY BASIDIOMYCETES 341

Smuts: Corn Smut, Oat Smuts, Onion Smut, Stinking Smut or Bunt of Wheat, Loose Smut of Wheat. Rusts: Nature of the Rusts, Stem Rust of Wheat, Crown Rust, Asparagus Rust, Cedar-Apple Rust, White Pine Blister Rust. Wood and Root Rots: Black Scurf of Potatoes, Mushroom Root Rot, White Heart Rot

XIV. DISEASES CAUSED BY SEED PLANTS 426

Dodder of Clover and Alfalfa, Mistletoe

XV. DISEASES CAUSED BY PLANT PARASITIC NEMATODES 439

Root Knot, The Stem Nematode

XVI. DISEASES CAUSED BY NON-PARASITIC AGENTS . . . 449

Bitter Pit of Apples, Apple Scald, Boron Deficiency, Graft Knot of Apple

GLOSSARY. 463

BIBLIOGRAPHY 471

INDEX 477

Elements of

PLANT PATHOLOGY

Chapter One

INTRODUCTION

WITHOUT plants the earth's surface would be devoid of life. The plant alone has the capacity to trap the sun's rays and use their energy to synthesize carbohydrates. As a result, man and all other animals are dependent on plants. They furnish food, clothing, shelter, drugs, ornaments and hundreds of manufactured articles. Consequently, plants have been cultivated since the beginning of civilization.

Any one who grows plants wants to grow healthy ones so that the increase may be desirable, whether it be grain, fiber or flowers. To further this objective, institutions of learning have developed special departments of plant science, known as horticulture, agronomy, forestry, etc., to teach the student the application of science to crop production. It has been learned that plants carry on catabolic and synthetic activities; they respire, manufacture carbohydrates, fats, proteins, and deposit certain inorganic salts as waste products. These activities are carried on in the protoplasm, the dynamic substance common to all living things. Plants function in many ways like animals.

Animal protoplasm is susceptible to disease, as in the case of hogs to hog cholera, cattle to lump jaw and man to tuberculosis. The fact that plants are similarly susceptible to diseases is not generally known. This is due, first, to the lack of knowledge of plants, and second, to the lack of study of the plant diseases. Few realize that about 40 specific diseases are known on the apple. Other cultivated plants, as well as those that grow wild, likewise are subject to many diseases. Some crops have had to be discontinued for a longer or shorter time in some sections because of certain diseases, for example,

spring wheat in the corn belt (wheat scab); the American chestnut in eastern United States (chestnut blight); cabbage in the corn belt (cabbage yellows); flax in the upper Mississippi valley (flax wilt); cotton in the southern states (cotton wilt); corn in parts of the Philippine Islands (downy mildew); sugar beets in California and certain parts of other western states (curly top of sugar beets). With some of these crops their extermination has been prevented by certain control measures, such as the development of resistant varieties of flax, cotton, cabbage and sugar beets.

Since there are many different kinds of plants and each species may have many different diseases, it will be apparent that it is a big task for one person to know all of them. In fact, when we consider only the diseases of agricultural crops, the magnitude of the subject may circumscribe our imagination as to the number and economic importance. Perhaps some idea of the scope of the field may be realized when it is known that it has been estimated that diseases of economic plants cause an annual loss of one and one-half billion dollars in the United States alone. Other countries suffer proportionate losses, although statistical figures in many countries are not available. Since many diseases are so seriously destructive, it is important that any one having to do with plants should become familiar with the *principles* and *practices* of plant pathology.

Chapter Two

PLANT PATHOLOGY AND HUMAN AFFAIRS

Ancient Records of Disease

PLANT diseases have had a profound influence on human affairs. It is this fact more than any other that has brought into existence the science of plant pathology. Plant diseases probably have existed since the beginning of plant life. They antedate man. The study of paleontology has given us many records of organisms attacking plants in early geologic ages. In the literature of paleobotany records of so-called spot diseases are common, as well as the organs of parasites such as conidia, mycelia and sex organs (oogonia and antheridia, probably of Peronosporales).

Although we think of plant pathology as a modern science, some of the more outstanding maladies date from early periods; e.g., some of the oldest records of plant diseases are to be found in ancient religious writings, chiefly the Bible. The Hebrew writers make frequent mention of blightings, blastings, rusts, mildews and smuts of the crops of this ancient people. The responsibility for these troubles was laid to the deity, of whose wrath or disfavor they were regarded as an expression. Blasting and mildew are mentioned in various books of the Old Testament from Genesis (4004 B.C.) to Haggai (520 B.C.).

> Amos 4:9. "I have smitten you with blasting and mildew: when your gardens and your vineyards and your fig trees and your olive trees increased, the palmer worm devoured them: yet have ye not returned unto me, saith the Lord."

Aristotle, who lived from 384 to 322 B.C., mentioned diseases of the fig, olive and the vine. Following Aristotle

came Theophrastus, a student of Aristotle, the first great botanist of record. Theophrastus, about 370 to 286 B.C., wrote of the diseases of the olive, the grape and the cereals. He sets forth his opinions of plant diseases as follows:

"As to diseases—they say that wild trees are not liable to diseases which destroy them. . . . Cultivated kinds, however, are subject to various diseases, some of which are, one may say common to all or to some, while others are special to particular kinds. General diseases are those of being worm-eaten, of being sunscorched, and rot.

"The olive, in addition to having worms (which also destroy the fig by breeding in it), produces also a 'knot' (which some call a fungus, others a bark blister), and it resembles the effect of sunscorch.

"The fig is also liable to scab. The fig is also often a victim to rot and to krados. It is called rot when the root turns black; it is called krados when the branches do so. Scab chiefly occurs when there is not much rain, after the rising of the pleiad.

"As to diseases of seeds—some are common to all, as rust, some are peculiar to certain kinds; thus chick-pea is alone subject to rot. Some again are liable to canker and mildew, as cummin.

"Generally speaking, cereals are more liable to rust than pulses, and among these barley is more liable to it than wheat; while of barleys, some are more liable than others, and most of all, it may be said, the kind called Achillean'. Moreover, the position and character of the land make no small difference in this respect; for lands which are exposed to the wind and elevated are not liable to rust, or less so, while those that lie low and are not exposed to wind are more so. And rust occurs chiefly at full moon."

Pliny in his *Natural History* tells of the diseases of the vine, the olive and the cereals, recording that the cereals were attacked more in the lowlands than in the uplands. The Romans had designated two gods to be propitiated to protect their crops from the blasting and the mildew. It seems logical to believe that the experiences of these people must have been of grave importance for they became a part of their religion. The people considered these diseases as a punishment of an avenging diety and as such, the only remedy was to appease the angry god. This led to many queer practices in an effort to stop the ravages of plant disease.

Modern Records of Disease

Ergot.—From the beginning of modern history the records are filled with accounts of the ravages, misery and desolation brought about as a result of plant diseases. With the exception of a few of the more malignant human diseases, probably no other disease has brought such misery and distress to so many people in a given period as has ergot poisoning, caused by *Claviceps purpurea* (Fr.) Tul. One can scarcely imagine the human anguish and suffering through sickness, famine and death tied up in the black ergot grains of the bread crop of the people of Europe during the middle ages. The "ignis sacre" or holy fire of the late Roman period has been credited to a type of gangrenous ergotism because later outbreaks of a similar nature are known to have been caused by the use of ergot-infected cereals. Several epidemics of gangrenous ergot poisoning swept through France and Spain during the middle ages. Ergot poisoning is so terrible as to be almost beyond description, for as the disease progresses, the fingers and toes and sometimes the ears and nose become gangrenous; and unless the cause is removed, the gangrenous condition progresses until death.

Such a condition would seem terrible enough in isolated cases, yet it occurred in epidemic form in France nine times in the seventeenth century, seven times in the eighteenth and three times in the nineteenth.

Germany experienced eleven outbreaks in the sixteenth century, ten in the seventeenth, twenty-one in the eighteenth and fifteen in the nineteenth. These epidemics in Germany, however, were of the convulsive instead of the gangrenous form. In the convulsive type of ergotism the extremities are not attacked by gangrene, but the nervous system is affected, resulting in convulsions and proceeding to delirium, melancholia and finally death.

Ergot has not assumed so destructive a role in this country, although it is known to cause ergot poisoning in man and animals. The bread crop of our country is wheat, while that of Europe was largely rye. Ergot, however, still takes its toll

from the wheat farmer. In some seasons the millers reject hundreds of cars of macaroni wheat where a trace of ergot exists. The presence of just an occasional black sclerotium is enough to leave an occasional black streak in the macaroni. Such discolored macaroni is rejected by the housewife and the manufactured product is returned to the miller.

LATE BLIGHT OF POTATOES.—Another plant disease that has had a profound effect on human welfare is the late blight of potatoes caused by *Phytophthora infestans* (Mont.) de By. While this disease did not cause as much misery and distress as ergot, it probably had more influence in shaping the course of human events. Late blight first made its appearance in Europe and America sometime about 1830 and, during the decade following, became generally distributed in Europe from southern Italy to the northern limits of the Scandinavian peninsula, and in America throughout the whole of the middle and eastern parts. Its prevalence gradually increased until 1843 when the first of three increasingly severe epiphytotics occurred, culminating in 1845 when it was estimated that five-sixths of the potato crop of Europe was destroyed. Inasmuch as the potato had come to be extensively cultivated throughout Europe and eastern America, serious hardship and distress fell everywhere. In Ireland, however, the blow fell hardest. In this little island of 8,000,000 people, the potato was the chief source of food, the annual consumption at that time being estimated at 210,125,000 bushels, or more than twenty-five bushels per person. In 1845, when the potato loss was almost 100 per cent, starvation stalked through every peasant home throughout the island and such accounts as the following were of weekly occurrence: "In this county men are dying of starvation at the rate of fifty per day," or, "Deaths in the Union workhouses alone amount to 1,500 per week."

These were the actual conditions in Ireland despite all the aid that charitable organizations and governments could give. At one time, 2,900,000 persons, or more than one-third of the population, were receiving rations at government expense. Indeed, 250,000 people died of famine and fever as a result of the loss of their potato crop.

Every one from layman to politician, from preacher to scientist, turned his attention to discovering the cause of the menace. This brought into the foreground the importance of plant diseases, and it may be said that modern plant pathology was born out of this world catastrophe to the potato crop. The scourge started an emigration from Ireland that lasted for fifty years, and between 1841 and 1851 the exodus totalled 1,640,000 persons. Successive losses of the potato crops led people to believe that the vitality of the potato was degenerating and to improve this they started potato breeding from seed. The 50 years following the great famine were the most active potato breeding years in history, and such standard varieties as Patterson's Victoria, Magnum Bonum and Early Rose were developed. Still another lasting and far-reaching effect of this catastrophe to the potato industry was the abolition of the protective duties that had previously been levied upon all foodstuffs imported from abroad into the United Kingdom.

Powdery Mildews.—At the same time that potato blight was wreaking havoc in Ireland, the powdery mildew of the grape, caused by *Uncinula necator* (Schw.) Burr., a native of the Orient, was slowly gaining a foothold in southern Europe By 1851 it had reached every grape growing country in Europe and in the next five years threatened the wine industry with extinction. The more exclusively a region was devoted to the grape industry, the greater was the disaster. In Madeira, where for 100 years the finest wines of the world were produced, the disease exacted the heaviest toll. On that small island of 240 square miles, mostly mountainous and untillable, the grape industry centered around Funchal where, prior to 1850, the landscape was covered with grape trellises and more than one and one-quarter million gallons of the finest wines were produced annually. In four years this disease swept away the industry. By 1857, only occasionally was a grape trellis to be found. The type of farming had changed to sugar cane and cochineal culture, which had been the industries in the island 200 years earlier.

Another effect of plant diseases on man, not quite so wide-

battle that is still waged today. Not once can it be let up, as was illustrated in France in 1915 when no labor was available for spraying and 70 per cent of the grape crop was lost. Likewise in new districts, where the disease appears for the first time, the lesson must be learned by sad experience, as was the case in 1918 in Victoria. The disease was first noted there in 1917, and the next year it took 90 per cent of the crop.

Coffee Rust.—In 1874 the coffee industry on the Island of Ceylon suffered the same fate as the grape industry in Madeira 20 years before. At that time the island had nearly 200,000 acres in Arabian coffee and was exporting annually about 100,000,000 pounds with prosperity reigning everywhere. Even then, however, the leaf disease was gaining a foothold, and in the next ten years it wiped out the island's coffee industry. So complete was the destruction that in 1913, only 18,700 pounds of coffee were exported and this of a new variety. As a result of this disease the whole industry of the country changed and now it is a land of cocoa and tea, crops whose every operation is different from coffee production.

Peach Yellows.—It is not necessary to go far afield to find an illustration of an industry destroyed by a plant disease. It has happened time and again in the agriculture of our own country. In the closing years of the last century peach yellows (a virus-disease) began to devastate the peach orchards in Maryland, Delaware and Michigan and in certain sections actually eliminated the peach industry. In Delaware, in 1920, there were only one-tenth as many trees as in 1890. In Michigan and Maryland a similar drastic reduction took place. In Maryland, the peach industry has been pushed into the mountainous region in the west, and the two counties, Kent and Queen Anne, which grew nearly one-half the peaches in 1890, grew only five per cent in 1920. In Michigan the situation was not so serious as in Maryland. The number of trees in one county dropped from 1,250,000 in 1900 to 56,000 in 1920. The loss to the peach grower did not end with the passing of the peach industry; the destruction of the peach orchards was a calamity, but the change in cropping practice brought further hardship to the orchardists concerned.

CURLY TOP.—The curly top disease of sugar beets, caused by a virus, has been fully as fatal to that industry as peach yellows was to the peach industry. The early history of sugar beet raising in the United States is one of serious losses from the curly top disease. In many instances, growers started growing beets and factories were built to process the sugar only to experience in a few years reduction in yields of 50 per cent or more from the curly top disease. For example, in Fremont County, Idaho, the acreage dropped from 8,682 with an average yield of 12 tons to the acre in 1910, to 490 acres and a yield of about seven tons in 1920. In sections of California and Utah, the loss from this disease is so great and so frequent that unless some way is found to combat it, these sections will be forced to give up the beet industry.

FLAX WILT.—The history of flax seed production in this country has been one of difficulties because of the flax wilt organism. Flax always has been a pioneer crop, for not only is it well adapted to growing on new land, but it consistently has failed to do well on the same land for more than a few years. For many years it was thought the depletion of nutrients or excreted substances were the cause of the so-called flax-sick soil, but it has now been established as an infestation by a fungus.

As a result of this disease, flax has been kept on the frontier where there was new land, but now new land is no longer available. Many states once leaders in flax production now produce only small amounts, for example, Iowa, Kansas, Nebraska, Minnesota, etc. Only by the continued utilization of resistant varieties is any promise given of a continued supply of flax seed for our commerce.

WATERMELON WILT.—Watermelon growing in this country has been kept continually on the move by the spread of the watermelon wilt organism. A few growers in a given region may find melon growing profitable, and the industry may build up rapidly. It has been the experience of many communities, however, that watermelon wilt may follow. Stands and yields become uncertain, and in a comparatively short time the growers find that they can grow the melons only on

new ground. When all the acreage becomes infested, watermelon culture must move elsewhere, leaving a disrupted industry and idle fields behind. Few other crops will grow profitably on typical watermelon soil so when the soil became infested, the land usually was allowed to grow up to weeds. In the watermelon districts of the southeastern states, watermelon growing has been gradually pushed back from shipping centers so that other centers have to be established by transportation companies, closer to the source of supply, only to be abandoned a few years later when the new fields become too heavily infested. Under other conditions where the area suited to melon growing is limited, the incidence of soil infestation means the almost complete abandonment of the fields. Such is the case in the Muscatine district in Iowa that was producing 8,000 acres of watermelons in 1900, but in 1930 produced fewer than 1,000 acres, with 5,000 acres of once profitable fields grown up in weeds.

This disease is not limited to any given area or section. Losses of 30 per cent or more have been reported from New Mexico, Texas, Illinois, Iowa and throughout the southeastern states, and there is practically no watermelon district not subject to its depredations.

CHESTNUT BLIGHT.—Furthermore, there is the chestnut blight or chestnut bark disease caused by *Endothia parasitica* (Murr.) A. and A. that for 25 years has swept unchecked from New York over the native chestnut timber in the Appalachian region. In 1904 it was discovered in the zoological park in New York City. In two years it was locally epiphytotic and in ten years it had swept the surrounding area and southward into New Jersey and Pennsylvania with 75 per cent destruction. By 1920 the pathogen had covered northern Virginia, with many local centers farther south, and at the present rate of spread it will cover the entire chestnut area by 1945. The destruction of the chestnut is complete wherever the pathogen gains a foothold. In 1920 it was estimated that there were 19,000,000 board feet of chestnut in the Appalachian region, two-thirds of it south of the Mason and Dixon line. The owners of the chestnut groves and timbered areas are not the

only persons affected by the destruction of the chestnut, for every wood-using industry in the region is hit by this disease. Telegraph and telephone companies in New York and Connecticut are even now having to ship in poles. Railroads in that region have been largely dependent on the chest-



FIG. 1. A once prosperous watermelon farm as it appeared after the wilt organism became established in the soil.

nut for ties. Mines use chestnut for timbering. All these industries are financially affected by the killing off of the quick-growing chestnut. But the worst hit industry probably will be tanning. There were forty-three plants manufacturing tanning extract from native material of which 90 per cent comes from the chestnut. When the chestnut is gone, the tanning industry will be dependent upon foreign supplies of which the principal one is quebracho, which is controlled by a monopoly. Thus one disease directly affects the telegraph, telephone, railroad, mining, tanning and leather companies in the United States. To date, no method

has been found of checking the destructive spread of the pathogen.

CURRENT CROP LOSSES.—Still another and probably more easily grasped effect of plant diseases is the one where only a part of the crop is lost. It may be only one per cent or it may be as high as ten per cent. Such losses occur annually with corn, potatoes, apples, wheat, oats and many other crops.

The state of Iowa is selected for illustration not because the losses are higher than in other states (they are in many cases much lower), but merely because the authors are more familiar with the diseases in this state. There was in Iowa in 1920 a total of 198,445 farms producing corn and 162,880 producing oats. Taking this number as a basis and dividing it into the estimated bushels of loss and multiplying by the price per bushel of the two grains, it will be found that each corn farmer experienced an average annual loss of $210 and each oat farmer a loss of $58 because of corn and oats diseases. Since most farmers also grow oats, the average annual loss per corn farmer was $268. This may be considered the normal loss over a period of years and is not at all representative of the loss incurred when epiphytotics occur.

This average annual loss did not include such losses as those of 1916 when black stem rust of wheat spread over the spring wheat belt leaving in its wake thousands of acres of wheat so shriveled that the farmers could not afford to harvest the grain. In North Dakota, for example, the spring wheat crop averaged only 5.5 bushels per acre in 1916, and in nine counties the average yield was less than 3 bushels per acre. For the state that year the loss was approximately 100,000,000 bushels valued at $220,000,000 on the basis of current prices. This represented an average loss of nearly $3,500 to every wheat farmer in the state. It should be emphasized, however, that losses of this magnitude do not occur annually, and they are not included in the annual average sustained losses.

Sustained losses may lead to the partial elimination of a crop from a particular section. All that is necessary is for

the losses to reduce the yield sufficiently to make the crop unprofitable. Such a case is well illustrated in the potato production in Iowa. Figures 2 and 3 show the price and yield trend of potatoes since 1866. The price has gradually

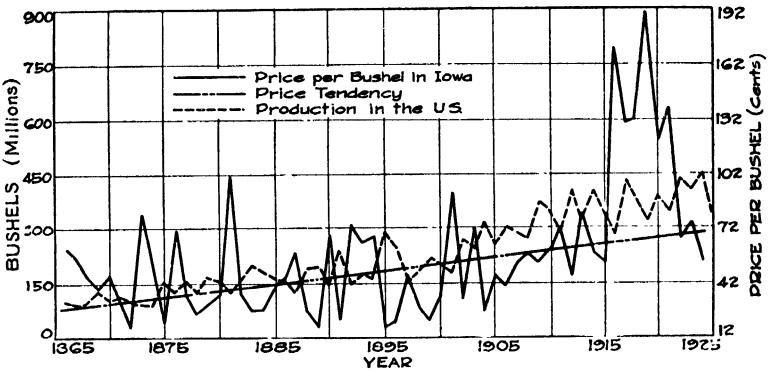


FIG. 2. The price tendency per bushel of potatoes was steadily upward after 1866.

advanced, while the yield has declined. It is believed that the chief cause for the decrease in yield per acre was the presence of certain diseases, as virus-diseases, black scurf, black leg, Colorado bettle injury and hopper burn. The lack

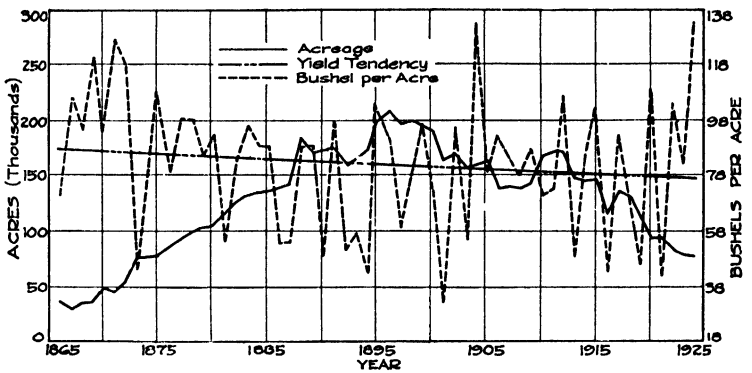


FIG. 3. The yield tendency and acreage of potatoes declined after 1866. The wide annual fluctuations in yield were probably in a large part caused by potato diseases.

of adequate understanding of the causal agents and available control measures for these pests caused the yield tendency to

decline and the acreage to decrease, indicating a shift away from potato growing to other more stable crops.

Figure 4 illustrates the situation in flax with reference to the increase and decrease in acreage and the general decline in the yield tendency per acre. The chief limiting factor was quite

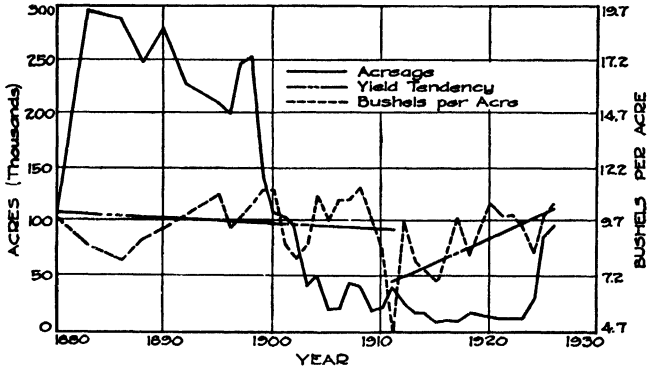


FIG. 4. Flax acreage increased very rapidly for about two decades following 1880. Subsequently it decreased rapidly until 1922, when it again increased. The yield tendency was steadily downward until 1910. Following 1910, when the acreage was very low and the new resistant strains were beginning to find their way into flax growing, the yield tendency moved upward.

probably the flax wilt disease. Figure 4 also illustrates the coming back of a crop through the discovery of effective control measures by the introduction of resistant strains, which occurred in 1910.

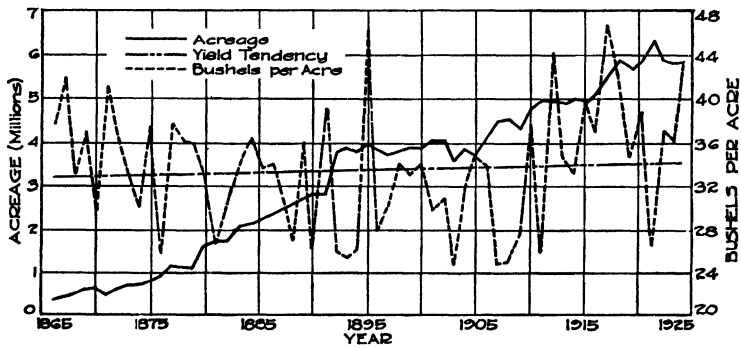


FIG. 5. The yield tendency and acreage of oats have steadily increased during the 60-year period. In this case it is apparent that oat diseases have not been sufficiently severe to bring the yield tendency downward.

These cases, shown graphically in Figs. 3 and 4, probably are sufficient to illustrate the type of loss resulting in changes in cropping practice brought about in part by plant diseases reducing the yield to a point where it was no longer economically practical to grow a given crop.

The oat crop illustrates a case where the yield tendency for the past 60 years has been steadily increasing, as shown in Fig. 5. The oat crop in the upper Mississippi Valley was not attacked by a disease that causes sufficient loss to induce a downward yield tendency. This is probably one reason why the state of Iowa ranks first in oat production in the United States.

Plant diseases are annually reducing the agricultural output of our country and disturbing our cropping practice far more than is generally appreciated. Little is gained by increasing our production through studies of soil productivity, adaptability of varieties and improved cultural methods as long as plant diseases are permitted to continue their destruction. It is obvious that more attention must be given to combating diseases and thereby saving that which has already been produced. New varieties and strains of plants not only must be desirable and productive, but also disease-resistant to deserve and maintain a place in crop production. Neglect in this direction leads to lack of stability and causes frequent costly readjustments for the farmer.

Chapter Three

DEVELOPMENT OF PLANT PATHOLOGY

BOTANY is the mother science of all such phases of plant study as agronomy, horticulture, forestry, bacteriology and plant pathology. Plant pathology may be said to be one of botany's youngest children, standing as a separate science. The science of plant diseases, or phytopathology, was in its early formative period between 1845 and 1860. During that time fundamental discoveries were made that laid a firm foundation for the science. Von Martius attributed the destructive late blight disease of potatoes to a fungus in 1845, and in 1853 de Bary demonstrated conclusively the phenomenon of parasitism in the smuts. Six years later Darwin advanced the theory of evolution, and in 1860 Pasteur succeeded in demonstrating to the scientific world the fallacies of the theory of spontaneous generation. These were great scientific events that have had a profound effect on all the biological sciences, and in particular animal and plant pathology. A new order of thinking was ushered in and a new era began.

Plant pathology, as a science of systematized knowledge of the facts and phenomena of disease as exhibited in plants, was directly clothed in our modern concepts of parasitism in a textbook prepared by Julius Kühn and published in 1858. Twenty-four years later, in 1882, Robert Hartig brought out his splendid treatise on forest diseases *Lehrbuch der Baumkrankheiten*, embodying many advances of our knowledge in this field. In 1886 Sorauer published his extensive treatises on plant pathology in two volumes entitled *Handbuch der Pflanzenkrankheiten*. These general books and much research work, especially in Germany, stimulated the growth of this

new science in every part of the world, the growth since 1886 has been so rapid that Sorauer's *Handbuch* is currently appearing in its sixth edition.

The significance of plant pathology was recognized first in this country by our educational institutions. It was probably first taught incidentally with botany by Burrill of the University of Illinois in 1873. Two years later it was taught as a special course by Farlow of Harvard (1875), who had been a student of de Bary in Germany.

Such outstanding events as Koch's discovery in 1876 that bacteria were the cause of anthrax in animals and man, Burrill's discovery in 1879 of bacteria as the cause of fire blight of apples and pears, Millardet's announcement of Bordeaux mixture for the control of the downy mildew of grapes in 1885 and Trillat's introduction of formaldehyde as a disinfectant in 1888 assured a certain place for plant pathology in relation to agriculture.

A direct effect of these discoveries in the field of research and service to the American farmer was the establishment of the section of vegetable pathology of the division of botany in the Bureau of Plant Industry, United States Department of Agriculture, in 1887 (see Fig. 6).

The same year saw the organization of the state agricultural experiment stations under the Hatch act, which gave another marked stimulus to the growth and development of plant pathology in several states. Fifteen thousand dollars were allocated to each state experiment station under this act, which provided for establishing, under the direction of the agricultural and mechanical college or colleges in each state, a department to be known as the agricultural experiment station, the purpose of which was the "acquiring and diffusing among the people of the United States useful and practical information on subjects connected with agriculture and to promote scientific investigation and experiments respecting the principles and applications of agricultural science." The duties of these stations are "to conduct original researches or verify experiments on: (1) the physiology of plants and animals; (2) the diseases to which they are severally subject,

with the remedies for the same; (3) the chemical composition of useful plants at their different stages of growth; (4) the comparative advantages of rotative cropping as pursued under a varying series of crops; (5) the capacity of new plants or trees for acclimation; (6) the analysis of soils and water; (7) the chemical composition of manures, natural or artificial, with experiments designed to test their comparative effects

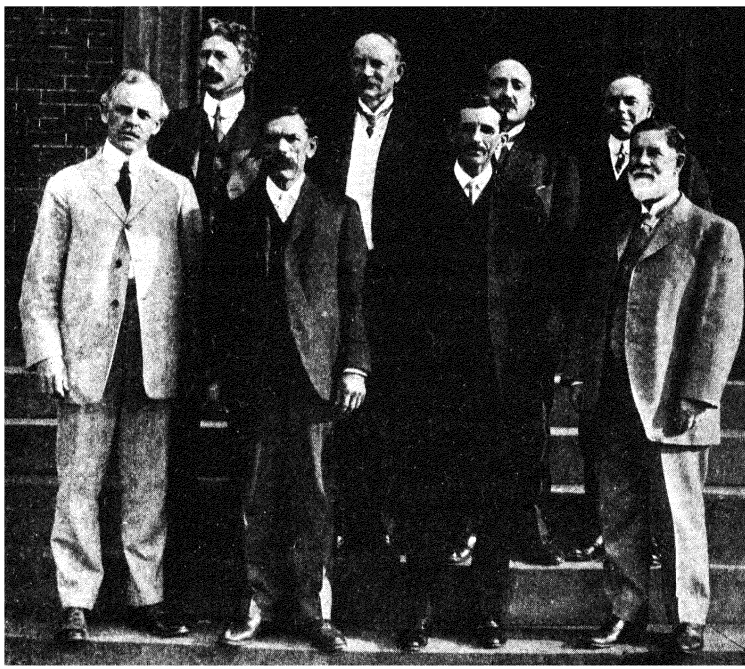


FIG. 6. These eight men organized the Bureau of Plant Industry and had a profound influence on the development of the science of plant pathology in America. They are, front row, left to right, D. G. Fairchild, P. H. Dorsett, B. T. Galloway and Erwin F. Smith; back row, left to right, W. F. Swingle, M. B. Waite, M. A. Carleton and A. F. Woods. (Courtesy of the Bureau of Plant Industry, U.S.D.A.)

on crops of different kinds; (8) the adaptation and value of grasses and forage plants; (9) the composition and digestibility of the different kinds of food for domestic animals; (10) the scientific and economic questions involved in the production of butter and cheese; and (11) such other researches or experiments bearing directly on the agricultural industry of

the United States as may in each case be deemed advisable." This act permitted the starting of investigations of diseases of plants in many states. Among the first were Indiana and New Jersey. Today nearly every state maintains a staff of experiment station workers in plant pathology.

Hand in hand with the research work in the experiment stations the teaching of plant pathology as a separate science was developed in the state colleges. The subject found fertile soil in our land-grant institutions, which have become the strongest centers of plant pathological teaching and research in this country.

From small beginnings, through teaching plant pathology in our universities, through research and service by the United States Department of Agriculture and the state experiment stations and through teaching and research in our land-grant institutions, plant pathology has come to have a permanent place as a science with more than 1,500 workers striving to safeguard the plants that afford food, shelter and happiness for mankind.

Chapter Four

DISEASE IN PLANTS

BEFORE going further, it is necessary to have some understanding of the conditions known as disease and health in plants. This task is made difficult because disease and health are not fixed, unchanging conditions. The state of disease and health in plants is made up of a large number of variables. The changes that occur in the healthy plant, however, may be thought of as regular or varying about a mean, the optimum of its development. In a diseased plant, the variations are irregular and more extensive, at times endangering the life processes of the plant.

Figure 7 represents the sum of a plant's activities in a state of health and disease as two spheres, one containing the other. The larger outer sphere (sphere of life) represents the range within which life processes of the plant may go on, and outside of which they will stop. Any diameter drawn through "c" to the periphery of the larger sphere may be considered the latitude of life. There may be as many diameters as there are conditions that influence the plant's activities. The optimum for the development of the plant naturally falls somewhere within the smaller enclosed shaded sphere (sphere of health), as at "c." The state of health changes gradually as the distance along any diameter (latitude of health, "aca" or "ece") from "c" increases, and the periphery of the small sphere marks the points at which the processes of the plant change from a state of health to a state of disease. Outside of the sphere of health, retrogression takes place, which constitutes disease.

Disease in plants is the sum of the deviations of the vital functions beyond the latitude of health. That field of biologi-

cal inquiry which treats of disease in plants is termed phytopathology.

Signs and Symptoms of Disease in Plants

The variations in plants by which the disease is recognized are designated as signs and symptoms. A few examples of each class are illustrated in Figs. 8, 9, 10 and 11. The sign of a disease is, strictly speaking, the manifestation of some struc-

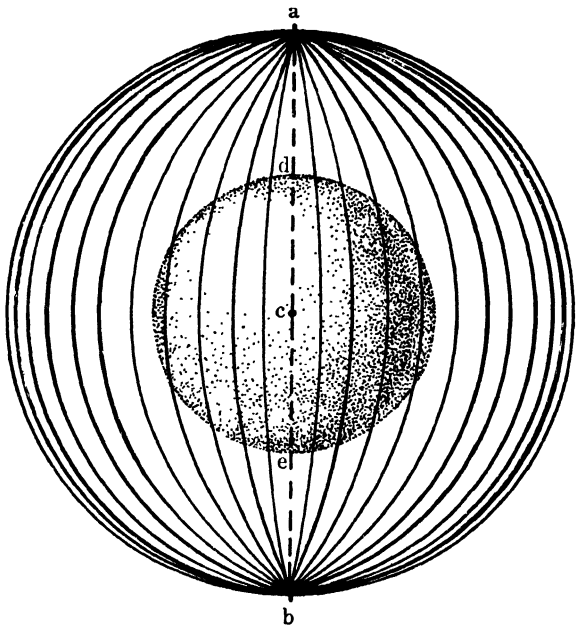


Fig. 7. A large sphere containing a smaller one illustrating the relationship between health and disease.

ture of the pathogen, as its mycelium or spores. These signs often are entirely external portions of the pathogen as in the case of the black color of the sooty molds on fruits or the white powdery growth of the powdery mildews. On the other hand, the signs may be stages that at first are covered by tissues of the host and exposed later, as in the immature stages of the smuts and the red or black stages of the several grain rusts.

The symptoms of a disease are any physiological or morpho-

logical changes from those exhibited by the healthy plant. These may be internal as well as external, pronounced or relatively inconspicuous, and never static, always changing. The symptoms are the visible effects of the causal agent acting on the host. In some cases the causal agent interferes with the normal functioning of the cells. In other cases the form, size and number of the cells are altered. These effects on the cells and tissues when sufficiently extensive become visible to the unaided eye as discolored living or dead areas, or overgrowths followed by death and dissolution of parts. For brief descriptions of the common symptoms that may be manifested in a plant disease, refer to the glossary.

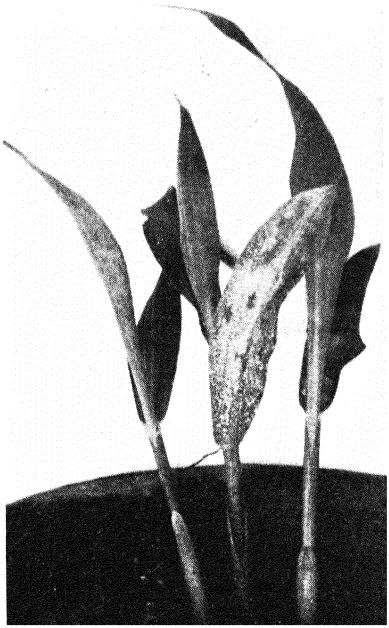


FIG. 8. Signs and symptoms of downy mildew of maize caused by *Sclerospora graminicola* (Sacc.) Schroet.

As the causal relationship progresses either to suppression of the attacking agent or to death of a portion, at least, of the host, the external manifestations portray the changing interrelation. Therefore, any classification, based on only one state of this changing complex, is purely arbitrary and artificial.

CLASSIFICATION OF DISEASE IN PLANTS

Many different classifications of plant diseases have been suggested in the past and even yet not all are agreed upon the most satisfactory system. There are several different bases upon which a classification can be made, and it would appear that the point of view or the use to which such classification is to be put should determine the basis on which it is made.

The basis most commonly used is that of cause. Other bases which have been used are symptoms and hosts. If one were interested chiefly in the etiology or cause of plant diseases, the logical basis on which to classify them would be that of the causal agent. If identification of diseases were the chief object in view, then one would be inclined to use symptoms

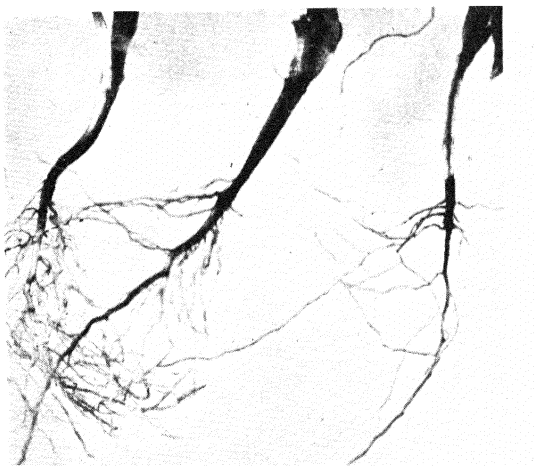


FIG. 9. Root symptoms of aster wilt caused by *Fusarium conglutinans* Woll. var. *callistephi* Beach.

as a basis. On the other hand, if one were already familiar with the different types of symptoms encountered in plant diseases and were interested mainly in the diseases of certain crops, then it might prove more convenient to group diseases according to crops affected.

Classification Based on Causal Agent

In this book diseases of plants are grouped as to cause or etiology and as to symptoms. The classification of diseases on an etiologic basis calls for a grouping according to the chief causal factor involved in each case. Many of the best known plant diseases are caused by microorganisms, such as the lower

and higher fungi. Still others are caused by the flowering plants, such as dodder and mistletoe, and others by environmental conditions. On an etiologic basis, diseases may be divided into two general categories, diseases of parasitic and non-parasitic origin.

DISEASES OF PARASITIC ORIGIN.—Diseases caused by parasites fall into three categories according to whether the pathogen is an animal, plant or virus. The former include those diseases caused by insects, nematodes and other animals. Diseases caused by insects are included in the science of entomology and generally are treated separately. The plant parasites which cause diseases are mainly representatives of the following groups of plants:

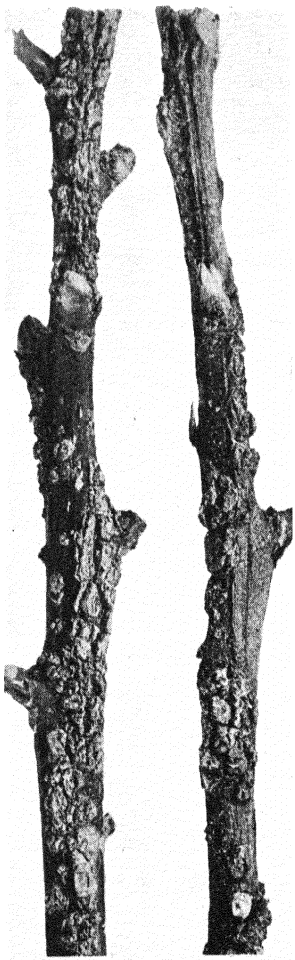


FIG. 10. Proliferation of the cortex, a symptom of citrus canker caused by *Pseudomonas citri* Hasse.



FIG. 11. Anthracnose fruit spot on cantaloup caused by *Colletotrichum lagenarium* (Pass.) Ell. and Halst.

A. Thallophytes.
   1. Fungi.
      a. Schizomycetes—bacteria, or fission fungi.
      b. Phycomycetes—water molds.
      c. Ascomycetes—sac fungi.
      d. Fungi Imperfecti—perfect stages unknown.
      e. Basidiomycetes—stalk fungi.

B. Spermatophytes.
   1. Angiosperms, or flowering plants.

It is known that in plants the viruses develop and reproduce at the expense of their hosts in a fashion similar to parasites.

DISEASES OF NON-PARASITIC ORIGIN.—Diseases caused by non-parasitic agents are deviations from the healthy condition of the plant induced by unfavorable growth conditions. Any excess or deficiency of water, of food substances, light, temperature, air in the soil medium, or other adverse environmental conditions may lead to diseases in plants. There are many diseases belonging to this group of which only a few are comparatively well understood. Some of these are blossom-end rot of tomatoes, black heart of potatoes, scald of apples, tip burn of lettuce, spray burn, frost injury and others.

Classification Based on Symptoms

It is recognized that any classification based on symptoms cannot be hard and fast, because symptoms are not static, but constantly changing as the causal agency continues its activity. In this moving picture, the most characteristic effect expressed in the plant is some form of necrosis. In many diseases necrosis is direct or immediate, in others it may be indirect or delayed. Since necrosis is the final symptomatic expression of the disease processes, the classification based on

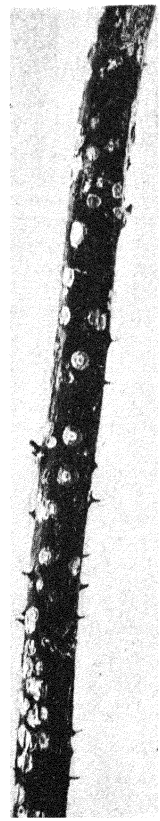


FIG. 12. Anthracnose of raspberry. Local necrosis caused by *Plectodiscella veneta* Burk., on a cane of black raspberry.

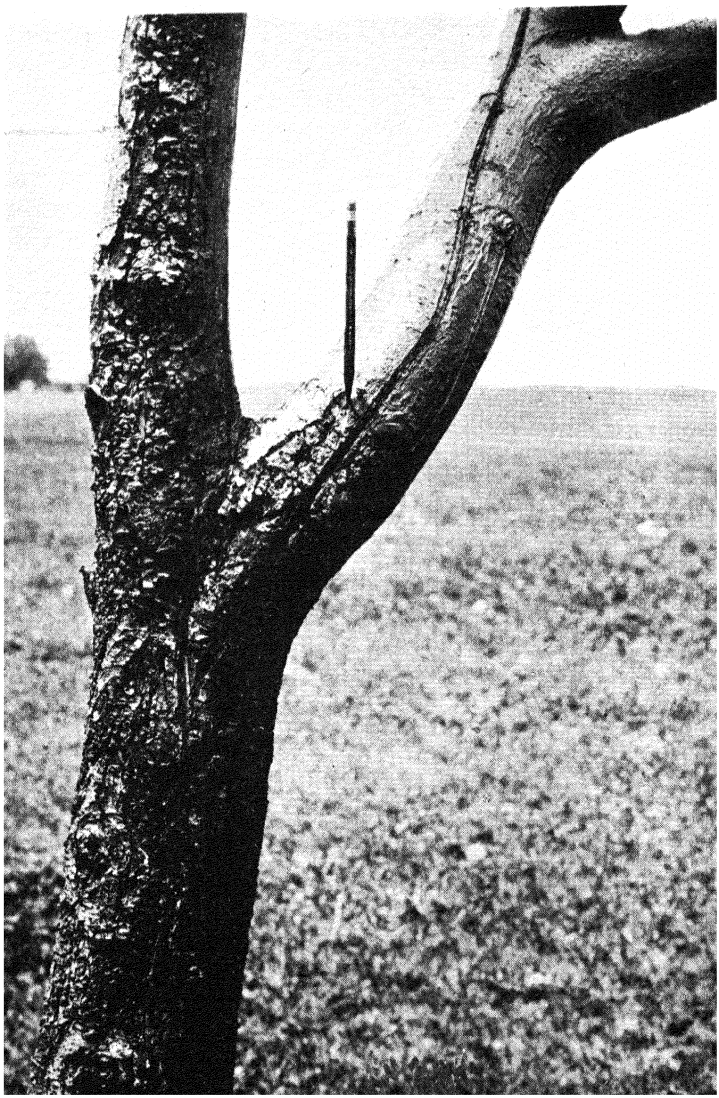


FIG. 13. Illinois blister canker. General necrosis caused by *Nummularia discreta* (Schw.) Tul., on the trunk of an apple tree.

necrosis is the best indication of the time factor in a disease as well as the effectiveness of the causal agent as a destructive factor. In choosing the most characteristic symptom as a basis for arranging plant diseases into two groups according to whether necrosis is direct or indirect, a simple scheme is devised that may be useful to the student.

A. DIRECT NECROSIS.—Direct necrosis occurs in any disease in which the causal agent induces a rapid and complete death or disintegration of the tissues as it advances in the host.



FIG. 14. Vascular necrosis associated with cucumber wilt caused by *Bacillus tracheiphilus* E. F. S. on cucumber.

1. *General necrosis.* General necrosis results where the causal agent induces the extensive and rapid progressive destruction of the host. (See Fig. 13.) A few examples are fire blight, damping-off of seedlings, Rhizopus rot of sweet potatoes, wood rots, chestnut blight, Illinois blister canker and spinach blight.

2. *Local necrosis.* Local necrosis results where the causal agent induces the death of cells and tissues in localized areas. (See Fig. 12.) Examples are numerous, as bean blight, anthracnose diseases, alfalfa leaf spot, crown rust of oats, Cercospora leaf spot of sugar beets, virus necrosis of tobacco.

B. INDIRECT NECROSIS.—Indirect necrosis occurs in those diseases in which the causal agent induces delayed death of the tissues following its activities in the host.

1. *Vascular necrosis.* Vascular necrosis occurs in those diseases in which the death of the tissues results primarily through the activities of a causal agent in the vascular elements of the host. (See Fig. 14.) This is the condition in cucumber wilt, flax wilt, Stewart's disease of sweet corn and cabbage yellows.

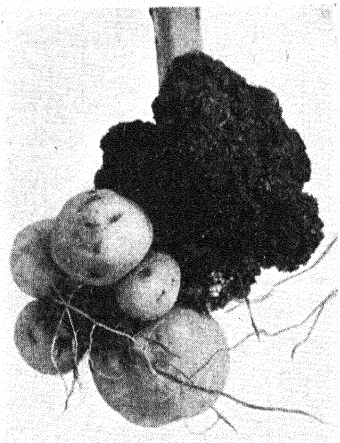


FIG. 15. Potato wart disease showing hypertrophy and cortical necrosis caused by *Synchytrium endobioticum* (Schilb.) Perc. on the tubers of the Irish potato.

2. *Cortical necrosis.* Cortical necrosis occurs in those diseases in which the death of the tissue results primarily through the activities of the causal agent in the parenchymatous tissues. (See Fig. 15.) Examples are plum pocket, white rust of crucifers, corn smut, crown gall, club root of cabbage, powdery scab of potatoes, spike disease of sandalwood, sereh disease of sugar cane, mosaic of tobacco and apple scab.

Chapter Five

PARASITISM

ESTABLISHMENT of the fact that parasitism occurs in plants by Anton de Bary in 1853 marks the beginning of the modern concept of plant pathology. Parasitism is best represented in the higher fungi and bacteria, but it also occurs in the other groups of plants, as the algae and flowering plants. The concept of parasitism was born early in the nineteenth century, but it was smothered by the ignorance, superstition and religious bias of the time. The introduction into Europe sometime between 1830 and 1840 of the late blight pathogen on potatoes stimulated unusual interest in the cause of the disease. Definite proof that this malady was of parasitic origin dealt another blow to the arguments of the proponents of the physiologic theory of disease.

Parasitism is the process of being parasitic. The term parasite is of Greek origin, "para" meaning "beside" and "sitos" meaning "food." The term was originally applied by the Greeks to the class of people who came to a feast to eat without being invited and gave nothing in return but flattery. A parasite is an organism that lives within or upon another living organism from which it derives nourishment and in which it may cause various degrees of injury.

Parasitism is a food relationship in which one organism, the parasite, gains its food from another living organism known as the host. It is the opposite of saprophytism, a relationship in which an organism derives its nourishment from dead organic matter. Parasitism is not rare among living things, being a common phenomenon prevalent in plants and animals. The parasitic habit probably arose as a necessity in the struggle for existence. It is believed that in the begin-

ning fungi, including bacteria, were all saprophytic or autotrophic in habit and that parasitism was a necessary development to afford a new source of food. It is also held that parasitism preceded saprophytism. Regardless of the sequence, it is clear that no sharp line delimiting these two phenomena can be fixed.

The boundary between saprophytism and parasitism is blurred. There are all gradations from partial to complete saprophytism and parasitism. The common black mold, *Rhizopus nigricans* Ehr., that occurs on moist bread in the summer and on over-ripe fruits, lives under these conditions a typical saprophytic existence. Under very favorable conditions for growth, some saprophytes may become parasites, that is, feed on living plant tissues. The common black bread mold under certain conditions may attack germinating corn, thickly sown radish, cabbage and tomato seedlings in the greenhouse, evergreen seedlings in shaded beds and potato tubers in the ground under humid, high temperature conditions. When this species of *Rhizopus* obtains its food from living plants, it leads a parasitic existence. It should be understood, however, that in a classification of this kind involving a very variable characteristic only the prevailing tendency has any real significance. On this basis, *Rhizopus nigricans* falls in the group that obtains its food generally from dead organic matter but may occasionally attack living plants. There are many other organisms that live as either saprophytes or parasites. In the same way the degree of parasitism of a given organism may vary with external conditions, as is commonly witnessed in the case of the organism causing the dry rot disease of corn (*Diplodia zeae* (Schw.) Lév.), the damping-off disease of seedlings (*Pythium debaryanum* Hesse) and many wood rots. These parasites also may derive much of their nourishment from their dead or dying host plants. Other plant pathogens are wholly dependent on living plant tissues as an immediate source of food for their development. Parasites limited in this way are known as obligate parasites. The rusts and powdery mildews are considered obligate parasites.

Species of parasitic organisms are not fixed and constant. They often manifest variations such as may be expressed in many higher plants as a result of changed environmental conditions or genetic deviations. These variations may be of different kinds and magnitudes; i.e., many pathogens show wide variation in cultural characteristics on artificial media, in form, in reaction to chemicals and temperature, and in pathogenic effect on closely related host plants. Where the variation in pathogenicity is consistent it is known as specialization. Its significance in disease control often assumes great importance. Variation has led to specialization in some pathogens and is designated by a variety of different terms as microspecies, biologic forms, biologic races, elementary species, strains, lines, biotype forms, physiologic forms or physiologic races, racial strains, etc. In plant pathology the specialization based on pathogenicity is of much importance and is well typified in the cereal rusts.

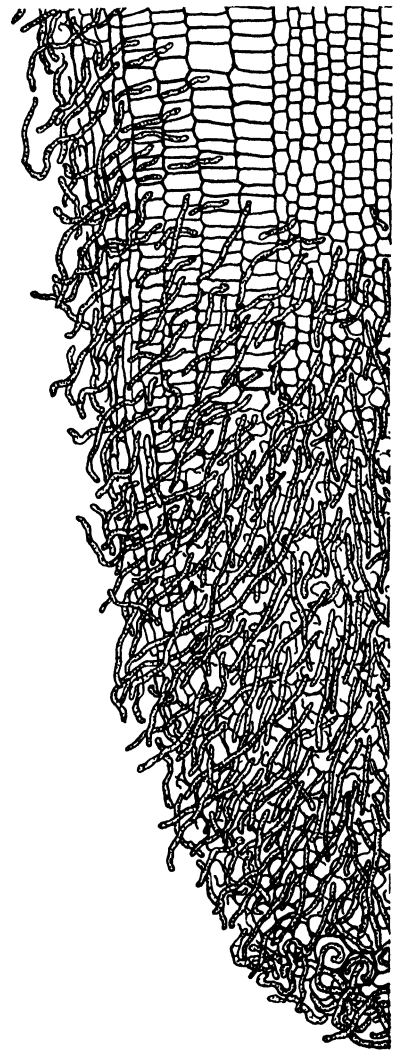


FIG. 16. A longitudinal section of a watermelon root tip infected with *Fusarium bulbigenum* Cke. and Mass. var. *niveum* (E.F.S.) Woll. The parasite penetrates inter- and intracellularly the root cap and the meristematic tissues in the zone of rapid cell division of the root.

Most plant parasites, when found inside their

hosts, are growing in the cells or between them. The former relationship is known as intracellular parasitism, and the latter as intercellular parasitism. In both cases the parasite draws its nourishment from the cell. Other parasites live largely on the surface of the plant or host, sending only slender branches down into the tissues. This is characteristic of the powdery mildews. Still others have a less direct contact with the host and may never actually penetrate it. Here the organism forms only a very close contact with the host from which it draws a part of its nourishment. The parasite in this case is epiphytic. The surface of fruits and leaves is sometimes disfigured or specked with mycelium or dense, hard, black or brown masses of some fungi. The parasite may become so general as to form a partial crust and interfere with the photosynthetic activity of the leaves

Infection Phenomena

For an organism to become a parasite it must be able to enter or to make intimate contact with another living organism, the host. Some organisms are able to enter the host through the unbroken epidermis, others through natural openings as the stomata or through injuries. The powdery mildews send hyphae through the unbroken epidermis of a susceptible host. Just how the delicate germ tube of a spore, the hypha, is able to penetrate the epidermal cells is not definitely known in all cases. It is held that the tube, when it comes in close contact with the host cells, may secrete an enzyme that partly dissolves the cell wall. In other cases it is recorded that the tubes

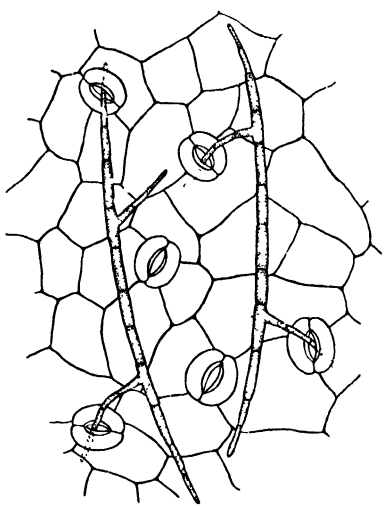


Fig. 17. Stomatal penetration of a sugar beet leaf by the hyphae of *Cercospora beticola* Sacc.

may force their way into the cells as in Botrytis. The germ tubes of the uredospore and accidiospore stages of many rusts enter the host only through the stomata. This is true also of the germ tubes of the spores of *Phytophthora infestans*, causing the late blight disease of potatoes. The wood and

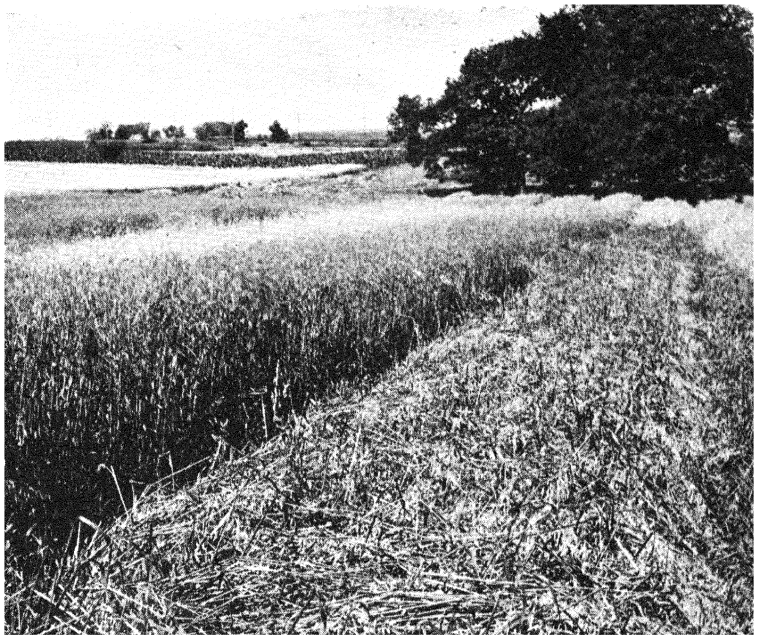


FIG. 18. A rust nursery showing two varieties of oats, one resistant and the other susceptible.

fruit rot fungi attack their hosts through wounds, seldom making their entrance through unbroken tissues. Some bacteria parasitic on plants are also dependent on wounds for infection, others enter through natural openings of the host as the stomata, hydathodes and lenticels. None of the bacterial plant pathogens is known to break through the uninjured epidermis. The causal agents of virus-diseases are thought to be dependent on some form of injury to gain entrance into the host.

In case of higher fungi and bacteria, parasitic invasion is favored by the presence of a film of water, but in the case of

the causal agent of the virus-diseases a film of moisture is not necessary. Once inside the host, the parasite may initiate growth, depending upon its ability to utilize the food material available and to resist toxins.

Resistance and Susceptibility

After the proper contact has been established between the parasite and the host, further growth on the part of the parasite is naturally dependent on its ability to obtain and utilize the food present. The terms resistance and susceptibility refer to the interaction of these two systems, the host on the one hand and the parasite on the other, in respect to the food materials presented to the parasite. The range of the resultant effect on the host may naturally vary widely; when it is marked, i.e., with the balance in favor of the pathogen, the host is said to be very susceptible; and when the reverse is true, the host is said to be very resistant. It is well known that not all varieties of a particular species are equally susceptible to a given pathogen. Some species or varieties may be very susceptible, while others are resistant.

Disease resistance is the sum total of the qualities of the host and the parasite that retard the activities of the causal agent. Resistance in most cases is only relative and the degree manifested by a plant is dependent upon a number of conditions, such as environment and the nature of the host or parasite. The disease condition of a given individual of a species may vary with the heterozygosity of the host or the pathogen as they affect each other individually and jointly. In terms of obvious pathological symptoms, we may consider susceptibility as the destructive development of a pathogen.

The fact that a plant is resistant to one pathogen does not necessarily mean that it is resistant to any other. Resistance is largely specific for a particular pathogen although there are instances where a plant is resistant to two or more pathogens. The resistance of a given host may be either mechanical or physiological in nature.

Mechanical Resistance.—A parasite may be dependent upon a natural or an artificial opening for its entrance into

the host. If this is not provided the parasite is unable to attack the host and the latter is said to be resistant. Potato varieties that have hairy leaves are more resistant to the late blight organism than those varieties that have smooth leaves. Varieties of wheat in which the seed is free from hairs or "brushes" are more resistant to the bunt organism, and barley varieties that fail to open their flowers are said to be resistant to the loose smut organism. Certain varieties of plums are resistant to the brown rot fungus because of the filling of their stomata with masses of parenchyma. Infection of the peach by the scab organism is prevented until the hairs

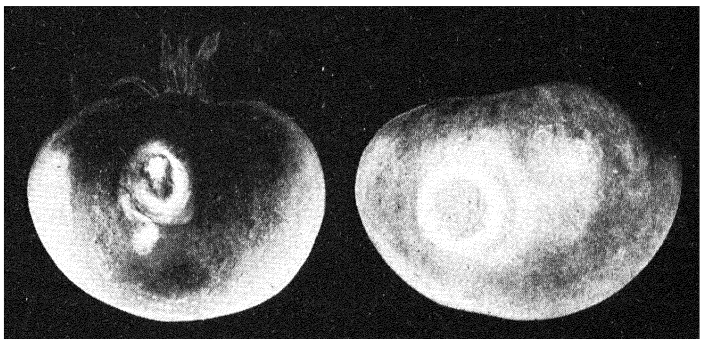


FIG. 19. These two tomatoes were both exposed to infection by *Phytophthora infestans*. The dark-colored (infected) fruit had the epidermis removed and the other did not.

covering the fruit wither, while the nectarine is susceptible because it is hairless. A waxy coating on the culms of some barley varieties insures resistance to the stem rust organism. The size and shape of the stomata may also influence resistance. The Mandarin orange is resistant to the organism causing citrus canker because its stomata are constructed so as to prevent the entrance of water and bacteria. In some cases where the parasite has been able to enter, the host lays down a mechanical barrier, such as layers of cork cells, which prevent the parasite from spreading throughout the tissues. The organisms causing the black leg of potatoes and the Thielavia root rot of tobacco are prevented from spreading

in the host tissues by the laying down of a barrier of cork cells. Varieties of tobacco that have the capacity of rapidly laying down wound cork cells show the greatest degree of resistance.

PHYSIOLOGICAL RESISTANCE.—Resistance in some plants is lodged in the protoplasm of the cells. The inhibition of the parasite may be brought about by some definite chemical substance in the cell or formed by the cell, as in the case of the presence of protocatechuic acid in the outer scales of red and yellow onions, which renders them resistant to the onion smudge organism, *Colletotrichum circinans* (Berk.) Vog. On the other hand, the cells of the host may not be rich in the specific substances needed by the parasite and it will fail to thrive. Such is the case with *Puccinia glumarum* (Schm.) Eriks. and Henn., which is able to enter the wheat variety Einkorn and attack the mesophyll cells, but fails to set up a harmonious food relationship; therefore, the spread of the mycelium is stayed. In still other cases, the parasite itself secretes some substance which kills the adjoining cells of the host. In such a case the parasite may make food available for itself, or, if an obligate parasite, bring about its own starvation through death of the host cells that it cannot use as food.

IMMUNITY

The relationship between a pathogen and a plant may vary from zero, or no infection, to complete infection. A zero relationship between a plant and a pathogen may be thought of as immunity, which is not common in plants. There are two forms of immunity in plants, natural and acquired. The former is better known than the latter. A plant is said to be naturally immune from a given pathogen when that plant, closely related genetically to the host or hosts of a particular pathogen, is impervious to infection. When the resultant action of the host on the one hand, and the parasite on the other is greater than zero the phenomenon is called resistance or susceptibility.

In animal and human pathology, the term immunity is not used in the sense described above. Animal pathologists speak of degrees of immunity when they mean actual degrees of resistance, according to usage in plant pathology. This unfortunate meaning of the term immunity has become fixed in animal pathology through long usage of the term in the relative sense. This difference should be borne in mind to understand the literature dealing with resistance and immunity in the two fields.

Although immunity in plants is rare, certain hop varieties are said to be immune to the hop mildew organism, *Sphaerotheca humuli* (DC.) Burr.; some potato varieties from the potato wart pathogen, *Synchytrium endobioticum* (Schilb.) Perc.; the Idaho and Wisconsin Refugee beans from bean mosaic virus; several wheat varieties from the flag smut organism, *Urocystis tritici* (Pers.) Rost., and three varieties of flax from the flax rust, *Melampsora lini* (Pers.) Lév.

Acquired immunity is well known in animals and man, but rare in plants. In man the recovery from certain diseases confers complete or partial "immunity" to another attack for a considerable time. Antibodies, or antitoxins, are elaborated in the animal body that inhibit or destroy the pathogen. In plants, recovery from disease fails to confer resistance to another attack. At least no good case is known at the present time. On the other hand, many researches indicate that some degree of acquired resistance may prevail. Serums, autotoxins, inhibitors and antibodies have been reported as conferring certain amounts of resistance. Recovery in the case of ring spot disease of tobacco is held to confer immunity to another attack. It may be shown, however, that the virus is present in the host in a form capable of producing the disease if transferred to healthy plants. A type of vaccination or immunization has been demonstrated in the hosts of certain viruses. It has been shown that inoculation with a mild strain of a systemic virus will protect the host from any visible effects following subsequent inoculation with a more virulent strain. Ordinary tobacco mosaic will protect the tobacco plant from the more severe aucuba strain; a mild strain of

cucumber mosaic virus when inoculated into zinnia will protect the plant from severe strains, and the presence of the little peach virus protects peach trees from peach yellows virus and vice versa.

Specialization in Parasites

Many of the common plant parasites are rather cosmopolitan in the range of host plants from which they may obtain their livelihood. Thus the organisms causing damping-off of seedlings, Texas (cotton) root rot, foot rot of cereals, and others are pathogenic to plants in many genera. Certain parasites are limited in their host range, occurring only on the varieties of a single species. This is true in the case of the organism causing watermelon wilt (*Fusarium bulbigenum* Cke. and Mass. var. *niveum* (E.F.S.) Woll.). This organism attacks all the varieties of the watermelon (*Citrullus vulgaris* Schrad.) but does not attack other species of the genus to which the watermelon belongs. Many other pathogens are known to have a very restricted host range as those causing blight of the chestnut, asparagus rust, Diplodia dry rot, alfalfa leaf spot, etc.

One of the most characteristic features of parasitic organisms is their tendency to become specialized. A morphological species may be divided into sub-species, a sub-species into varieties and a variety into races or strains. It is well known that all the individuals of a given species of plant or animal are not absolutely alike. For instance, maize (*Zea mays* L.) consists of several varieties, as dent, sugar, flint and pop corn. These varieties, again, are made up of several races. One of the well known races of dent corn is yellow dent, made up of several strains as Reid's yellow dent, Iodent, etc. There are many races of sweet corn as Golden Bantam, Country Gentleman, etc. Each variety and race has certain constant characteristics that differentiate it from all others.

If the higher plants may show such marked differences, separating them into groups, there is no reason why a comparable grouping might not occur in the organisms that parasitize plants. Such a grouping within species of plant

parasites has been known for more than half a century. Plant parasites may be separated into groups within a species through differences in form, color, cultural characteristics on artificial media, temperature reactions and pathogenic effect on host plants. These variations have led to specialization in some pathogens and such specialized groups have been designated by several different names, as strains, races, biologic forms, physiological species, physiologic races, physiologic forms, varieties, races, etc. In this discussion, the last two mentioned groupings will be used, the former denoting a broader grouping than the latter, similar to the grouping already described in maize.

The basis for the grouping pathologically is the degree of development of a particular parasite on host plants in different genera, species or varieties of plants serving as hosts. This difference in degree of development permits the separation of a given parasitic organism into physiologic varieties and races. Varieties may be alike morphologically, but they differ from one another in the degree of development on some of their host plants. The degree of development may vary on different genera of host plants; i.e., *Puccinia graminis* Pers. consists of seven varieties, one each on wheat, oats, rye, timothy, bluegrass, bent grass and hair grass. The variety on oats develops very sparsely on wheat and vice versa. Again each of these varieties of rust may consist of many races distinguished from one another by their pathogenicity on different varieties of oats, wheat, barley, etc. A very remarkable race development exists in the fungus causing cotton wilt. There are three races of the cotton wilt organism, one in the United States, one in India and another in Egypt. Cotton varieties susceptible to the race of *Fusarium vasinfectum* Atk. in the United States are very resistant to the races in India and Egypt. In the same way the cotton varieties susceptible in India are very resistant in the United States. Varieties are known in many parasitic fungi belonging to the rusts, smuts, downy mildews, ascomycetes and fungi imperfecti.

In the rusts, etc., there are races within varieties. These different races may be separated by the extent to which they

develop on certain varieties or races of their host plants. For instance, the variety *Puccinia graminis tritici* Eriks. and Henn., which occurs on all varieties of wheat, is made up of a large number of groups or races that show different degrees of development on certain varieties or races of wheat. It is possible to separate these races by using certain varieties of wheat as differential hosts. Races are known to occur in

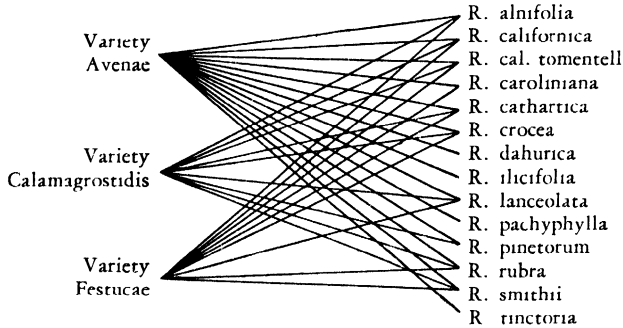


FIG. 20. The Rhamnus hosts of three varieties of *Puccinia coronata*. (After S. M. Dietz.)

several of the grain rusts, as *Puccinia graminis* Pers., *P. coronata* Corda, and *P. rubigo-vera* (DC.) Wint. var. *tritici* (Eriks. and Henn.) Carl. There are a number of races in the variety *Puccinia graminis tritici*, of which about 150 already have been described. It is probable that this number will be increased through subsequent studies.

Varieties and races show differences in degree of development not only on the cereal hosts but also on the alternate hosts. Note the specialization on the genus *Rhamnus*, the alternate host of the organism causing crown rust of oats in Fig. 20. On *Rhamnus* species, functioning as alternate hosts, the degree of development of a variety of *Puccinia coronata* may be altered through new associations incident to reduction division and fertilization, affording new combinations in the inheritance mechanism. Races probably are rust biotypes that remain constant as a result of being propagated vegetatively through the uredospore stage. This type of parasitism is of great importance in the control of certain diseases through the selection of varieties to be grown in a given

locality and in connection with the development of disease-resistant varieties or strains of the host plant. The degree of development of a given race remains rather constant and stays within the range of already existing forms. New forms have been produced by making crosses of two races of *Puccinia graminis* on the common barberry. New races of this and other fungi may also arise by mutation.

Polymorphism and Heteroecism

In many plant parasites in addition to specialization it is not uncommon to find several different spore forms produced on the same mycelium. Organisms having two or more asexual spore forms are said to be polymorphic. It is important in connection with disease-producing organisms to know all the spore forms. With some plant parasites one spore form is produced on the living host and the other on dead tissue.

Polymorphic fungi may develop all their spore forms on a single host. In other instances different spore stages develop on separate hosts. When an organism requires two or more hosts to pass through all of the stages of its development, it is said to be heteroecious. The first case of heteroecism was established by de Bary in 1865 when he demonstrated that *Puccinia graminis* alternated from cereals to barberry. Heteroecism is best known in the rusts, where there are more than 264 cases. As a rule, the two host plants grow near one another in the same plant community. The aecidial stage of many rusts having their uredo and teleutospore stages on grasses, sedges, rushes (monocots) occurs on dicotyledonous plants. In the same way the aecidial stages of many rusts having their uredo and teleutospore stages on dicotyledons occur on certain conifers. No general rule can be laid down, however, without the introduction of many exceptions. If the two hosts of the different spore stages of the parasite are far apart, the possibilities of the parasite completing its life cycle are remote. For instance, in the case of the cedar-apple rust fungus which parasitizes the apple and the red cedar, it has been shown that if the two hosts are two miles apart the chances of the parasite completing its life history are greatly

reduced. Where the apple and the cedar stand close together, the rust, under favorable conditions, develops in epiphytotic form. The following rusts are heteroecious: stem rust (*Puccinia graminis*), crown rust (*P. coronata*), corn rust (*P. sorghi* Schw.), white pine blister rust (*Cronartium ribicola* F. de Wal.), cedar apple rust (*Gymnosporangium juniperi-virginianae* Schw.). If the rust completes all of its stages on a single host, it is said to be autoecious. Such rusts as asparagus rust (*Puccinia asparagi* DC.), bean rust (*Uromyces appendiculatus* (Pers.) Fr.), clover rust (*U. trifolii* (Hedw.) Lév.) and flax rust (*Melampsora lini*) are autoecious.

Perennial Mycelium

The mycelium in some plant parasites may remain alive throughout the life of the host without entirely killing it. In such cases the action of the mycelium in certain parts of the host seems in no way to injure the adjoining cells. When the mycelium of the parasite enters the vegetative tissues of the host during the growing season, it often is able to live through the winter in the mycelial stage. In such instances the parasite is said to have a perennial mycelium. There are a number of examples of this kind known and in most cases parasites that are able to adapt themselves in this way are widely destructive. In several of the downy mildews, rusts and smuts, this condition exists.

One notable instance of the functioning of the perennial mycelium is the case of the pathogen causing the late blight disease of potatoes. When infected tubers are placed in storage where the temperature is low, for instance between 0° and 5° C., the mycelium becomes more or less dormant. In the spring when such tubers are planted, the mycelium becomes active and grows up the stem of the young shoot, following the cortex. The parasite may sporulate either in the soil or at the surface. The spores initiate secondary infection on adjoining plants. In this manner the perennial mycelium functions in initiating the annual occurrence of the disease. The spread of the pathogen from the infected tuber up the stem is shown in Fig. 21.

The disease known as black knot of the plum, caused by *Dibotryon morbosum* (Schw.) Theissen and Syd., which attacks the twigs and branches of the plum, may remain alive and



FIG. 21. Two shoots developed from a potato seed piece infected with *Phytophthora infestans* when planted. The pathogen grew up the stem of the smaller shoot to the surface of the soil where it sporulated over the area between a and x. The other shoot remained healthy.

grow in the cortical tissues for five years or longer. In this case, periodic reinfection is not necessary as it is in the case of the pathogens causing the late blight of potatoes and white rust of crucifers. Among the rusts *Gymnoconia interstitialis* (Schl.) Lagh., the causal agent of the orange rust of raspberries

and blackberries, stands out as a striking example of this class. In this case the pathogen pervades the crown, and when new shoots come forth the mycelium of the parasite follows and later forms great quantities of spores on the young foliage. Such plants seldom bear any fruit, and in most instances they die after two or three years. Where the mycelium of the pathogen becomes perennial, the infected parts seldom recover and, as a rule, are finally killed.

The pathogen *Plasmopara viticola*, causing the downy mildew of grapes, may invade the buds, survive the winter and produce a crop of spores on the young canes and leaves in the spring when the host becomes active. This relationship is most pronounced in this country on the wild grape. With *Albugo candida* (Pers.) Kuntze, causing the white rust disease of crucifers, the pathogen often attacks the plants that come up in the fall and become winter annuals. One of these is Shepherd's purse (*Capsella bursa-pastoris* (L.) Medic.). The white rust pathogen hibernates in the crown of the host until spring.

Mycorrhiza

The living roots of some plants are often partly or wholly covered with a dense mat of fungous mycelium. This mycelium may be wholly superficial, replacing the epidermis, or it may in part be lodged inside the cells. When such a relationship between a fungus and a root prevails, the combined growth is known as a mycorrhiza.

There are two kinds of mycorrhizae. Where the feeding part of the root is covered by a felt of fungal hyphae the coalition is called an external or ectotrophic mycorrhiza. The roots of such trees as beech, hornbeam, oak and Scotch pine are often coated with mycelial mats. Seemingly, the fungi do little damage to these trees, although the rootlets may be thick, fleshy and brittle. In other cases the mycorrhiza seems to be necessary for the normal growth of the plant. The second type of mycorrhiza is called an internal or endotrophic mycorrhiza. In this case the mycelium of the fungus penetrates through the cell walls of the host forming dense masses

inside the cells of the cortex and stimulating cellular hyperplasia, which results in enlargements as are shown in Fig. 22.

It is held that the fungus assumes a symbiotic relationship with the plant in that it takes minerals and water from the soil, and these are absorbed in turn by the root from the fungous mat. In return the fungus receives its supply of carbohydrates and probably other nutrients from the root tissues. The fungi participating in this relationship are held to be annual in their development. Probably more of the known mycorrhizal fungi belong to the basidiomycetes than to any other group.

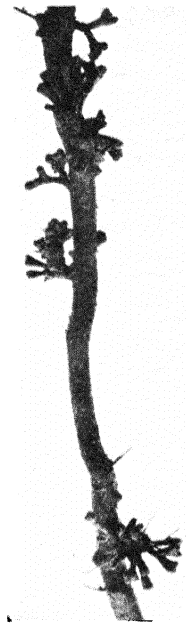


FIG. 22. Mycorrhizal formations on a root of alder.

On the other hand, organisms having such a relationship to the roots of plants may well be considered as parasites. The fungi concerned are weak pathogens whose parasitic activity is curbed by the reaction of the host cells. Plainly the fungus obtains its food and produces more or less injury and hence may be considered as a parasite.

EPIPHYTOTIC DISEASES

When a parasite of either animals or plants becomes destructive to the life of a large number of individuals in a community, district, country or continent the disease becomes epidemic, epizootic or epiphytotic. (These three terms are of Greek origin, "epi"—upon; "demos"—people; "zoon"—animals, and "phyton"—plant. An epidemic disease means a disease on people; epizootic disease, a disease on animals, and an epiphytotic disease, a disease on plants.)

The use of the term epidemic is not, from the standpoint of its derivation, applicable to plant diseases, but it is very generally used in most books and publications relating to plant

diseases. This is plainly a case where the early students of plant pathology borrowed a term from the medical profession. In plant pathology it is more nearly correct to use the term epiphytotic.

It is well known that epiphytotics of the stem rust occur only when the humidity and temperature are high. A plant pathogen cannot induce an epiphytotic merely because the weather conditions are favorable for the development of the host and the causal agent. If the pathogen causing stem rust is not present, favorable weather and abundant susceptible host tissue fail to bring about an epiphytotic. In the same way the rust pathogen may be present and the weather ideal for the destructive development of the rust organism, but if the host plant is absent an epiphytotic cannot come into

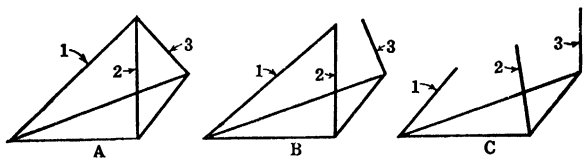


FIG. 23. Three conditions must be fulfilled for the development of an epiphytotic: (1) the general prevalence of the causal agent; (2) a large number of susceptible host plants, and (3) the presence of favorable environmental conditions.

existence. All of this means that three conditions need to be fulfilled at the same time before a parasite can initiate an epiphytotic. (See the graphic representation in Fig. 23.) These are (1) the general prevalence of the causal agent; (2) a large number of susceptible host plants spread generally over districts, countries or continents, and (3) the presence of favorable environmental conditions for the destructive development of the causal agent. If only two of these conditions are fulfilled, (B, Fig. 23), an epiphytotic fails to materialize. The extent to which these three conditions develop simultaneously governs the amount of injury sustained by the plant or crop involved.

It is probable that in the beginning epiphytotics start from single or a few contiguous infected plants. From such a center the pathogen may through sporulation or spread of the

mycelium infect other adjoining plants and in this way move progressively until whole fields and even districts, counties, states and countries become involved. The beginning of a local epiphytotic of late blight of potatoes is shown in Fig. 24. In this case the plants in the field of some 60 acres later became generally infected.

Soil-inhabiting plant pathogens as *Fusarium lini* Bolley may become established in localized centers through the use of infected seed. In such cases the pathogen may spread to

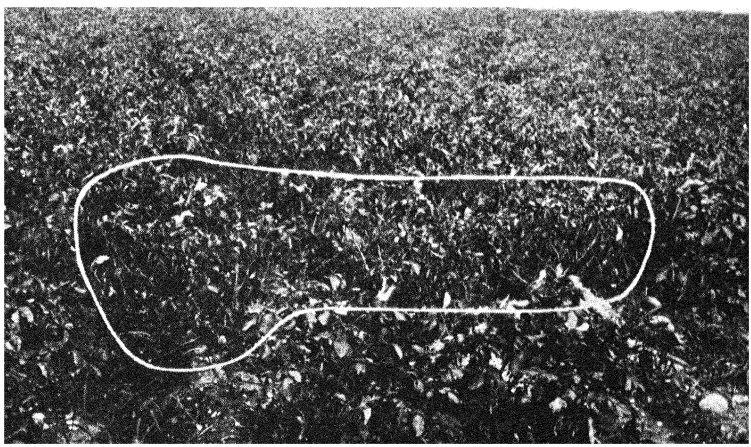


FIG. 24. The white line encircles the areas within which potato plants have become infected by *Phytophthora infestans* through the planting of an infected tuber. The pathogen from such localized areas of infected plants may initiate an epiphytotic.

adjoining healthy plants in the soil as mycelium and as spores borne in the soil and in the aerial parts of infected plants. (See Fig. 25.) When several crops of flax are taken in succession on a field, the pathogen may become abundant and the crop be destroyed.

Epiphytotics may also be initiated through resting spore stages of a particular pathogen as in the case of the apple scab organism. If the foliage is generally infected in the fall and favorable environmental conditions prevail in the spring, the amount of ascospore infection may become sufficiently

general to constitute an epiphytotic. Similarly, local epiphytotics frequently result from the alternate stages of certain rusts. The initial area of infected plants is never large but may rapidly increase through secondary infection resulting from the successive crops of uredospores.

Losses incident to epiphytotics may be small or large, depending upon the value of the crop and its size and distribu-



FIG. 25. The black line marks an area within which all the flax plants are infected by *Fusarium lini*. The pathogen in such centers of infection may initiate a local epiphytotic. (U. S. D. A. Farmers' Bulletin 1747.)

tion. If the area involved is small, the condition is known as a local epiphytotic. Several extensive epiphytotics have been described in an earlier chapter. The best known extensive epiphytotic pathogens have all been exotic or introduced parasites, for example the downy mildew of grapes from the United States to Europe, the chestnut blight organism of the American chestnut from China to the United States, and the late blight fungus of potatoes from America to Europe. Local epiphytotics occur annually and much phytopathological practice is designed to retard or prevent one or more of the three conditions required to bring about an epiphytotic.

Chapter Six

THE INFLUENCE OF ENVIRONMENT ON PLANT DISEASES

THE influence of environment on the incidence of plant diseases has been known for a long time. Environment consists of all the conditions that surround the plant, such as soil, temperature, moisture, light and winds. The experienced grower appears to sense when these conditions are favorable or unfavorable for the development of the plant. Those who have been to the mountains have observed that the trees grow tall and straight in the deep valleys, while up near the snowline they are low, crooked and scraggly. In the corn belt corn grows six to eight feet tall and yields 40 to 100 bushels to the acre, while 300 miles north corn is much shorter and yields much less. This difference in the response of the forest trees and of the corn is due partly to the effects of environment. Just as environment influences the growth of plants, it may also cause them to vary in their response to plant pathogens. Under certain conditions a plant may be very susceptible, while under others it may be highly resistant.

The environmental factors that may affect the host and parasite are such attributes of the soil as the physical and chemical nature, biological condition, temperature and water content, and such attributes of the air as temperature, moisture, sunlight and wind. All these conditions influence the development of the parasite and the host, singly and in combination. The problem presented is, then, to evaluate the effect of these components separately and collectively. For convenience and simplicity we shall deal separately with a few of the environmental factors as they relate first, to the parasite,

second, to the host and last, to the interaction of the host and parasite.

Environment and the Parasite

Environment largely controls the development of a parasite. If conditions are favorable, the pathogen increases rapidly and becomes prevalent; on the other hand, if conditions are unfavorable, the parasite fails to flourish and may be destroyed. Among the environmental attributes that are best understood are temperature, moisture, soil texture, soil composition and reaction, wind, and various combinations of these. A discussion of a few of these conditions follows.

Soil Temperature.—Soil temperature has a noticeable effect on the multiplication and invasive ability of many pathogens. In many cases the organisms require a low soil temperature, as in the case of the organisms causing bunt of wheat and loose smut of oats. In the former the optimum temperature for spore germination ranges from 16° to 18° C., and for infection 9° to 12° C. Unless these latter temperatures prevail in the soil, infection of the young seedlings cannot occur to any appreciable extent. The onion smut pathogen, which is so prevalent in the northern onion growing regions frequently has been introduced into the south on onion sets, but has never become destructive. This is explained by the fact that the pathogen readily infects the onion seedling at 10° to 12° C., but it fails to infect when the temperature is higher. Similarly, *Sclerotium cepivorum* Berk., causing white rot of onions, takes a heavy toll at 12° to 18° C. but is inhibited at 26° C. The optimum soil temperature range for the black scurf pathogen is 15° to 18° C. At higher temperatures the amount of infection falls off rapidly. The pathogen causing Thielavia root rot of tobacco also is favored by a medium or low soil temperature. The pathogenic action of the fungus is held to have an optimum range of 17° to 21° C., with a minimum of 15° C. and a maximum of 30° C.

On the other hand, *Fusarium conglutinans* Woll., the parasite causing cabbage yellows, flourishes at high temperatures and not at low. The cabbage crop in Iowa was practically ruined

in 1916 when the summer was hot, while in 1915, with a cool season, the disease was almost non-existent. The greatest number of plants was found to become infected and show yellows symptoms at 26° to 29° C., while relatively few were attacked at 14° to 17° C. (See Fig. 26.) Similarly the flax wilt organism, *Fusarium lini*, is most severe at high soil temperatures.

A further example of the varying influence of the action of

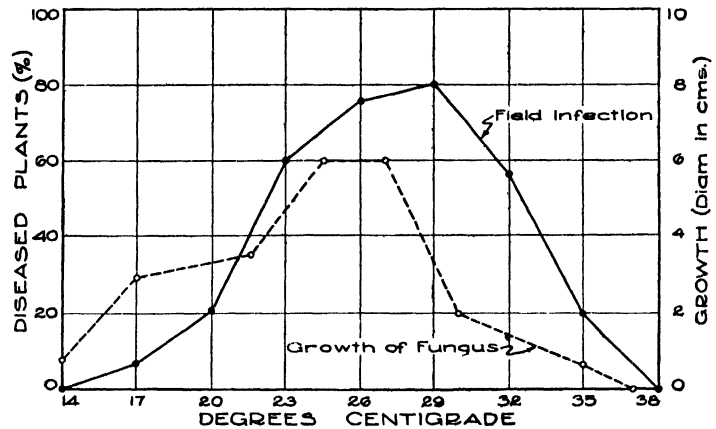


FIG. 26. Graph showing the similarity of the influence of temperature on the field infection of cabbage by *Fusarium conglutinans* and on the growth of the organism in culture. (After W. H. Tisdale.)

soil temperature on the development of a parasite is apparent with the watermelon wilt pathogen, *Fusarium bulbigenum* var. *niveum*. In culture the optimum temperature is 24° C. In field trials it has been found that watermelon wilt was much more severe at 25° to 28° C. than at 16° to 18° C. At the lower temperatures the watermelon wilt organism is hindered in its growth, while at higher temperatures the wilt typical of *Fusarium* spp. is developed.

SOIL MOISTURE.—Soil moisture has a marked effect on the development of many plant parasites. Some develop best at a low, others at a high soil moisture. Thus the causal agents of bunt of wheat, common scab of potatoes, loose smut of oats, bean mosaic and seedling blight of wheat are favored by

low soil moisture, while the pathogenic agents of club root of cabbage, black leg of potatoes and Fusarium wilt of tomatoes require a high soil moisture content.

The wheat bunt organisms, *Tilletia tritici* (Bjerk.) Wint. and *T. levis* Kühn, require a soil moisture of about 22 per cent. At lower or higher soil moistures the parasites are inhibited. Common scab of the potato is very severe where the soil moisture is around 14 per cent, while at 34 per cent hardly any scab is present. Soil moisture is said to be a much more determinative factor in the growth of this organism than the presence of favorable soil temperature. *Plasmodiophora brassicae* Wor., the causal organism of club root of cabbage, causes the greatest damage when the soil moisture is more than 60 per cent. The action may be explained by the fact that the organism needs water for spore germination and migration of the zoospores, as well as for infection.

Soil Reaction.—The effect of soil reaction on plant patho-

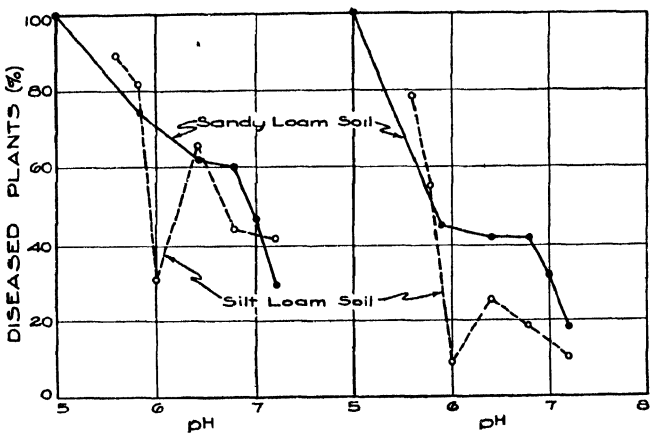


Fig. 27. Graph showing the increase in tomato wilt caused by *Fusarium lycopersici* as the soil acidity increases. (After E. C. Sherwood.)

gens is not so marked as temperature and moisture. *Corticium vagum* B. and C., causing black scurf of potatoes, and *Fusarium bulbigenum* Cke. and Mass. var. *lycopersici* (Brushi.) Woll. and Rg., the cause of tomato wilt, are favored by high soil acidity, as shown by Fig. 27. In the case of the causative

organism of club root of cabbage, acidity is favorable, while alkaline soils inhibit its development. This soil reaction has such marked effects that liming acid soils is a common control practice in the United States. Recent work has shown that the practice of liming soils for legumes may be effective in inhibiting the growth of the pythiaceous fungi that cause damping-off of seedlings in acid soils, and in counteracting the actual soil acidity.

Other Soil Factors.—Various other factors in the soil may have an effect on the growth of plant pathogens. The physical nature of the soil probably plays an important part in determining the distribution of many of the pathogens. *Thielavia basicola* (B. & Br.) Zopf is recorded as preferring a compact soil and seldom produces serious injury in light, well aerated and well drained soils. Many fungi, as *Pythium* spp., are recorded as occurring most abundantly in the top few inches of soil. This is undoubtedly partly determined by the physical nature of the soils. The size of soil particles, determining the type of soil, aeration and moisture-holding capacity, have, in part at least, a direct bearing on the presence or absence of various pathogens.

Another factor that may be of great importance is the biological condition of the soil. Various larvae are reported as important factors in the distribution through the soil of pathogens such as *Bacillus carotovorus* L. R. Jones (black leg of potatoes) and *Bacterium stewartii* (E. F. S.) Stev. (Stewart's disease of corn), and they are undoubtedly of importance in opening avenues of infection for various soil-borne pathogens. Even the earthworm has been accused of disseminating the club root organism throughout a field and from one field to another.

An interesting development in the soil portion of the environmental complex lies in the recent work on the effect of one organism on another, or of two or more organisms uniting to produce a disease. Certain parasites of fungi such as *Trichoderma lignorum* (Tode) Harz are thought to be of some help in controlling pathogens in the soil. Other cases indicate that various organisms of the soil such as *Ophiobolus*

graminis Sacc., *Helminthosporium gramineum* (R.) Erik. and *Gibberella saubinetii* (Mont.) Sacc. may not produce a serious condition of disease separately, but together or with other saprophytic organisms they may be the cause of a great deal of damage to gramineous hosts.

Finally, the condition of the parasite in the soil and its population have a great influence on the amount of disease produced. Many cases have been cited in an earlier chapter of cropping practices being forced from one section to another because of the ravages of parasites living in the soil, i.e., flax from the east coast to semi-arid regions of the west Mississippi valley. Watermelons have been forced out of many sections by *Fusarium bulbigenum* var. *niveum; Plasmodiophora brassicae* has ruined cabbage land, while *Phymatotrichum omnivorum* (Shear) Duggar has prevented much of the land of the south and southwest from being used for production of cotton and other crops.

Air Temperature.—The effect of air temperature on plant parasites has long been known. In fact, it was thought to be the direct cause of many diseases in ancient times. That temperature, high or low, should be so interpreted is not surprising when the marked dependence of disease processes upon certain temperatures is understood. Often this relationship of temperature and the incidence of disease is so pronounced that it determines the distribution of a given disease. Late blight of potatoes occurs chiefly in the northern part of the United States, and it seldom occurs in the southern part except where potatoes are grown as a winter crop. This temperature relationship is due to the relatively low temperature (12° C.) required for abundant spore germination, as seen in Fig. 28, and for infection by *Phytophthora infestans*. The apple scab disease is most prevalent in the cooler parts of the United States. The pathogen that causes apple scab requires temperatures from 13° to 20° C. for the most extensive development of its ascospore stage. Unless the ascospore stage of the pathogen is permitted to bring about infection, little apple scab occurs, except in regions where the organism overwinters in the limb. Many similar illustrations might be

cited where the general occurrence of a disease is determined by low temperatures.

Let us inquire next into the possible relationship of higher temperatures to the incidence of parasitic diseases. The

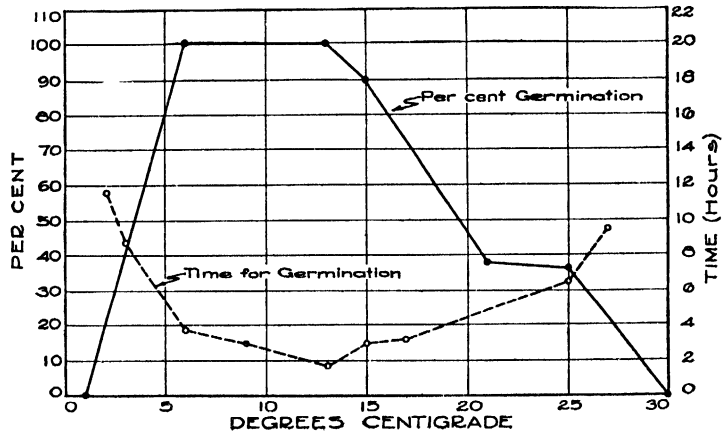


FIG. 28. Graph showing the relation of temperature, percentage germination and the time required for germination of the conidia of *Phytophthora infestans*.

Physoderma disease of corn occurs prevalently in the southern part of the United States. Its occurrence in the corn belt is rare. The parasite that causes the Physoderma disease of corn requires a high temperature for germination of its spores, minimum 23° C., optimum 28° to 29° C. It is held that night temperatures as high as the optimum for the pathogen do not prevail in combination with a film of water in the corn belt, while they do in the southern states.

The rapid spread of stem rust in the upper Mississippi valley occurs only when the temperature and moisture are relatively high. It is generally appreciated by the wheat farmer that hot, muggy weather materially encourages the development of a rust epiphytotic. The high temperature in this case facilitates the destructive development of the parasite, *Puccinia graminis tritici*. *Cercospora beticola* Sacc., causing the beet leaf spot, requires high temperatures coupled with high moisture conditions for its most active development. High

air temperatures are essential before the development of numerous other parasites may reach epiphytotic proportions.

MOISTURE.—The great majority of plant pathogens requires conditions of rather high relative humidity if not the actual presence of free moisture on the surface of the plant before they are capable of producing infection. One of the few exceptions to this rule is the group of Erysiphaceae or powdery mildew organisms that, for the most part, require hot, dry weather. These parasites, especially on peas, apples and oaks, appear to prefer the dry conditions of the more exposed plants. Apples and oaks exposed to the sun and under hot, dry conditions, are much more liable to have mildew than those in shaded and moist places.

The situation found with the Rhizopus soft rot of sweet potato may be cited as an intermediate condition. Here the amount of infection varies with the relative humidity. At 24° C. a relative humidity of 75 to 84 per cent may cause 100 per cent infection, while a relative humidity of 93 to 99 per cent yields only a trace of infection.

Many diseases (peach leaf curl, white rust of crucifers, brown rot of stone fruits, etc.) are recorded as occurring in epiphytotic proportions under high humidities. *Plasmopara viticola* and most other downy mildew organisms as well as most bacterial plant pathogens seem to require the actual presence of a film of water before infection can occur. In the case of the cedar-apple rust organism the teleutospore horns are produced only during periods of rain or continued periods of foggy weather. The basidiospores of this rust are not liberated, however, until the humidity has decreased slightly below the saturation point.

The period of infection of the apple by the anthracnose organism occurs during the autumn rains. This produces a condition of continuously wet bark on the trees with a surface film of water in which germination of the spores may take place. In the case of the stem rust and the bean anthracnose organisms a relative humidity of 95 per cent or more is required before they are capable of infecting the host. The crown rust organism causes 100 per cent infection in a satu-

rated atmosphere and 6 per cent infection when the relative humidity is 93 per cent.

Environment and the Host

The effect of the environment on the host, as on the parasite, may be attributed to the factors of the soil and air. The various attributes that induce rapid, succulent growth of the host may usually produce a plant highly susceptible to pathogenic invasion. Pathogenic agents will be especially encouraged either through the weakening of the various mechanical or physiological factors, limiting the entrance of the

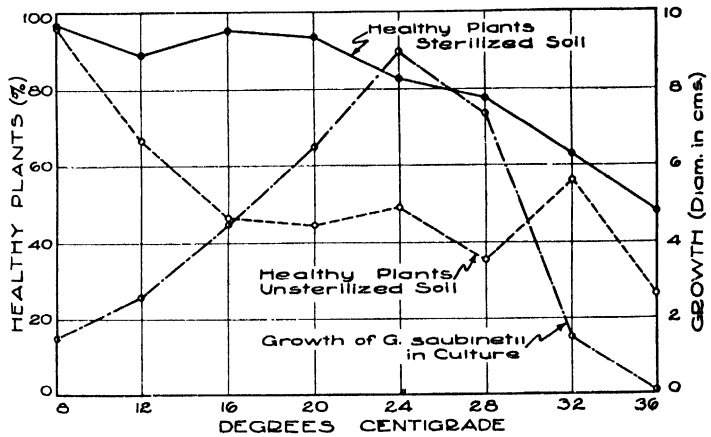


Fig. 29. Graph showing the relation between temperature and the growth of *Gibberella saubinetii* in sterile soil and in unsterilized soil. The optimum temperature for the seedling blight of wheat is the same as the optimum for growth of the causal agent, but above the optimum for the growth of the host. (After J. G. Dickson.)

potential parasite, or, through the presentation to the parasite of excessive food supply. Likewise, conditions, as lack of light or excess of moisture, that cause a plant to grow poorly may favor the production of a diseased condition. On the other side are such conditions as strong light or conditions of high transpiration, low soil nutrients or low soil moisture that produce a highly differentiated plant that has a high carbohydrate content and is resistant to disease. Mention will be made of a few cases where the environment makes the plant more susceptible or resistant.

Soil Characteristics.—An illustration of the effect of environment on the host is that of the relation of the seedling blight of wheat and corn, caused by *Gibberella saubinetii* The optimum soil temperature for the growth of spring wheat is 16° to 20° C., winter wheat is 12° to 16° C., and corn, 24° to 28° C. The maximum percentage of infection of wheat by Gibberella is at soil temperatures of 22° to 28° C., whereas with corn, the maximum injury is produced by the pathogen at 8° to 20° C. Thus, in each case, as shown in Figs. 29 and 30, the pathogen is most destructive at temperatures other than those most favorable to the growth of the host. This is said to be due to the lack of formation at these temperatures of the complex carbohydrate fractions of the cell walls that make the host cells resist the action of the parasite and the failure of the host to form pentosans on which the fungus lives. Hence, the southern part of the corn belt and the northern part of the wheat belt are free from the scab disease. *Diplodia zeae*, causing dry rot of corn, has a similar relation to its host. The temperature range for growth of *Diplodia zeae* is 10° to 35° C. When Diplodia-infected seed is planted in cool, moist soil; i.e., under 18° C., the corn plant is retarded in its development, while the fungus continues its growth and kills the seedlings at or following emergence. The stand obtained is much higher at 20° to 24° C. than that obtained at 15° to 19° C. In late summer, however, the temperatures become favorable for the host and the pathogen, and moisture becomes the determining factor. The parasite continues its activities in the corn plant until the tissues have a moisture content of less than 21 per cent.

Often the rate of growth of the host is retarded by temperature. With wheat the time of emergence may vary from two days at 30° C. to 22 days at 5° C. The wheat bunt pathogen requires a low temperature for germination and, if the low temperature prevails, the host is held longer in a susceptible condition that may facilitate infection by the bunt organism.

Air Temperature.—In the study of the virus-diseases there has probably been more emphasis placed on the effect of weather conditions than in any other group, since their

recognition at present depends mainly on symptoms. The action of temperature on these disease complexes may be on the causal agent. Considering the remarkably resistant nature of most of the virus extracts, in vitro, however, the effect

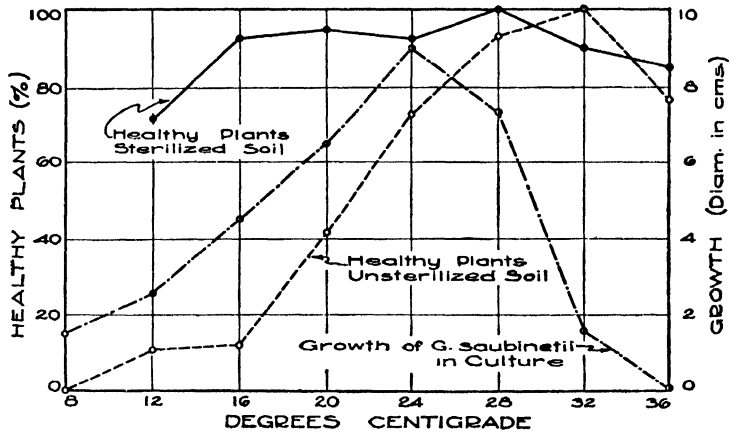


FIG. 30. Graph showing the relation between temperature and the growth of *Gibberella saubinetii* in sterile soil and in unsterilized soil. The optimum temperature for seedling blight of corn is below that for the growth of the causal organism or the host. (After J. G. Dickson.)

must either be one of direct action on the host or at least on the interaction of the host and the infective principle. The action of temperature in producing masking, which is so general at high temperatures, may or may not harm the virus particle. Although the tobacco mosaic virus resists a temperature of 85° to 90° C. for ten minutes "in vitro," disease symptoms are masked in the host at 36° to 37° C. or above. Under these conditions the new leaves fail to develop any of the disease symptoms, while even the older leaves tend to retain their normal appearance. On lowering the temperature the symptoms become apparent, indicating that the virus has not been harmed. The raspberry mosaics show a gradation in this respect. The mild raspberry mosaic fails to appear at 25° C. or above; the red mosaic is very mild at these temperatures, while the yellow raspberry mosaic seems to exhibit no divergence with temperature variations. As indicated later, in the consideration of the viruses, the mild potato

mosaic and spindle tuber of potatoes are just opposite in their manifestation of symptoms. The symptoms of the former are apparent at 15° C. and become masked at 25° C., while the reverse is true of the symptoms of spindle tuber. That this masking at higher temperature is not the rule is shown by the fact that the mosaic of the Cruciferae, curly top of sugar beets and others are more severe at high temperatures. In bean mosaic a partial masking is obtained at 28° to 32° C., complete masking at 12° to 18° C. and optimum expression of symptoms at 20° to 28° C.

The importance of growth condition of the host is shown in such diseases as the bacterial wilt of cucurbits. Since dissemination and infection by the pathogen are dependent on insect vectors, conditions producing a succulent host favorable to insect attack are the most conducive for the development of epiphytotics. The action here is entirely on the host.

Another type of host reaction favorable to the establishment of disease processes occurs in ergot. Infection of the host depends on the availability of open flowers. Therefore, conditions leading to a continued production of flowers and large heads, which are open for a long period, are optimal. These relations are present in periods of warm, wet weather, which are also optimal for the production and germination of the ascospores and conidia of the parasite.

Humidity.—Conditions favorable for ergot epiphytotics include relatively high humidities, although the temperature relations are, of course, primary. More exact evidence of the action of humidity may be seen in various leaf diseases. *Plasmopara viticola*, which is dependent on stomatal openings for entrance into the host, may be cited. A relative humidity of 80 per cent or more is said to produce opening of the stomata of the grape leaves, regardless of soil moisture. If the soil moisture is above 20 per cent, however, the stomata will remain open and permit entrance of the germ tubes of the pathogen even with a relative humidity as low as 40 per cent. The organism producing Cercospora leaf spot of sugar beets is also claimed to be dependent on stomatal openings for the

entrance of the germ tubes. The older leaves are heavily infected in close stands where the humidity is high.

In the soft rot of sweet potatoes, the humidity relationship appears definitely to affect the host. The formation of cork layers by exposed cortical cells of the sweet potato proceeds most rapidly at relative humidities of 95 to 100 per cent and decreases with the humidity. Optimum infection of sweet potatoes by *Rhizopus nigricans* at 23° C. occurs at 75 to 84 per cent relative humidity. Above 85 per cent relative humidity the cork layers apparently form rapidly enough to prevent infection by the germ tubes of the pathogen. Below 85 per cent relative humidity the germ tube develops more rapidly than the cork layer and infection results.

LIGHT.—The effect of light is obvious since it is one of the major factors in the successful growth of green plants. The nature of its influence may be questioned in many cases as the effect may be confused with that of other factors. It is reported that cucumbers grown under glass are much more susceptible to the causative agents of powdery mildew, stem rot, etc., because of the weakened condition of the plants as a result of the low light intensity. Likewise, lettuce drop, caused by *Sclerotinia* spp., is more prevalent on plants grown under glass. The prevalence of powdery mildews in greenhouses may be aided partly by the weakened condition of plants.

Light also may be too intense for the normal growth of the plant. High light intensities often result in high rates of transpiration, which may become injurious. Indeed, this is often given as a cause of the blossom-end rot of tomatoes. Bean mosaic symptoms are reported as much more pronounced when the infected plants are grown under continuous 1,000-watt light than under normal daylight in the greenhouse. On the other hand, moderate light, as in continuous 200-watt light, is said to produce stronger seedlings, more resistant to damping-off fungi than those grown under normal conditions. An interesting case is the report that in the Rocky Mountains at an altitude of 8,000 feet or more there are no potato mosaic symptoms. This is reported as resulting from the increased

stimulation of the production of chlorophyl by the intensity of the short light waves at these altitudes.

Effect of Environment on Disease

In considering the combined action of the characteristics of the environment on the host-parasite complex, it is well to recall the three primary conditions essential to the successful development of epiphytotic mentioned in Chapter V: (1) the general prevalence of the causal agent; (2) a large population of susceptible host plants spread generally over districts, countries or continents and (3) the presence of favorable environmental conditions for the destructive development of the causal agent.

The most usual type of interaction may be illustrated in club root of cabbage. The causal agent, *Plasmodiophora brassicae*, must have water to further the germination, dissemination and infection of the host by the spores. Temperature also has a direct effect on the germination, period of swimming, etc. The optimum conditions are a temperature of 18° to 24° C. in soil of 60 per cent moisture-holding capacity. The cabbage plant also is dependent on definite relations of water content and temperature of the soil. The host requires a soil moisture-holding capacity of 40 to 50 per cent. The favorable environmental effects on the disease relation, however, are shown by the fact that after the initial conditions favorable to the establishment of infection, the further development of the club depends on the age of the host and the conditions favorable for its root growth. The size of the club is directly correlated with the growth of the host's root system.

Another type of relation is shown in oat smut. The growth of the oat plant occurs over a temperature range of 3° to 33° C., with the optimum at 16° to 24° C. The spores of the pathogen, *Ustilago avenae* (Pers.) Jens., are favored in their germination by a temperature of 16° C., while the growth of the mycelium so produced reaches its optimum at 20° C. For the establishment of a fully diseased state of the host, the environmental conditions must be such that favorable spore

germination and mycelial growth of the pathogen coincides with the production of a susceptible seedling in order that the fungus may reach the growing point before the nodes of the seedling are differentiated and so prevent its progress. A temperature of approximately 15° to 18° C., fulfilling these requirements, is optimum for the establishment of the disease.

A similar relationship occurs in fire blight, caused by *Bacillus amylovorus* (Burrill) Trev. High temperature and humidity are not only favorable to the growth and dissemination of the parasite but also induce succulent, susceptible growth of the host. The conditions that supply the most abundant inoculum, enable its spread and favor its development, also result in the formation of slightly protected tissue especially susceptible to the physiological and mechanical effects of the growth of the parasite. Hence, a warm, wet spring is liable to be fatal to many trees, especially when such conditions bring about rapid development. The variations in disease expression are dependent on the environmental conditions and alternate with these conditions. An example of this may be found in the various evidences of the attack of *Fusarium bulbigenum* var. *niveum* on watermelons. Under the normal conditions of growth of watermelons, where the temperature ranges between 25° and 28° C. or above, the fungus attacking the roots enters the vascular system and through a plugging and toxic action causes a severe and often rapid wilting of the host plants. This expression is at the optimum conditions of growth for host and parasite. If, however, infection occurs and the plants are subsequently maintained at a temperature of 18° C., the fungus may enter the host and produce a seedling rot. The hypocotyl and radicle of the seedlings are rotted off either before or shortly after the appearance of the plants above the ground. This variation apparently is caused by a change in the environmental conditions under which the plants are grown. It also indicates that the environment must be considered in any discussion of a disease relationship.

The relation of temperature and moisture is well known in the disease of corn caused by *Diplodia zeae*. The entire life

cycle, production of spores, their extrusion, germination and development, is dependent on the moisture available. Since low soil temperatures at the time of planting cause a slow growth of the seedling and the abundant moisture necessary for germination favors the fungus, the stand is reduced. Later in the summer when the temperature has risen to where it is optimum for host and parasite, the amount of moisture is the determining factor. If the moisture content of the soil is moderately high, the fungus gains the upper hand and a severe infection will result. The penetration of the ear continues until the moisture content of the cob reaches 20 per cent or lower and the activity of the fungus may be traced through the ear as it dries to this point.

Another example of the effect of temperature on the interrelation of host and parasite is found in the sore-shin disease of cotton, caused by *Corticium vagum* B. and C. Both the host

TABLE I. OPTIMUM TEMPERATURES OF GROWTH AND INFECTION OF ELEVEN CROP PLANTS AND EIGHTEEN PLANT PATHOGENS

Host Plant	*Diseases*	*Causal Organisms*	*Optimum Temperature in Degrees Centigrade*		
			Host	Parasite	Infection
Potato	Common scab	*Actinomyces scabies*	24	28	20-25
Flax	Wilt	*Fusarium lini*	20-21	26-28	24-28
Potato	Stem canker	*Rhizoctonia solani*	24	23-27	15-21
Corn	Dry rot	*Diplodia zeae*	24-28	28-30	15-19
Wheat	Seedling blight	*Gibberella saubinetii*	12-16	24-28	26-30
Corn	Seedling blight	*Gibberella saubinetii*	24-28	24-28	12-16
Rice	Brown spot	*Helminthosporium oryzae*	32-36	28	16-20
Wheat and barley	Root rot	*Helminthosporium sativum*	12-16	25	28-32
Cabbage	Yellows	*Fusarium conglutinans*	20	25-27	26-27
Tomato	Wilt	*Fusarium bulbigenum* var. *lycopersici*	28-30	26-28	26-28
Tobacco	Black root rot	*Thielavia basicola*	20-25	28-30	17-23
Wheat	Take-all	*Ophiobolus graminis*	12-16	19 24	12-16
Oats	Smut	*Ustilago avenae*	20	20	16-21
Wheat	Bunt	*Tilletia tritici*	12-16	18-20	9-12
Onion	Smut	*Urocystis cepulae*	20-25	18-22	15-20
Onion	Smudge	*Colletotrichum circinans*	20-25	24-26	26
Onion	White rot	*Sclerotium cepivorum*	20-25	20-24	12-18
Tomato	Crown gall	*Pseudomonas tumefaciens*	30	22	22

and a mass selection was made from this progeny. This population became the new variety Powdery Mildew Resistant Cantaloup No. 45. This melon has come into extensive use in the Imperial Valley of California where the powdery mildew had become a limiting factor in cantaloup production.

Many similar cases illustrating the utilization of hybridization in combining resistance and quality in a new strain or variety might be cited, but it seems more worthwhile to show how resistance to more than one pathogen has been bred into a single strain. In this case we may choose the diseases caused by the rusts and smuts of oats. A variety, Victoria, introduced from South America in tests for crown rust resistance proved to be resistant to all known races of the crown rust organism except No. 41, encountered only once. In addition Victoria proved to be immune to the pathogens causing loose and covered smut of oats. Unfortunately this unusually resistant oat lacked adaptability to the upper Mississippi oat growing region. Another oat variety, Richland, isolated by line selection from Kherson in 1906, is an early maturing, short, stiff straw, high yielding oat very resistant to the stem rust organism.

These two oat varieties were crossed in 1930 and the F_1 increased the following season. Plant selections were made in the next three generations for quality, productivity and resistance to the two rusts and two smuts. An F_5 line known as No. 509 was increased during the next four generations. The F_9 was designated as the variety Boone. In field tests its resistance equals its pedigree record, and its yielding capacity exceeds that of Richland by 25 per cent.

Chapter Eight

DISEASES CAUSED BY PHYCOMYCETES

PHYCOMYCETES, or alga-like fungi, are the lowest group of the true fungi. They resemble some of the green algae in their method of sexual reproduction. Many of the representatives of this group still live only in water, either as saprophytes or parasites on water plants, insects or young fish. Others have acquired terrestrial habits and are saprophytes on dead organic matter or parasites on living plants or animals on land. Many of these fungi are highly destructive parasites on a large number of cultivated plants. The two classical examples are the late blight fungus of the potato and the downy mildew fungus of the grape. Their advent in Europe emphasized for the first time the great destructive capacity of the parasitic fungi.

GROWTH AND DEVELOPMENT OF THE PHYCOMYCETES.—To understand just how the members of this group live and grow as parasites, it becomes necessary to know something of their form and function. The main body (mycelium) consists of a single branched cell. Each cell is surrounded by a cell wall except in the case of some of the very simplest forms where no cell wall is present, and many very small cells may fuse to form a body known as a plasmodium. When the cell is branched the individual cylindrical filaments are called hyphae (singular, hypha).

These cells may vary in size from a fungous body so small that it can complete its whole life history inside of a single cell of a leaf, and is visible only with a microscope, to one so large that it is easily visible to the naked eye and large enough to handle, similar to a colony of the bread mold. The mycelium of the Phycomycetes differs from the mycelium

of other groups of the true fungi in that it has many nuclei scattered through the cytoplasm. In most of the parasitic species the mycelium is inside the host between the cells. Frequently the mycelium is provided with special branches for food absorption from the adjoining cells. These branches may be called haustoria or rhizoids. In many instances the mycelium does not have these special food-absorbing appendages, and absorption is directly through the mycelial wall.

In addition to absorbing food, this mycelium bears a large number of reproductive organs asexual or sexual. In the lower Phycomycetes the whole vegetative body may round up into a sphere and the contents divide into a large number of motile spores as in *Physoderma zeae-maydis* Shaw, the organism causing brown spot of corn. In the damping-off fungus, *Pythium debaryanum*, a piece of the mycelium may surround itself with a thick wall. This body is called a chlamydospore. In others a stout branch of the mycelium, called a conidiophore, grows out to the surface of the host and bears a crop of spores. These spores are called conidia and may germinate by sending out a tube or by the breaking up of the cytoplasm into many small spores. In some members of this group the spores formed in the conidium are motile by means of one or two long cilia. These spores are known as zoospores or swarm spores.

These asexual spores are in most cases very easily killed by dry air, sunshine and high temperature. They must have conditions favorable for germination shortly after they mature or they will perish. A film of water is essential to their germination. Before the spores can germinate by zoospores they must be in water for one to five hours at a temperature ranging from 1° to 20° C. The zoospores maintain their motility only for a short time, when they lose their cilia, round up and send out a germ tube that penetrates the host.

In addition to these asexual spores, two types of sexual spores are produced. In one of these types branches of mycelium of unequal size are cut off by cross walls. The

larger of these cells is the oogonium, which produces internally a uninucleate or multinucleate female gamete. The smaller, the antheridium, produces a number of uninucleate male gametes, which are liberated into the oogonium. These gametes fuse inside the oogonium forming one large cell, which, as it matures, thickens its wall. The cell is known as an oospore and serves to carry the fungus over periods adverse to its growth. It is often produced inside the host and is liberated only when the tissues rot. With the advent of favorable conditions for its growth, the contents may either break up into a large number of zoospores, develop a conidiophore bearing a conidium or send out a germ tube, which may branch and become a mycelium. These oospores or resting spores are very resistant to high and low temperatures, dry air, sunlight and moisture. They may germinate immediately after they are ripe or lie dormant, awaiting the advent of favorable environmental conditions. In many cases they remain alive in the soil for two years and probably longer.

In other members of this group, the mycelium produces short side branches, from the ends of which small, multinucleate gametangia are cut off. Two gametangia meet and their nuclei fuse in pairs following the disintegration of the confluent end walls between them. The new cell formed by this process enlarges and thickens its wall. This structure is known as a zygospore. It germinates under favorable conditions by the production of a short hypha bearing a terminal conidium. As in the case of the oospores, these zygospores are very resistant to adverse conditions and may germinate with the advent of favorable conditions after a short or long rest period.

PATHOGENICITY OF THE PHYCOMYCETES.—Most Phycomycetes require a humid environment to flourish. A film of water is actually necessary for the germination of the spores. Regions where humidity is low are rather free from the ravages of this group of organisms. In a temperate climate, as prevails in the central United States, they are most prevalent and destructive in the fall and spring of the year. Plants

may be attacked in any stages of their development, from seedling to maturity. Herbs, shrubs and trees (either wild or cultivated) may serve as hosts. Many of the Phycomycetes live part of the time as saprophytes and the rest of the time as parasites. They probably secrete enzymes or other metabolic products destructive to host tissues. Some of the Phycomycetes are obligate parasites; that is, they grow only on the living host.

Two general types of symptoms are induced by representatives of this group: (1) direct necrosis, a rapid killing and softening of the tissues as typified in the soft rot disease of sweet potatoes, and (2) an indirect necrosis following hypertrophy and hyperplasia as in the white rust on radishes and in downy mildew on the stems of the grape.

To understand more fully how the Phycomycetes attack their host plants, three pathogens causing direct necrosis and five inducing indirect necrosis are presented in some detail.

Soft Rot of Sweet Potatoes

Rhizopus nigricans Ehr.

Rhizopus nigricans is the common bread mold that often may develop saprophytically on moist bread and other foods. In general botany this plant was studied purely as a saprophyte growing on bread or artificial media. This fungus may, under certain conditions, become a parasite and cause serious injury to growing seedlings and stored vegetables and fruits. Its effect on the tissues of the sweet potato is rather typical of that on potatoes, strawberries and many different kinds of vegetables and fruits.

Although this fungus is one of the saprophytic molds, its damage to vegetables may begin in the field and later extend into the storehouse, market and kitchen. It is estimated that previous to 1920 *Rhizopus nigricans* annually destroyed 20 to 30 per cent of the sweet potato crop in the United States. In 1917 the estimated loss in dollars was fixed at $9,000,000. Since 1920 the loss has not been so great because of better sweet potato storage conditions, but

still from 5 to 10 per cent of the crop has rotted in storage or en route to market.

Although many crops are severely injured, the loss to the sweet potato crop is of most economic importance. Strawberry shipments from the south are often almost a total loss as a result of Rhizopus rot. In 1924, the Texas strawberry growers suffered a 20 per cent loss from this organism. It

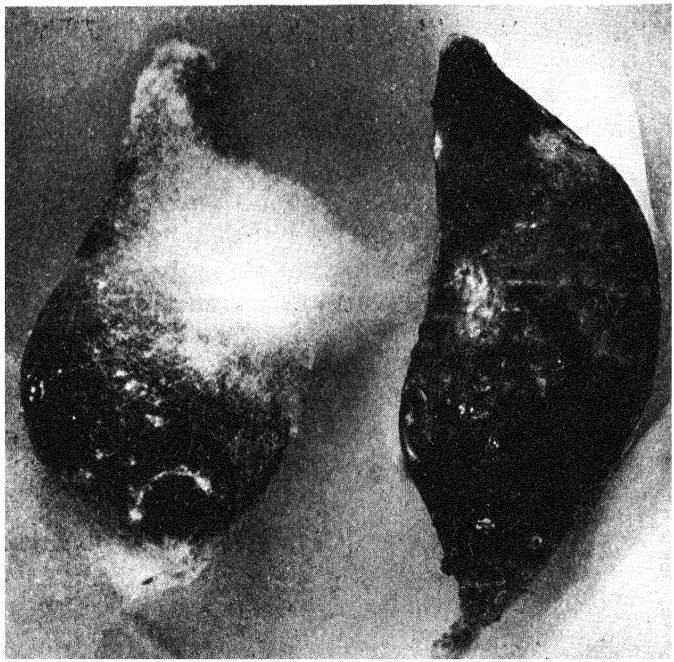


FIG. 42. Two sweet potatoes, one destroyed by the soft rot organism and the other healthy.

occasionally becomes destructive in shipments of peas and peaches; quinces suffer materially in storage, and figs in the south may be rotted on the trees.

SYMPTOMS.—The soft rot organism, *Rhizopus nigricans*, enters the host only through open wounds, and once inside the tissues spreads rapidly, causing them to become soft and watery. The tissues may be so completely dissociated by the action of the enzymes formed by the pathogen that

a badly infected root falls to pieces in handling. If there are several infected roots in a hamper the pressure of adjoining roots may cause the skin to rupture and the infected roots to leak or run. The enzymes produced by the pathogen have no effect on the skin of the roots.

In the early wet stage the roots have a pleasant odor characteristic of the wild rose; later, when bacterial action has set in, there is a soured smell. At first the color of the infected tissues is unchanged, but later they become cinnamon or chocolate brown. If the skin is broken in a moist atmosphere, the pathogen grows to the surface and forms a thick, bushy mass of mycelium and conidiophores bearing the large black conidia. Where soft rot develops in storage and the tubers are undisturbed, they dry down, forming hard, shriveled, shrunken mummies. This is known as the dry rot stage. The pathogen does not always destroy the whole root, its attack being confined to limited areas, sometimes extending in a narrow band around the tuber, the ring rot stage.

ETIOLOGY —The mycelium of *Rhizopus nigricans* may occur either in the tissues or on the surface. There are no cross walls in the mycelial strands, which branch freely and ramify in all directions, forming a tangled mass in the host tissues. A diagram of the host relation of the pathogen is shown in Fig. 43.

On the surface of the host the pathogen may spread by means of long hyphae coming in contact with the substratum some distance from the original colony. The hyphae branch at the tip, penetrate the tissues and initiate a new colony held fast to the host. Conidiophores, which grow into the air from these rooting points of the mycelium in the host tissues, bear at their tips large black conidia filled with the asexual spores. These spores are liberated by the bursting of the conidium and are scattered far and wide by the wind. In addition to these asexual spores, *Rhizopus nigricans* produces sexual spores, which are formed by the fusion of two gametangia. The resulting spore, known as a zygospore, is larger than the asexual spore and is thick walled and brownish black

in color. It serves as a resting spore. Under favorable conditions the asexual spores will germinate readily. The asexual spore germinates by a germ tube that becomes a mycelium, while the zygospore produces a short hypha bearing a conidium at its tip. The infection of the roots

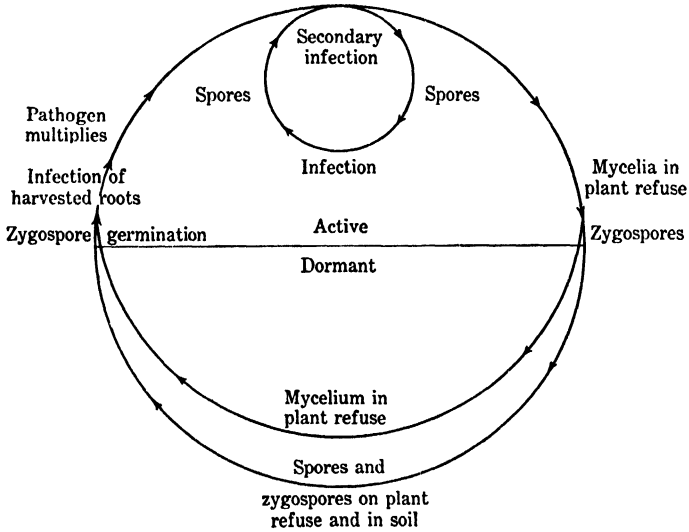


FIG. 43. A diagram of the host relation of *Rhizopus nigricans*. This organism survives the winter in the sexual and asexual spore stages on plant refuse and in the soil.

occurs only after the young mycelium has invaded the injured cells of a wound and produced pectic enzymes.

The softening of the tissues is brought about by the enzyme protopectinase, which dissolves the middle lamellae. The product of this dissolution is then converted into a form of food available to the fungus by a second enzyme, pectinase. The cells of the host are killed and separated by the action of the enzymes slightly in advance of the fungous mycelium. As a result, the sap diffuses out of the cells into the intercellular spaces, causing the necrotic tissues to become very wet. This explains the brownish water-soaked appearance and watery rot so characteristic of soft rot. The carbohydrates made available are utilized by the pathogen for its growth and development. Alcohols and acids are left behind as waste products.

The optimum temperature for infection by this parasite lies between 18° and 24° C. The extreme temperature range over which infection will take place is 3° to 32° C. At 23° C., the greatest amount of infection occurs at a relative humidity of 75 to 84 per cent and decreases as the humidity is either raised or lowered.

CONTROL.—The control of the soft rot organism involves two principles: first, sanitation; and second, proper curing and storing of the sweet potatoes. The storage house should be thoroughly cleaned in the fall before the crop is stored. It is recommended that after thoroughly washing the walls and floor they be sprayed with a strong copper sulphate solution consisting of one pound of copper sulphate in five gallons of water.

When the crop is dug it should be allowed to lie on the ground exposed to the sunshine during the day until the roots are dry, placed in baskets or hampers in the field, hauled directly to the storage house and stored in these containers. Care must be exercised to see that the roots are not bruised in harvesting. They should be cured rapidly at a temperature of 24° to 26° C. for ten days or two weeks. After the crop has been thoroughly cured, hold the temperature at 10° to 15° C., and avoid chilling or rehandling, either of which facilitates infection and frequently leads to serious losses.

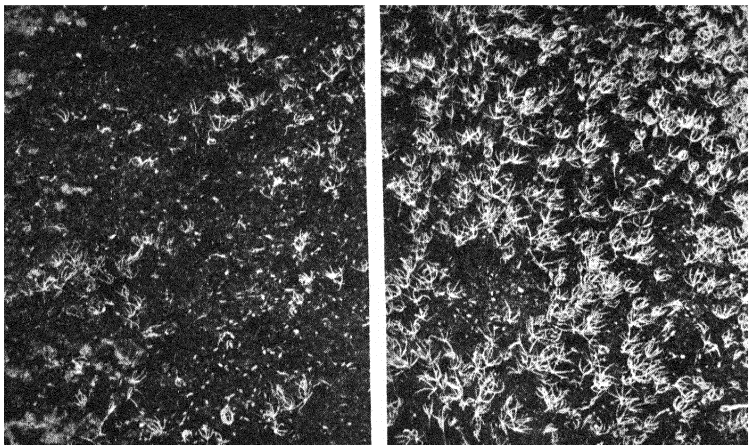
DAMPING-OFF OF SEEDLINGS

Pythium debaryanum Hesse

Damping-off is a disease common to seedlings grown under glass. It occurs also on plants in the open, for example, on pine, spruce and fir seedlings, tobacco, alfalfa, sugar beet, cabbage seedlings and on many other garden and field crops. Damping-off may be caused by several different pathogens, of which *Pythium debaryanum* is one of the most cosmopolitan and destructive. The organism is world-wide in distribution and frequently causes serious losses to crops, especially those in hot beds, cold frames, shaded plant beds and in greenhouse propagating beds. Often the seedling stand is

decreased to the point where replanting is necessary. In other cases the crop has to be replaced by another.

SYMPTOMS.—The most characteristic symptom of the damping-off disease is the falling over of the seedling as a



A B

FIG. 44. A, imperfect stand of pine seedlings on soil harboring the damping-off fungus; B, nearly perfect stand of pine seedlings on treated soil.

result of being girdled at or just below the soil line. The pathogen penetrates the cortex of the hypocotyl or lower portion of the stem and causes direct necrosis of the cortical tissues. Before necrosis occurs, the cortical tissues appear waterlogged and glassy. Soon the cells of the cortex collapse and turn brown. The progress of the pathogen up the stem in young seedlings seems to be limited only by the presence or absence of a saturated atmosphere and favorable temperature. A dry atmosphere inhibits the development of *Pythium debaryanum* in seedlings. When the soil is heavily infested with the pathogen the roots may be attacked. In such cases necrotic areas develop, some of which may completely girdle the root of older seedlings that are not killed.

Probably the most destructive effect of Pythium occurs on seedlings that have not emerged. When infection takes place in this stage general necrosis of the hypocotyl and cotyledons

develops, causing the whole seedling to turn brown and die. It is not uncommon in susceptible crops to find half the plants killed before emergence. If the temperature and moisture are high, the surface of the dead tissues and the surrounding soil may show an ashy white mycelial growth of *Pythium debaryanum*.

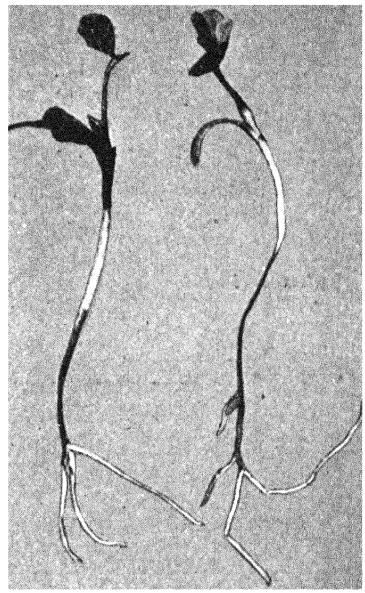


FIG. 45. Damping-off disease on alfalfa seedlings. The dark, discolored areas are lesions caused by *Pythium debaryanum*.

Although the roots of older plants may become infected, the injury is much reduced as the cortical tissues grow older. The aboveground portions of plants other than the base of the stem are seldom attacked by this pathogen.

ETIOLOGY.—Damping-off may be caused by a water mold known as *Pythium debaryanum*. Its host relation is shown in Fig. 46. The mycelium consists of coarse, non-septate, highly granular, irregularly branched hyaline hyphae. The younger hyphae are finely granular; the older ones are coarsely granular and sometimes empty. The hyphae penetrate the host and become inter- or intracellular in the tissues. *Pythium debaryanum* does not fruit freely on or in the living host. The fruiting of the pathogen is better observed when it is grown in pure culture on natural media or when infected seedlings are placed in water at room temperature. Under normal conditions the fungus produces two forms of spores, conidia and oospores. The conidia are hyaline, round to slightly pear-shaped swellings formed at the tips of the hyphae by the deposition of a cross wall. These conidia readily break off from the hyphae and on germination produce

zoospores. These zoospores are provided with two long cilia that aid them in swimming in a film of water. After a short period of motility the zoospore becomes quiescent, rounds up and sends out a germ tube that may enter the host. Sometimes the conidia are not terminal but intercalary in a hypha, the spore being formed through the deposition of two septa, and the delimited segment rounds up into a spore.

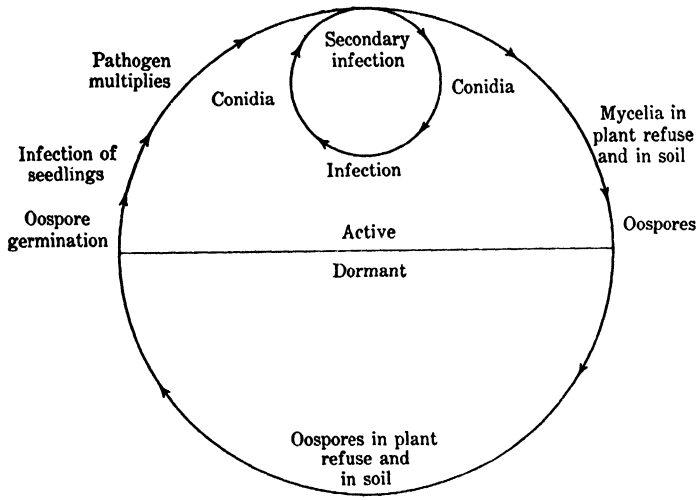


FIG. 46. A diagram of the host relation of *Pythium debaryanum*. This organism survives the winter in the soil as oospores.

Oospores are more common than conidia. These sexual spores may form inside the tissues of the host and be liberated when the tissues decay. The oospores have thick walls that fit them to survive periods unfavorable for their growth and development. Under favorable conditions they may germinate by zoospores or by a germ tube that develops into a mycelial colony in the soil or penetrates the host. Pythium is probably most prevalent in the top six inches of soil, at temperatures of 20° to 30° C., when there is an abundant supply of moisture, causing slight damage at soil temperatures outside this range. The effect on the seedling is most severe, however, when infection is followed by a warm, dry

period. Under such a condition a mild infection may kill the seedling.

CONTROL.—Since the pathogen attacks so many different crops involving widely different cultural practices, only the principles utilized in practice are set forth under this heading.

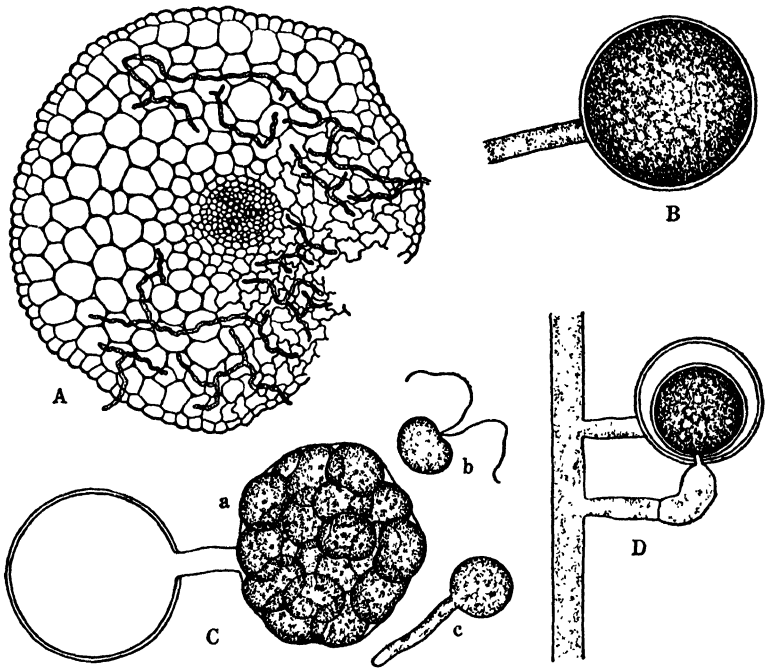


FIG. 47. *Pythium debaryanum:* A, a cross section of an alfalfa seedling root showing necrosis of the cortex and mycelium in the tissues; B, a mature conidium; C, conidium germinating; a, vesicle; b, zoospore; c, spore with a germ tube; D, oospore with the antheridium still adhering.

Cultural practice should be modified so as to avoid high soil moisture and temperature, and high atmospheric humidity. In seedbeds or cold frames, it is frequently desirable to apply a thin layer of dry clean sand or peat soil over the surface after the seed has been sown. Where conditions will permit, the beds may be advantageously crowned to permit surface drainage. Thick seeding and excessive shading should be avoided. For many pines the use of sphagnum moss or burlap as a mulch and covering is held to increase the germi-

nation of the pine seeds and decrease damping-off. In the case of sugar beets, flax, alfalfa, etc., it is well to sow early to permit germination and seedling emergence while the soil temperature is less than about 10° C. Rotation, using less susceptible crops such as corn, oats and potatoes, probably reduces the Pythium population. Plowing where the soil is infested sometimes permits the seedlings to become established without serious stand losses.

In crops grown under greenhouse, garden and nursery conditions, soil sterilization by heat and chemicals is generally effective and practical. Heat is used in baking and steaming the soil. Chemicals having fungicidal value may be used in dilute solutions, as formaldehyde and aluminum sulphate. Seed treatment may protect the germinating seed during its early stages of growth, but it has little or no beneficial effect after seedling emergence.

Late Blight of Potatoes

Phytophthora infestans (Mont.) de By.

Late blight is primarily a disease of the potato, although it does occur on several other species of plants belonging to the nightshade and figwort families. The tomato may be as seriously injured as the potato when conditions are highly favorable, although in practice tomatoes very often escape serious injury.

Late blight was unknown prior to 1830. At least, no reference is found to this destructive pathogen's attacking the potatoes until sometime between 1830 and 1845. At that time late blight appeared almost simultaneously in Europe and North America.

In 1845 the potato was the chief source of food in Ireland, and a majority of the 8,000,000 people were dependent upon the potato crop. In 1844 and 1845 when the crop was swept away by late blight, famine resulted, and suffering was intense. Norton, writing from Edinburgh, in 1845, said:

"To the poor (in these European countries) the potato may be considered the staff of life. In many parts, thousands of families

rarely obtain any other food from one year's end to another. The disease became a natural calamity. The fear of famine became universal and every energy is aroused to avert the danger. Something must be done or the potato bids fair to become extinct. Scientific men in various countries have accordingly turned their attention to it and in most cases have been aided either by their respective governments or by agricultural societies. Among the first on the continent

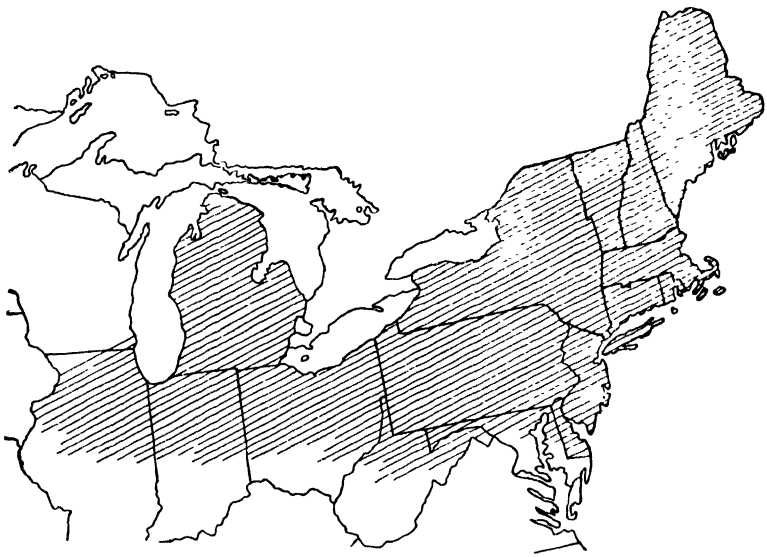


FIG. 48. Map showing the distribution of late blight in the United States in 1845. (After N. E. Stevens.)

was a commission from Holland and Belgium. In Germany, Liebig, among others, has turned his attention to the subject. M. Payen has lately published three or four reports containing the results of elaborate microscopical and chemical researches. Boissingault, A. Peroz and others have also made public their opinions. In Britain and Ireland a great portion of the best scientific and practical men are now uniting their efforts. The English government has sent to Ireland three competent chemists with Dr. Lindlay as Botanist and Physiologist."

At first there was the most extreme difference of opinion as to the cause of the disease, which is also apparent from Norton's writings:

"Some ascribe the disease to electricity, some to atmospheric influence, some to wet season, some to wet, drought and frost combined, some to insects or animalculae, some to ruptures of the cell, some to decomposition of proteins, others to fungi, others to a disease and vitiated constitution in the potato weakened by long and high cultivation, others unite nearly all of the above, others still ascribe it to a direct visitation of Providence, and yet another class declare that they know nothing about it. I think these last are the safest at present."

The late blight disease was prevalent and destructive in the United States in 1843, 1844 and 1845. It was not reported in 1842, but it did occur in the years 1846 to 1851, inclusive, being most severe and extensive in 1845. In 1844 the late blight pathogen caused the following losses:

BLIGHT LOSSES

New Hampshire	25 per cent
Vermont	25
Massachusetts	25 to 30
Rhode Island	10
Connecticut	25 to 30
New York	50
New Jersey	15
Pennsylvania	20 to 25
Delaware	25 to 30

Late blight occurs on every continent in the world and annually destroys immense quantities of potatoes. The reduction in yield in the United States was 30,998,000 in 1928; 1,753,000 in 1929; 5,169,000 in 1930; 9,000,000 in 1932, and 3,409,000 bushels in 1934. The wide fluctuations in loss are largely determined by the seasonal weather conditions.

SYMPTOMS.—Upon the leaves of the potato this fungus develops characteristic necrotic spots that increase until the whole leaflet is destroyed. Necrosis frequently begins at the edge or tip and then spreads over the leaf. In the initial stages, the leaf infections appear as yellow spots, which soon become waterlogged and have a slight purplish tint. In drier weather the necrotic areas are dark brown, bordered by a narrow, light-yellow zone. The moist appearance of the spots accompanied by the wilting of the leaf, or of that

portion affected, offers an easy diagnostic symptom. The pathogen produces a glistening-white mass of conidiophores and conidia on the infected areas. When the environmental conditions are not highly favorable there is little accompanying stem injury, but when conditions are very favorable the necrotic areas may extend to the stem. The collapsed cortical tissue is shown in Fig. 50.

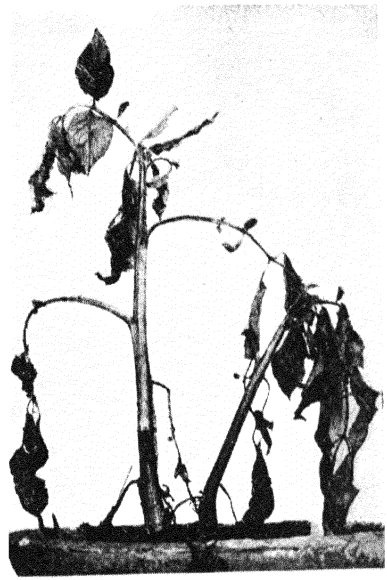


FIG. 49. A young potato plant nearly destroyed by the late blight fungus. This plant was artificially infected with the late blight organism.

On white-skinned tubers early stages of infection show as pink irregular areas, varying in size depending upon the spread of the pathogen in the tissues. If the epidermis is removed, the flesh is also pink instead of white. If such infected tubers are subjected to temperatures above 15° C. in a moist atmosphere, the color of the infected areas changes to dark brown as necrosis occurs in the underlying tissues. Soft rot bacteria frequently follow the late blight organism, causing a complete disintegration of the tissues. This condition is known as the wet rot stage. When infected tubers are held at low temperatures in a dry atmosphere, little bacterial action follows the invasion of the pathogen. Under these conditions the tissues shrink, forming conspicuous, light-brown, depressed necrotic areas. This is the dry rot stage.

ETIOLOGY.—The mycelium of *Phytophthora infestans*, like that of the other members of this group of fungi, is coenocytic. It ramifies between the host cells (intercellularly) and produces its long, coiled, filamentous haustoria within the host cells. The optimum for the growth of the mycelium is 16°

portion affected, offers an easy diagnostic symptom. The pathogen produces a glistening-white mass of conidiophores and conidia on the infected areas. When the environmental conditions are not highly favorable there is little accompanying stem injury, but when conditions are very favorable the necrotic areas may extend to the stem. The collapsed cortical tissue is shown in Fig. 50.



FIG. 49. A young potato plant nearly destroyed by the late blight fungus This plant was artificially infected with the late blight organism.

On white-skinned tubers early stages of infection show as pink irregular areas, varying in size depending upon the spread of the pathogen in the tissues. If the epidermis is removed, the flesh is also pink instead of white. If such infected tubers are subjected to temperatures above 15° C. in a moist atmosphere, the color of the infected areas changes to dark brown as necrosis occurs in the underlying tissues. Soft rot bacteria frequently follow the late blight organism, causing a complete disintegration of the tissues. This condition is known as the wet rot stage. When infected tubers are held at low temperatures in a dry atmosphere, little bacterial action follows the invasion of the pathogen. Under these conditions the tissues shrink, forming conspicuous, light-brown, depressed necrotic areas. This is the dry rot stage.

ETIOLOGY.—The mycelium of *Phytophthora infestans*, like that of the other members of this group of fungi, is coenocytic. It ramifies between the host cells (intercellularly) and produces its long, coiled, filamentous haustoria within the host cells. The optimum for the growth of the mycelium is 16°

to 18° C. The conidiophores arise singly or in groups of two or four from this mycelium spreading beneath the epidermis. The conidiophore is branched, and at the tip of each branch a conidium is produced. After the conidium is formed, it is pushed to one side and the growth of the branch continues. The continuation is, however, larger than the tip that produced the conidium, so that this further growth is marked by an enlargement of the branch, making a characteristic form of conidiophore. The conidia germinate readily when mature at low temperatures, optimum 12° C., by the production of numerous zoospores, or at higher temperatures, optimum 24° C., by sending out a germ tube. The zoospores are motile for a brief time, depending upon the temperature, and finally they come to rest and round up. Germination follows readily, and the germ tube penetrates into the leaf through the stomata. The spores lose their vitality in a few hours in an atmosphere of less than 80 per cent humidity. The conidia not only serve to spread the pathogen rapidly from leaf to leaf, but they also fall upon the soil, where they may infect the tubers as readily as the leaves, causing a dry or wet rot depending upon the moisture and temperature conditions. No oospore stage of *Phytophthora infestans* has been found in the potato plant. The sequence of its developmental stages is shown in Fig. 53.

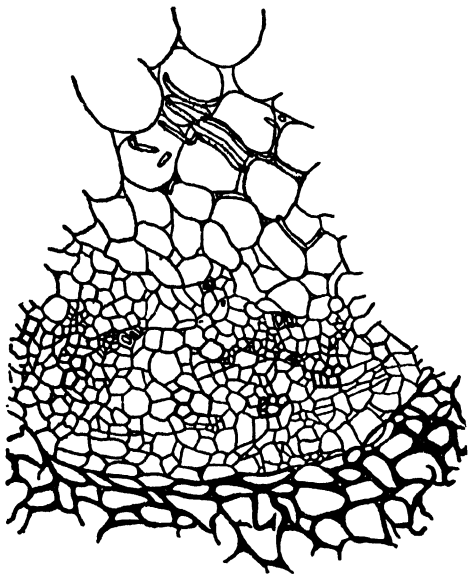


FIG. 50. Cross section of a potato stem showing the mycelium of *Phytophthora infestans* in among parenchyma cells. The cortex has been destroyed.

Many conidia naturally fall upon the soil and may be washed down near the young tubers during a rain. Such conidia may germinate either by a germ tube or by zoospores, and the hyphae enter the tubers through the buds or "eyes." The mycelium in these newly infected tubers may live within the tuber until the next season. In such cases the mycelium is said to be perennial. In the spring when the infected "seed pieces" are planted, the mycelium grows up the dwarfed stems produced by such infected tubers. At the surface of the soil the mycelium in the stems forms its conidiophores and conidia on the surface that may serve as a source of inoculum for the foliage on adjoining plants. Under favorable conditions the infection may spread until all the plants in a field or district become affected.



FIG. 51. The dry rot stage of late blight in a potato tuber.

Recently it has been shown that the late blight organism is composed of three races—one that prefers the tomato and two that occur on potatoes, distinguished from one another by their growth and development on certain wild species of *Solanum*. As far as is known at present, only two races occur in this country—one on the tomato and the other on the potato. The other race on the potato only occurs in Germany at the present. Very recently it has been shown that the virulence of *Phytophthora infestans* growing on susceptible varieties may be built up by repeatedly passing the pathogen through highly resistant varieties.

CONTROL.—The control of this pathogen involves (1) the selection of healthy seed, (2) modification of cultural practice, (3) spraying with Bordeaux mixture and (4) utilization of resistant varieties and hybrids. Clean seed is best obtained

by maintaining a seed plot, which receives special attention to insure its freedom from disease. Considerable loss can be avoided by delaying the time of lifting by high hilling and by allowing the tubers to lie in the sun after being lifted. They should be stored at 0° to 4° C.

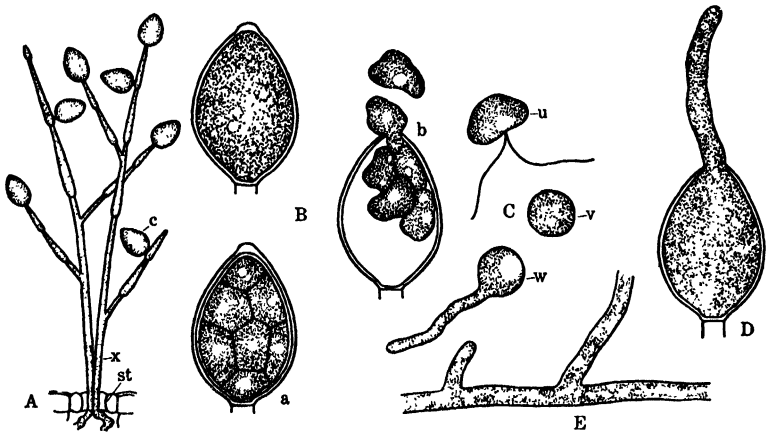


FIG. 52. *Phytophthora infestans:* A, the conidiophores x, bearing conidia c, arising from the internal mycelium through a stoma, st, B, conidia, zoospores are being formed in a and extruded in b; C, zoospores; swimming, u; resting, v; producing germ tubes, w; D, direct germination of conidium; E, mycelium.

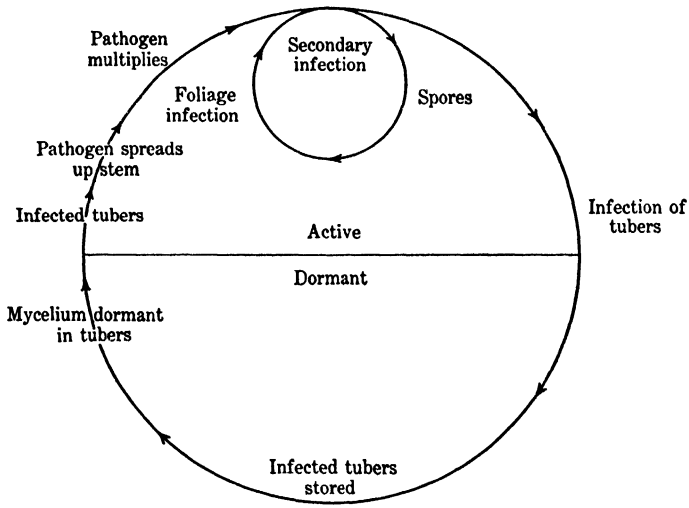


FIG. 53. A diagram of the host relation of *Phytophthora infestans*. This organism produces only asexual spores on the potato plant and its mycelium survives the winter in stored tubers.

Spraying with 4–4–50 Bordeaux mixture, starting when the vines are from six to eight inches tall and continuing every ten days until harvest time, is the only effective way of combating this pathogen in sections where late blight occurs annually. Most of our American varieties are rather susceptible, while many of the European varieties, German and British, are rather resistant. Unfortunately these

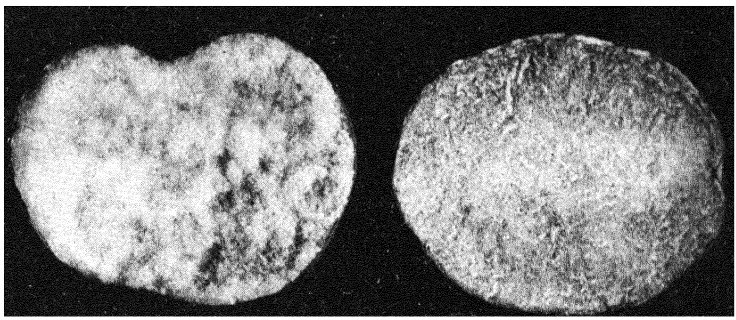


FIG. 54. One of these two cut potatoes shows a dense, white mass of conidiophores and conidia of the late blight organism.

foreign resistant varieties are not well adapted to our American growing conditions and markets. During the last few years several hybrids have been developed in this country and Germany that are resistant to the late blight organism and also are of good quality and high yield. When resistant hybrid potatoes such as the variety Sebago are made available to the grower, the cost of production will be greatly reduced in the sections where the pathogen develops in destructive form.

WHITE RUST OF CRUCIFERS

Albugo candida (Pers.) Kuntze

White rust is a common disease on cultivated and wild crucifers occurring generally throughout the world. This disease is not, as the name implies, a rust. The rusts are all caused by a group of the Basidiomycetes, which we will study later. The only way the white rusts resemble the

true rusts is in the manner in which the spores are borne. In both cases the spores develop from the mycelium forming pustules beneath the epidermis. The pathogen causing white rust is an obligate parasite which reproduces by abundant asexual and sexual spores. Its mycelium may become perennial in winter annuals and biennial host plants.



FIG. 55. White rust on the lower surface of a radish leaf.

SYMPTOMS.—White rust may occur on any of the aboveground parts of the plant. Early stages of infection are first visible as light-yellow spots. Later the epidermis over the yellow area is raised, changing the color of the spots to a creamy white surrounded by a narrow yellow zone. Finally the epidermis over the white area ruptures and rolls back, exposing a white, flour-like mass consisting of the asexual spores or conidia of the parasite. When the pustules burst, localized necrosis results. The symptoms on the stems in the early stages are the same as those described on the leaves. As the pathogen spreads in the tissues of the stem, hypertrophy and hyperplasia of the cells result, causing a material enlargement of the stem.

The enlargement frequently causes an inverted U- or S-shaped bend in the inflorescence stalk. The swollen parts are at first dark green, later changing to pale green. Pustules may be sparse or numerous on such hypertrophied areas. Later the infected tissues are killed and become brown. The flowers and pods show symptoms similar to those that develop on the stem only more pronounced as to the extent of enlargement and distortion. In wet weather the infected tissues soften, following the invasion of secondary organisms. In dry weather the distorted tissues become mummified.

ETIOLOGY.—White rust of crucifers is caused by the fungus known as *Albugo candida*. Its mycelium is intercellular and obtains its food from the adjoining cells by sending haustoria into them. The parasite probably secretes some substance, which induces the hypertrophy and hyperplasia already referred to in the paragraph on symptoms. From the mycelium in the tissues there develop later, just beneath the epidermis, short-stalked conidiophores. From the ends of these conidiophores are borne chains of conidia, which raise the epidermis, forming a typical pustule. Finally the epidermis ruptures, rolls back and exposes the conidia. The conidia may be blown about by the wind, and if they fall in a film of water upon a susceptible host each conidium produces from six to 18 motile zoospores. After a period of motility these zoospores round up and send out a germ tube, which can enter the host only through the stomatal openings.

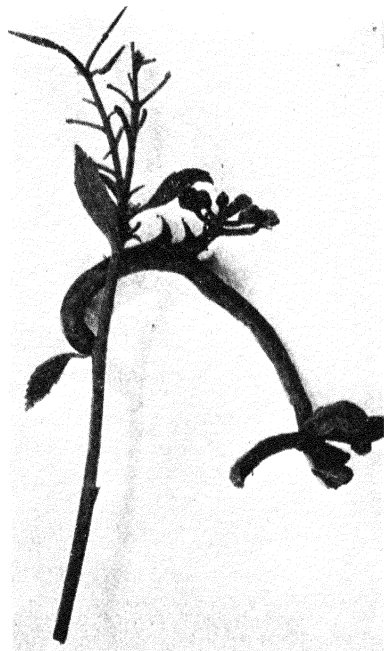


FIG. 56. White rust on floral organs of the radish showing hypertrophied structures.

When the tissues start to die, the same intercellular mycelium produces oospores. The oospores are several times the size of the conidia, thick walled, dark brown and tuberculate. They are very resistant to adverse conditions, which permits the pathogen to bridge unfavorable periods for its growth and development. With the advent of favorable growing conditions, the spores burst open, liberating a sac containing from 50 to 100 zoospores. These in turn may also bring about infection in the same way as that described for the zoospores

produced by the conidia. See Fig. 58 for a diagram of the host relation of *Albugo candida*.

In addition to producing conidia and oospores, the mycelia may become perennial in the crowns of some of its hosts and live over winter. *Albugo candida* requires damp, humid conditions for its most rapid growth and spread. In the

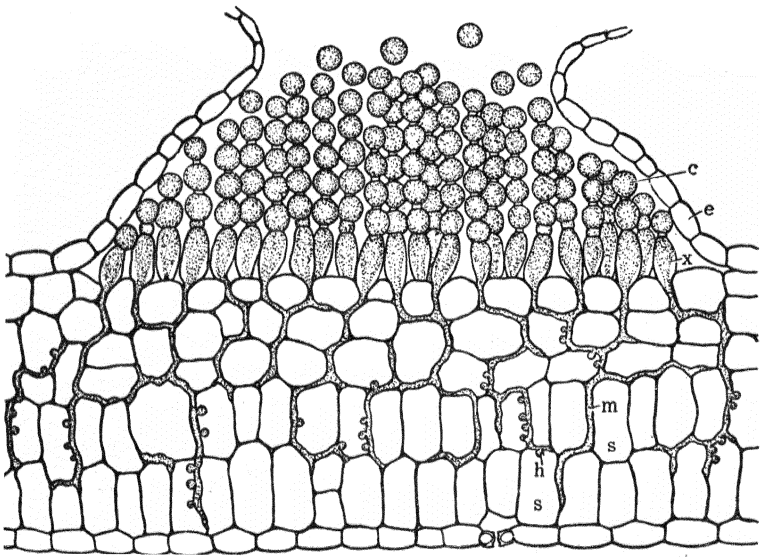


FIG. 57. Cross section of a radish leaf infected with *Albugo candida*. The intercellular mycelium, m, produces the haustoria, h, in the mesophyll cells, s. The conidia, c, are produced in chains from the conidiophores, x, and escape after the rupturing of the epidermis, e.

middle west it is most prevalent in the spring and fall when there usually is greater rainfall and lower temperatures than in the summer. During July and August its development is seriously inhibited.

CONTROL.—Little work has been done on control of the white rust fungus since the rust seldom causes serious damage. In the light of the life history of this pathogen a long rotation and sterilization of the soil in garden patches should prove worth while as control measures. The destruction of wild crucifers growing in the vicinity of gardens and avoidance of thick seeding should do much to prevent the pathogen's prevalence and destructiveness.

Downy Mildew of Grapes

Plasmopara viticola (B. & C.) Berl. and de T.

Plasmopara viticola has been known in this country since 1834; it reached France in 1875, and spread from there throughout the vineyards of Europe. In the United States the disease generally is more destructive to varieties of European origin,

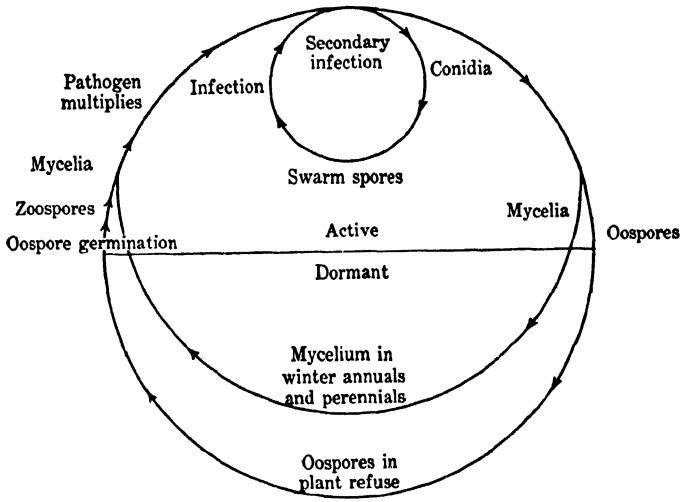


FIG. 58. A diagram of the host relation of *Albugo candida*. This organism survives the winter in the form of oospores in plant refuse and as perennial mycelium in some of the crucifers that are winter annuals or perennials.

although no variety is notably resistant under all conditions. Smooth and pubescent, wild and cultivated varieties are affected. The downy mildew occurs also on the five-leaved ivy, a close relative of the grape. It is probably native in the Mississippi valley on the wild grape. The pathogen was introduced into Europe from America on root stocks, sought because of their resistance to the insect disease caused by Phylloxera. This insect, also imported from America, was seriously destructive to the grape industry. Grape growers long had been cautioned by French scientists of the danger of importing the downy mildew, yet no special precaution was

exercised and the pathogen was introduced. Within a decade it had spread throughout Europe, which affords a striking illustration of the rapidity of spread of a plant parasite. In 1905 the downy mildew appeared in the island of Java, having been introduced through the mails, on fresh grapes. More recently it has also spread to the west coast of our own country and is reported as causing heavy losses in California.

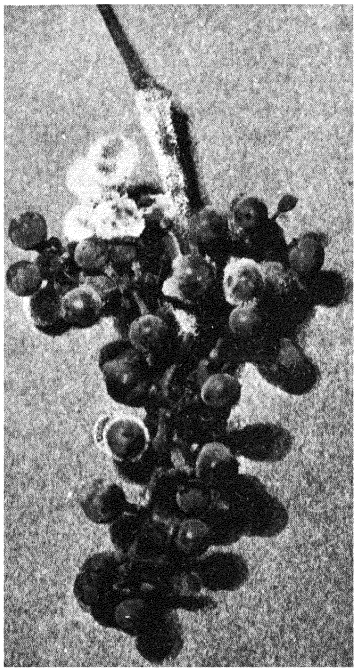


FIG. 59. Downy mildew on a cluster of green grapes. The pathogen appears as a white downy growth on some of the berries and on the inflorescence stalk.

Losses from the downy mildew pathogen have been more severe in Europe than in America, because of the greater susceptibility of the European sorts. This organism not only injures the vines, but also destroys the fruit. In 1884 it caused a loss of 18,000,000 francs in Puy de Dome in southern France, and in 1886 the crop was a total loss in Dordogne. The ravages of this pathogen were so severe in France during the eighties of the last century that many grape growers turned to other pursuits. France did not suffer alone; Germany, Italy and Austria had similar experiences with this new pathogen of the grape. Had it not been for the discovery of Bordeaux mixture in 1885, the grape growing industry of Europe would have been destroyed. The practice of spraying gave some relief from this scourge, although it is recorded that the loss in Germany in 1906 amounted to $7,500,000 and in 1925 to 40 per cent of the crop.

In the United States losses resulting from the downy mildew

are greatest east of the Mississippi River. They are seasonal and range from a trace to 10 per cent.

SYMPTOMS.—This pathogen attacks all parts of the plant above ground, leaves, young stems and fruit. The leaf

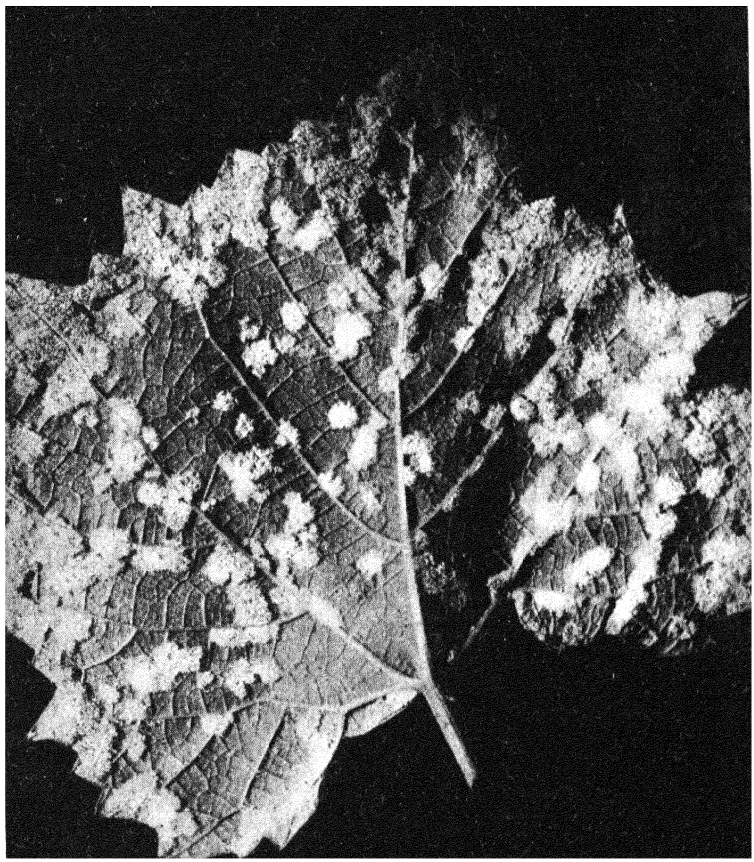


FIG. 60. The undersurface of a grape leaf showing the downy mildew

infection appears conspicuously as light-yellow areas on the upper surface. These areas may be large or small. If several areas fuse, the spots may vary from one-eighth to two inches in diameter. Soon the cells of these yellow areas become necrotic, causing a spot of dead, brown cells. On the lower

surface of these areas the fungus produces a profuse, glistening-white growth, the conidiophores and conidia of the parasite. It is not uncommon when the weather is favorable to find most of the lower surface of the leaf covered with a dense growth of the fruiting stage. On the young stems first evidence of infection consists of water-soaked areas. Later these are covered with a similar glistening-white growth. Leaves on such stems are dwarfed and distorted. The shoots are often swollen and light yellow in appearance. This type of infection is found chiefly on native hosts in the Mississippi valley in the spring and on those shoots that originate from the crown near the surface of the ground.



FIG. 61. Downy mildew on a young grape shoot. The white, downy growth of the systemic pathogen appears generally on the stem, petiole, tendrils and undersurface of leaves.

The green grapes are very susceptible, the first sign being a change in color from light green to red. Infected fruit hardens and becomes covered with a dense mass of conidiophores. After some time the fruit drops or shells, leaving only a naked fruit stalk. The pedicels of the grapes also may be attacked.

ETIOLOGY.—The downy mildew of grape is caused by one of the algal fungi, *Plasmopara viticola*. The sequence of its

host relations is shown diagrammatically in Fig. 62. As the other fungi of the downy mildew group, the mycelium of this fungus is situated within the tissues of the host, between the cells. The mycelium is nourished by means of haustoria, which penetrate the cell walls. Through them the pathogen has a direct contact with the protoplast of the host cells. From the mycelium in the tissues the thick conidiophores finally emerge through the stomata. They are much-branched, tree-like and bear conidia on their tips. These conidia are

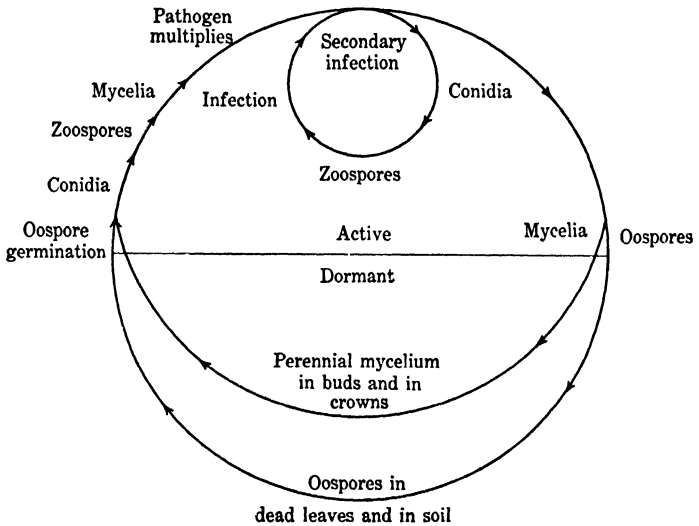


FIG. 62. A diagram of the host relation of *Plasmopara viticola*. This organism survives the winter in the form of oospores in plant refuse.

easily detached and blown by the wind to neighboring leaves or vines where they germinate and cause new infection. While the conidial stage is being produced on the surface, the oospore stage is developing inside the leaf. The mature oospores are brown, thick-walled resting spores which may germinate and renew infection in the spring. The oospores on germinating send out conidiophores bearing single large conidia that may be wind blown to the new foliage. The conidium, under favorable conditions, germinates by zoospores and causes primary infection. The mycelium may also serve as a means of carrying the fungus through the winter. It is

known to live over as a perennial mycelium inside the buds and in the crowns of the plant.

The conidia germinate in the dew and rain on the leaves by forming swarm spores; that is, the contents of the conidium

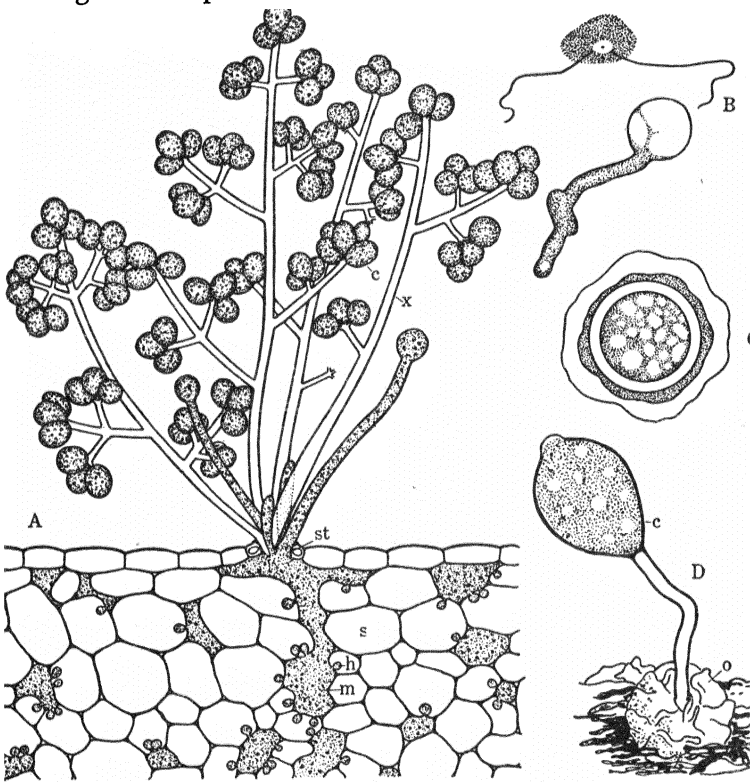


FIG. 63. *Plasmopara viticola:* A, the intercellular mycelium, m, bearing the haustoria, h, inside the host cells, s, produces conidiophores, x, which extend through a stoma, st, and bear conidia, c; B, motile and germinating zoospores; C, dormant oospore inside of the old oogonial wall; D, large conidium, c, produced on the germination of an oospore, o. (Figures B, C and D redrawn from C. T. Gregory.)

separate into six or eight small naked portions. Each portion has a definite shape and two cilia by means of which it swims about in the moisture on the leaf. After a short period of motility these swarm spores round up, put down a wall and germinate by formation of a tube. It is this tube that penetrates the leaf by way of a stoma. *Plasmopara viticola* grows best at 25° to 27° C. Shaded and moist situations are the most

favorable for its development, and most of the injury occurs during humid weather. While long dry periods do not kill the pathogen, they seriously check its development.

CONTROL.—Since the dead leaves carry the oospore stage it is advisable to rake up or plow under the leaves in the spring, thereby preventing the primary infection resulting from the oospores. An effective method of control for the downy mildew of the grape is to spray the vines with Bordeaux mixture 4–4–50, making five to six applications during the growing season. The first application should be made just before the blossoms open in the spring, and the other applications should follow at intervals of about two weeks. It is usually advised that in addition to the spraying of the vineyard, infected buds or canes should be removed as soon as they are detected.

BROWN SPOT OF CORN

Physoderma zeae-maydis Shaw

Brown spot of corn is caused by one of the phycomycetous gall fungi. It is prevalent on corn in the southern Gulf States and in the Orient. The only other known host plant is teosinte. This disease rarely occurs in the central and northern corn belt section of the United States. Physoderma has probably existed in the southern states for a long time, although the first published record of its occurrence in this country was in 1913.

It is most destructive in Alabama, Georgia, South Carolina, Mississippi and Florida. Outside this area the injury to corn has not been of any consequence. Under very favorable conditions for the pathogen, however, losses of 10 per cent may occur. All varieties of corn are susceptible, and some inbred lines in Illinois have been noted as more susceptible than others.

SYMPTOMS.—Brown spot occurs on the blades, leaf sheaths and stalk of the corn plant. The early symptoms are small, yellowish spots that, within a few days, turn a darker yellow and later become brown. In general, these necrotic areas are

circular in shape and the surrounding tissues tend to take on a reddish tinge. The epidermis becomes bulged, blister-like and is finally broken, liberating a fine powdery mass consisting of the resting spores of the parasite. The spots may be numerous or sparse. If plentiful, the spots may



FIG. 64. Brown spot of corn. The black spots are brown lesions in which may occur some of the pathogen. (Reproduced from *Jour. Ag. Res.*, Separate G-163.)

coalesce and form much larger yellowish-green streaks thickly sprinkled with light-brown pustules. In general appearance the sori, where the spots are numerous, resemble corn rust, where the uredospore pustules are about to break through to the surface. Infection is most abundant on the lower part of the plant.

The parasite causes larger spots on the midribs and leaf sheaths than on the blade. When the infection takes place

on the inside of the leaf sheath, reddish spots develop on the outer side of the sheaths. Physoderma is more prevalent about the nodes than elsewhere, and severe infection at this point will lead to shredding of the sheath and a disintegration of the cortical tissues, that may cause the stalk to break or "lodge."

ETIOLOGY.—Brown spot of corn is caused by *Physoderma zeae-maydis*. In the brown spot lesions there are a number of

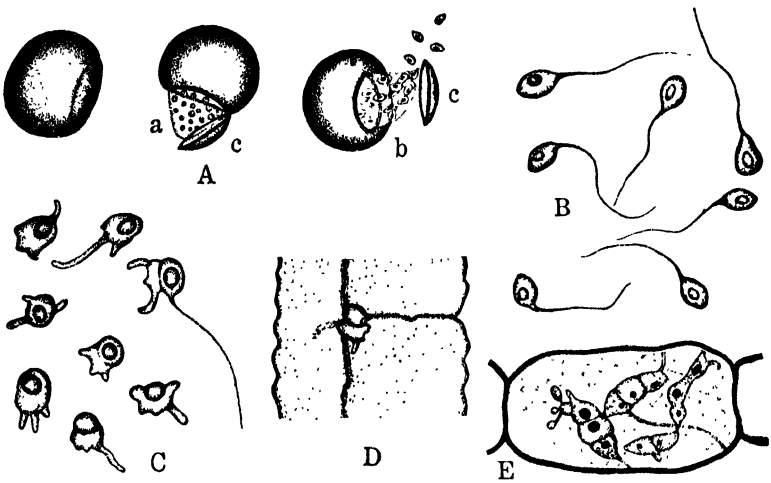


FIG. 65. *Physoderma zeae-maydis:* A, sporangia showing escape of spores at a and b after opening of lid, c; B, zoospores; C, amoeboid spores; D, infection of host cells by amoeboid spore after coming to rest; E, turbinate cells of the pathogen within a host cell. (Redrawn from W. H. Tisdale.)

thick-walled, smooth, yellow resting spores provided with circular lid-like openings on one side. These spores may be blown about by the wind or fall on the ground and become mixed with the soil. With favorable growing conditions, these resting spores germinate and produce zoospores, 3 to 4 by 5 to 7 microns in size, with one cilium three to four times the length of the zoospore. It is these motile zoospores that bring about infection. They produce a crop of thin-walled spores on the surface of the host or send out a fine rhizoid-like tube that penetrates into a cell of the epidermis, where it may branch repeatedly. Later, infecting rhizoid-like tubes pene-

trate the adjoining cells forming relatively large, branched, swollen hyphae-like structures. This vegetative part of the parasite may enlarge and form the resting spores in the cells, which are liberated through the death and disorganization of the tissues.

The thin-walled spores may form when the zoospores come to rest on the surface of a young corn leaf by the development

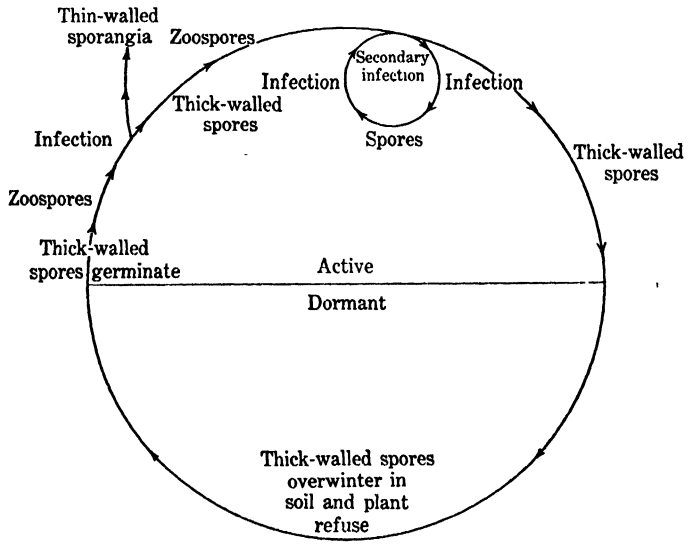


FIG. 66. A diagram of the host relation of *Physoderma zeae-maydis*. This organism forms its thick-walled resistant spores by all the protoplasm passing into the swollen portions of the hyphae.

of an irregular slipper-shaped structure somewhat larger in diameter than the original spore. This body enlarges and becomes a spore in which there may be formed 300 smaller zoospores similar to those developed in the resting spores. The role of these thin-walled spores is not well understood, but their importance in the life history, spread and destructiveness of the pathogen may well be significant. A diagram of its host relation is shown in Fig. 66.

Physoderma is intracellular and limits its activity to one or a few cortical cells. The cells adjoining those attacked by the pathogen are stimulated to increase in number and size. The

effect is a tumor-like enlargement in which necrosis later results.

This pathogen is greatly dependent upon proper temperature and moisture conditions. The resting spores require a minimum of 23° C. for germination. The optimum is 28° to 29° C. In addition to high temperature, the pathogen needs a film of water for the spores to germinate and for infection to take place. This may explain the high amount of infection inside the leaf sheath where such conditions occur naturally. Temperature and moisture conditions probably limit this pathogen to the southeastern United States and prevent it from developing in the cooler northern or arid western portions of the corn belt.

CONTROL.—Little work has been done on the control of this disease, but its life history and biology suggest that sanitation through destruction of infested refuse, through deep plowing and crop rotation should reduce the damage. Where corn is fed as a fodder, it is well to avoid applying the manure to fields to be used immediately for corn.

CLUB ROOT OF CABBAGE

Plasmodiophora brassicae Wor.

Club root is a disease that has been known in Europe for more than two centuries. It was first recorded in England in 1736. The disease is caused by one of the lower Phycomycetes that attacks the roots of cabbage, radish, turnip, cauliflower, rape and other Cruciferae, producing swellings or tumorous enlargements. It is from this symptom that the disease has received the names of club root and finger-and-toe disease.

Club root of cabbage occurs in U. S. S. R., Germany, France, British Isles, Spain, Scandinavia and in many other countries. It is probably more destructive in Europe than elsewhere. As early as 1876 it is said to have caused a loss of $225,000 in the vicinity of St. Petersburg, Russia. In many instances in northern Europe it has become necessary to abandon lands for cabbage and turnip culture for five to ten years.

It is generally distributed over the north central and eastern portions of the United States, extending as far west as Wisconsin, Iowa and Minnesota. The heaviest losses have been reported from Vermont, Virginia, New York and Wisconsin. Market gardeners near our larger eastern cities probably have suffered the most serious losses. The fact that the pathogen is soil-borne and is capable of attacking such a wide range of cruciferous hosts makes Plasmodiophora a serious limiting factor in intensive cabbage growing sections.



FIG. 67. Two of the three young cabbage plants show club root. Note the spindle-shaped primary root and the knob- or finger-like laterals. (Courtesy of B. F. Lutman.)

SYMPTOMS.—The club root organism may attack cabbage and other crucifers in the seedling stage in seedbeds, as well as more mature plants after they are transplanted into the field. The pathogen gains entrance into the cortical tissues where it multiplies and induces rapid hyperplasia and hypertrophy, forming the characteristic root enlargements. These swellings at first are smooth, pale yellow and brittle. Later they become dark colored, rough, crooked, soft and flabby. The clubs on the roots are spindle-shaped, while those on the lower part

of the stem become spherical. The broken surface of the swellings permits the entrance of soft rot organisms that may cause a rapid decay of the diseased root.

Infected plants gradually lose their lower leaves, become stunted and distorted. The plants fail to head and only a naked, leaf-scarred stem remains. In other cases the leaves turn yellow and wilt or flag during the hottest part of the day, because of the underdevelopment of the vascular system of the roots, coupled with the continued growth of the tops. As the season advances and days become warmer, the vascular system is unable to convey adequate water to the leaves and the plant wilts and dies.

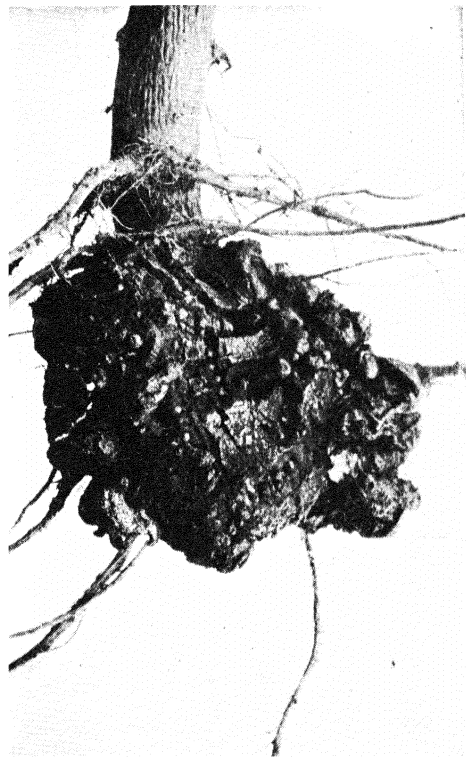


FIG. 68. A large club on the root of an old cabbage plant showing the rough surface of the gall.

ETIOLOGY.—Early students of the club root erroneously attributed the disease to many different causes, as insects, nematodes, soil and climate. Woronin, a Russian, first showed, between 1873 and 1877, that a Phycomycete, *Plasmodiophora brassicae*, was the real cause. The host relation is outlined diagrammatically in Fig. 69. This organism lives in the soil or diseased roots in the spore stage. These resting spores swell and liberate swarm spores that have a single anterior flagellum. Germination occurs only in a film of water at

temperatures ranging from 6° to 28° C. The optimum range is 18° to 25° C. The swarm spores enter the host either through the base of the root hairs, through the rupture caused by the roots developing on the stem or through

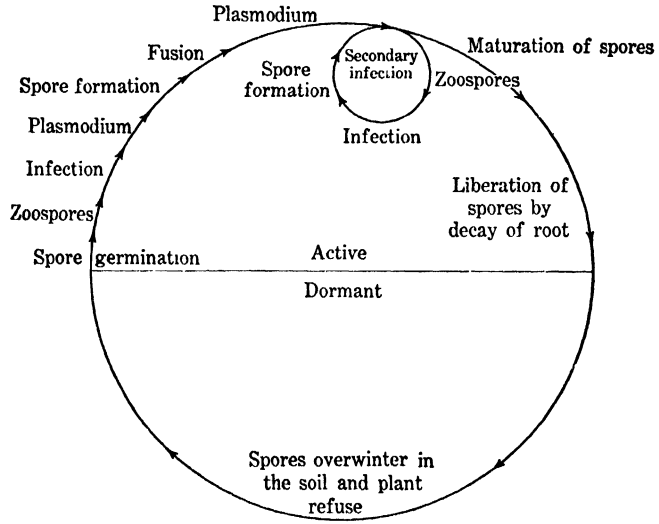


FIG. 69. A diagram of the host relation of *Plasmodiophora brassicae*. This organism produces spores capable of remaining dormant for about seven years.

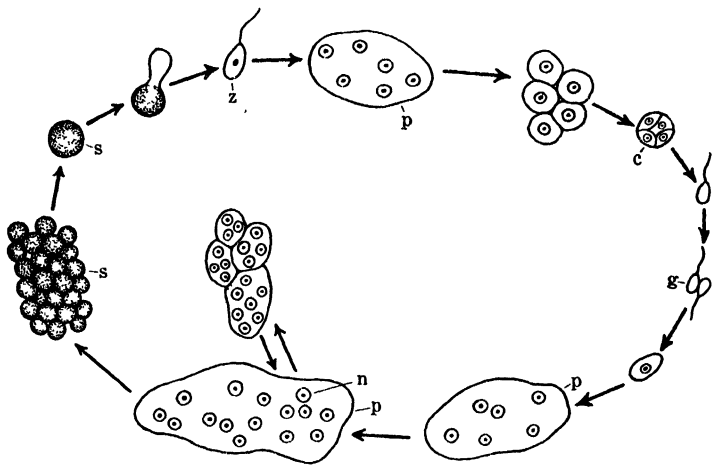


FIG. 70. A diagrammatic life cycle of *Plasmodiophora brassicae*. See discussion under etiology; s, spores; z, zoospores; p. plasmodium; c, conidium; g, gamete; n, nuclei. (Redrawn from W. R. Ivimey Cook.)

stem wounds. Once inside the host, the swarm spore forms a small, naked, multinucleate vegetative body, the plasmodium. This later divides into a number of separate thin-walled bodies in which are formed from four to six small zoospores that may migrate from the root hairs into the cortical tissues where they may fuse in pairs, forming a swarm spore of the same size as the one that originally attacked the host. Such swarm spores produce the plasmodia that occur generally in the host cells.

These plasmodia may migrate as a whole or separate into parts and spread up and down the root in the cambial region, stimulating spindle-shaped swellings of the tissues. Once the pathogen has reached the cambium, it spreads inward and infects the medullary rays or moves outward from the cambium into the cortex. The migrating plasmodia grow and divide as they are carried from cell to cell. Finally the plasmodia break up into thick-walled, brown resting spores. The roots soon decay, liberating the resting spores in the soil where the organism may remain alive for at least seven years.

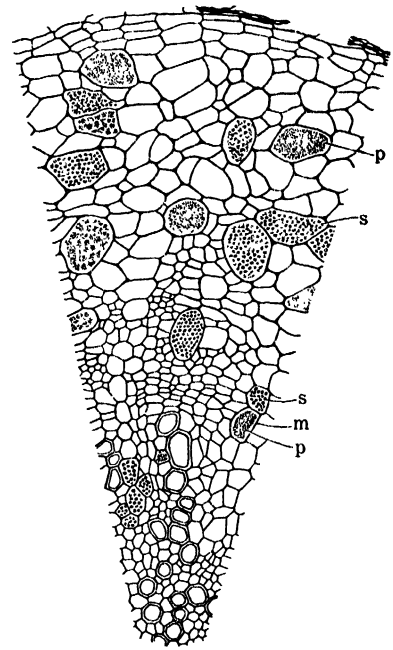


FIG. 71. Partial cross section of clubbed cabbage root. The hypertrophied cells, m, contain a plasmodium, p, or a spore mass, s, of *Plasmodiophora brassicae*. Note the abnormal separation of the few vessels present.

This organism is most prevalent in cool, wet soils. The moisture content of the soil must be greater than 45 per cent of the water-holding capacity for the club root organism to cause infection. The pathogen prefers an acid soil and is inhibited in a neutral or only slightly acid soil.

It develops rapidly after an excessively rainy season, especially on poorly drained soils. The most favorable temperature for infection lies between 18° and 25° C. After a plant becomes infected its death is hastened by hot weather.

The pathogen is disseminated by feeding the clubbed roots to stock and distributing the manure on the land, or by transfer of contaminated soil on hoofs of animals, shoes of men, tools, worms, etc. It also may be carried on non-susceptible host transplants such as celery roots. The association with worms complicates the control measures because of the movement of the worms from place to place.

CONTROL.—Great care should be exercised to prevent transfer of the pathogen from an infested soil to one not infested. Tools should be thoroughly cleaned in passing from one field to another; animals should be prevented from passing through the fields that are infested; care should be taken that manure does not become infested. Seedlings for transplants should be grown on soil known to be free from *Plasmodiophora brassicae*. Some varieties of cabbage are more resistant to the pathogen than others. Certain cruciferous weeds are immune, while others are susceptible. The weed hosts should be destroyed in infested fields. The application of one to two tons of hydrated lime per acre, worked into the soil enough in advance of planting to permit the reduction of the acidity, has proved effective in some localities.

POWDERY SCAB OF POTATOES

Spongospora subterranea (Wallr.) Johnson

Powdery scab occurs on the potato, tomato and several other species of the family Solanaceae, but is of economic importance only on the potato. The disease is well known throughout northern Europe, northern United States, Canada, the British Isles and South America. It was first found in the United States and Canada in 1913. It is possible that the pathogen was carried into the United States from Canada through the adjoining potato section of the state of Maine.

Powdery scab is not a serious disease in this country. The

prevalence of the pathogen is limited to humid, cool climates. The pathogen is probably more prevalent in the British Isles, northern Europe and Canada than anywhere else. The losses that it entails are caused by the lesions in the flesh of the tubers and the unsightly appearance of the scabby surface that reduces the market value of the potato for table and seed purposes. Under favorable conditions it is not uncommon to find from 10 to 50 per cent of the crop showing scab sori. Severe attacks of powdery scab organism develop when potatoes are grown on the same soil year after year.

SYMPTOMS.—The earliest symptoms are small, light-brown, circular raised spots. These spots may be surrounded by a



A B C

FIG. 72. Powdery scab of potatoes showing the scabby lesions, A; the cankerous stage, B; and dry rot, C.

translucent ring one to two mm. in diameter, having a very definite margin. The presence of the pathogen in the cortical tissues causes rapid cell division in localized areas. The aggregate effect is a white, spherical, tumorous growth protruding through the broken epidermis of the tubers, roots or stolons about one-half centimeter in diameter. (See Fig. 73.) Finally the infected host cells die, leaving behind an open sorus filled with a brown debris consisting of dead host tissue and spore balls. The bottom of the sorus is devoid of cork cells, which means that each sorus remains an open wound. The torn skin standing about the periphery of the

sorus is one of the characteristic symptoms of powdery scab, which often enables one to distinguish it from common scab, described in a later chapter. Frequently several sori occur in close proximity, forming brown scabby patches on the surface of the tubers. As the result of the loss of water when infected tubers are placed in dry storage, shrinking and shriveling may develop around the sori. The injury to the tubers may be considerable, depending on storage conditions. A dry rot may develop about the sorus and in the adjoining shriveled tissues through the activity of Spongospora plasmodia or by the infection and spread of storage rot organisms as species of *Fusarium* and *Phoma*. In such cases brown or black depressed necrotic areas develop that may be limited or widespread in the tuber.

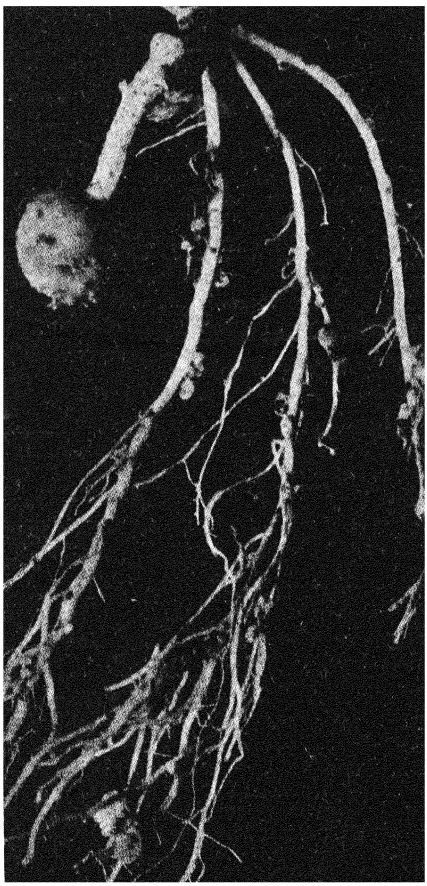


FIG. 73. Galls on potato roots produced by the powdery scab organism. (After I. E. Melhus, J. Rosenbaum and E. S. Schultz.)

When conditions are highly favorable for Spongospora it may remain active in the plasmodial stage after the tubers are mature and before they are lifted. In such cases the plasmodia penetrate deep into the flesh of the tubers leaving behind large cavities and malformed tubers. This is known as

the cankerous stage. It is not common in this country and develops only where the crop is permitted to remain in wet soils following maturity.

ETIOLOGY.—The powdery scab of potatoes is caused by the phycomycetous fungus *Spongospora subterranea*. Its host relation is illustrated in Fig. 74. The spore balls found in the old sori, which are responsible for the powdery nature of the scabby area, are made up of numerous spores, each of which on germinating gives rise to a single uninucleate amoeba. The amoebae move around by pseudopodia and

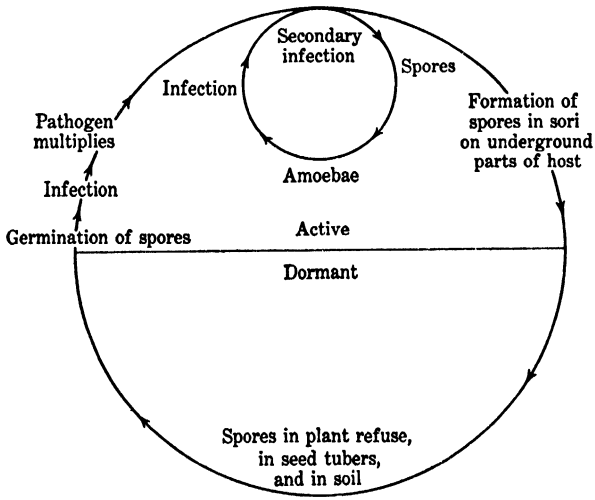


FIG. 74. A diagram of the host relation of *Spongospora subterranea*. This organism survives the winter in the spore stage in the soil and in diseased roots and tubers.

fuse to produce small plasmodia. The plasmodium penetrates through and between the epidermal cells, killing many in the process, and comes to rest below the epidermis. The epidermal cells so killed produce the light-brown spot typical of the earliest symptoms. The plasmodium develops and separates the epidermis from the cortex, causing the translucent ring-like appearance. From the lower surface of this plasmodium, pseudopodia are pushed down between the cortical cells, dissolving the middle lamellae and softening the cellulose of the cell walls. This penetration of the

plasmodial branches may extend inward from two to six layers of cells.

The cells in contact with these plasmodial branches are stimulated to growth and cell division. During this growth there are produced from the plasmodial branches small



FIG. 75. A cross section of a sorus of *Spongospora subterranea* on a potato. Spore balls and other debris still remain in the sorus. The bottom of the sorus is actually an open wound because no cork cells have formed. (After I. E. Melhus, J. Rosenbaum and E. S. Schultz.)

"penetrating pseudopodia" that push through the softened cellulose cell wall into the growing cell. Once within the host cell, the protoplasm of the parasite seems to pass into that of the host cell surrounding the host nucleus. The

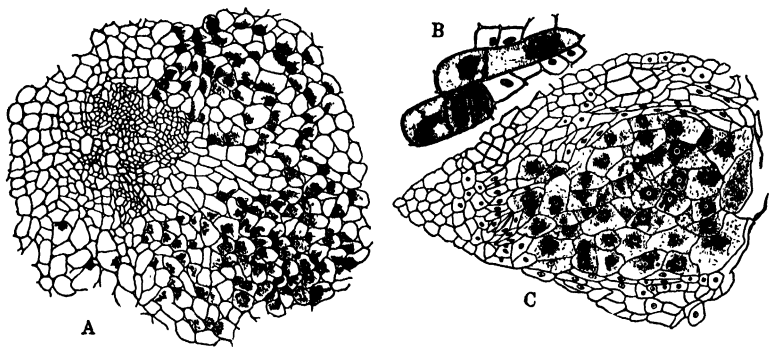


FIG. 76. Cross section of infested potato roots showing *Spongospora subterranea* in the cells. At A the infected portion of the root is enlarged. In B the pathogen is bunched around the host nucleus. At C the amoebae are in the hypertrophied cells. (After I. E. Melhus, J. Rosenbaum and E. S. Schultz.)

portion of the plasmodium within the host cell separates from the parent plasmodium. The host nucleus may become lobed and distorted or it may remain normal until the spores are produced. Meanwhile the infected host cells have enlarged to approximately ten times their normal size, and these giant cells then undergo nuclear division and form five to six small cells. These small cells are always infested by a plasmodium and never appear to be stimulated to any further growth. The giant cells in their elongation spread radially, rupture the epidermis and expose the white cortical tissues of the host. The plasmodium also enlarges until it fills the cell. The nuclei divide and some of the cytoplasm of the plasmodium collects around each nucleus, forming the uninucleate spores of the spore ball.

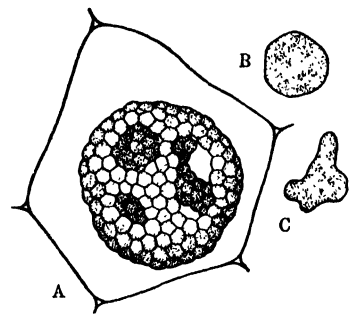


FIG. 77. A, spore ball of *Spongospora subterranea* within a host cell; B, a single spore; C, amoeboid spore.

In the older sori the plasmodia produced by the germination of spore balls, in situ, enter and kill the surrounding host cells. There is no cell stimulation as in the case of infection in the young cells. This may produce the dry rot stage of the disease. If penetration is radial, the deep, cankerous rot of the tuber results.

The spore balls escape from the infected cells after the sori rupture and overwinter in the soil. The spores produce amoebae which if they fail to fuse or enter the host may encyst on the advent of unfavorable conditions and liberate an amoeba on the return of favorable conditions. The amoebae again cause infection on young formative potato tissue of the tubers, roots and stolons.

The prevalence of *Spongospora subterranea* can be rather definitely correlated with soil types in some of which the organism is thought to survive for at least 14 years. The organism flourishes in a soil that is poorly drained and neutral or slightly alkaline in reaction. Experiments have shown that

the application of lime favors the organism, while acid phosphate and potassium sulphate tend to inhibit it. Thick-skinned varieties of potatoes have been shown to be more resistant than the thin-skinned ones.

CONTROL.—The control of Spongospora may be effected by avoiding the use of infected seed and infested soil. Where this is not possible, the land should be thoroughly drained before it is used for potatoes. The seed stock should be selected for freedom from disease and treated with formaldehyde before planting. If it is common practice to use a commercial fertilizer, it is desirable to have it carry a higher percentage of acid phosphate and ammonium sulphate than normally used.

Chapter Nine

DISEASES CAUSED BY BACTERIA

DISEASES of plants may be caused by the fission fungi, or bacteria, technically known as the Schizomycetes. An understanding of diseases caused by the bacteria had to await the development of the microscope and the overthrow of the theory of spontaneous generation of disease. Bacteria were discovered by Leeuwenhoek in 1683, and in 1860 Pasteur showed that bacteria did not originate spontaneously.

In 1863 Devaine showed that anthrax of cattle was caused by a specific bacterium and that the disease might be transmitted by a transfer of the organism to a healthy individual. These results were fiercely attacked for the next ten years. It remained for Robert Koch in 1876 to establish definitely that *Bacillus anthracis* (Cohn) emend. Koch was the cause of anthrax in animals and man. He (1) cultured the bacterium of anthrax from the blood, (2) inoculated susceptible animals, (3) reproduced the disease and (4) recovered the bacterium. In this way he fulfilled the cardinal requirements for the proof of the pathogenicity of the organism *Bacillus anthracis*. Not only did he establish the pathogenicity of the bacterium concerned, but he also fixed the requirements for all future practice in establishing the causation of a disease. These are now known as Koch's Postulates and are always carried out in determining the causal relationship of a pathogen in diseases of plants.

Only three years after the germ theory had received this indisputable experimental support on the animal side, Burrill, in 1879, showed that fire blight of apples was due to a specific bacterial organism. This means that almost all of our knowledge of diseases of plants caused by bacteria has been obtained

within the last half century. During the first half of this period progress was very slow and controversial. Strong impetus in this field came through the leadership of Dr. Erwin F. Smith who was an active worker in the field for over 40 years. The work of this man laid a thorough foundation for our knowledge of this group of diseases.

PLANT PATHOGENIC SPECIES

The number of known species of bacteria pathogenic to plants is not large, although diseases caused by bacteria are now known to occur in more than 150 genera of flowering plants. The bacteria causing diseases are almost all in the family Bacteriaceae. This family includes all true bacteria that are rod-shaped and do not produce endospores. The plant pathogenic species are gram-negative, white or yellow, non-spore-forming, non-acid fast, aerobic rods. Most of them are motile, however, some few are not. A fluorescent pigment is produced by some species. Bacterial plant pathogens differ from animal pathogens in that the former are usually favored by an acid medium and low temperature (20° to 30° C.) and the latter by an alkaline medium at the temperature of the blood of man (37.6° C.).

There are six species of bacteria pathogenic to plants not included in the Bacteriaceae. Three of these belong in the genus *Spirochaeta* in the order Spirochaetales. This family includes spiral bacterial forms. The pathogenicity of these three species is somewhat questionable. They are usually found limited to the latex vessels of milkweeds. In addition there are three very important species in the genus *Actinomyces*. More recently they have been placed in a separate order of bacteria, Actinomycetales. The most important of these three is *Actinomyces scabies* (Thax.) Güssow, the cause of the common scab of the potato.

The student will discover in consulting reference books that the same bacterial pathogen may have two or more scientific names. The different names that apply to the organisms discussed in this chapter are as follows:

Bacillus amylovorus (Burrill) Trev., 1889, or *Erwinia amylovora* (Burrill) Com. S.A.B., 1917.

Bacillus carotovorus Jones, 1901, or *Erwinia carotovora* (Jones) Holland, 1920.

Pseudomonas phaseoli (E.F.S.) E.F.S., 1901, *Bacterium phaseoli* (E.F.S.) E.F.S., 1905, or *Phytomonas phaseoli* (E.F.S.) Bergey et al., 1923.

Pseudomonas malvacearum E.F.S., 1901, *Bacterium malvacearum* (E.F.S.) E.F.S., 1905, or *Phytomonas malvacearum* (E.F.S.) Bergey et al., 1923.

Bacillus tracheiphilus E.F.S., 1895, or *Erwinia tracheiphila* (E.F.S.) Holland, 1920.

Bacterium stewartii (E.F.S.) Stev. 1925, *Aplanobacter stewartii* (E.F.S.) McCull., 1918, or *Phytomonas stewartii* (E.F.S.) Bergey et al., 1923.

Pseudomonas campestris (Pammel) E.F.S., 1897, *Bacterium campestre* (Pammel) E.F.S., 1905, or *Phytomonas campestre* (Pammel) Bergey et al., 1923.

Bacterium tumefaciens Smith & Towns., 1907, *Pseudomonas tumefaciens* (Smith & Towns.) Duggar, 1909, or *Phytomonas tumefaciens* (Smith & Towns.) Bergey et al., 1923.

Pseudomonas rhizogenes R. B. W. K. & S., 1930, or *Phytomonas rhizogenes* R. B. W. K. & S., 1930.

Actinomyces scabies (Thaxter) Güssow, 1914, or *Oospora scabies* Thaxter, 1890.

The student naturally wishes to know why an organism should have more than one name. It should be recalled that the role of bacteria in plant disease is a comparatively recent discovery and this confusion illustrates one of the numerous obstacles that must be overcome in the growth and development of a new science.

The bacterial plant pathogens may gain entrance to the host through natural openings as stomata, water pores, lenticels and wounds. They may migrate in the host through the intercellular spaces, intracellularly and in the vessels. Some are known to secrete enzymes that lead to dissociation of the cells of the tissues. In other cases it is held that the cells and vessels of the host burst as a result of the pressure exerted through the increase in number of bacteria. Bacterial plant pathogens are disseminated by insects, meteoric water (rain and dew), plant parts and man. They may survive from year

to year in plant parts, soil or insects. Modification of cultural practice, sanitation and the use of resistant strains and varieties have proved effective control measures.

Many bacterial pathogens rank among the most destructive plant parasites known. We shall proceed to study a few diseases in some detail, choosing representatives according to the following classification based on pathological effects. (See Chapter IV.)

Direct Necrosis.

(1) *General.* Fire blight (*Bacillus amylovorus* (Burrill) Trev.) of apples and pears; Jones' soft rot (*Bacillus carotovorus* L. R. Jones) of vegetables, and black leg (*Bacillus carotovorus* L. R. Jones) of potatoes.

(2) *Local.* Bean blight (*Pseudomonas phaseoli* (E.F.S.) E. F.S.) and angular leaf spot of cotton (*Pseudomonas malvacearum* E.F.S.).

Indirect Necrosis.

(1) *Vascular.* Cucumber wilt (*Bacillus tracheiphilus* E. F.S.), black rot of cabbage (*Pseudomonas campestris* (Pam.) E.F.S.) and Stewart's disease of sweet corn (*Bacterium stewartii* (E.F.S.) Stev.).

(2) *Cortical.* Citrus canker (*Pseudomonas citri* Hasse), crown gall on apples and other hosts (*Pseudomonas tumefaciens* (E.F.S. and Town.) Duggar), olive knot (*Pseudomonas savastanoi* (E.F.S.) Stev.) and potato scab (*Actinomyces scabies* (Thax.) Güssow).

Fire Blight of Apple and Pear

Bacillus amylovorus (Burrill) Trev.

Fire blight was first described from the highlands of the Hudson in New York state by Denning in 1794. William Coxe, author of the oldest American book on fruit culture, published in 1817, describes fire blight in America on the wild apple. Fire blight, therefore, probably is a disease native in America. It occurs on many different rosaceous plants as the pear, apple, quince, apricot, plum, mountain

ash, hawthorn, Juneberry, strawberry, etc. The wild species of Rosaceae suffer less than the cultivated.

The intensive development of the orchard industry favored the spread and development of the fire blight organism. This disease assumed much importance in the central west in the

FIG. 78. Destruction of the tips of the branches of an apple tree by *Bacillus amylovorus*.

middle of the last century. It crossed the Rocky Mountains into California about 1900, but only recently did it enter the Old World. In 1919 it occurred in New Zealand, and in 1924 an outbreak on the pear occurred in Italy.

Fire blight occurs generally throughout the apple and pear growing regions of the United States and Canada. It is said to be most destructive in the southern and Gulf States and the far west, where the losses have been enormous. During a period of 20 years about one-third of all the full-grown pear orchards in California were destroyed. From 1901 to 1905

inclusive the loss was estimated at $10,000,000. In the San Joaquin valley nearly 500,000 pear trees were killed. It is said that "one of the greatest industries of the San Joaquin valley vanished like a dream." In Sacramento County from April, 1930, to April, 1931, it was found necessary to remove more than 150,000 pear trees and 2,000 apple trees on account



A B

FIG. 79. Fire blight on apple: A, twig blight; B, blossom and spur blight.

of fire blight alone. The number of trees removed would make an orchard of approximately 1,500 acres.

The Bartlett pear suffers so severely that the growing of this variety has been abandoned in many sections. The damage caused by the fire blight organism also is frequently very severe in nurseries and farm orchards.

SYMPTOMS.—*Bacillus amylovorus* flourishes in succulent, rapid-growing tissues. The symptoms depend upon the part or parts of organs of the host attacked. They may be grouped as follows: leaf blight, blossom blight, fruit blight, twig blight, body blight and collar blight.

Leaf blight is the injury produced by the pathogen when it enters the leaf through the stomata and causes limited or extensive necrotic areas. It is evidenced by the blackening of the tissues as the pathogen advances. Blossom blight is the sudden wilting and turning brown of the flowers. This may occur so generally as to include nearly all the flowers on a tree. In other cases only a comparatively few flowers are killed. The attack may also occur on the green fruit, producing a fruit blight. Water-soaked areas that rapidly soften and turn black appear at the points of infection. These areas generally enlarge until the whole fruit is included.

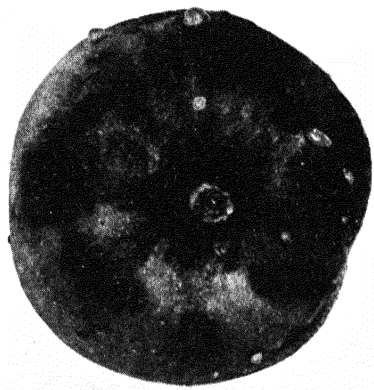


FIG. 80. Discoloration and milky exudate on apple infected with *Bacillus amylovorus*.

From all three of the above types of blight the bacteria may spread rapidly into the young branches and produce a twig blight. A wilting and blackening of the tissues similar to that on the foliage is produced in all points of the young twigs and their appendages. Sometimes the twigs are attacked directly through natural openings or wounds, where the resulting injury is the same. These infected twigs curl at the tip and are conspicuous in winter with the adhering brownish-black leaves.

The pathogen may also gain entrance into the body of the tree through water sprouts, causing necrosis of the bark. This condition is known as body blight. The dead tissue dries out, forming a shrunken area or canker. When conditions become less favorable for the pathogen in the tissues, the line of demarcation between the living and dead tissue is marked by a ridge of callous tissue. Later a crack in the outer bark shows the outline of the canker.

Where the pathogen finds its way through suckers or wounds into the trunk at the ground line and destroys the growing tissues the bark may ultimately loosen and fall away. This effect of *Bacillus amylovorus* is known as collar blight. The cankerous condition may extend up the trunk



A B

FIG. 81. Twig cankers following spur blight: A, milky exudate from "hold-over" canker, B, drying and marginal cracking of old canker. (Figure A courtesy of Wisconsin Experiment Station.)

or down below the ground line in the larger roots, which is often fatal to the tree.

In the spring of the year, trees with large cankers tend to "bleed," which permits the bacteria to come to the surface in a white gummy exudate.

ETIOLOGY.—Burrill in 1877 first reported the presence of "minute oscillating particles" in the diseased tissue. The next year he announced that these particles were bacteria and named the pathogen *Micrococcus amylovorus*. As the name indicated, he believed the pathogen to be globose in form and capable of digesting starch. Later it was shown that

the organism was a short peritrichiate rod, hence a bacillus. The bacteria may be motile in the diseased tissues. Their cell walls gelatinize, causing the bacteria to stick together in slimy masses, when they ooze to the surface of cankers. This stickiness is an important factor in their distribution.

Infection takes place mainly through nectaries, stomata, lenticels and wounds. The bacteria are best able to produce

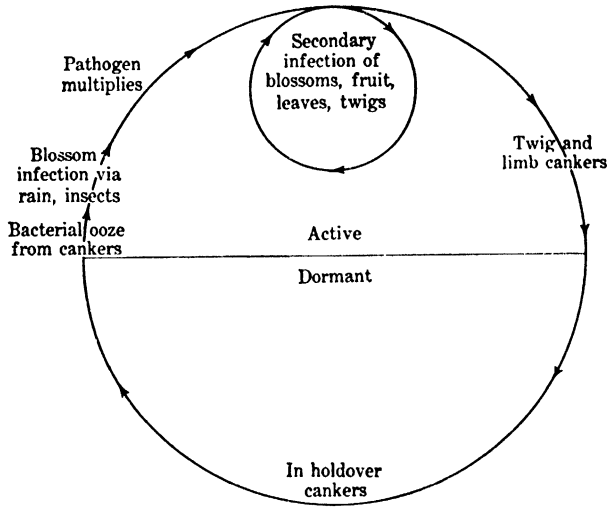


FIG. 82. A diagram of the host relation of *Bacillus amylovorus* This organism survives the winter in holdover cankers

rapid necrosis when they gain entrance to young, succulent tissues. Thus the flowers or young leaves are usually the first points of infection. Moderate temperatures and a high humidity favor infection and rapid spreading of the pathogen in the tissues.

The dissemination of the bacteria is mainly by insects and meteoric water, although wind and the activities of man also are important. Pollinating insects probably cause the primary spread of the bacteria, while biting insects are said to be more active in spreading the pathogen than sucking insects. Rain has recently been shown to be an important distribution agent.

The bacteria remain active only in host tissues. They are

unable to divide or remain virulent very long in the soil. The bacteria can live five or more days in the nectaries of the peach, plum and cherry blossoms. They can live seven days in honey dew on the surface of plants and about 72 hours in honey. Exposure to hot, dry weather or direct sunlight quickly kills the pathogen.

Since the host plants are perennial they afford a splendid habitat for the organism. The overwintering is apparently confined to the holdover cankers. In the spring, with the flow of sap, the bacteria of these cankers become active and ooze out to the surface of the tree. The organism may be carried from these holdover cankers to the blossoms, from blossoms to fruit, from blossoms and fruit to twigs, shoots, water sprouts and suckers, and from these into the bark of the trunk or root where it may overwinter. The complete host relation of the organism is represented diagrammatically in Fig. 82. The organism is said to double its numbers every 30 minutes. This leads to a rapid increase in the destructive effect on the host. It is said that the advancement through the tissues of the host, as well as a large percentage of the damage, is caused by the mechanical pressure exerted by the large number of organisms. In addition to this mechanical effect, the bacteria produce cytolytic enzymes. The action of the enzymes and mechanical pressure probably work coordinately, since the organisms remain intercellular at first. The cells of the host are apparently not entered until after the first bacteria have advanced some distance beyond the point of original infection. Following the action of the cytolytic enzymes the cells soon become filled with a black amorphous substance, which gives the typical darkening of the diseased tissues. As a result of this combined action and the rapidity of increase of the bacteria, the period of incubation is only three or four days, while the symptoms are well developed in from six to eight days. The rapidity of the development depends on the weather conditions in addition to the number of bacteria.

CONTROL.—The control of this pathogen involves the practice of sanitation, use of resistant varieties, modification

of cultural practice and spraying. Much can be done to hold the disease in check by inspecting all trees in autumn and again in the early spring before the blossoms open, cutting out and treating all cankers in main limbs and body, and dressing the wounds with some approved wound dressing.

The application of a dilute Bordeaux spray during blossoming has proved a very beneficial control measure. A weak Bordeaux spray of 1–3–50 or 2–3–50 is usually applied when 90 per cent of the flowers are in full bloom.

Throughout the summer, beginning with the falling of blossoms, it is well to make periodic inspections of all the young apple trees and remove all infected twigs. The pruning shears should be disinfected periodically by wiping with a rag moistened in a solution consisting of one part formaldehyde in nine parts of water to avoid spreading the organism. Old worthless trees full of cankers should be cut down and burned.

All water sprouts should be removed from the bodies and main limbs at least two or three times during the season. The blossoms should be removed from young trees growing in the nursery. Early stages of limb or trunk infections may be checked by application of a solution prepared by dissolving nine pounds of zinc chloride in one quart of water to which three ounces of hydrochloric acid has been added. After the zinc chloride is dissolved, the solution is added to three and one-half quarts of ethyl alcohol. The solution may be applied directly upon the surface of cankers. The wound is dressed with the above named bactericide without scarification.

Varieties differ materially in their resistance, although no variety of apple or pear is immune. Under nursery conditions there is evidence that the Yellow Transparent, Golden Russet, Sutton, Fameuse, Wagener, Tompkins King and Rhode Island are more affected than Ben Davis, Red Astrachan, Duchess of Oldenburg, Northwestern Greening, Gravenstein, Jonathan or Delicious. In the orchard, such varieties as Tompkins King, Baldwin, Grimes, Alexander, Imperial, York, Yellow Transparent, Willow Twig and Transcendent

Crab are susceptible. Genet and Duchess of Oldenburg do not suffer seriously. In the pear orchard the more resistant varieties are Duchess, Seckel, Kiefer and Aragon, while the more susceptible are Flemish Beauty, Bartlett and Clapp's Favorite. Pear and apple trees should never be interplanted in the same orchard.

As far as practical, adopt cultural practices such as sodding the orchard and avoiding heavy nitrogenous fertilizers which tend to prevent the tree from making a rapid, soft, succulent growth during June and July.

Jones' Soft Rot of Vegetables

Bacillus carotovorus L. R. Jones

Bacillus carotovorus causes a rapid wet rot of vegetables. It is general in the north temperate zone where it occurs in various crops, especially carrots, turnips, cabbage, rutabaga, radish, salsify, parsnip, lettuce, Irish potatoes, tomatoes, eggplant and peppers. This disease is not only very serious on crops in the field, but it also is responsible for heavy losses of vegetables en route to market in carload lots. Head lettuce, cabbage and celery suffer probably more than any of the other crops when the temperatures are not controlled.

Symptoms.—The typical symptom of this disease is, as its name indicates, a soft, slimy rot of the infected tissues, usually the roots. As a result of the apparent resistance of the epidermis to the bacteria producing the rot, the organ infected may be completely rotted internally while on first glance appearing normal from the outside. On closer inspection, however, the grayish, brownish or clay-colored discoloration of the disintegrated tissue becomes apparent. In the early stages the cells take on a water-soaked appearance. The soft, slimy condition is present only after the collapse of the cells, resulting from the destruction of the middle lamella.

In the advanced stage of decay, the disintegrated internal tissues may vary in color if the epidermis is broken. There is usually a very disagreeable odor of putrefaction. In the

case of carrots and cabbage, the inner vascular tissue decays the most rapidly, and the resultant mass in the very advanced stages is often highly colored by the carotinoid pigments that seemingly are not destroyed. When the rot occurs in the field, especially if it is in the crown of the plant, the outer older leaves turn yellow and wilt. These symptoms are closely followed by similar reactions of the inner leaves The rot may develop rapidly in warm damp conditions of storage when the effect on the host is the same as those described in the field.



FIG. 83. A longitudinal section of a carrot showing soft rot

ETIOLOGY.—Soft rot of vegetables is caused by a small, white, rod-shaped organism, *Bacillus carotovorus*, which is actively motile by means of two to five long peritrichiate flagella. The colonies produced in culture are circular, grayish-white, raised, smooth and usually glistening. There are apparently a large number of strains of this organism differing in the range of the hosts attacked, in physiological tests, in cultural characteristics and in serological reactions. It is a pertinent question at present as to whether these are to be called strains of a single organism or separate species of a genus or genera.

The organism seems to overwinter on stored parts or on roots, tubers and other plant parts left in the field, as well as in manure from contaminated feed and in the soil. The organism is easily spread by contact. The most important methods of dissemination are by handling, by tools, by insects and various other related means. This ease of dissemination leads to a ready source of inoculum sufficient to cause

a serious epiphytotic when conditions are favorable. The absence of such an epiphytotic under favorable conditions results in that infection is possible only through freshly wounded tissue. The host relation of the organism may be diagrammed as in Fig. 84.

As in the case of most bacterial plant pathogens, the organisms live in the intercellular spaces. An enzyme, pectinase, is produced, which effects an hydrolysis of the

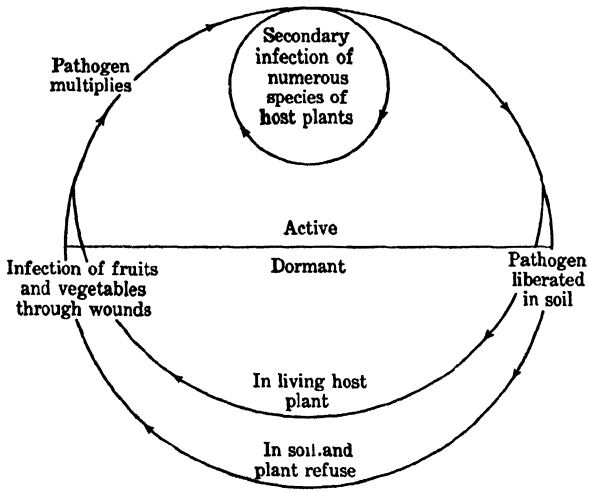


FIG. 84. A diagram of the host relation of *Bacillus carotovorus*. The organism survives the winter in the soil and in the living host plant.

middle lamella or pectic portion of the cell wall. Either this action or further enzymes produced result in a toxic effect on the host protoplasm. It is only in the later stages following the death of the protoplasm and dissolution of the walls that the bacteria enter the cells.

The soft rot organism seldom attacks well-developed green parts, nor does it develop vigorously in storage tissue unless these tissues are turgid. The attack of the organism in the field or in storage is favored by warm, damp conditions. The greatest bacterial action is said to occur in high humidities at a temperature of 25° to 30° C. The minimum is held to be 4° C., and the maximum 38° C. The loss is not great,

however, at temperatures below 25° C. Exposure of the organism for ten minutes to direct sunlight and for two hours to diffused light causes it to die. It is also extremely susceptible to desiccation.

CONTROL.—The pathogen may be controlled by rotation, by avoiding manure containing soft rot bacterial refuse, by drying the surface of roots and exposing them to sunshine before storage and by maintaining a constant low temperature (4° C.) during storage.

The practical importance of crop rotation in this disease is obvious. The fact that this organism is capable of attacking a variety of vegetables makes such measures particularly necessary. Infested soil should be devoted for a period of years to such crops as corn, other grains and grasses. If desired for garden use, the bush fruits could probably be grown safely, or seed crops as peas or beans.

The danger of introducing and perpetuating the soft rot pathogen with compost and manure is greater than is generally understood. The careful grower, therefore, should always aim to guard against this danger. On susceptible crops, only manure from grain- and hay-fed animals, or compost into which garden refuse has not entered, should be used.

One of the most striking characteristics of the organism is its susceptibility to sunlight and desiccation. Since the bacteria must be conveyed to the storage cellar almost exclusively on the surface of the roots, it follows that if these surfaces can be thoroughly air-dried before their storage, practically all the bacteria will be killed. They should be left on the surface of the ground in the field for a day after pulling and be stored at 4° C. in dry storage.

BLACK LEG OF POTATOES

Bacillus carotovorus L. R. Jones

Black leg, black stalk, stem rot—as it is variously known—was first described in Germany in 1889. In 1905 it was first found in this country in Vermont. The disease occurs generally in the United States and is frequently destructive

in the field and in storage. It is probably world-wide in distribution.

This disease is sporadic in its development. Some seasons it is prevalent and destructive, in others it is not. A wet soil and high temperatures favor its development. The black leg organism frequently destroys from 10 to 50 per cent of the crop in individual fields. In addition to reduced yield, serious losses often are encountered in storage and transit. In Minnesota the annual average loss is estimated to be about two per cent of the crop. In the maritime provinces of Canada the loss in 1915 was 10 per cent. From one to two per cent of the crop is destroyed in Maine. Often the early crop in the southern states is more seriously damaged than the late crop in the north. Although black leg often becomes serious in isolated fields and larger districts, its estimated loss for the whole United States from 1918 to 1927 was less than one per cent.

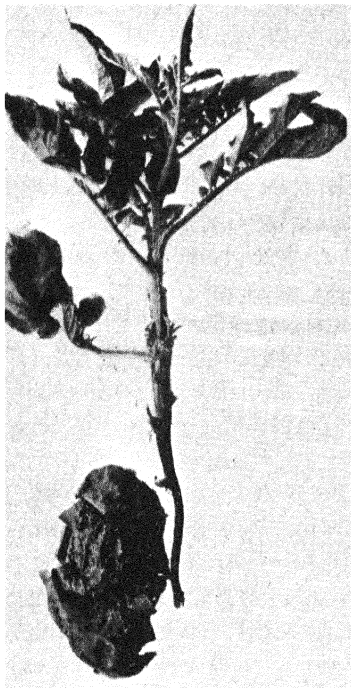


FIG. 85. Potato plant showing black leg following the spread of the pathogen from the diseased seed piece.

SYMPTOMS.—The symptoms of this disease are most conspicuous on the vines. In the early part of the growing season the leaves stand erect rather than spreading. The leaflets become light yellow, roll upward and the internodes are shortened. Where the stems are invaded by the pathogen late in the development of the plant, the foliage symptoms may be partly or wholly wanting.

A constant symptom of black leg is the progressive necrosis

of the stem, starting at the seed piece and extending upward. The infected tissues are soft and vary in color from light brown to shiny black. This black discoloration may be limited or extensive, depending upon soil conditions. If the seed piece is infected when planted or becomes infected later and soil conditions are such that the formation of a cork layer cannot take place, decay advances rapidly. When the "set" has decayed the pathogen advances into the stem

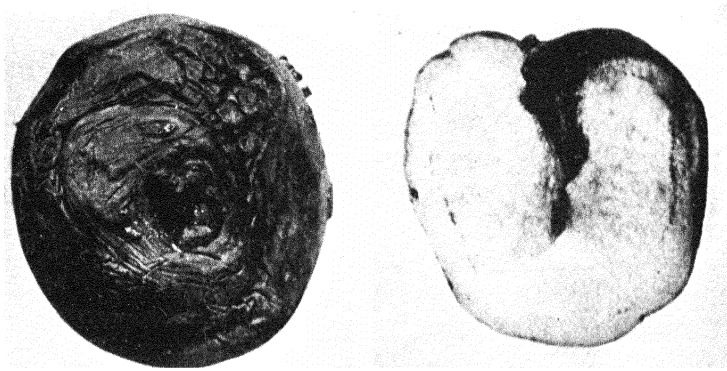


FIG. 86. Symptoms of black leg in a potato tuber.

and general necrosis takes place, causing the aerial portion of the plant to collapse.

Under favorable conditions the pathogen is known to enter the stolons and find its way into the stem end of the tubers. In such cases the vascular ring turns black and the surrounding tissues collapse. With less favorable environment the only symptom in the tubers is a slight black vascular discoloration. When such infected tubers are dug early and shipped to market in hot weather, the pathogen becomes active and causes a pronounced soft rot. Under favorable conditions, symptoms develop in three days. If slightly infected tubers are placed in dry cold storage, the diseased tissues shrink and a depression may develop about the stem end. Under proper storage conditions the parenchymatous tissues lay down a layer of cork cells that inhibit the further spread of the pathogen.

ETIOLOGY.—Black leg of potatoes is caused by a strain of *Bacillus carotovorus*. The pathogen may live over winter in the soil, in the pupal stage of the seed corn maggot and in the stem end of infected tubers. *Bacillus carotovorus* is a wound parasite that secretes an enzyme, known as pectinase, capable of dissolving the middle lamellae, which permits the parenchymatous cells to fall apart. At the same time the protoplasmic membranes of the cells are destroyed, and the sap

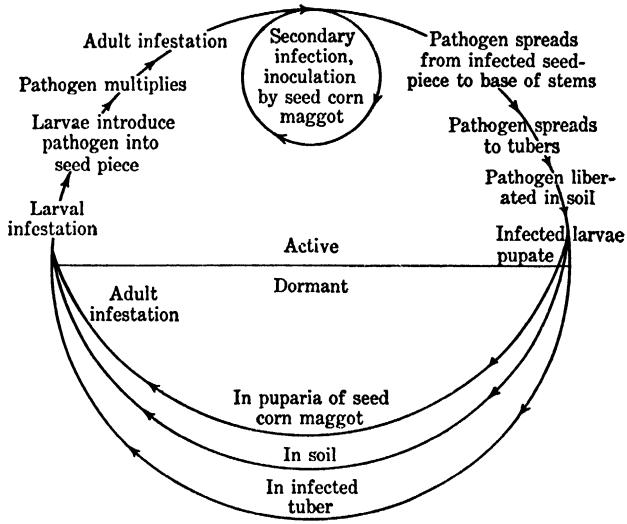


FIG. 87. A diagram of the host relation of *Bacillus carotovorus* in potatoes. This organism may survive the winter in the stem end of the infected tubers, in the soil, and in the digestive tract of the hibernating puparia of the seed corn maggot.

diffuses into the intercellular spaces, affording abundant food for the bacteria. During this bacterial action, a brownish or black pigment is formed, which diffuses into the adjoining normal tissues and vessels. The black leg pathogen grows best at about 26° C., but it is capable of withstanding extremely low temperatures, −28° C. for several hours. The pathogen flourishes in a wet soil or humid atmosphere at comparatively high temperatures. In its growth it utilizes the several sugars, but not starch. Seed pieces in the field that have been drained of all starch by the young plant may

be converted into a soft rotted mass in two to three days. Otherwise, the "set" seems to have the capacity to cork out the progress of the pathogen.

There are three ways that infection of the growing plant may take place. (1) Infection of the seed piece may take place when seed corn maggots (*Hylemyia cilicrura* Rond.) contaminated with the pathogen feed on the "sets" in the field. In feeding, the maggots cut away the cork layer or burrow into the set and leave the pathogen in the wounds, where it may induce a rot. (2) The pathogen in the soil may infect the seed piece when planted under conditions preventing the formation of wound cork, which inhibits the spread of the pathogen in the tissues of the seed piece. (3) When the pathogen is present in the seed piece it may make its way into the base of the stem of the young plant and cause a progressive necrosis.

It recently has been shown that the larvae of the seed corn maggot serve as vectors. The adult flies have been observed to lay their eggs either on or near the seed pieces in the field. When these hatch the larvae use the seed pieces for food. When young they cut through the callous tissue on the cut surface of the "set" and feed on the inner tissue. If the soil is wet no callus is formed and the soft rot pathogen may enter and develop. This condition is favorable for the development of the larvae which may feed on the bacteria and decayed tissue. Later in the development of the larvae they burrow deep into the set and carry the bacterial pathogen with them. In these deep wounds the host is unable to lay down a cork layer and the pathogen develops rapidly and finds its way into the developing stem where the characteristic stem necrosis takes place. The larvae carry the pathogen not only on their surface and appendages, but also in their digestive tracts. They feed on the decaying tissue. It is held that the presence of the bacteria is necessary for the growth of the larval stage of the insect. The bacteria remain in the intestinal tract as well as on the outside of the new stages throughout the metamorphosis from larva through pupa to the adult fly. The only stage of the insect that is

not internally infested is the egg stage. This may, however, become contaminated on the surface when the adult fly deposits its eggs. The newborn larva, in making its way out of the egg, may become contaminated by contact with the pathogen on the surface of the egg, which enables the larva to become a vector. The pathogen may survive the

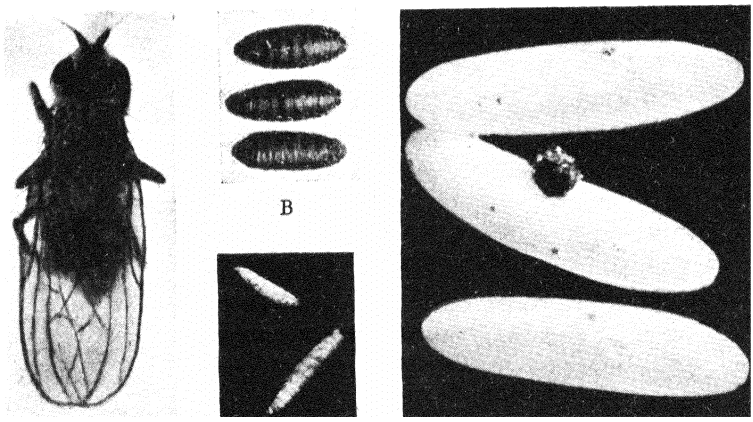


FIG. 88. The seed corn maggot (*Hylemyia cilicrura* Rond.): A, adult female fly; B, puparia; C, larvae; D, eggs. (After J. G. Leach.)

winter in the puparium, as indicated in the host relation in Fig. 87.

CONTROL.—The control of the black leg pathogen is difficult when conditions are favorable for its development. Crop rotation, field roguing and use of resistant varieties will reduce its ravages. Crop rotation is of first importance because the pathogen and the insect vector exist in soil that has recently produced a crop of potatoes. The seed should be cut and planted immediately after treatment. Field roguing consists of going through the field and pulling out infected hills as they appear during the growing season. This can frequently be accomplished when cultivating the crop. Soil that is poorly drained should not be used for potatoes. The varieties Rural New Yorker and Green Mountain are less susceptible than such early sorts as Bliss Triumph and Irish Cobbler.

Bacterial Blight of Beans

Pseudomonas phaseoli (E.F.S.) E.F.S.

Bacterial blight of beans, first described in 1893, occurs throughout all humid climates in North America and annually reduces the crop yield about five per cent. The causal agent of bacterial blight was first attributed to a single species of

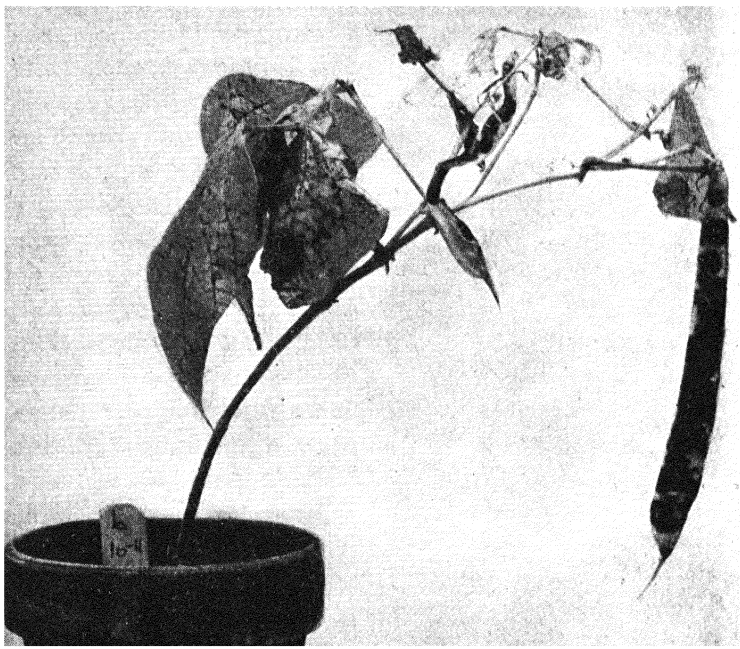


Fig. 89. Lesions on leaves and pods of bean caused by *Pseudomonas phaseoli*.

Pseudomonas, but during the past decade it has been shown that seven closely related species of *Pseudomonas* may cause blight on beans. The first of these to be described and the best known is *Pseudomonas phaseoli*.

Symptoms.—Bean blight is primarily a spot disease although the pathogen may become systemic in the vessels of the host. The most characteristic symptom is the local development of necrotic areas on the leaves, stems and pods.

On the leaves the local lesions start as small, water-soaked, translucent spots or light-green wilted areas that enlarge and become dry, brown and brittle. Surrounding this area is a yellow zone or border and around this yellow zone a narrow light-green area. One or several of these spots may enlarge until nearly the entire leaf is involved. Another type of leaf lesion often occurs on young leaves. Small angular islets between veins become water-soaked and light green, then dry and brown leaving small angular necrotic spots.

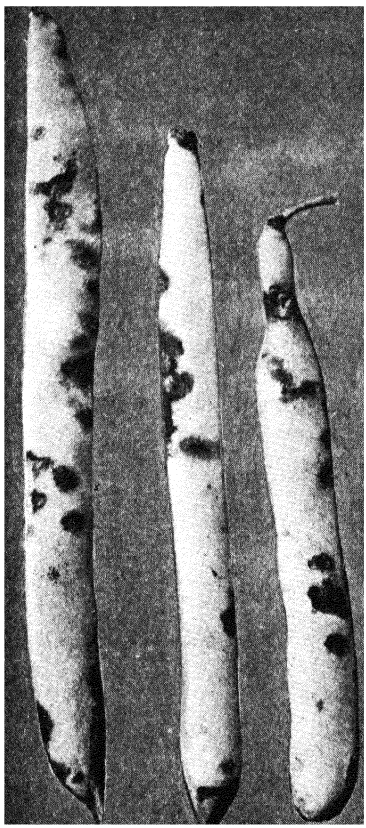


FIG. 90. Bacterial blight lesions on the bean pods.

On the stem of bean seedlings water-soaked areas similar to those on the leaves occur, but as the lesions grow older they dry down and have the appearance of reddish-brown streaks. Under certain conditions, the pathogen may become systemic in the xylem vessels. Another type of injury to bean seedlings is known as "snake heads." The terminal bud is killed and only the cotyledonary leaves remain. Older stems may exhibit still another type of injury known as stem-girdle, a transverse lesion at one of the nodes, usually the cotyledonary node. This so weakens the plant that it breaks over. On the pods the lesions start very much as those on the leaves, but as they enlarge they dry from the periphery toward the center, often resulting in water-soaked areas with brown dry borders.

On these necrotic spots it is often possible to see a yellowish incrustation of dried bacterial ooze If the lesions on the pods result from the bacteria moving in the vessels, they may occur as long irregular breaks along the suture, otherwise they are similar to those caused by local infection. *Pseudomonas phaseoli* causes yellowish spots on the light-colored seeds and if the entire seed is infected it may become wrinkled and yellow. Such seeds seldom germinate. Most primary infections in the field result from systemic infection. When white seeds become infected through the movement of the bacteria from the vessels they become yellowish because of the spread of the bacteria beneath the seed coat. In other cases the infection may show up only as a yellow spot at the hilum. This spotting is not apparent on beans with colored seed coats.

ETIOLOGY.—*Pseudomonas phaseoli*, the organism causing bacterial blight of beans, is a short rod, motile by a single polar flagellum. Though it has been shown that the organism can overwinter in the soil in old bean refuse, infected seed is the chief way this pathogen survives from one year to the next. The organism is resistant to drying and has been known to live on or in the seeds for three years. Severely infected seed may be prevented from germinating or may produce "snake heads." Less heavily infected seed may produce plants bearing cotyledonary infection from which bacteria spread to other parts of the same plant and to other plants, producing the characteristic lesions. Wounds are not necessary for infection, but moisture and a high temperature are essential. From the cotyledonary lesions or any local lesions the pathogen may enter the xylem vessels and in this manner penetrate various parts of the plant. Dissemination is brought about by the scattering of drops of water containing the pathogen, as dew or rain drops being blown from infected parts to new areas or by drops of water carrying the pathogen being transferred to new leaves by brushing in the wind or by man in picking and cultivating. Infected seed or plant debris will also spread the organism. Warm weather is necessary for rapid spread, since infection takes

place much more rapidly at 27.5° C. than at 18° C. The host relation of the organism is shown in Fig. 91.

CONTROL.—The bean blight may be controlled by using disease-free seed, the use of resistant varieties and avoiding spreading infection in cultivating, hoeing or picking the crop. Since the seed may be infected without visible symptoms, it is unwise to use seed grown in a region where bean

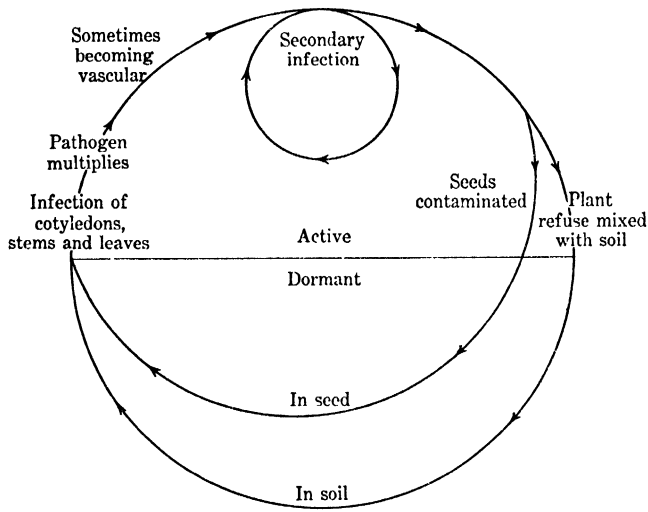


FIG. 91. A diagram of the host relation of *Pseudomonas phaseoli*. This organism survives the winter in or on the seed, or in the soil.

blight occurs. One should turn to the more arid sections of the United States for seed stocks. Certain western sections where blight does not occur make a business of producing disease-free seed. It has been found that certain varieties are more resistant than others, i.e., the Robust Pea, Yellow Eye, White Marrow and Great Northern. When infection has started in a field one should avoid cultivating, hoeing or picking when the leaves and the surface soil are damp as the bean blight pathogen is easily spread by brushing against the vines. The bacteria are capable of surviving in the soil for over a year and rotation is, therefore, important in controlling this type of inoculum.

Angular Leaf Spot of Cotton

Pseudomonas malvacearum E.F.S.

The most important bacterial disease of cotton is known by different names in various cotton growing regions, i.e., angular leaf spot, black-arm, gummosis, etc. The organism produces a rapid necrosis of localized areas on all parts of the host aboveground. The organism occurs in most of the cotton growing regions of the world, in some of which it is very destructive. It is more serious in the southeastern part of the United States, where it annually causes from three to eight per cent reduction in the crop. The average annual reduction in the United States during the last ten years was 250,000 bales. Nearly all species of the genus *Gossypium* to which cotton belongs are susceptible.

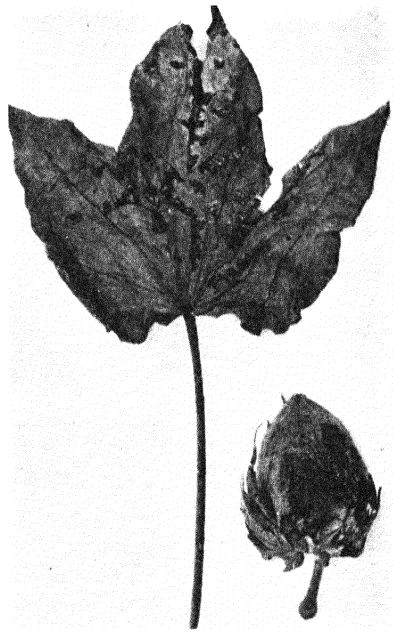


Fig. 92. Lesions of angular leaf spot of cotton on the leaf and boll.

Symptoms.—The disease is recognized by characteristic necrotic lesions on the leaves, bolls and stems. On the leaves the disease appears first as small, water-soaked, translucent, variously angled spots. The small necrotic spots may coalesce, forming larger dead areas. From this injured tissue the bacteria escape and collect on the surface in the form of viscid, creamy drops. The spots often become so numerous as to cause the leaves to fall prematurely. When young succulent leaves are infected, the bacteria may spread for some distance in the large veins, producing V-shaped,

necrotic areas. Lesions may occur on the bracts and veins. The green bolls also are attacked and show brown or black shrunken spots. If very young bolls are attacked they may be shed or may fail to develop. If the bolls are larger when infection occurs, one-sided bolls may develop, or the necrotic areas may extend into the lint and seeds, interfering with the development of the seed. On the stem the lesion appears

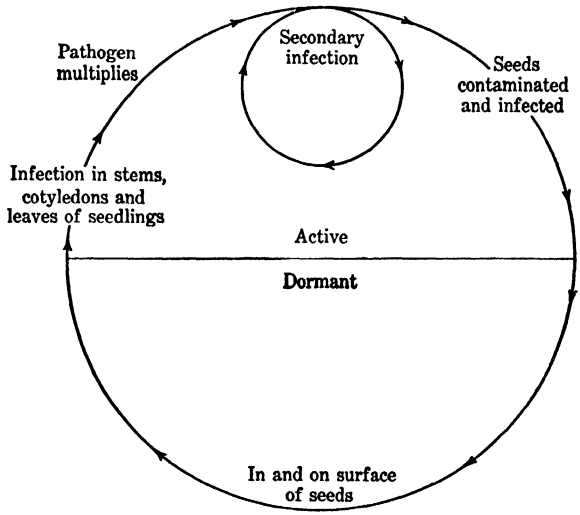


FIG. 93. A diagram of the host relation of *Pseudomonas malvacearum* on cotton. The pathogen survives from one growing season to the next on the surface and within the seed.

as long water-soaked areas in the cortex, which later become long, shrunken black strips from which the bacteria ooze as a slimy, creamy exudate in damp weather. The ooze dries in the form of yellowish-white granules, scales or crusts. This form of infection on the stems, branches and petioles is known as black-arm.

ETIOLOGY.—*Pseudomonas malvacearum*, the organism causing angular leaf spot of cotton, is a short, yellow, one-flagellate rod-shaped bacterium occurring in pairs or very short chains. The yearly host relationship is shown in Fig. 93. While this organism is susceptible to desiccation when placed on a

microscope slide, it can live on cotton lint for at least four months. The pathogen overwinters probably in or on the surface of seeds. When the seedling develops, infection of the cotyledons takes place and the newly formed leaves become infected either by direct contact or by splashing of rain drops containing the pathogen. Wounds are not necessary for infection, but there must be moisture present before the pathogen can make its way into the stomatal cavity. Wind during rain storms is probably the most important means of spread. Insects are relatively unimportant in the dissemination of the pathogen to the leaves but are probably an important contributing factor in boll infection, which is aided by wounds. Under the optimum environmental conditions of a temperature of 30° to 40° C. and a relative humidity of 60 to 75 per cent, the incubation period is three to ten days. Under less favorable conditions, the period of incubation lengthens to seven to ten days. A soil temperature of 25° to 30° C. and soil moisture of 40 per cent seem to be the optimum conditions for infection. The maximum infection occurs when there is a decrease in the soil temperature 24 to 48 hours after sowing.

CONTROL.—Since the main source of primary infection seems to be from seed-borne organisms, necessity for planting disease-free seed is clearly indicated. Delinting by the use of sulphuric acid also will serve as a means of surface sterilization of the seed. There is, however, no treatment that will remove or destroy the bacteria borne internally. Delinting not only kills the organisms on the lint, but also increases the germination of the seed. The treatment is as follows: (1) delint for three to four minutes in concentrated sulphuric acid; (2) wash in water. Dusting with DuBay ceresan is also held to be effective. The best control is by the planting of disease-free seed, and using the more resistant varieties.

WILT OF CUCURBITS

Bacillus tracheiphilus E.F.S.

Cucurbit wilt is manifested as a rapid wilting of the plant through the activities of a bacterium in the vascular system.

Since the first report of the disease in 1893, it has been known that the bacteria causing the wilt are closely associated with insects that feed on the cucurbits. The host range is mainly in the Cucurbitaceae including, cantaloup, squash, pumpkin, melon, cucumbers and others. The watermelon is said to be almost immune.

The organism is present in 31 states of the United States and to some extent in Europe, South Africa, China, India

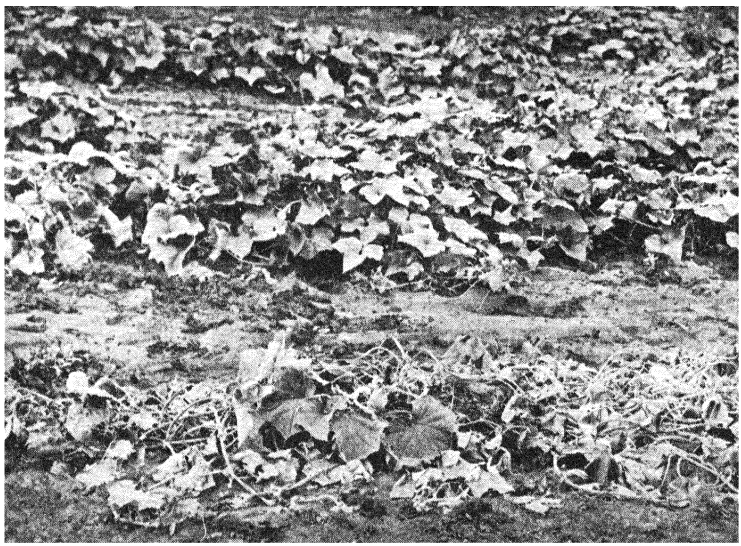


FIG. 94. Wilt of cucumber caused by *Bacillus tracheiphilus*.

and Japan. The pathogen is one of the chief limiting factors in the growing of melons and cucumbers in many sections. The bacteria are not dependent on weather for infection since they are disseminated by insects. The host may be attacked at any stage of its development, and frequently five to 75 per cent of the crop is destroyed.

SYMPTOMS.—The first symptoms recognizable are a dull-green color and loss of turgescence of the leaf immediately around the injury. This condition usually occurs on two or more leaves that may droop at once. The dull color, followed shortly by wilting, progresses from the foliage

down the petioles and into the stem and the infected areas wither and become brown. In the stem the disease spreads rapidly in all directions, until the entire plant becomes wilted and dies within a few days If fruit has been set, it wilts and dries up. In less susceptible plants, as squashes and melons, the host is dwarfed and flowers and branches are produced excessively. The same external symptoms are produced, however, by mechanical injury, root rot or borer injury.

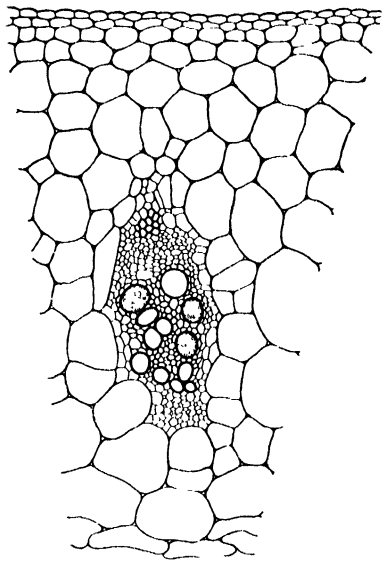


FIG 95 Cross section of vascular bundle of a cucumber plant showing plugging of vessels by bacterial wilt pathogen.

An easy way of diagnosing bacterial wilt is to test the sap viscosity. The sap of an infected plant is markedly stringy, and may adhere to the fingers when touched, and on drawing away the hand the sap is drawn out in delicate threads. When the bacteria are abundant and the infection severe, the sap is milky white instead of colorless as in a normal plant. If the cut end of a diseased stem is placed in water, the sap may ooze out in droplets.

ETIOLOGY.—Wilt of cucurbits is caused by a white, peritrichiate rod-shaped organism, *Bacillus tracheiphilus*, so named from its special preference for the vessels of the plant. The organism is most frequently found in the spiral vessels. There is apparently a large number of intergrading strains, each of which differs slightly from the others in its virulence on different hosts. Infection apparently takes place only through wounds that pierce the vascular bundles. The pathogen is, therefore, easily spread artificially by the injection of pure cultures of the organism into the leaf blade

of the susceptible plant. The incubation period is from 3 to 15 days depending on environmental conditions. In nature the dissemination of the pathogen is limited to and seems to be dependent on insect vectors. The vectors especially concerned are the twelve-spotted and striped cucumber beetles (*Diabrotica duodecimpunctata* Oliv. and *D. vittata* Fabr.). The beetles

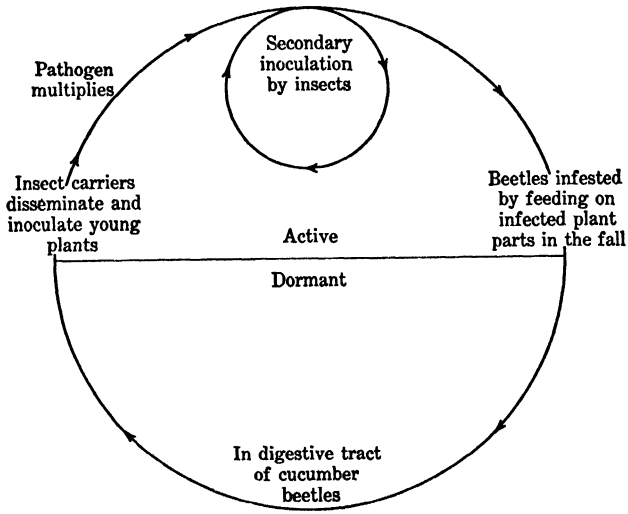


FIG. 96. A diagram of the host relation of *Bacillus tracheiphilus*. This organism survives the winter in the digestive tract of the 12-spotted or striped cucumber beetles.

feed on wilted leaves, thereby contaminating their mouth parts. In feeding on another susceptible plant, the beetle places the bacteria in a wound, which may extend to the xylem and expose the plant to attack. Feces deposited by the insects may also serve to spread the pathogen. Infection is said not to take place through the stomata. The beetles feed on infected tissue in the fall after the living plants have been killed, and the organism overwinters in their alimentary tracts. In the spring these beetles serve as vectors of the wilt organism. This hibernation in the digestive tract of an insect is a very unusual and striking adaptation. The bacteria overwinter in the insect only and are not reported as living over on the seed or in the soil. The yearly host relationship may be represented as in Fig. 96.

Infection by the wilt organism apparently is not directly influenced by temperature or rainfall. The favorable condition of the host seems more important than optimum conditions for the pathogen, as succulent tissues are apparently more easily invaded. The minimum temperature requirement of the organism is 8° C., the optimum 25° to 30° C., the maximum 35° C., and the thermal death point is 43° C.

CONTROL.—The control of the cucumber wilt organism can only be effected by controlling the insect vectors, the cucumber beetles. As yet, entomologists do not have a simple practical method. Up to the present time, exclusion by the use of cages or dusting with calcium arsenate diluted with some filler such as air-slaked lime or talc is held to be the most practical method of control. Nicotine sulphate (Black Leaf 40) is also of value as an insecticide if diluted so as not to harm the foliage.

The use of a catch crop, such as squash, that comes on a little earlier than the main crop is said to be a good practice. One or two later plantings of squash in the same field to keep available a supply of succulent tissues is said to help. In addition it is advisable to pull out all infected plants and either burn or bury them. Commercially desirable resistant varieties are not known, although such varieties are greatly needed.

STEWART'S DISEASE OF SWEET CORN

Bacterium stewartii (E.F.S.) Stev.

This is a vascular disease, principally of sweet corn, although it also occurs on field corn. The causal agent has not been found outside of North America, except in an isolated area in Italy. This disease was first found in New York state in 1897 on early varieties of sweet corn, and previous to 1932 it was considered of minor importance except in the eastern and southern parts of the United States. In 1932 and 1933, however, serious epiphytotics developed in the middle-western corn growing section. During these two seasons many fields of sweet corn showed losses ranging from 2 to

80 per cent. Recently several high-quality varieties have been developed that are remarkably resistant to the pathogen.

SYMPTOMS.—The pathogen produces indirect necrosis in which the most typical external symptom is a wilting of the plant. In the seedling stage general infection results in wilting of the water-soaked leaves and dwarfing of the plant. The young plants may be killed, while in stunted plants the leaves become yellow, then brownish and shriveled. Often only the lower three to four nodes of the plant are discolored. If infection is local the diseased condition is disclosed by water-soaked streaks on the foliage, which may extend a few millimeters or the entire length of the leaf. These water-soaked streaks later turn yellow, finally become brown and dry. The areas may shrink, crack and fall out. The infection showing in this way may become progressively more severe and at flowering result in wilting of the leaves, browning and death of the plant. If the tassel appears, it is dwarfed and bleached, and the pollen is aborted. The bacterial slime may exude into the spiral whorl, seal the leaves over the tassel and cause an arching in the stem in its later elongation. The elapse of time between the first appearance of the disease and the death of the plant varies from four days to a month.



FIG. 97. Wilting of sweet corn seedlings following infection with *Bacterium stewartii*. Healthy seedling on the right.

The most distinctive characteristic of the disease is revealed when the stem is cut lengthwise. The fibrovascular

bundles appear as yellow streaks in the white parenchyma; but in the stems of plants that have been dead for a time some of the bundles may be black instead of yellow. If the stem is cut crosswise and the cut surface exposed to the air for a few minutes, a yellow slime charged with bacteria exudes in droplets from the ends of the vessels. This yellow substance is a sign by which the disease may be easily identified. In the very young plants, however, the yellow slime is detected less easily than in older plants with well-developed vascular systems. Microscopic examination will show the presence of the organism in the vessels and before there is any outward manifestation of the disease except in the dwarfed condition of the plants. In some cases the organisms may come to the surface of the husks through the stomata and coat the kernels, while in others they may occur inside the kernels.

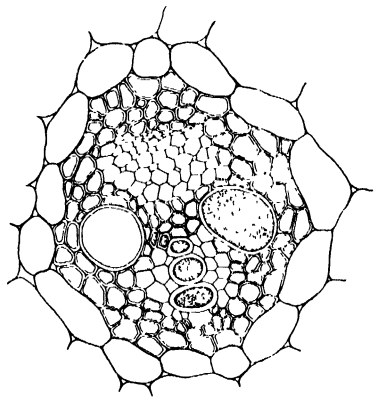


FIG 98. Cross section of vascular bundle of sweet corn seedling showing plugging of vessels and tracheids by the bacteria causing wilt

Fields of sweet corn affected with the disease are very uneven, particularly when the ears are forming. Plants of different sizes and in various stages of disease may be intermingled with apparently healthy plants. It is common to find diseased plants in the same hill with healthy ones that may continue in a healthy condition to the end of the season. There are no indications that the pathogen is communicated by contact from one plant to another. The pathogen does not spread from an infection center but is scattered throughout the field. Usually, the small plants are the first to succumb, which suggests that the organism may be the cause of their slow growth.

The bacterium invades the vessels in all parts of the plant,

including the roots. Plants, which do not succumb to the disease until after the ears form, show the bacteria in abundance in the vessels of all parts of the stem, tassel and ear, where it may ooze out among the kernels and the inner husks, though the ears show no tendency to decay.

Etiology.—This disease is caused by a short, yellow, non-motile, rod-like bacterial organism, *Bacterium stewartii*, which flourishes in the vessels of the host. The host relation is shown diagrammatically in Fig. 100. In cross sections of the stalk, drops of a yellow, viscid substance, composed chiefly of bacteria, exude from the fibrovascular bundles. The bacteria, although confined to the vessels, cause various disturbances in the surrounding cells.



Fig. 99. Stunting of sweet corn caused by a moderately pathogenic strain of *Bacterium stewartii*.

The organism is said to be disseminated chiefly by the seed, or by manure, implements, the southern corn rootworm and flea beetles. It has been shown that *Bacterium stewartii* may live free in the soil and subsequently cause infection if the roots are injured by white grubs, *Phyllophaga* spp. Primary infection may be initiated through the planting of infected seed which may furnish the inoculum for insects. The southern corn rootworm, the larval stage of *Diabrotica longicornis* Say., is capable of transmitting the pathogen from diseased to healthy plants, and it has been shown that the bacteria overwinter chiefly in the digestive system of hibernating adults of the flea beetle, *Chaetocnema pulicaria* Melsheimer. The flea beetles, in feeding on the tips of young leaves, initiate infection by placing the

bacteria in wounded tissue. Thus the typical streaking of the tips of young leaves in the spring is explained. Further evidence indicates that the flea beetle, *Chaetocnema denticulata* Ill., and the twelve-spotted cucumber beetle, *Diabrotica duodecimpunctata*, are able to transmit the bacteria from diseased healthy plants. The organism requires a high temperature

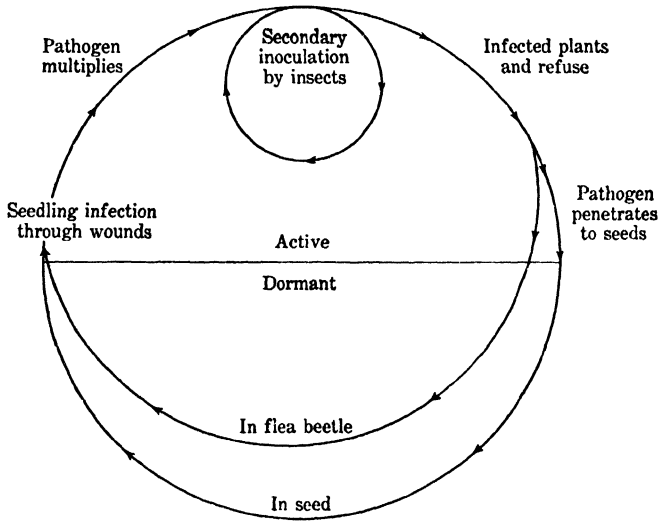


FIG. 100. A diagram of the host relation of *Bacterium stewartii*. This organism survives the winter in the seed corn and in the intestinal tract of the flea beetle.

for its most rapid growth, resulting in the disease becoming more prevalent in seasons of hot weather. Temperature is the most important factor, although any variation in the environment that tends to promote growth of the host also increases the growth of *Bacterium stewartii*.

CONTROL.—The ravages of this disease may be reduced by the use of resistant varieties, seed disinfection, rotation and destruction of infected refuse. The organism on the seed is killed by soaking in 1–1000 mercuric chloride for 15 minutes. The $HgCl_2$ treatment does not control the internal infection. Seed grown in the north is less liable to carry infection than seed grown farther south. It is also held that infected seed can be made safe for planting by dry-air pasteurization at

60° to 70° C. for one hour. Early varieties of sweet corn are susceptible, while late ones are usually more resistant. This probably explains the nominal amount of wilt in states where late varieties as Country Gentleman, Stoles Evergreen, Zigzag, etc., are grown. Varieties of dent and pop corn are resistant, while flint and sweet corn varieties are more susceptible. New hybrids showing a high degree of resistance have been developed in Iowa, Illinois, Indiana and Minnesota.

Black Rot of Cruciferous Crops

Pseudomonas campestris (Pam.) E.F.S.

This disease was first reported on cabbage in 1891 in Kentucky, but its infectious nature was not definitely demonstrated until 1893. This is one of the most serious diseases occurring on cruciferous crops. The losses are frequently 40 to 50 per cent and at times the loss may be complete. Reports from Long Island, western New York, Ohio, Illinois, Michigan, Wisconsin and Texas are often to the effect that entire fields have been destroyed. The pathogen occurs in Europe where it is fully as destructive as in the United States. Its ravages do not stop with the damage done in the field, as it may continue in storage and has been reported as developing in sauerkraut.

Among the more prominent hosts are cabbage, cauliflower, kale, kohlrabi, rape, turnip, rutabaga, radish, black mustard, wild mustard and Brussels sprouts. It is important to note that not all the hosts are domesticated forms for wild mustard, a common and widespread weed, is a host plant. Not all the host plants are equally susceptible; cauliflower is very susceptible, while rutabaga, radish and turnip are more resistant.

Symptoms.—The plant may become infected at any time from the seedling to the mature plant. When the pathogen occurs on the seed, black rot symptoms may develop on the seedlings in the seedbed. The first symptom is a yellowing or wilting of a part of the leaf followed by a blackening of the

veins. In the end, the diseased area becomes brown and parchment-like, with black interlacing veins standing out clearly. The diseased leaves eventually fall from the stems so that only a small tuft of leaves may remain at the top. The stem bears the conspicuous leaf scars showing the row of blackened bundles. The black discoloration of the vascular bundles is one of the chief diagnostic characteristics of this disease. On slender petioles, the blackened bundles show through as dusky stripes. Usually the bark and pith are free from bacteria and are normal in appearance. Sometimes the axillary buds are forced into activity, resulting in secondary shoots appearing on the stem. Plants that are attacked early may be killed in a few days. Dwarfed plants and misshapen heads are common. The roots and basal parts of the stem are woody and, therefore, they are not usually attacked except when the plants are young.

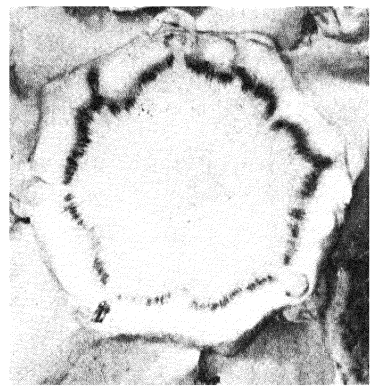


FIG. 101. Discoloration of the vascular system of a cabbage stem by *Pseudomonas campestris*.

ETIOLOGY.—The cause of the disease is *Pseudomonas campestris*. It is a yellow, one-flagellate organism, which is sometimes motile when taken from the plant, but more often not. It is rather small and variable in shape, often resembling a coccus. The organism does not withstand desiccation long, and experiments have shown that the pathogen will not remain viable in pure culture for over eight weeks unless transferred.

Most of the infections take place through water pores, which occur in groups on the margins of the leaf. Drops of water that may persist for hours are formed by the extrusion of excessive moisture through these pores. Through these droplets the bacteria may gain access to the host. As there

is a waxy bloom on many cruciferous leaves, the ordinary stomata are rather well protected and infection through them is seldom observed, except in very young seedlings. The pathogen does not appear to be one that is spread through the air. The cabbage butterfly and aphids transmit the pathogen as do slugs and other mollusks. The host relation is shown in Fig. 102.

The period of incubation is from 11 to 21 days. At times there may be a slight darkening after the sixth day. From the

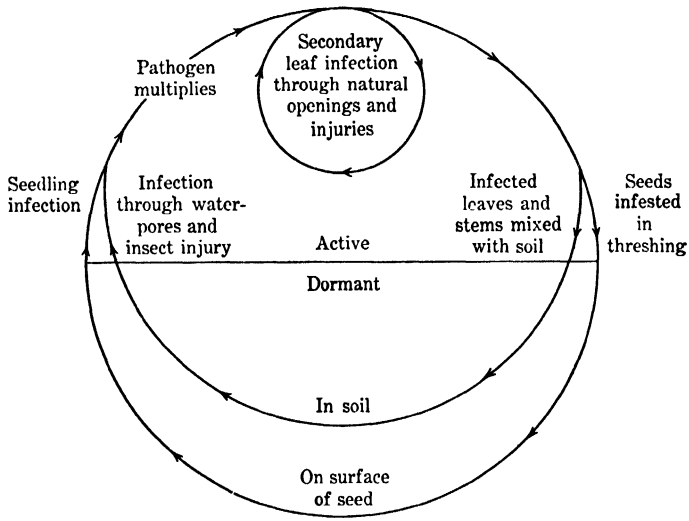


FIG. 102. A diagram of the host relation of *Pseudomonas campestris*. This organism survives the winter on the surface of the seed or may lie dormant in the soil four to five years.

leaf blade, the pathogen passes into the leaf traces of the petiole and thence into the stem. Subsequently, the organisms pass up and down the stem and out into other leaves, always by way of the vascular system.

CONTROL.—Since leaf-eating insects carry the organism from one plant to another, it is well to use some insecticide such as the arsenates. Manure from animals fed on diseased plants is known to be a source of inoculum, but the greatest source of inoculum is from the bacteria on the seed. The seed may become contaminated by the threshing of diseased plants.

Threshing releases the organisms, which lodge on the slightly oily seed. This seed, through seed trade practices, may be distributed widely. The seed should be treated with mercuric chloride, 1–1000, for 15 minutes. All cruciferous weeds that might harbor the pathogen should be destroyed. Careful attention should be given to the growing of transplants. The seedbed should be made from soil that has not grown a crop of cabbage within the past five years.

Crown Gall of Apples

Pseudomonas tumefaciens (E.F.S. & Town.) Duggar

Apple growers and nurserymen have known this disease for at least half a century. The organism occurs in every state in the union and is doubtless world-wide in distribution. Crown gall is found most commonly on fruit crops. The pathogen is common and destructive to nursery stock and is often carried to the orchard, vineyard, garden and hothouse on nursery stock. On some plants, however, the destructive effect is marked, as raspberries, grapes, peaches and roses. Infected trees are restricted in trade by federal and state laws.

Crown gall was first thought to be caused by certain physical agents, as frost, mechanical injuries, etc. An Italian scientist was probably the first to show that crown gall on grapes was caused by a bacterial organism, but most of our knowledge of this disease may be attributed to Dr. Erwin F. Smith, of the United States Department of Agriculture. Not all overgrowths on apple trees, however, are caused by the crown gall organism. The most important of these other types are graft knot and infectious hairy root. Graft knots are more prevalent and destructive than crown gall and infectious hairy root.

Field examinations made in 32 nurseries in 13 states during 1925, 1926 and 1927, involving 55,669 two- to three-year-old piece-root grafted and 30,632 budded nursery apple trees, showed from 0.35 to 1.26 per cent and 0.04 to 1.02 per cent crown gall, respectively. Another survey made in 1925 and reported in 1934 shows a trace to two per cent on budded and

grafted nursery apple trees grown in the north central and northeastern United States. Less crown gall occurred on the budded than the grafted trees.

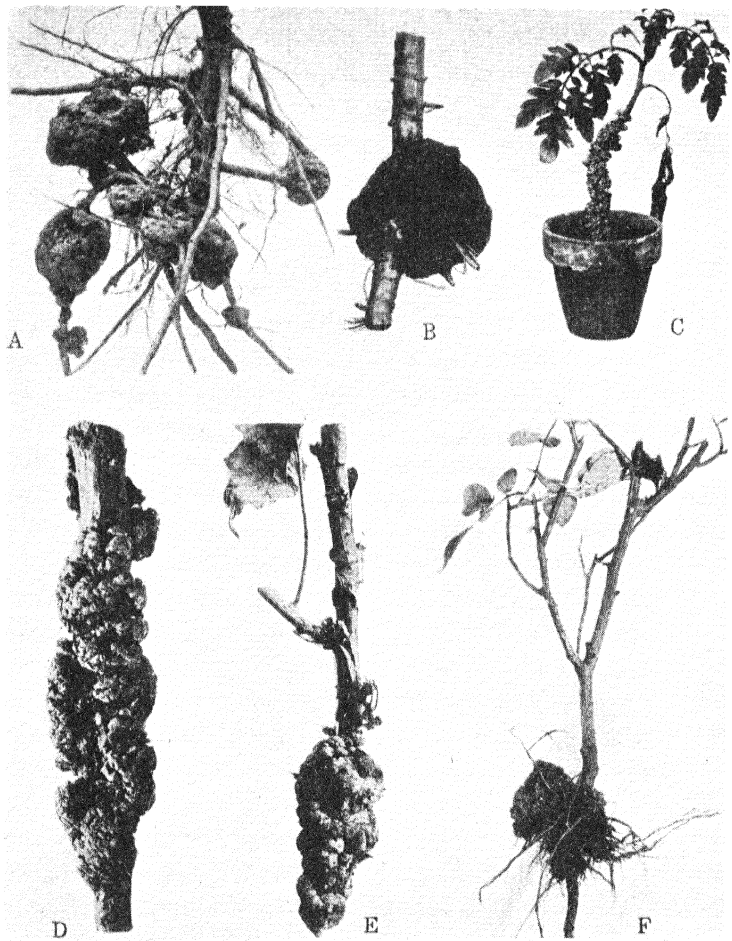


FIG. 103. Crown gall on various host plants: A, apple, B, peach, C, tomato; D, grape, E, raspberry; F, rose.

SYMPTOMS.- The crown gall organism induces overgrowths or tumorous swellings on roots and stems. The swellings or overgrowths are most prevalent on the underground parts of plants. Since the organism is a wound parasite, it is often associated with the union of piece-root grafted apple stocks.

It causes primarily a disease of the cortical parenchyma where it induces the cells to increase in size and number. The result is an imperfectly vascularized, naked, irregular, soft overgrowth or tumor, composed in part, at least, of masses of rapidly dividing, round or spindle-shaped cells. Incident to the tumorous growths, the vascular elements are frequently

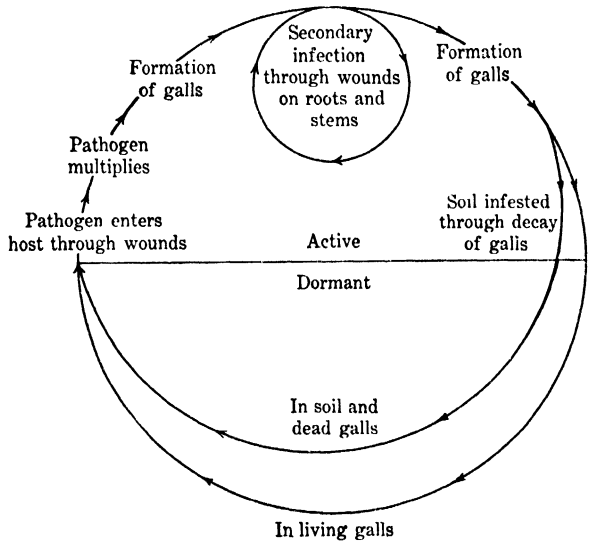


FIG 104 A diagram of the host relation of *Pseudomonas tumefaciens*. This organism survives the winter in the soil and in galls

distorted and displaced, resulting in an interference that prevents normal water flow upward in the plant.

The young galls appear first as small creamy white, soft or spongy swellings that turn light brown when removed from the soil and exposed to the air. As they enlarge on the living plant they turn gradually to a dark brown. A bark layer is not formed on such gall tissue, which permits its rapid decay caused by the easy invasion by saprophytic organisms present in the soil. Roots are rarely formed on galls induced by *Pseudomonas tumefaciens*. The galls produced by this pathogen are largely local at the point of infection, but under special conditions secondary galls may be formed at a considerable distance from the point of primary infection.

The secondary galls are like the primary except for size.

Until recently the tumorous growths caused by the crown gall pathogen were confused with the overgrowths, or masses of excess callous tissue, associated with the union of piece-root grafted apples. These overgrowths, known as graft knots, form when continuity of the stock and scion at the union is imperfect. Likewise, excessive root development, known as hairy root, was at one time considered a symptom of the crown gall organism. It is now known that hairy root may be induced by another bacterial pathogen known as *Pseudomonas rhizogenes* R.B.W.K. & S., which resembles *Ps. tumefaciens* in many respects.

ETIOLOGY.—Crown gall is caused by the white, motile bacterium *Pseudomonas tumefaciens*, whose host relation is outlined in Fig. 104. The pathogen is rarely vascular or intracellular. It occurs in abundance on the surface of young galls and to a less extent in the enlarged distorted tissues. In addition this pathogen survives in a free state in the soil. It enters the host through wounds, chiefly in the roots and other underground parts. Once in the tissue, it may migrate in the intercellular spaces in the zone of sap diffusion about the wound. If the adjoining cells are meristematic and dividing rapidly, the bacteria may be carried along by the lengthening of the intercellular spaces. In such cases secondary galls may form at some distance from the point of initial infection. Under favorable conditions of moisture and temperature, tumorous swellings manifest themselves in ten days to two weeks after the organism is inoculated into the host.

CONTROL.—Crown gall on young apple trees can be rather effectively controlled by (1) choosing land that has never grown plants infected with *Pseudomonas tumefaciens;* (2) surface disinfecting the stocks and scions used in grafting with corrosive sublimate (1–1000); (3) wrapping unions of well-matched wedge or double-tongue grafts with nurseryman's adhesive tape.

Land that has recently grown a crop of roses, raspberries or peaches should be avoided for growing apples. Fields that have recently been in sod or land that is wet may produce

a better crop of trees than clean-cropped, well-drained land. Too much emphasis cannot be placed upon cleanliness in the grafting sheds and upon avoiding the dissemination of the pathogen in the process of grafting. Only well-matched double-tongue or wedge grafts should be planted. The amount of crown gall can also be reduced by growing budded instead of piece-root grafted trees.

Every effort should be made to avoid injuring the stem or roots of young trees in the nursery because such wounds offer an avenue of infection. Little is known regarding the relative resistance of varieties of apples, except that many of the commercial sorts are susceptible.

Potato Scab

Actinomyces scabies (Thax.) Güssow

Common scab of potatoes is caused by a species of Actinomyces. Related species are common in rich soil, constituting an important part of the soil flora, where they play a significant role in liberation of plant food. In addition to being general in soils, *Actinomyces* spp. are known to occur in great abundance on roots of almost all sorts of plants, including oaks, beeches, hazel nuts, elms, elders and various ferns. Potato scab occurs in Europe and America and probably wherever the potato is grown.

The first mention of the disease is made in Loudon's Encyclopedia of Agriculture (1825) as follows:

". . . scab, that is to say the ulceration of the surface of the tubers, has never been explained in a satisfactory manner. Some attribute it to the ammonia from the dung of the horse, others to alkali, and certain others to the use of wood ashes on the soil. Not using diseased seed and planting in other soil are the only known means of preventing the malady."

The causal agent was first described in 1890. It occurs not only on potatoes, but also on beets, turnips, mangels, rutabagas, carrots and radishes. The damage caused by potato scab consists of unsightliness, extra thick paring, changed taste and odor, decreased yield, increased liability to tuber

decay and failure to meet seed certification regulations. In the chief potato growing sections of the United States, many thousands of bushels of scabby potatoes are left in the field annually. In northern Maine the potato-starch industry owes its existence in part to this waste caused by the common scab organism. The scabby potatoes are used for manufacturing starch.

It is probably safe to estimate the loss from this pathogen at 5,000,000 to 24,000,000 bushels a year. In Iowa, a minor potato growing state, scab causes a loss ranging from 5 to 10 per cent annually. In 1922 the loss in Iowa was estimated at 15 trainloads each consisting of 50 cars, which carried 500

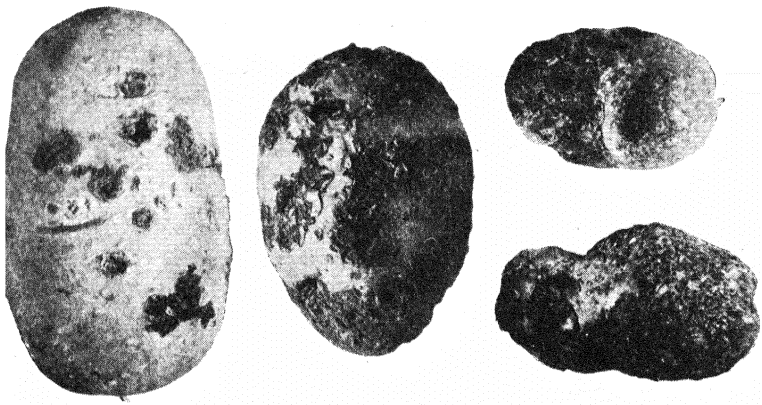


FIG. 105. Potato tubers showing various degrees of infection with *Actinomyces scabies*.

bushels per car. In 1934 when the total production was low for the state because of an unusually dry, hot season, the loss was 10 per cent or 558,000 bushels. The same year the three leading potato-producing states, Maine, New York and Michigan, produced 123,134,000 bushels, and scab caused a loss of 4,797,000 bushels, a quantity greater than the amount produced in each of 30 other states.

SYMPTOMS.—Scab infection at first appears as minute reddish or brownish surface lesions on the young potato tubers, stolons, roots and underground parts of the stem. These spots start most frequently at the lenticels. The lesions increase in diameter and change to a dark-brown color. The

dead host cells accumulate in the lesions, forming a thick layer of dead tissue over the cork, which has been regenerated from the inner cells of the starch parenchyma. This response of the host is caused by the organism on its surface and in its outer cell layers. Chemical substances are formed which cause the cork cells to increase in size and number. If scabby potatoes are carefully removed from the soil and examined immediately, an evanescent grayish film is visible on the lesion. This gray film consists of the scab organism ramifying on the surface of the host. This sign of the disease is only present in the early stages.

ETIOLOGY.—Potato scab is caused by *Actinomyces scabies*, one of the higher bacteria. The aerial growth of this organism consists of long prostrate filaments on which abundant lateral branches arise at short intervals. The mycelium segments to

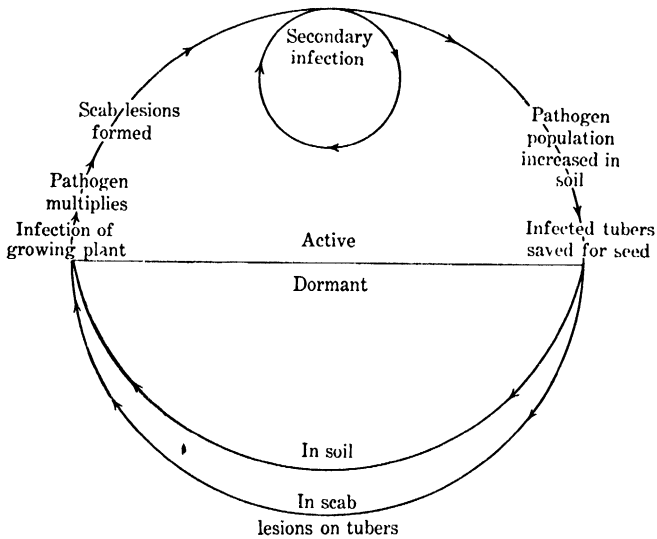


FIG. 106. A diagram of the host relation of *Actinomyces scabies*. This organism survives the winter in the soil and in infected tubers.

form spores that are cylindrical, about one and one-half microns in diameter and nine microns long. These spores germinate, producing one to four germ tubes.

The parasitism of the organism is facultative and may be

favored by alkaline conditions of the soil in the presence of moisture and abundance of organic matter. Its host relation may be outlined as in Fig. 106. Only young tissue is susceptible. Scab lesions cannot form on the more mature tubers. Some acid soils inhibit the development of *Actinomyces scabies*, hence the beneficial effects associated with applications of sulphur and ammonium sulphate. The organism increases in soils continuously cropped to potatoes. It is favored by rather high soil temperatures, 21° to 24° C., and retarded by low soil temperatures. Potato scab is more prevalent in regions having a high summer temperature than in those of lower tempera-

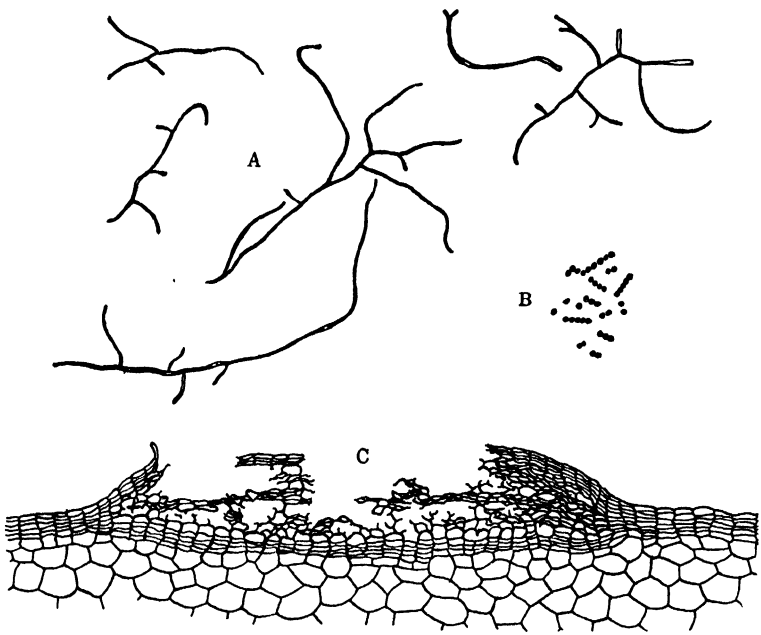


FIG. 107. A, drawings of *Actinomyces scabies* showing branching and irregular segmentation; B, groups of conidia; C, section of an old lesion. (Redrawn from Lutman and Cunningham.)

tures. Dry soils are more favorable for the scab organism than wet soils. The unfavorable effect of moist soils is apparently caused by the lack of aeration, as *Actinomyces scabies* is very sensitive to low oxygen concentrations. There is some evidence that *Actinomyces scabies* is present in soils that have

probably never grown a crop of potatoes. Feeding experiments show that the pathogen is not killed by passing through the alimentary tract of the horse or cow.

CONTROL.—The control of common scab consists in using clean seed, new land, soil treatment, resistant varieties and seed treatment. Clean seed is best obtained by selection. New land is not always possible in many parts of the United States, and as the country grows older its availability will decrease, but a long rotation may lower the amount of soil infestation. As pointed out earlier, the scab-producing organism is favored by certain soil reactions, as a neutral or slightly alkaline soil. Such soils should be made slightly acid by changes in the cropping practice. A green manure in some soils is said to have this capacity. Ammonium sulphate also inhibits the development of the scab organism. Sulphur at the rate of 400 to 800 pounds per acre on light soils reduces the amount of scab. Lime and fresh stable manure often cause the pathogen to flourish and should not be used on potato land.

Not all varieties are equally susceptible. Some degree of resistance exists in the thick, rough-skinned varieties of the russet group. The semi-russet varieties also show some scab resistance, but the white, thin-skinned varieties seem to be very susceptible. Some of the russet varieties are Burbank's Russet, Cambridge Russet, Dibbles Russet and Scab Proof Russet. It is held that the thickness of the skin determines to some extent the resistance. The susceptible varieties are thin-skinned, as Bliss Triumph, Early Ohio, Rural New Yorker, Early Rose, and Irish Cobbler. The susceptible sorts are the only varieties used extensively in the middle west and the eastern part of the United States.

Seed treatment is effective in killing the pathogen on the surface of the tubers. When a soil is relatively free from virulent strains of the scab organism, seed treatment offers effective control. Hot formaldehyde, corrosive sublimate or organic mercurial treatments are the most effective, economical and practical for the average grower.

Chapter Ten

VIRUS-DISEASES OF PLANTS

PERHAPS the most important development in plant pathology since the establishment of parasitism and the demonstration of bacteria as the cause of disease in plants was the recognition of the group of maladies known by such names as "virus-diseases," "chloroses," "degenerative diseases," "viruses" and "mosaics." The most generally accepted name at present is "virus-diseases." The original meaning of the word "virus" was liquid poison. For a long time virus has been used to denote the causal agents of such animal diseases as hog cholera, foot and mouth disease of cattle, dog distemper, etc. There are also several well-known human diseases caused by viruses; i.e., yellow fever, smallpox, infantile paralysis, mumps, trench fever, etc. Plants may be parasitized by agents belonging to the same classes as those attacking man and animals.

In plant pathology the term "virus" in most cases designates an infectious, filterable, obligate parasitic entity, having a definite incubation period, capable of multiplication and causing characteristic symptoms in the host. It should be realized that there are viruses that are not of a parasitic nature that do not pass through certain filters or that do not induce disease symptoms. If a description of a virus without any exceptions were to be formulated then we would be limited to size only. In the light of such a premise a virus is an entity too small to be seen with the microscope using white light. Thus it is felt that the first description affords a clearer working concept. This working concept will need changing as our knowledge of viruses increases, e.g., we may soon be able to add the essential nature of the agent.

Discovery of Virus-Diseases

It is probable that virus-diseases existed a long time before their infectiousness was established. The highly prized variegated flowers of tulips ("tulip break," which has recently been shown to be a virus-disease) were pictured about the middle of the eighteenth century and described in 1758 in Gardener's Dictionary. Peach yellows was known in America as early as 1791, and six serious outbreaks occurred between 1791 and 1888. Virus-diseases of the potato seem to have been known in Europe previous to 1757; at least, degenerative diseases of the potato were described in a book "Practical Husbandman" published in 1757. A disease known as potato curl was recognized as a serious disease in 1764. The mosaic disease of tobacco was described in 1857, but at that time its true nature was not known.

Although virus-diseases were recognized, our understanding of them had to await the establishment of the theory of parasitism in 1853. The causal relation of the higher fungi and bacteria to disease in plants and animals dominated scientific thought during the last half of the nineteenth century with only meager efforts directed to this mysterious and unknown group of diseases. Our first definite knowledge of virus-diseases began with Adolf Mayer's work in 1886. He described a tobacco disease that he called "Mosaikkrankheit," (mosaic disease). In his studies he was able to take the juice from diseased plants and induce similar symptoms in healthy plants. He showed, too, that the incubation period was 10 to 11 days, that heated juice lost its capacity to initiate the disease and that all parts of the plant were infected. It was his opinion that the causal agent was some sort of a bacterium.

The next important discovery in this field was by Erwin F. Smith, in 1888, when he showed that the peach orchards of Michigan, New York and the Atlantic seaboard were suffering from a disease named peach yellows. Smith was not able to transmit this virus by inoculating with the juice, as Mayer had done, but he was able to transmit it by budding infected buds

into healthy trees. He observed that the virus was, under certain conditions, highly infectious and that it had a rather long incubation period. Smith says "the failure to isolate any pathogen, yeast or bacterium suggests the possibility that the cause is some amoeboid organism living in the protoplasm and so much resembling it as to be difficult to detect." Iwanowski reported new studies of the tobacco mosaic disease in 1892. In the main these studies confirmed those already published by Mayer, except that he found that the causative agent was able to pass through filters, which are known to hold back most bacteria. This capacity of the virus to pass through filters has become an important means of diagnosing a virus-disease. Credit for first demonstrating the filterability in virus-diseases rests with Iwanowski. Like Mayer, Iwanowski believed the causative agent was a visible microorganism. Credit for definitely removing the viruses from the realm of visible known organisms must be given to Beijerinck, who in 1899 advanced the theory that the causative agent was a "contagium vivum fluidum," a contagious living fluid.

Most of the host plants of virus-diseases are angiosperms. No case has yet been reported on the gymnosperms, ferns, mosses, liverworts or algae. Bacteria are known to be attacked by an entity known as a bacteriophage. Among the angiosperms many families of plants appear to be susceptible to one or more viruses. More than 15 different virus-diseases have been described on tobacco, 18 on the potato and three on the cucumber. Each virus may consist of strains having characteristic symptoms differentiating them from other strains. Fifty-four strains have been described for common tobacco mosaic virus, and the different potato viruses also consist of many different strains. In 1935 it was reported that virus-diseases were known on 1,100 species in 450 genera distributed in 90 families of plants.

Economic Losses

Virus-diseases seem to be increasing, although many of them probably are of long standing. Herbaceous plants are attacked more commonly than are woody plants, most forest

trees being free as far as known at present. Plants are seldom killed outright by viruses, though in most cases they are dwarfed or stunted. A few virus-diseases cause little or no injury, as latent potato mosaic, while others are very destructive, as curly top of sugar beets, spinach blight,



FIG. 108. The effect of potato mosaic virus on size and yield of infected plants. Healthy plant at left, virus-infected plant on right. Note the dwarfed tubers and vine.

mosaics of potatoes, mosaic of beans, mosaic of cucumbers, peach yellows, spike disease of the sandalwood tree, bunchy top of the banana, yellow dwarf of onions and aster yellows.

Losses from virus-diseases may vary from a trace to nearly the whole crop. It has been estimated that peach yellows caused an annual loss of 450,000 trees in New Jersey, Pennsylvania, Delaware and Maryland. The losses caused by virus-diseases in the potato are variable, ranging from very slight to complete crop failures, while the annual loss caused by tobacco mosaic exceeds one million dollars. The sugar beet crop in the Yakima valley in Washington in 1924 was reduced to one-fourth of the normal crop by the curly top virus and in the state of California between 1899 and 1915

the same disease caused a loss of $10,000,000. Crops of tomatoes, beans and species of cucurbits have frequently been seriously damaged in the northwest by the curly top virus.



FIG. 109. These two bunches of onions, one healthy and the other infected with the yellow dwarf virus, are of equal number and were taken from the same row when about half mature. The ultimate yields were fully as markedly different at harvest.

In crops that are propagated vegetatively, the injury or loss grows progressively. The spindle tuber virus of potatoes (Bliss Triumph) decreased the yield over a four-year period as follows: 100, 70, 26 and 12½ per cent, respectively. In 1928 yellow dwarf of onions developed in epiphytotic form in a small onion growing district in Iowa, consisting of about 1,000 acres made up of small tracts varying from less than 1 to 12 acres. The decrease in yield in different fields varied from a trace to more than 95 per cent. The average loss for the district was estimated at 40 per cent of the crop.

SYMPTOMS

The most obvious symptoms of virus-diseases are usually seen on the foliage, the symptoms being most marked on young leaves. The macroscopic symptoms are much more perfectly known than the microscopic. In fact the internal symptoms of virus-diseases have received relatively little attention and until such studies are made, no scheme of classification

can be fully justified. For convenience, however, the symptoms of virus-diseases are divided into three classes, which may appear separately or in various combinations. The expressed symptoms are not constant, being greatly in-

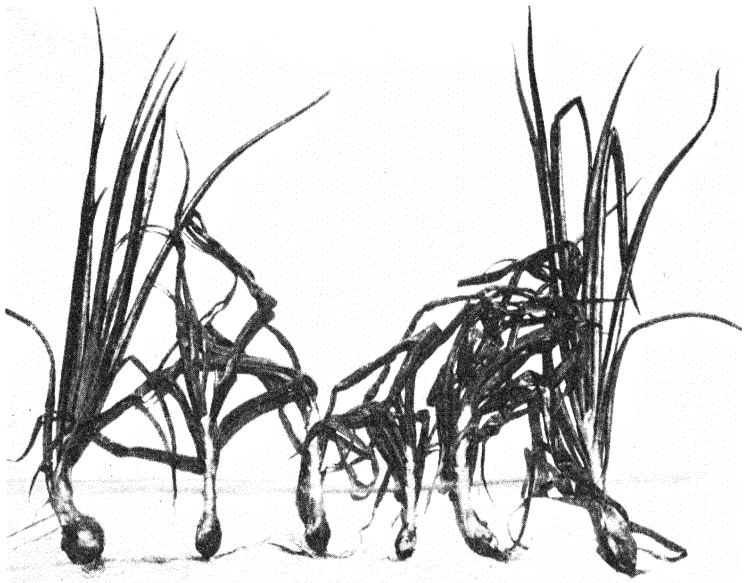


FIG. 110. The yellow dwarf disease of onions. The two end plants are healthy and the four center ones are diseased. The conspicuous symptoms are yellowing and dwarfing of the plants.

fluenced by environmental conditions and by hosts of different varieties and species, for instance, the tobacco mosaic virus causes mosaic pattern on tobacco and necrotic lesions on beans.

HYPOPLASIA.—Most plants attacked by viruses are underdeveloped either as a whole or in some parts and these abnormalities may manifest themselves in a variety of ways as yellowing, mosaic, chlorosis, dwarfing and distortion in one or more of the plant organs.

Many virus-diseases are characterized by *yellowing*, a limited chlorophyl development or its partial destruction in the plant. This symptom may be noticeable not only in the leaves, but

also on the stems and fruits. In peach yellows, aster yellows and yellow dwarf of onions, yellowing is the most conspicuous symptom.

Sometimes only certain portions of a leaf are yellow, while the rest is normal in color. The yellow and green areas may vary in size and pattern. This uneven distribution of the chlorophyl gives rise to the characteristic mottling known as *mosaic* (Fig. 111) and is one of the most general symptoms of plant viruses. The mosaic viruses induce not only macroscopic symptoms, but also internal histological changes in the leaf. The light-green areas are thinner than the dark-green ones in about the ratio of two to three as a result of the smaller size of the cells. This difference in thickness of the leaf apparently is not uniform in all mosaic diseases. In the sugar cane and corn mosaic the light-green areas are the thicker. In severe cases of mosaic the chloroplastids may be reduced or disintegrate into hyaline particles. Starch has a tendency to accumulate in mosaic-infected leaves and in certain mosaic diseases (corn mosaic) X-bodies occur in the cytoplasm usually near the host nucleus. These X-bodies are variable in size and shape, and their content is denser than the host cytoplasm in which they are embedded. In some tissues they are sufficiently conspicuous to be seen with the low power of the microscope without staining the tissues. Inclusion- or X-bodies do not occur in all plants affected with virus-diseases.

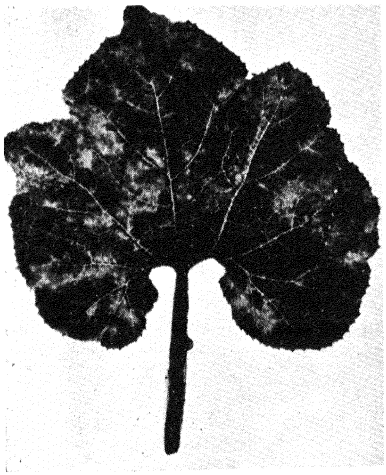


FIG. 111. Yellow and darker green leaf areas typical of cucumber mosaic.

Parts of leaves or fruits may have practically no chlorophyl, a condition called *chlorosis* (Fig. 112), which may be manifested as a white or cream-colored mottling. White

pickle of cucumber and chlorosis of abutilon show this type of symptom.

Virus-infected plants are frequently stunted or spindly, with stems sometimes longer and sometimes shorter than normal. The spindle tuber virus of potatoes causes the

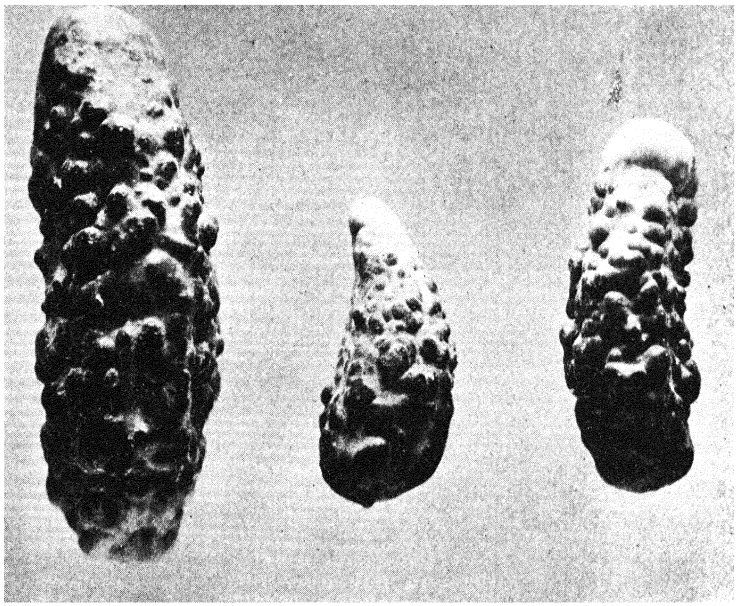


FIG. 112. White pickle, symptoms of cucumber mosaic on the fruits. Frequently the tissues carry little chlorophyl.

dwarfing of the underground and aerial stems and markedly decreased yields of tubers. The shortening of the internodes causes a rosette-arrangement of the leaves. This condition prevails in curly dwarf of potatoes, bunchy top of bananas and wheat rosette. The leaves, flowers and fruits may be dwarfed as extensively as the stems.

The uneven development in different parts of the plant, as the leaf, causes puckering, curling, rolling and twisting of all or part of the leaf. Some form of *distortion* (Fig. 114) usually is associated with every type of hypoplasia induced by viruses. One of the potato mosaics is described as a

rugose mosaic because of the distortion of the leaf blade. In the case of curly top of beets and the leaf roll of potatoes the common name of the disease refers to the particular form of distortion. In general, these different forms of hypoplasia occur in combination, which increases and modifies the effect on the host.

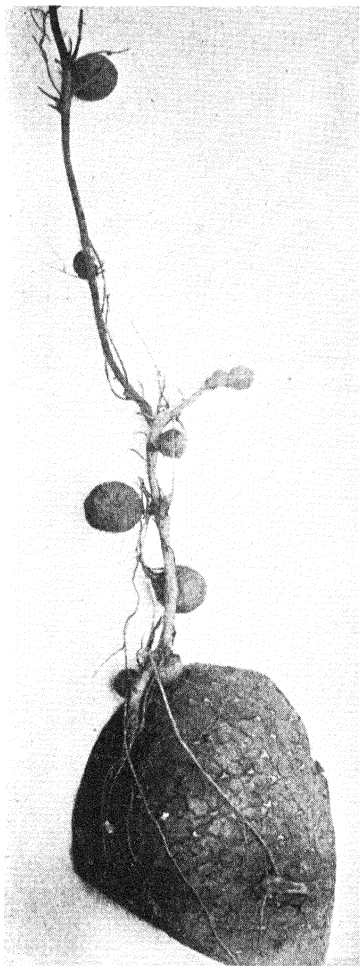


FIG. 113. This virus disease is known as spindle tuber of the potato. Infected seed pieces develop long, slender, dwarfed plants that seldom yield many marketable tubers.

HYPERPLASIA.—In other virus-diseases of plants, overgrowth results. The whole plant or some of its parts grow larger than normal. A virus-disease of sugar cane induces the formation of *galls* on the stems and leaves. The swellings are due to proliferation of the parenchymatous tissues in the fibrovascular bundles. In some plants a virus stimulates the latent buds to undue activity, leading to the development of many new branches, most of which are spindly, yellowed and distorted. The aster yellows virus causes many of the axillary buds of the aster plant to become active. The most conspicuous symptom of the spike disease of the sandalwood tree is the formation of many *witches' brooms*.

NECROSIS.—Some viruses rapidly kill groups of cells in isolated areas on stems, leaves, fruits or tubers of the

host. Necrosis of definite areas may result in the absence of either mottling or distortion. Sometimes the necrotic areas occur as distinct isolated spots in the leaf blade; in other cases the dead areas are linear, following the veins of the blade ex-



FIG. 114. Two potato plants, one shows rolling of the leaflets and dwarfing characteristic of leaf roll, while the other plant is healthy.

tending down the petiole to the stem. The former condition prevails in ring spot and the latter in the streak disease of tomato and tobacco. In the leaf roll disease of potatoes the necrotic areas manifest themselves in the phloem of the vascular bundles of the tubers and stems. In spinach blight, necrosis tends to become general in all the leaves, causing rapid death of the whole plant.

NATURE OF CAUSAL AGENT

As stated at the beginning of this chapter, the cause of virus-diseases of plants is relatively unknown. We do not know whether it is animate or inanimate, i.e., living or dead. Some students of this group hold that the causative agent is living, because it has many of the properties of living things, while others believe it is not living in that the causative agent responds like inanimate things. In each case the reasoning is largely by analogy rather than by demon-

strated facts. However, this need give the student of plant pathology no great concern. It is the properties, methods of spread and infection, and the influence of environmental conditions rather than the nature of the causal agent that need to be known to combat these agents most effectively.

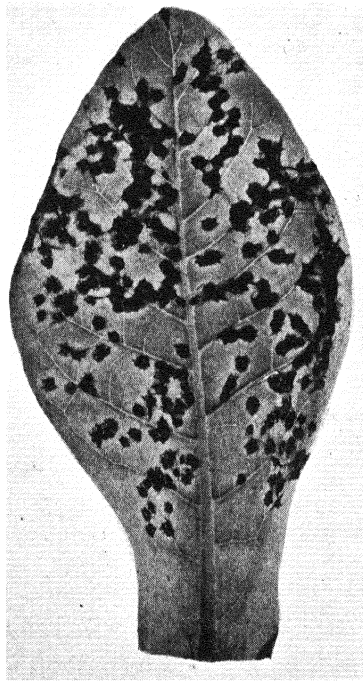


FIG. 115. Local necrosis at the point of inoculation with the tobacco mosaic virus. (Courtesy of L. O. Kunkel.)

ANIMATE.—The theory that virus-diseases of plants and animals are caused by living organisms is as old as our definite knowledge of this group of maladies. Mayer, who first described tobacco mosaic, reported the finding of bacteria and amoeboid-like bodies in the palisade cells in the yellowed areas of diseased leaves. The same year, Loeffler and Pfeiffer, working with smallpox, demonstrated minute bodies in the pox lesions, which they thought resembled protozoa. Since that time many other students have announced bacteria and protozoa as the cause of some virus-diseases as the result of finding such organisms in diseased tissues. In many virus-diseases there are dense bodies in the cytoplasm of infected plant cells. These dense bodies, called inclusion- or X-bodies, superficially resemble living microscopic organisms and have been thought to be the causal agent. The evidence presented so far has been largely circumstantial and not conclusive.

But the advocates of the animate concept go further than living organisms of the order that we now know. They

believe that a virus is alive but of a different order as to size, structure, complexity and life history, i.e., an organized body simpler and more primitive than bacteria. They find support for such an hypothesis in the fact that the causative agent has many of the characteristics of living organisms as reproduction or multiplication, adaptability, modification by attenuation, obligate parasitism, fixed thermal death point, production in the host of characteristic symptoms after the elapse of a quite constant period of time, etc. These characteristics of viruses will be discussed in greater detail in later paragraphs.

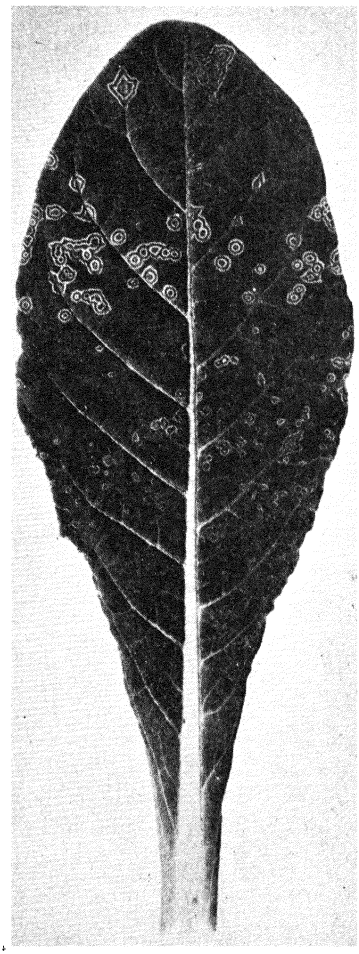


FIG. 116. Ring-like necrotic areas following inoculation with virus. (Courtesy of L. O. Kunkel.)

INANIMATE.—On the other hand, the fact that viruses have never been shown to assimilate food or respire may place them among inanimate things. All known disease-producing microorganisms can be seen with a microscope. So far the highest resolving power of the microscope using a white light has failed to reveal viruses. This fact, coupled with their response to chemicals, suggests they have properties differing from living things. The causative agent has been held to be an enzyme, i.e., oxidase, free hydrogen peroxide, "or some residuary by-

product of plant metabolism which has been allowed to remain unassimilated in the cells of the leaves through the excess development and retardation of enzymes." The idea has been ventured that a virus is like a catalyst which "upsets the cell's metabolism, and leads to a functional derangement that vastly increases the catalyst, which, being set free, infects other cells." It has been suggested, too, that a virus is analogous to growth-promoting substances. Under suitable conditions these may multiply and manifest characteristics common to living organisms. Recently a comparatively stable, highly infectious, crystalline protein, which is held to be the causal agent of the common tobacco mosaic, has been isolated from infected plants. These crystals are autocatalytic proteins, which increase in the host protoplasm. The molecules of at least this protein are of great size, possess distinctive physical properties and are thought to have a molecular weight of 17,000,000. Nothing is known of their chemical structure, how they reproduce or how they incite the host responses.

Properties of Viruses

The virus is lodged in the juice of infected tissues, although the sap is not as infectious as the crushed tissue. The transfer of the virus by juice is known to be most easily accomplished in the mosaic group. Many viruses are systemic in the plant, but they are not equally abundant in all of the organs. The seeds as a general rule are free, except in members of the Leguminosae, notably the garden bean, and in a few plants in certain other families. Roots and other underground parts of plants seem to contain less virus than the above-ground parts. Some viruses are known to be strictly local in some plant organs. Systemic symptoms usually manifest themselves only in tissues that develop subsequent to inoculation. The causative agent is closely associated with growth phenomena and has a deleterious effect on photosynthesis and the use of stored food.

Some viruses are very infectious, the most extreme case of this being the virus of tobacco mosaic while others are much

less so, as the virus of aster yellows. In tobacco mosaic the virus can be conveyed to a healthy plant merely by stroking the leaves enough to break the trichomes (leaf hairs). The juice of an infected tomato plant can be diluted one part in 1,000,000 without losing all of its infectiousness. No other virus will tolerate this extreme dilution. The virus of potato crinkle mosaic can only be diluted 1–10; bean mosaic virus 1–1000; curly top virus 1–1000; and cucumber mosaic virus 1–10,000. The viruses of peach yellows, spike disease of the sandalwood tree, infectious chlorosis of abutilon, peach mosaic, etc., seem to be much less infectious. In these cases, actual contact of diseased and healthy tissue is necessary to bring about the transfer of the virus to healthy tissue. Recently it has been shown that the viruses of peach yellows and the spike disease are spread by insects. The common tobacco mosaic virus and infectious chlorosis of abutilon represent the two extremes of infectiousness, and all the other viruses fall between these two extremes.

In general, when a plant becomes systemically infected it never recovers, and in perennial plants the new growth each year carries the characteristic symptoms. There are, however, a few instances where plants recover; i.e., sugar cane will recover from the grass mosaic virus, the cucumber (var. Chinese Long) from a cucumber mosaic virus, and tobacco from tobacco ring spot virus. Recovery, however, is more common in animals and man than in plants.

Death from virus-diseases in most cases is slow, requiring in some cases many years (some exceptions are ring spot on some species of tobacco and legumes and blight on spinach). Virus-infected plants are often more susceptible to fungous and bacterial organisms, insects and adverse environmental conditions than healthy plants. Potatoes suffering from leaf roll may show more hopper burn (induced by the potato leaf hopper) than healthy ones. In the same way, onions infected with yellow dwarf virus are injured more by hot, dry weather than healthy ones.

In many animal viruses a certain degree of immunity is acquired by the host on recovery, but in plant viruses this

condition is not so common. Also, an animal seldom if ever suffers from two virus-diseases simultaneously, while two or more viruses frequently exist in a plant. The combination of two or more viruses is known as a virus complex, and where such complications prevail, the symptoms are unlike those resulting from either virus alone. Tomato streak may be



FIG. 117 The plants in front of the white labels are milkweeds that may serve as carriers of the cucumber mosaic virus. This land had been planted to cucumbers two years previously.

caused by two viruses acting simultaneously, namely, the tomato and potato mosaic viruses.

The host range of different viruses varies. Some seemingly are restricted, while others have a wide host range. Leaf roll of potatoes is restricted to potato and tomato, while cucumber mosaic attacks species of every genus in the Cucurbitaceae as well as numerous plants in other families. One of these is the milkweed, a common weed in many cultivated fields, pastures and fence rows. There are many weeds that are known to be virus hosts. Such infected plants are known as virus carriers. (Fig. 117.) They may play an important role by carrying a virus over from one growing season to the next and in serving as a source of the virus throughout the current growing season.

Influence of Environment

The symptoms of many viruses can be prevented from expression by varying the environmental conditions. A change in the temperature or amount of light will interfere with their expression. In such cases the virus-disease is said to be masked. Mild mosaic of potatoes expresses itself at 15° C., but it is masked at 25° C. Spindle tuber symptoms, on the other hand, show at 25° C., but are entirely absent at 15° C. Again, a plant may carry a virus without showing any characteristic mosaic or necrotic symptoms. Such a host may be considered a symptomless carrier; e.g., a virus prevails in the potato and seemingly in no way interferes with its growth and productivity. When this virus is transferred to tobacco, however, characteristic mosaic symptoms develop in the tobacco plant.

Viruses may be attenuated by inoculating them into resistant hosts and then taking them back to susceptible plants. This has been demonstrated in the case of the curly top virus of sugar beets. When this virus is inoculated into the nettle-leaved goosefoot (*Chenopodium murale*, L.) and then into the sugar beet, the symptoms are less marked and the injury less severe than the original. If, however, this attenuated virus is passed through species of chickweed (*Stellaria media* (L.) Cyrill.), its virulence is said to be restored. In other cases they can be passed through a resistant host without losing any of their virulence. The optimum conditions for the plant usually favor the development of symptoms. Temperatures above the maximum or below the minimum for the growth of the host tend to mask the symptoms. Any condition that retards photosynthesis and growth tends to inhibit the expression of virus symptoms.

Possible Size of the Virus Entity

The exact size of the causative agent of virus-diseases is unknown. These agents are so small that they cannot be seen with the highest magnification available in our microscopes utilizing white light, i.e., objects less than 0.1μ. The

virus particles are so small that they pass through earthen filters that hold back nearly all living organisms. Thus, it is said that a virus is a "filter passer" or "filterable." Filterability in viruses was first discovered in connection with the plant virus causing tobacco mosaic by Iwanowski in 1892. Six years later two animal pathologists accidentally discovered the filter-passing capacity of the agent causing the foot and mouth disease of cattle. Presumably all plant viruses are filterable, although this has actually been demonstrated only in a limited number of cases, i.e., tobacco mosaic, cucumber mosaic, yellow dwarf of onions, curly top of sugar beets, etc.

A particle that can pass the finer grade earthen filters is probably less than 0.2μ in diameter. Certain data are available that approach rather accurately the size of these particles. The problem of size has been studied by five different methods.

TABLE IV. THE COMPARATIVE SIZE OF TWO BACTERIA, SOME PLANT AND ANIMAL VIRUSES AND FOUR PROTEIN MOLECULES

Entities	*Name*	*Size in Millimicrons*
Bacterium	*Escherschia coli*	1500
"	*Serratia marcescens*	750
Virus	Psittacosis of Parrot	275
"	Potato y	250
"	Potato x	200
"	Hyoscyamus mosaic	150
"	Rabies	125
"	Tomato spotted wilt	110 170
"	Rous sarcoma of fowls	100
"	Large bacteriophage	60
"	Aucuba mosaic	40-50
"	Medium bacteriophage	25
Protein	Haemocyanin	24
Virus	Yellow fever	22
"	Tobacco mosaic 1 & 6	15
"	Cucumber mosaic	15
"	Tobacco ring spot	15
"	Bacteriophage (small) (*Bacterium prunt*)	11
"	Tobacco mosaic (purified)	11
"	Poliomyelitis (Infantile paralysis)	10
"	Foot and mouth disease	10
Protein	Serum globulin	6.3
"	Serum albumin	5 4
"	Egg albumin	4.34

First, by using the ordinary microscope, second, through differential ultra-filtration; third, through photomicrography with a single ultraviolet wave length; fourth, by ultra-centrifugation; and fifth, by X-ray. If these measurements are indicative of the size of the entities causing virus-diseases, then they vary rather widely in diameter from 10 to 275 mμ. Small visible organisms such as the bacterium *Serratia marcescens* Bizio are two and one-half times larger than the largest virus entity. Normal protein molecules, on the other hand, are mainly smaller than most virus entities. In table IV some common protein molecules are listed that range from about 4 to 24 mμ. It is probable that some virus entities may be as large as a mass of several protein molecules.

Influence of Physical Agents

Viruses in expressed plant juice are inactivated by temperatures ranging from 42° to 90° C. Thermal inactivation of the virus of yellow dwarf of onions occurs at 75° to 80° C., after ten minutes; tobacco mosaic virus, 90° C., after ten minutes; cucumber mosaic virus No. 1, 60° to 70° C., after ten minutes; bean mosaic virus, 44° to 56° C.; tomato spotted wilt virus, 42° C., after ten minutes; ring spot virus of tobacco, 70° C., after ten minutes; leaf roll virus, 45° to 50° C., and tomato mosaic virus 60° C., after ten minutes. As indicated above, the range in thermal inactivation point of viruses ranges from 42° C., in tomato spotted wilt to 90° C., in tobacco mosaic. In the living plant, they may not be as tolerant as the host, as in the case of the sereh virus, which it is claimed can be killed in the cane by treatment with hot water at 45° to 50° C., for 30 minutes followed by an exposure at 50° to 53° C. The virus of peach yellows is inactivated in the host at 38° C., after 11 hours, and that of spotted wilt will frequently disappear from plants grown in a warm greenhouse.

Viruses are more tolerant of low temperature than high. The tobacco virus has been subjected to −185° C., without destroying its infectivity. It is held that this virus is more tolerant of ultraviolet light and direct sunlight than either the vegetative or spore forms of bacteria. Radiations from a

mercury vapor arc in quartz having a wave length shorter than 285 mμ completely inactivated tobacco mosaic virus in 15 seconds when the virus was comparatively free from the crushed leaf tissues.

The tobacco mosaic virus will remain alive in dried leaves for many years and may survive from one year to the next in infected refuse in the soil. The bean mosaic virus has been reported to survive in stored beans for 30 years. The tobacco ring spot virus, on the other hand, is readily destroyed by desiccation. Some viruses maintain their infective capacity in the presence of decay. The tobacco virus is reported to maintain its infective capacity for 15 months in fermenting tobacco juice.

Influence of Chemicals

To separate a virus from plant juices has proven a difficult task. Until recently, the best that had been accomplished in this direction was to free the mosaic juice of tobacco of about 90 per cent of the leaf tissues without destroying the infectivity of the virus. The dye known as safranin will precipitate the tobacco virus and cause it to become inactive. When it is released from the safranin with amyl alcohol, however, its infectivity is restored. Two parts of acetone or alcohol in one volume of the juice at 0° C. gives a precipitate high in virus content. Ammonium sulphate, calcium chloride, and magnesium sulphate may also be used to precipitate the virus.

The tobacco mosaic virus is destroyed in 75 to 80 per cent ethyl alcohol, but not in 45 to 50 per cent. Ether, acetone, chloroform, carbon tetrachloride, toluene and glycerine are less toxic to the virus than alcohol. The tobacco virus in the crushed leaf tissue and juice is also remarkably resistant to various alkaloids, salts of alkaloids, glucosides and ethereal oil. Two per cent mustard oil and five per cent digitalin destroyed the virus in five days. Four per cent formaldehyde quickly destroys the infectivity of the virus. The potato mosaic viruses are thought to be less tolerant of chemical agents than the common tobacco mosaic virus. Twenty-five per cent alcohol and a 1–500 dilution of nitric acid destroyed

the infectivity of the viruses of potato crinkle mosaic and rugose mosaic in one hour. In general it may be said that the tobacco virus is more resistant to chemicals than are vegetative stages of known microorganisms.

Movement and Multiplication of Viruses

The curly top virus requires at least one and one-half hours to travel through a beet petiole seven inches long. The causative agent has been found in the foliage and in the roots. Tomato mosaic virus travels in tomato stems at the rate of one to two centimeters per hour. The virus causing the streak disease in corn has been found to travel 40 centimeters in two hours. Low temperatures retard the movement.

In general, viruses do not multiply, except in living plants associated with growing tissues. Many attempts have been made to grow viruses in pure culture on artificial media in a manner similar to bacteria and the higher fungi, but no one has succeeded to date. Tobacco mosaic virus, however, has been found to multiply in detached tissues held under certain favorable conditions. Recent investigations with viruses show that they increase rapidly in concentration at the point of inoculation before increasing to any appreciable extent in other parts of the host. This situation may be followed by dissemination of the virus to all parts of the plant.

Dissemination

Viruses may be disseminated in many ways; i.e., by the seeds of some plants as the pea, red clover, bean, lettuce, wild cucumber and tomato; by cuttings, as in potatoes and sugar cane; by grafts and buds, as in chlorosis of abutilon; possibly by pollen in the bean; by harvesting green cucumbers; and by insects. In the case of highly infectious viruses, such as the tobacco mosaic virus, it may be transmitted by human agencies—tools, clothing, hands of persons working in infected fields and the chewing of mosaic-diseased tobacco and spitting on the young leaves. Wind and rain are not known to disseminate viruses. So far, soil has not been shown to be a carrier of the viruses, except where it contained infected refuse.

Wheat rosette virus is held to be transmitted in some unknown way through the soil.

It should not be understood from the preceding discussion that the method of dissemination is known for all viruses. Their dissemination is known in comparatively few cases even though the disease produced by the virus has been known for many years. It is only recently that the method of dissemination of the viruses of peach yellows and the spike disease of sandalwood was discovered. The former is held to be spread by *Macropsis trimaculata* Fitch and the latter by *Moonia albimaculata* Distant.

Insect Vectors

As more is learned concerning virus-diseases, the important role of insects in their transmission is being more fully realized. Insects that aid in the spread of viruses are known as vectors, and those with sucking mouth parts are more effective vectors than those with chewing or crushing mouth parts. Species of aphids and leaf hoppers comprise the majority of virus vectors. Viruses probably are not spread from one insect to another, although the virus can be taken from one insect and inoculated into another, making the second insect a true vector. This condition prevails in the hopper carrying the virus of the streak disease of corn in South Africa. Insects generally become vectors only through feeding on virus-infected material.

Some insects can transmit several different viruses, while others are known to transmit only one. The peach aphid (*Myzus persicae* Sulz.) may transmit at least 14 different viruses, but the beet leaf hopper (*Eutettix tenellus* Baker) carries only the curly top virus. Just what relationship exists between the virus and the insect is not known. In the light of information now available, however, it would seem that in the majority of cases the insect is only a mechanical vector. It can hardly be different, where one insect carries many viruses; e.g., the peach aphid may transmit the viruses of sugar beet mosaic, spinach mosaic, tobacco mosaic, potato leaf roll and others. In other cases an insect carries only a single virus and

becomes capable of transmitting the virus only after the lapse of two hours to many days. In such cases the relationship may be different from that in the peach aphid. This interdependence of the virus and the insect is said to be biological, implying that the virus undergoes some change in the insect.

In the case of the raspberry viruses the aphid *Amphorophora sensoriata* Mason rarely transmits any of the viruses attacking raspberries. On the other hand, *A. rubi* Kalt. may transmit the viruses of three raspberry mosaic diseases but not a fourth known as curl. Another aphid, *A. rubicola* Oestl., transmits curl and rarely the others. Sometimes an insect may carry two different viruses simultaneously, as the potato aphid that carries the viruses of cucumber and tobacco mosaic. In this specific instance the aphid transmits only the cucumber mosaic when allowed to feed on the tomato. The peach aphid may also carry two viruses at the same time, i.e., leaf roll and mosaic of potatoes. This vector may transmit only the leaf roll virus to healthy potatoes and only mosaic virus to tobacco plants. (Tobacco is not a host to the leaf roll virus.) This and other similar instances suggest that insect vectors may serve to separate a virus complex made up of two or more viruses.

The vector capacity of some insects has been shown to increase for a period after feeding on diseased plants. The beet leaf hopper has been found to be better able to transmit the curly top virus 24 hours after feeding on diseased plants than directly afterward. Some insects have been shown to harbor the virus as long as they live after becoming viruliferous. This is true in the beet leaf hopper, and there is only one known case where the virus is carried in the egg stage from one generation to another; namely, the rice stunt disease, where the leaf hopper *Nephrotettix apicaulis* var. *cincticeps* Uhler is said to retain the virus through the egg stage for three generations. In aphids reproducing parthenogenetically (similar to vegetative propagation in plants) the young may be as potent vectors as the stem mother.

The length of time an insect may remain a vector varies. In cases where the insect merely transmits the virus mechanically, the virus may be lost in a comparatively short time

when the insect is moved from infected to healthy tissue; in other instances, particularly where the insect has a biological relation, it may be retained for a comparatively long time. The hopper *Cicadulina mbila* Naude, which is the vector for the streak disease of corn, has been reported as retaining its vector properties after five months of starvation or feeding on healthy plants. The incubation period varies from ten days to three weeks, depending upon whether the insect is an adult or nymph. Environmental conditions, as temperature, may also influence the incubation period in a vector.

Control

Recovery in virus-diseases probably is rare, and no way has yet been discovered whereby the tissues can be freed of the virus except in the sereh disease of cane and peach yellows. In these two cases treating the stems of cane in water held at 52° C. for 30 minutes to one hour, and young dormant peach trees at 50° C., for ten minutes, has proved effective. Since cure is so rare and still impractical commercially, prevention of infection assumes great importance in practice. The most generally practiced control measures consist of preventing transmission and spread through eradication of carriers, roguing infected plants, rotation of crops, indexing seed stocks, control of vectors, utilization of disease-resistant varieties and strains, seed certification and plant quarantine.

The viruses of tobacco mosaic and cucumber mosaic are known to be spread by laborers cultivating the crop. Tobacco mosaic virus is spread in the process of topping, and the cucumber mosaic virus when the green cucumbers are harvested. In such cases it is well to remove the infected plants before starting the topping and gathering of the green fruit. The number of insect vectors should be reduced to a minimum through spraying and seasonal planting to avoid as far as possible the peak of the insect population.

Some success has been obtained in reducing the amount of the aster yellows by enclosing the plants with a fine screen wire fence (18 wires to the inch) four to six feet high. The growing of asters in muslin (22 x 22 threads per inch) houses

in the field has proved effective in reducing aster yellows. In general, it may be said that insect vectors are difficult to control because of the small number required to initiate general infection in the field. Control from the standpoint of insect injury is not adequate from the point of view of insects as virus vectors. Sometimes wild plants and weeds growing in fence rows or waste places serve as centers for the spread of infection. This is especially true in the case of tomato mosaic and cucumber mosaic viruses. The former virus may overwinter in perennial species of wild ground cherry and the latter in the swamp milkweed. Recently the wild Wandering Jew (*Commelina nudiflora* L.) has been shown to be a carrier of the celery mosaic virus in Florida. The aster yellows virus has many weed hosts. Such host plants may serve as sources for initiating aster yellows. Weed hosts should be destroyed, thereby preventing them from functioning as a source of the virus. Where such carriers occur in fields, rotation, using an immune crop coupled with clean cultivation is recommended. Where a virus as the tobacco mosaic virus may live in the soil in plant refuse, rotation of crops not only in the fields, but also of the seedbeds is good practice. A singular type of control of virus is practiced in celery mosaic; e.g., in Florida the removal of all weed hosts within 170 feet of celery fields shows a marked reduction in incidence of the disease in the protected fields.

If the virus is seed-borne or carried in vegetative parts used for planting purposes, the stocks should be indexed for the presence of virus symptoms. This has been used effectively in the control of the virus of yellow dwarf of onions. Seed certification of potatoes makes necessary the elimination and destruction of all infected plants. In still other cases the whole seed source, if badly infected, is destroyed or used for food purposes.

In the case of beans and other legumes the presence or absence of viruses readily can be determined by growing representative samples under conditions favorable for the expression of virus symptoms. This practice applies not only to seeds, but also to plants propagated by tubers, bulbs or

cuttings. Roguing and the destruction of infected individuals tends to keep down the source of the inoculum. This method is practiced in the case of peach yellows, virus-diseases of raspberries, etc. Where a virus is just invading a district, quarantine and eradication may afford relief. In the latter half of the last century, several states enacted laws for the eradication of peach yellows. Eradication is still the only effective control measure known for the virus of peach yellows and is faithfully followed by peach orchardists. More recently the federal government and certain states in the south have established quarantines and instituted eradication measures against the viruses of the phoney disease and mosaic of peaches.

The most practical and effective means of control, however, lies in the direction either of finding or making very resistant sorts. This has been accomplished in several cases; i.e., varieties Idaho, Wisconsin and U. S. No. 5 Refugee beans resistant to the bean mosaic virus, varieties P. O. J. No. 2,878, 2,871 and 2,696 resistant to sugar cane mosaic virus, variety Shamrock resistant to cucumber mosaic virus, variety Ambalema resistant to common tobacco mosaic virus, variety Red Rock resistant to wheat rosette virus, and varieties U. S. No. 33, 35 and 600 resistant to the curly top virus of sugar beets.

Common Tobacco Mosaic

The tobacco mosaic virus is universally distributed in all parts of the world where tobacco is cultivated. More than 200 different species of plants in 12 families are known to be hosts of the mosaic virus, which consists of strains having different symptoms and host ranges. The infectious nature of mosaic virus to cultivated tobacco was first demonstrated by Mayer in Holland in 1886, and it was the first plant disease to be attributed to a virus.

The tobacco mosaic virus does not kill the tobacco plant, but it modifies the leaves and dwarfs the plants so that the quality and quantity of the crop is seriously reduced. The extent of the damage to the crop depends upon the time the plants become infected, the percentage of infected plants in a given field and the prevailing weather conditions Often

the loss in quality and quantity may reduce the returns on the crop 20 to 40 per cent.

SYMPTOMS.—The external symptoms of tobacco mosaic are manifested only on the aboveground parts of the plant.

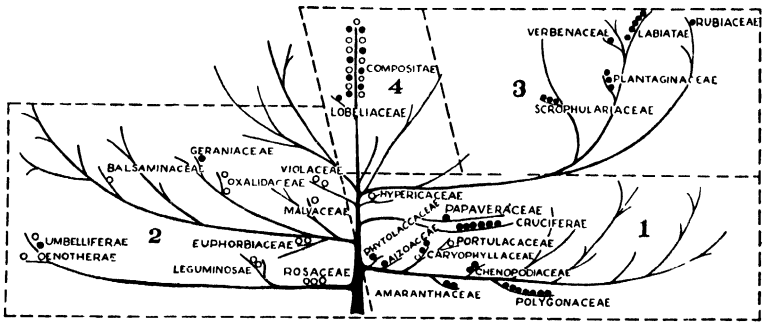


FIG. 118. Taxonomic relationships of plants susceptible to infection by common tobacco mosaic virus. Families of tested dicotyledonous plants diagrammatically arranged according to the plan of the Königsberger stammbaum of Mez and Ziegenspeck. Species tested by inoculation with the virus are indicated by circles; black circles, susceptible species; white circles, no symptoms. (Redrawn from Francis O. Holmes)

The age and the vigor of the host at the time of its inoculation and subsequent environmental conditions influence the manifestation of the many symptoms. The most striking symptoms occur on the foliage as a mosaic pattern of two shades of green. The leaves curl, become distorted and blistered. Sometimes the plant as a whole may be dwarfed and the flowers distorted, blotched and bleached. Infected suckers often show marked mottling. Necrosis, evident as dark specks, may follow the earlier symptoms. The symptoms may become masked at temperatures above 37° C. and below 11° C.

On young plants the first visible symptoms occur on the innermost or bud leaves as light-yellow and dark-green spots, arranged in a mosaic pattern. If older leaves are exposed to infection, all the new leaves may show "mottle top" or "grey top" with light and green areas involving the entire surface. In addition, yellow spots or lesions may form on the more matured inoculated leaves. In cross sections of the leaf, the palisade cells and the spongy parenchyma of

the light-colored areas are underdeveloped, while in the darker areas they are larger and contain more chlorophyl than normal leaf tissue. In the individual cells there appear

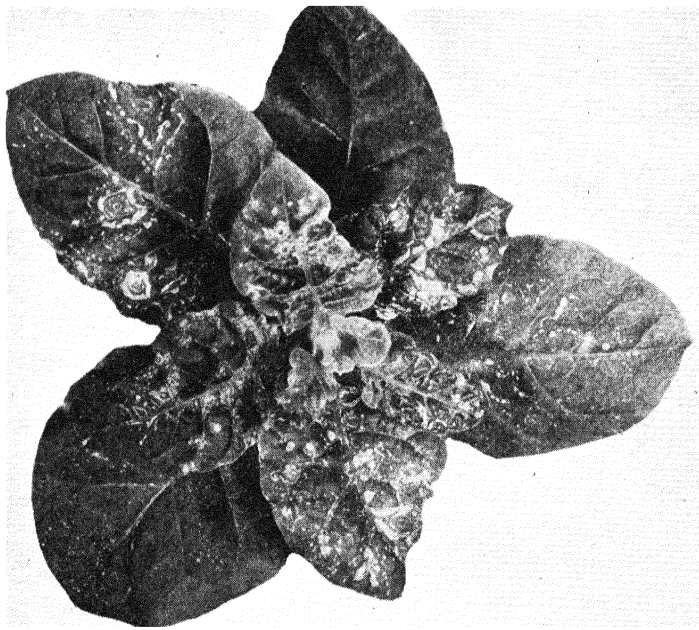


FIG. 119. Tobacco mosaic showing mottling, blistering and distortion of younger leaves. (Courtesy of L. O. Kunkel.)

two types of inclusions—X-bodies and striate material. The X-bodies are numerous, vacuolate and more dense than the cytoplasm of the cell. These are often in close proximity to the nucleus and are regarded as merely the product of the diseased cell. The striate material is an amorphous substance with readily visible striate markings probably generated by the diseased living cell.

ETIOLOGY.—The causal agent of tobacco mosaic is held to be a protein existing as liquid crystals or long mesomorphic fibres in which the particles are about 15mμ in diameter. The extracts of tobacco mosaic virus taken from different

hosts are still infectious when diluted 1–1,000,000 and sterile filtered juice may retain its infectivity for several years. Extracted sap will not remain infectious as long as the sterilized sap. The virus is very tolerant of high temperatures; 8 minutes at 90° C. are required before it is destroyed; it will retain its infectivity for 70 minutes at 85° C. and is not entirely inactivated in 10 minutes at 100° C. dry heat. No enzyme has been found that will attack virus protein. Tobacco mosaic is readily transmissible and is one of the most infectious viruses known at the present time. It may be communicated by breaking a trichome with an object carrying the virus. Seemingly, the host is most susceptible to infection through a wound, although there is some evidence that the virus may enter through the stomata.

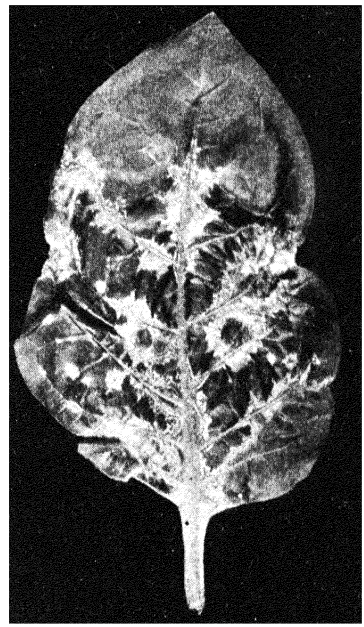


FIG. 120. Mottling and blistering of a leaf of tobacco caused by common tobacco mosaic virus. (Courtesy of L. O. Kunkel.)

The tobacco mosaic virus has an incubation period of 14 to 17 days within the tobacco plant. Within seven days after inoculation, however, the virus may be recovered from any portion of the stem. Roots, although showing no evidence of the virus, possess a high concentration of the virus protein that may, in fact, be recovered from the roots earlier in the incubation period than from the stem. The virus consists of a large number of strains that may be shown by immunological methods to be closely related. These strains vary from those producing mild mottling of the leaf to those producing severe yellowing, distortion, dwarfing

and local necrosis of the leaf and stem. It is held that antisera can be produced and that precipitins may form in the presence of the homogenous antigen.

No vector of the virus is known, although it is extremely infectious, and aphids occur commonly on tobacco. The aphids *Myzus pseudosolani* Theob, *M. circumflexus* Buckt. and *Macrosiphum solanifolii* Ushm. can transmit the tobacco mosaic virus from tomatoes to tobacco, but they cannot transmit it from tobacco to tobacco. There is considerable doubt as to whether the virus is seed-borne, and if it is, such instances are rare.

CONTROL.—In devising control measures for the tobacco mosaic it must be borne in mind that the virus is extremely infectious and that it is disseminated largely by man in growing the crop. Sanitation, therefore, is probably the most effective control measure. All infected plants should be removed from the plant bed and field as soon as they appear. Perennial solanaceous weeds growing in or about plant beds and fields should be destroyed, regardless of whether they are infected with the virus. Tobacco stems or any other form of trash known to carry the virus should not be returned to the field or plant bed.

Laborers working about the plant beds should avoid chewing infected air-cured or flue-cured tobacco. Since clothes that have been in contact with infected tobacco may serve to contaminate the hands, it is well for all laborers to wear clean clothes and to wash their hands in soap and water after handling infected tobacco refuse, roguing or in any other way working with growing tobacco.

Recently a variety of tobacco, Ambalema, resistant to the mosaic virus has been developed, but it is not of sufficiently high quality to find a place in practice. Although this variety is not immune, the symptoms are mild and of short duration, finally disappearing. Breeding work in the production of varieties which limit the activity of the virus to the formation of local lesions also shows promise, but no high quality resistant variety or strain has been made available to the growers to date.

The losses may also be reduced by a process held to be a type of vaccination or immunization. By this process tobacco plants are inoculated with a mild strain of the virus, which may protect them from further injury by certain other destructive, leaf-distorting strains.

Sugar Cane Mosaic

Sugar cane mosaic originated in eastern Asia from where it has been distributed in sugar cane cuttings to practically all sugar cane growing countries of the world. Not only was sugar cane mosaic the most widespread disease of sugar cane, but it was also the most destructive until mosaic-tolerant and resistant varieties came into general use. Before the mosaic-tolerant P. O. J. varieties were used in Louisiana the sugar industry was definitely in distress. The more recent resistant varieties have still further reduced the mosaic losses and thereby materially aided the stability of the sugar industry. Recently, in a comparative test made in Louisiana using infected and healthy plants of eight varieties resistant to mosaic, the yield ranged from 3.8 per cent increase to 49.5 per cent decrease in the virus-infected plants compared with the healthy plants. Five of the varieties showed a decrease of more than 12 per cent, two showed less than 6 per cent decrease and one showed an actual increase of 3.8 per cent.

Sugar cane mosaic first was recognized in 1892 as a transmissible virus disease in Java. This malady had been known in Java for a long time and was believed to be caused by a bud variation. In this country, sugar cane mosaic was unknown until 1919, although there also is evidence that it existed in this country before it was definitely identified.

Symptoms.—Sugar cane mosaic causes the plants to appear conspicuously yellowish-green throughout. This color effect, however, is due to the numerous irregular light-colored spots or streaks on the leaves. The light-yellow areas predominate and they are uniform in size, shape and arrangement for a given variety of cane. Later these light-yellow areas may lose their color, becoming small opaque

spots and streaks that finally dry up and become a whitish color. These areas may comprise from 20 to 30 per cent of the leaf.

In older plants a striping or cankering of the stalks may develop. Incipient cankers which appear as discolored or water-soaked patches or streaks on the internodes are exposed if the leaf sheath is pulled away from the node. In severe



FIG. 121. Two sugar cane plants, Co. 281, one healthy and the other showing symptoms of mosaic virus strain No. 3. (Courtesy of E. M. Summers.)

cases these streaks become sunken and the internodes spindle-shaped and attenuated. Long cracks may appear permitting the tissues to dry out. The diseased cane may have shortened internodes and show premature root development and excessive stooling at the nodes.



FIG. 122. Dwarfing associated with planting of virus-infected cuttings of sugar cane, CP 29/291. (Courtesy of E. M. Summers.)

ETIOLOGY.—The causal agent is not as infectious as the tobacco mosaic virus and is transmitted mechanically with some difficulty. The corn aphid, *Aphis maidis* Fitch, is the chief insect vector, although the rusty plum aphids, *Hysteroneura setaria* Thos. and *Toxoptera graminum* Rond., may occasionally serve as vectors. The corn aphid is known to be distributed long distances by the wind, which naturally aids in the dissemination of the virus. The expressed juice of the infected plant loses its infectivity when exposed to the air for a day at room temperature, when diluted 1–100 and when passed through filter paper or an L_3 Chamberland candle. This virus is unlike many other viruses in that it has a low thermal death point, 53° to 54° C. Infected plants may apparently recover, although in some cases the virus is present and may induce infection when inoculated into healthy canes. The virus is made up of several different strains that vary in the degree of injury caused to the host. The yellow strain causes greater reductions in yield than the green strain.

Inoculated susceptible plants, when growing rapidly, show the first symptoms of secondary infection about 15 days after inoculation. These symptoms and the course of the disease resemble in every way those following inoculation through the agency of the insect vector. Replanting diseased stools with cuttings obtained from healthy plants does not excite the disease, since the virus is not transmitted through the soil. Cuttings obtained from infected plants usually produce diseased stools, but seeds of corn, sorghum, sugar cane, crab grass, bull grass, etc., produced on mosaic-diseased plants are always free from the virus. This virus can be transmitted to different species of wild and cultivated grasses as follows: Maize, sorghum, pearl millet, sudan grass, Guatemala grass and Eulalia as well as several weed grasses, as giant foxtail, yellow foxtail, quack grass, bull grass and wild sugar cane.

CONTROL.—Control of sugar cane mosaic consists primarily in the elimination and destruction of infected plants and the use of resistant varieties. Only selected, healthy cuttings

should be used for planting purposes. If mosaic occurs in the fields, it is well to rogue infected canes. Since the virus attacks many weed grasses, these should be destroyed in and about the sugar cane plantation. Within recent years, much has been accomplished through the utilization of resistance

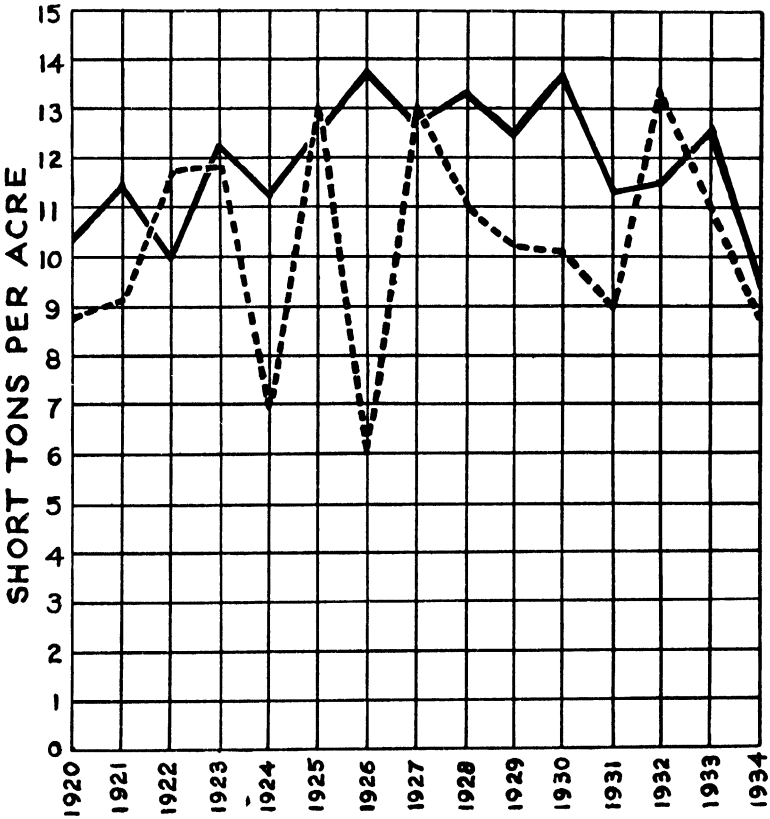


FIG. 123. Annual fluctuations of sugar beet yields in Colorado and Idaho. Over 90 per cent of the crop grown in Colorado (solid line) is outside the curly top area. In Idaho curly top is general and prevalent. The sharp fluctuations in the Idaho yields (broken line) are caused by the destructive development of the curly top virus in 1924 and 1926. (After Hartley and Rathbun-Gravatte.)

in the control of the mosaic virus. Varieties of sugar cane vary widely in susceptibility and the degree of injury manifested in the plant; i.e., the varieties P. O. J. 36, 213, 228 and 234 developed in Java were very susceptible, but the

growth of the plants was not seriously injured, a phenomenon sometimes designated as "tolerant." The varieties Kassoer, P. O. J. 2,714, 2,725 and 2,727 are known to be highly resistant, and P. O. J. 2,725 has been used extensively in the development of such commercially useful resistant varieties as P. O. J. 2,878, 2,871 and 2,696.

Curly Top of Sugar Beets

The curly top disease was first recognized as of major importance in California in 1899, and since that time many epiphytotics have occurred in practically all of the sugar beet growing areas west of the Rocky Mountains. The disease also has become a serious factor in Argentina, the only region outside of Western North America in which the causal virus occurs. The curly top disease has jeopardized the sugar beet industry in the United States for nearly 40 years. Curly top has often been so destructive to the crop that beet growing has had to be discontinued in whole districts and sugar factories abandoned, resulting in huge additional financial losses.

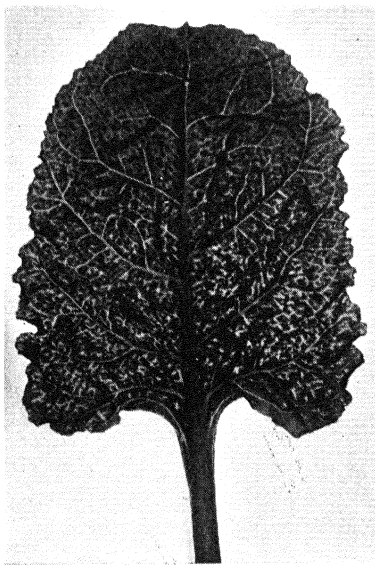


Fig. 124. Clearing of the smaller veins of a young sugar beet leaf. This vein clearing is one of the earliest symptoms of curly top. (After H. H. P. Severin.)

The curly top virus has a very wide host range and the symptoms produced on most of the host plants have a general similarity. During the last decade it has been shown that the curly top virus causes vast losses of tomatoes (western yellow blight), beans, squash, pumpkins, spinach, etc., throughout the western sugar beet growing region.

Symptoms.—Characteristic symptoms in the early

stages of the disease are the clearing or transparency of the minute veins on the youngest or innermost leaves, and in the latter stages wart-like protuberances on the lower surface of the leaves. The increase in the number of rootlets, which is



FIG. 125. Curly top of sugar beets showing the crinkling and curling of the younger leaves of the infected plant. (Reproduced from *Jour. Ag. Res.* Separate G-384.)

sometimes described as "hairy" or "woolly" root, is another important symptom.

The earliest symptom of curly top is an inward rolling of the outer margin of the youngest leaves, while later the entire blade may show a pronounced inward curling. The diseased leaves are dark, dull green in color, thick, crisp and brittle, and an exudate may appear on the petioles. The lower surface of the leaves becomes roughened, and protuberances usually develop after the veinlets have become transparent.

The virus is fatal to young beet seedlings, dwarfs more mature plants and causes "dead heads" or blighted or dwarfed seed stalks. In all cases the virus induces phloem degeneration, evident as dark, concentric rings in cross sections of the

roots. The phloem parenchyma cells adjacent to the sieve tubes show hypertrophy followed by necrosis and discoloration.

Etiology.—The virus passes the ordinary earthen filters such as Berkefeld V, N and W, and the porcelain Chamberland filter candles to L_{13}. The expressed juice from infected plants may be diluted 1–20,000 and still induce curly top

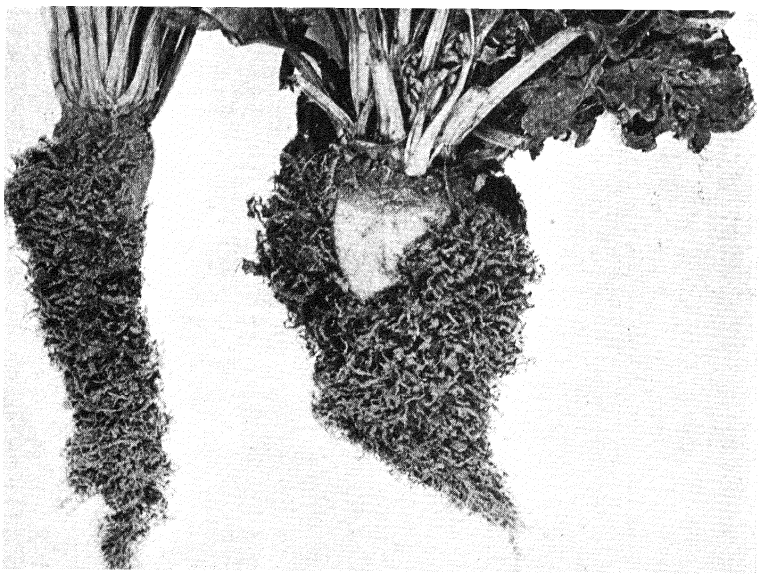


Fig. 126. Tap roots of beets infected with curly top virus showing woolly root development. (After H. H. P. Severin.)

when inoculated into healthy plants. The virus is quite resistant to desiccation, remaining active after 10 months in dried phloem exudate, two months as an alcoholic precipitate of infected beet leaf juice and six months in dried viruliferous beet leaf hoppers.

The incubation period of the virus within the plant varies from 4 to 18 days under average conditions. This may be extended by varying the environmental conditions. The virus has been shown to move through the petiole at a rate of 60 inches per hour, and it apparently moves rapidly only

in tissues into which food is moving. Movement into regions from which sugar is being translocated is extremely slow. The virulence of the virus may be varied by passing it through certain hosts; i.e., the nettle-leaved goosefoot (*Chenopodium murale*), and certain resistant types of sugar beets reduce its virulence, but such attenuated strains may be restored to their original virulence by inoculating them into chickweed (*Stellaria media*). The virus is transmitted mechanically only with difficulty although it has been done by pricking the virus from extracted phloem sap into the crowns of healthy beets.

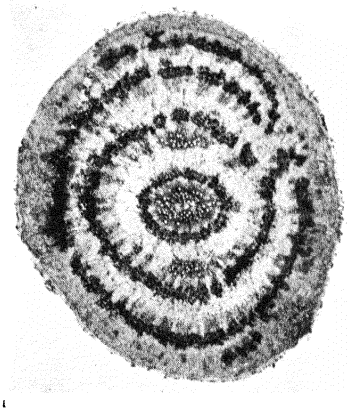


FIG. 127. Cross section of tap root of sugar beet infected with curly top virus showing primary hyperplasia and necrosis. (After K. Esau.)



FIG. 128. Adult beet leafhopper. (After H. H. P. Severin.)

The vector is a leaf hopper, *Eutettix tenellus*, that is distributed generally west of the Rocky Mountains. It is primarily a phloem feeder, which is probably the basis of its ability to transmit the virus. There appear to be two types of transmission of the virus by the vector: In the first type infection occurs after short periods of feeding on diseased and then on healthy beets, which is considered to be purely a mechanical transmission of the virus on the contaminated mouthparts of the vector; and in the second type, infection depends upon the development of the infective power within the insect, which

necessitates a delay of 21 to 24 hours after feeding on an infected host plant or a virus containing extract.

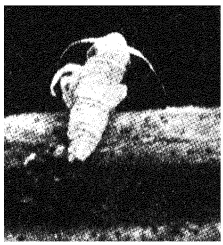


FIG. 129. Recently hatched nymph ready to crawl away. Such nymphs do not transmit the curly top virus. (After H. H. P. Severin.)

The virus has been shown to overwinter in living beet leaf hoppers without any diminution of virulence. In addition, the virus may overwinter in winter annuals which serve as hosts for the first brood leaf hoppers in the spring. These winter annuals seem to serve as a source of virulent inoculum. Many of the plants are symptomless carriers, and some of these seem to attenuate the virus.

CONTROL.—The most practical control of the curly top virus is of very recent development; it consists of using resistant varieties. Through extensive mass selection the United States Department of Agriculture discovered and made available to sugar beet growers the resistant variety known as U. S. No. 1. This resistant strain has already been used extensively although released less than five years ago. Since 1936 the United States Department of Agriculture has released three other resistant strains known as U. S. 33, U. S. 35 and No. 600. The first two strains were obtained by mass selection from U. S. 1. The two strains U. S. 33 and U. S. 35 were more resistant to the curly top virus than U. S. 1, resulting in higher yields where opportunity for serious infection prevailed. The strain No. 600, which originated as a triple hybrid, is resistant and probably higher-yielding than either U. S. 33 or U. S. 35, but is slightly lower in percentage sugar.

Frequently the loss from curly top may be reduced by planting the beet crop early enough to permit the beets to pass the critical seedling stage before the beet leaf hopper migrates from its weed hosts to the beet crop. Early planting of the resistant varieties is necessary if they are to have a chance to develop their full resistance before the leaf hopper infestation occurs. In like manner, it is possible to decrease

the leaf hopper population by decreasing the weed hosts about sugar beet fields; and by replacing introduced plants by native vegetation, which is less favorable to the leaf hopper. The native vegetation returns in the normal succession if the exotic plants are destroyed.

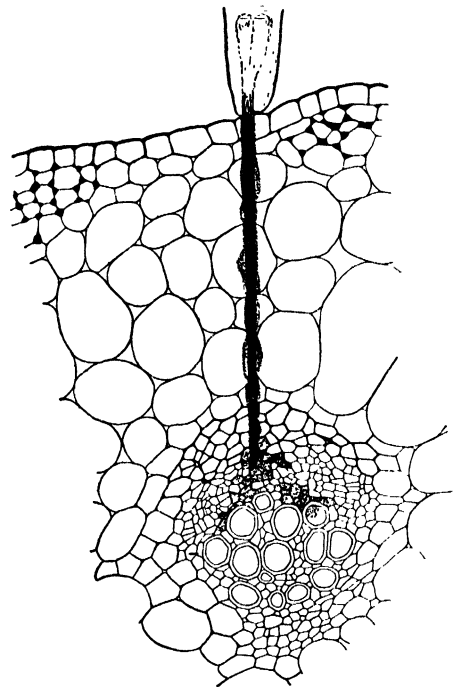


FIG. 130. Stylets of *Eutettix tenellus* showing their relation to the tissues of the beet petiole during feeding (X180). (Reproduced from *Jour. Ag. Res.* Separate G-891.)

ASTER YELLOWS

Aster yellows is prevalent in Europe, Japan and America, but it is most destructive on the China aster in the United States. The virus of aster yellows differs from the mosaic viruses just described in that it cannot be transmitted mechanically, and it requires a ten-day incubation period in the leaf hopper (*Cicadula divisa* Uhl.) before it may be transmitted by the vector. There are two strains of the aster yellows virus that cause similar symptoms but differ somewhat in their host range. One of these virus strains causes a disease of celery in California known as celery yellows.

Aster yellows is one of the chief limiting factors in the growing of asters in the midwestern states. It is not uncommon to find 90 per cent of the plants infected, and although such plants may not die they seldom produce merchantable flowers or viable seed. Celery and lettuce are the only other crop plants known to sustain any loss from the virus.

SYMPTOMS.—The aster yellows disease is characterized by marked changes in the habit and color of affected plants. The leaves show diffuse and distinct yellowing but no mottling. The first symptom appearing on a young plant is a slight yellowing along the veins in the whole or in a part of a single young leaf. This symptom, called "clearing of the veins," is a specific and distinct characteristic symptom of yellows in asters. The spread of symptoms seems to be hindered by the larger veins which act as a barrier to the movement of the virus. Yellowing does not appear in leaves that are mature when the plant is attacked, but younger leaves may show symptoms of the disease. Nearly a total lack of chlorophyl may occur in young leaves when the attack is severe. Yellowing may be exhibited in all the normally green portions of the plant.



FIG. 131. Aster yellows, showing stunting and excessive branching.

Diseased leaves are frequently somewhat deformed, having longer petioles and smaller leaf blades than leaves of healthy plants. The leaves tend to grow more erect, giving the infected plant a constricted and upright appearance. These plants nearly always remain dwarfed and often show a "rosetting" caused by the increase in the number of branches and the shortening of the internodes.

Probably the most striking symptom of the disease is in the form and color of the flowers, which become green or yellowish-green. Sometimes only a portion of the flower is affected, giving it a distorted appearance. In other cases

individual flowers may develop into stems which may or may not bear small flower heads. In affected flowers the stamens have a tendency to abort, and the anthers are usually small or shrivelled producing very little or no pollen. The pistil may enlarge and elongate, extending beyond the corolla tube. Trichomes or hairs are often reduced in numbers and may develop into leaf-like structures. The root systems of



FIG. 132. The two plants at the left show the effects of aster yellows. The plant at the right is healthy. Note the excessive branching and upright habit of the plant at the left and the dwarfed condition of the center plant.

diseased plants appear normal, but are sometimes smaller than those of disease-free plants. In advanced stages of the disease, tissues below the apical growing point may become necrotic or even leaves and side shoots may turn brown and dry up.

ETIOLOGY.—The causal agent of aster yellows is a virus that may be transmitted from plant to plant by two different species of leaf hoppers, *Cicadula divisa* and *Thamnotettix*

montanus Van D. The former is the most effective vector. The celery yellows strain of the virus is known also to be transmitted by the mountain leaf hoppers, *T. geminatus* Van D. and *T. montanus*. The aster yellows virus is not carried in the seed and cannot be artificially transmitted except by grafting or budding, and hence little is known of the nature of the virus.

The adult leaf hopper *Cicadula divisa* is unable to transmit the virus until a ten-day period has elapsed after the initial feeding on an infected plant. Nymphs of the leaf hopper may take up the virus, but because of the ten-day period necessary for the development of their infective power, never transmit the virus until they become adults. Some insects are able to retain the ability to transmit the virus for 100 days or as long as they live, while others lose it after a much shorter time. The insect remains a vector only as long as the temperature of its environment does not go above 31° C. for 12 days. Such exposure results in entire loss of infectivity, while exposure for shorter periods results in partial loss of the viruliferous nature. The virus cannot be transmitted from one insect to another, and it is not transmitted through the egg or in the faeces.

The incubation period in the host varies from 10 to 39 days, and the average is about 18 days. The aster yellows virus produces symptoms on more than 150 different hosts distributed in 31 families, and the following hosts may serve as carriers: Chrysanthemum, sow thistle, wild carrot, etc. The host range of the celery yellows virus is not as extensive, only ten host species are known to date.

CONTROL.—Aster yellows virus is difficult to control because of its wide host range, the prevalence of carriers and preponderance of the vector on many weed hosts. The most effective way of controlling aster yellows virus is to grow the plants in tobacco shade cloth houses (22 x 22 threads per inch). Screen wire (18 mesh to the inch) or tobacco shade cloth fences six feet high built around the aster beds reduces the amount of infection but is not as effective as actually enclosing the asters in cloth houses. A late crop

of asters transplanted the first of July in the upper Mississippi valley often entirely escapes aster yellows, perhaps as a result of the high temperature effect on viruliferous leaf hoppers, while an early transplanted crop generally becomes infected.

Chapter Eleven

DISEASES CAUSED BY ASCOMYCETES

THE Ascomycetes or sac fungi comprise approximately half of all the fungi known. Most of these are saprophytes, but many are plant parasites, causing such diseases as peach leaf curl, brown rot of stone fruits, powdery mildew, apple scab, chestnut blight, ergot and many others.

The organisms vary widely as to form and size. Some of them are extremely simple, consisting of a single cell, as the yeasts; others are more complex, as the species of *Sclerotinia*, having a profuse mycelium and several spore forms. As to size, most of them are microscopic, but some are visible to the naked eye; e.g., one of the largest is the common morel.

The chief distinguishing characteristic of this group is the production of a spore-containing sac, the ascus. The ascus is formed directly from, or on hyphal cells taking part in or simulating the sexual process. The ascus may originate directly from the mycelium, as in species of *Taphrina*, or from a part of the mycelium differentiated into an ascogenous layer. This layer may be a loose or compact mass of hyphae (stroma) underneath the epidermis, as in the peach leaf curl fungus; a flat disc (apothecium) as in the brown rot fungus; a thick-walled, flask-shaped ascus-containing structure (perithecium) as in the apple scab fungus; or a closed, thick-walled spore case (cleistothecium) as in the powdery mildews. In the majority of cases the ascogenous stage, which may serve as a resting stage, occurs after the host is dead, completing its development during the winter and spring.

The number of ascospores in each ascus varies from 2 to 256, but in most of the species each ascus contains eight. The mycelium may be sparse or profuse, many-celled, uni-

nucleate, inter- or intracellular and endophytic or superficial. The conidia may be borne singly or in chains on simple or branched conidiophores. These conidia are usually short-lived and serve to aid the rapid spread of the fungus. It is usually the conidial stage that functions in aiding the spread and destructiveness of the parasite during the growing season.

The general effect produced on the host by different Ascomycetes is variable, as rapid general necrosis in the Sclerotinia rots of rosaceous fruits, and Thielavia root rot of tobacco; local necrosis in alfalfa leaf spot, or indirect necrosis in peach leaf curl and the apple scab. In some cases the mycelium stimulates abnormal and excessive branching of the host. In other cases the mycelium may live in the tissues over long periods without causing apparent injury.

Chestnut Blight

Endothia parasitica (Murr.) A. and A.

Chestnut blight organism was probably introduced into this country on nursery trees from the Orient sometime previous to 1904. The pathogen spread rapidly throughout the range of the chestnut forests.

The rate of spread and destructiveness of the chestnut blight organism is probably without a parallel in plant pathology in the United States. Although there were only a few scattered infections in 1904, in 1908 the parasite occurred in Connecticut, Massachusetts, Vermont, New York as far north as Poughkeepsie, and south into New Jersey, Pennsylvania and possibly Delaware. It is now distributed throughout the range of the chestnut to the west and south. By 1911 the estimated loss was $25,000,000, and nothing has occurred to prevent its continued destructiveness. It now appears certain that the chestnut tree in this country is doomed. The destruction of the chestnut tree will adversely affect many industries, particularly lumbering and mining.

The seriousness of this disease led the legislature of Pennsylvania in 1911 to enact a law setting up a five-man com-

mission to ascertain the most efficient and practical means of prevention, control and eradication of chestnut blight and to enforce a quarantine against the parasite. The commission began its work late in July, 1911, by placing scouts in the field to locate diseased trees. During the summer of 1912, the commission had in its employ about 200 field agents and scouts locating diseased trees and directing their removal under proper sanitary conditions. Later it became apparent that the pathogen was so generally distributed that eradication was impractical.

SYMPTOMS.—The first evidence of the disease is a loss of color of the leaves borne above the cankers. As the pale-green color extends inward from the margin the outer parts become yellow. About this time the leaves begin to wilt, become entirely yellow and later brown. These brown shrivelled leaves are particularly noticeable in young and older trees in a forest stand. If the limb is not entirely girdled by fall, the young leaves in the spring remain small and yellowish or pale green. The yellowed leaves do not reach mature size, but soon wither and turn brown. These symptoms may develop on trees of all ages.

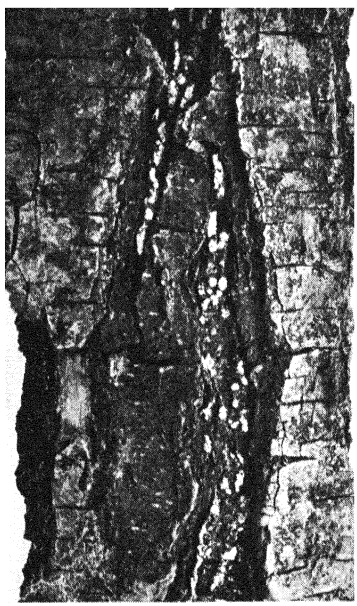


FIG. 133. Bark from old blight canker of chestnut. The small, raised, light-colored areas are perithecial stromata of *Endothia parasitica*.

The earliest symptom of the disease on twigs is a yellow to yellowish-brown discoloration of the bark. Later this twig infection leads to either a reddish, sunken canker on the smooth-barked limbs, or hypertrophy of young vigorously growing twigs. In heavily infected limbs the

withered brown leaves and underdeveloped burs hang on the dormant trees through the winter.

Infection of old limbs or stems first appears as longitudinal splits in the bark with pustules in the deep crevices. In old cankers the bark becomes rough and creviced with the yellowish-orange pustules in the crevices. When the bark is stripped from these cankers there are exposed tawny fan-shaped mycelial mats. The bark often falls away leaving the unprotected discolored wood of the cankers, and from the base of such dying trees numerous sprouts may develop.

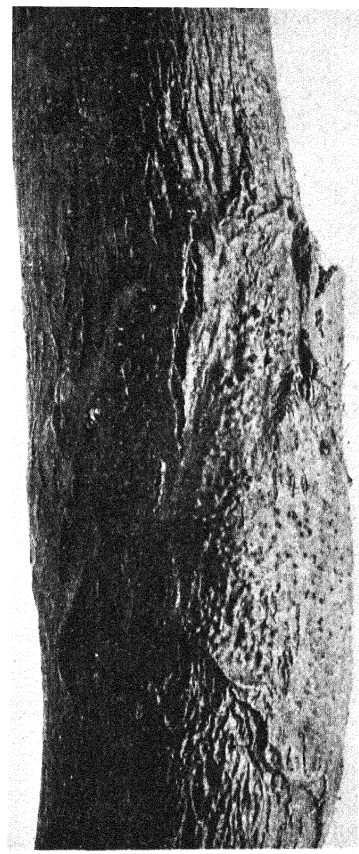


FIG. 134. Limb canker of chestnut showing smooth bark canker bearing pycnidia of *Endothia parasitica*.

The first signs of the disease are small reddish pycnidia, which appear on the cankers within two or three weeks after infection. Following a rain these pustules become surmounted by a yellowish, waxy tendril of spores. Later there develop larger yellowish pustules bearing numerous papillae on the outer surface. These are the stromata with the protruding ostioles of the perithecia. As the perithecia mature the ostioles turn orange, then reddish-brown and finally black.

ETIOLOGY.—The hyphae of *Endothia parasitica*, the causative agent of chestnut blight, are branched septate threads. They produce characteristic fan-like mats of mycelium in the bark and cambium. Each mat consists of a number of bundles of

parallel hyphae diverging from a single point as the rays of a fan, pycnidia are produced from the mycelium in the bark and the spores are extruded from the pycnidia in yellow, waxy tendrils. The thin-walled, one-celled, pycnidiospores do not germinate in pure water. The spores may live one year in

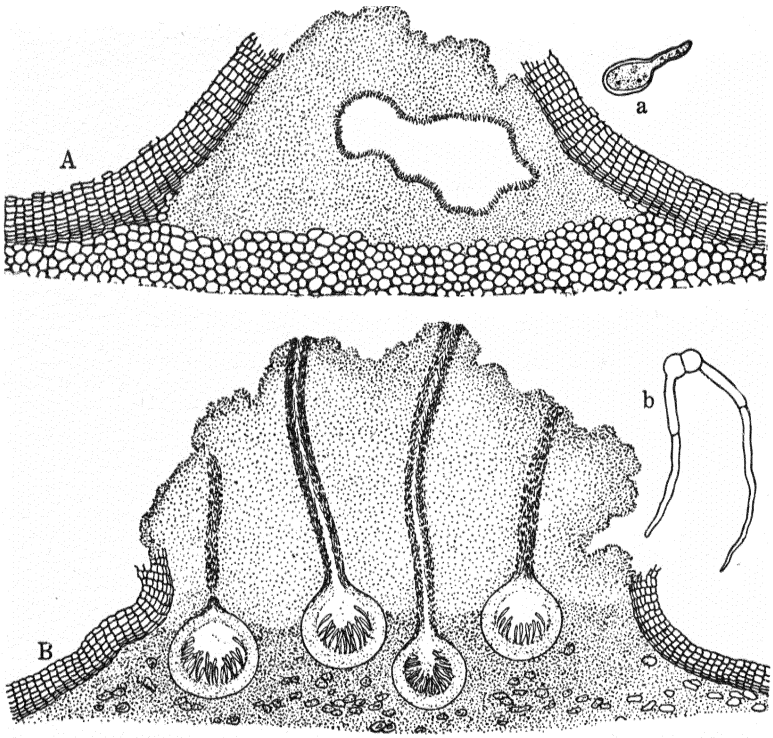


FIG. 135. Fruiting structures and spore stages of *Endothia parasitica:* A, diagram of a section cut through a pycnidium; a germinating pycnidiospore, a; B, a section of the bark showing the long-necked perithecia containing asci; a spore germinating, b.

the dry air of the laboratory or 119 days in the soil and have been observed to survive the winter, like the ascospores.

When the conidia germinate they may cause infection only through wounds. A mass of saprophytically growing mycelial threads is first produced in the wounded tissue. Wefts of this mycelium then penetrate the middle and inner bark, causing immediate death of the tissues as they advance.

Single hyphae apparently do not invade living tissues. A successful infection results in the formation of the mycelium on which may be produced stroma bearing pycnidia and later perithecia. The numerous bottle-shaped perithecia develop in submerged layers of the stroma under the previously active pycnidial layers and send their long necks up through the surface layers. The asci, which may be continuously formed

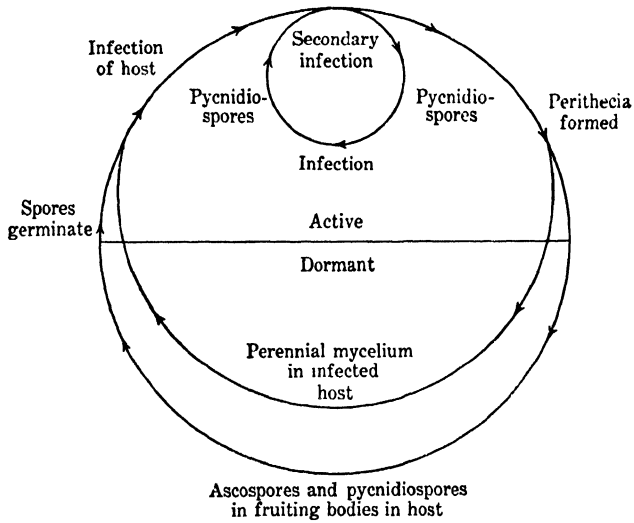


FIG. 136. A diagram of the host relation of *Endothia parasitica*. Both types of spores may survive the winter in their respective fruiting bodies. The mycelium is perennial in the living host.

over long periods within a single perithecium, contain eight two-celled spores with thicker walls than the pycnidiospores. These spores normally survive the winter and reproduce the pycnidial stage through new infections in the spring. The relation of the different stages of the pathogen is shown in Fig. 136.

Wind is probably the principal agent for the dissemination of the ascospores, but they are no doubt also carried long distances by birds, animals, insects and by shipment of infected material (bark, nuts and timber). Weather conditions seem to have little effect upon the development of the spores,

providing they fall into a wound on the chestnut. This is effected, especially in the spring, when the weather is damp. The infection always takes place through wounds made by some agent.

CONTROL.—All hope of saving the present stand of the American chestnut has been abandoned. The efforts of the Pennsylvania Chestnut Blight Commission already have been narrated. Protection from this pathogen in the future may best be secured by hybridizing, using some of the highly resistant species as one of the parents. Some progress in this direction has been made, but it is bound to be slow because of the long time required to grow forest trees. Although the Chinese chestnut (*Castanea mollissima* Bl.) and the Japanese chestnut (*Castanea crenata* Sieb. and Zucc.) have shown marked resistance, they are not as valuable commercially as the American chestnut (*Castanea dentata* Borkh.).

BROWN ROT OF STONE FRUITS

Sclerotinia fructicola (Wint.) Rehm.

In America the peach, plum, cherry, apple and apricot are the important economic hosts of the brown rot pathogen. The brown rot of fruit in the Old World recently has been shown to be caused by a different species of *Sclerotinia*, *S. laxa* Alder. and Ruh. Until recently it was believed that this species of *Sclerotinia* did not occur in this country. Evidence has been advanced, however, showing that the *Sclerotinia* occurring in the Pacific coast states may be the same as that in Europe.

The brown rot fungus is especially destructive in the middle west on such plum varieties as Lombard, German Prune, Blue Damson, etc. Brown rot generally occurs throughout the United States wherever the stone fruits are grown.

The brown rot organism causes one of the most destructive diseases of the peach crop, and under favorable conditions it progresses with great rapidity and, unless controlled, may destroy a crop within a few days. The pathogen is especially favored by hot, damp weather, which may account for its

destructive occurrence in the peach growing districts of the South. This disease also may develop on fruit en route to market. It has been estimated that prior to the development of satisfactory control measures (1907–10) brown rot often occasioned the loss of $1,000,000 annually in Georgia and at least $5,000,000 annually in the United States.

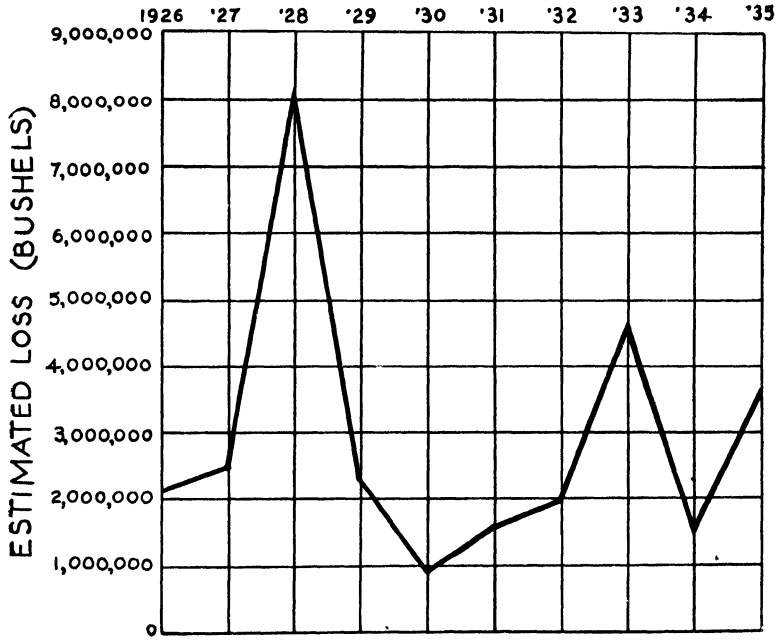


FIG. 137. Estimated loss of peaches caused by the brown rot organism in the United States (reporting area) 1926 to 1935, inclusive. (After N. E. Stevens and Jessie I. Wood.)

SYMPTOMS.—*Sclerotinia fructicola* may attack the flowers, foliage, twigs and fruit. The flowers may become infected by conidia produced from mummies hanging on the tree or by ascospores discharged from apothecia developed on the mummies lying on the ground. The pathogen may enter either the petioles or pistil. The mycelium travels between the cells causing rapid browning and blackening of the tissues, such that infected flowers often have the appearance of having been touched by frost. In fact, on plums, frost is often the erroneous explanation of the blighting of the flowers. The

real cause usually can be readily ascertained by searching during damp days for the glistening young conidiophores. If such flowers are incubated in a moist chamber for 24 hours, the brown tufted conidiophores may completely cover the necrotic tissues. Blossom blight often is very destructive



FIG. 138. Brown rot of plum. Infected plums showing shrivelling of the fruit and the causal agent, right.

when the weather is hot and damp, but it is inhibited by dry weather.

The leaves and twigs are injured much less than the flowers although under favorable conditions necrosis of the leaves and young twigs does occur. When this form of injury is prevalent the twigs, superficially, at least, suggest the symptoms of the apple blight disease except for the presence of the conidial stage of the brown rot organism. When less

favorable conditions for the development of the pathogen follow, however, the dead leaves fall away and the necrotic areas on the twigs crack. The advance of the pathogen in the host is marked by the formation of ridges of callous tissue. These cankers may become holdover cankers for the pathogen, which may sporulate in the spring and initiate primary infection.

It is on the fruit that the brown rot organism is best known and most destructive. The fruit is most susceptible

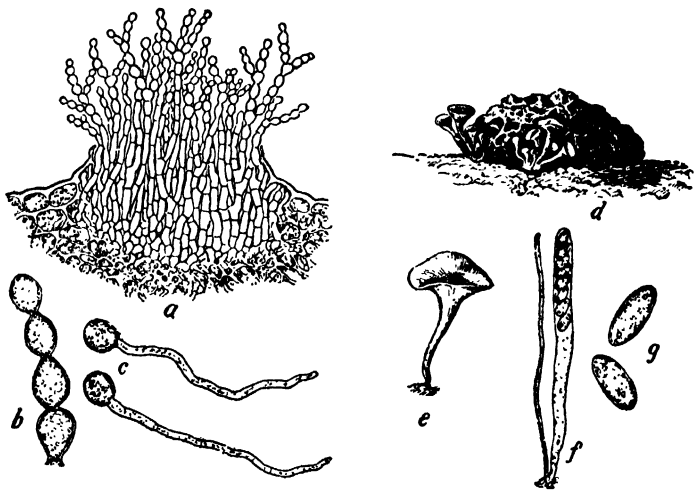


FIG. 139. Sclerotinia on plum: *a*, section showing a spore pustule and chains of conidia; *b*, part of a spore chain; *c*, spores germinating; *d*, a mummy plum and apothecia; *e*, an apothecium; *f*, ascus; *g*, mature spores. (After Longyear.)

when ripe. The pathogen may gain entrance through natural openings or through wounds. Lesions caused by the scab organism or more open wounds induced by the curculio are especially vulnerable. These two avenues of infection are so important that the spray schedule for the control of brown rot is designed to prevent scab and curculio injury. Once the fruit is invaded by the pathogen a brown spot is formed which increases rapidly until the whole fruit is discolored. Such fruits are soft and watery and later become covered with the brown tufts of conidiophores. These symptoms may

develop in the orchard, in the car en route to market or in the local market. In the orchard the generally infected fruits dry up gradually, and the surface wrinkles, forming the mummies so common in the fall in an orchard where brown rot was prevalent.

Not all of the generally infected fruits fall to the ground, and any of these mummies can produce spores the next spring and give rise to primary infection. The mycelium in the fruit that falls to the ground continues to live saprophytically in the necrotic tissues. By spring a sclerotial mass of mycelium has formed just below the "skin" of the fruit. It is this dense mass of mycelium that gives rise to the apothecial stage.

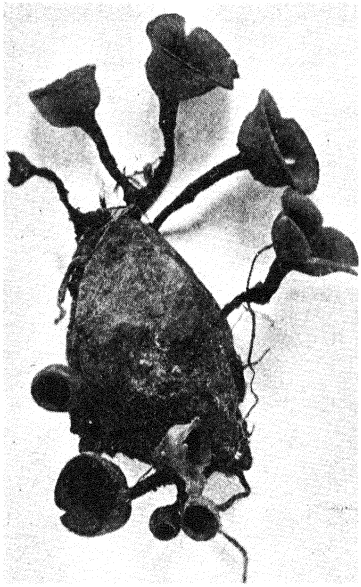


FIG. 140. Apothecia of various ages of *Sclerotinia fructicola* which grew out from a mummified plum. (Reproduced from U. S. D. A. Farmer's Bulletin No. 1410.)

ETIOLOGY.—Brown rot of stone fruits in America is caused by *Sclerotinia fructicola*, a fungus that has three spore stages. The asexual is produced in great profusion on infected tissues as tufts of gray mold over the surface. The tufts of mold, made up of conidiophores and conidia, arise from the mycelium in the tissues. The spores are short rods borne in chains at the tips of the conidiophores. In the presence of moisture and favorable temperature these may germinate immediately and produce infection of the host. In addition to these rod-shaped conidia, smaller spherical-shaped spores have been observed in pure cultures and on plums. It is believed these may function as the spermatia that fertilize receptive bodies before the apothecia may be produced.

The sexual spore stage is much more seasonal and less abundant. It develops from the mycelium in the mummies lodged in the surface mulch below the trees when the mummies are from one to three years old. From 1 to 20 stalk-like structures may grow out from the mycelium in the mummies and bear at their distal ends small, brown, cup-shaped organs, the apothecia. The inside surface of the cup bears a continuous compact layer of cylindrical hyaline asci and paraphyses.

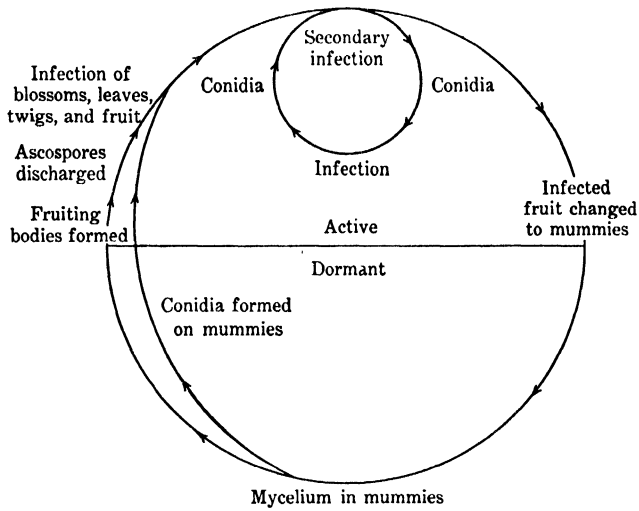


FIG. 141. A diagram of the host relation of *Sclerotinia fructicola*. This organism survives the winter as mycelium in mummies of diseased fruits.

The paraphyses that occupy the space between the asci are slender, septate and branched. The asci contain eight ascospores, which are discharged at maturity through an apical pore. The ascospores are discharged forcefully when ripe through the sudden expansion and contraction of the asci and paraphyses. This spore discharge or "spore puffing" is readily witnessed by allowing the drier air currents to come in contact with the apothecial cups, which have been in a moist chamber overnight. The shooting of the spores doubtless aids their distribution by raising them up into the air currents. The ascospores are ready to germinate as soon as

discharged and bring about infection of the host when a film of water is present and suitable temperature conditions prevail.

The spores, conidia and ascospores on germination form large unbranched tubes that may enter the host through natural openings, wounds or may even penetrate directly through the epidermis of cherries and peaches. Once inside the host the mycelium ramifies between the cells destroying the middle lamella through the action of pectase, pectinase and protopectinase, which dissolve the pectin compounds. In addition, peptinase and oxidase are formed abundantly. This dissolution of the middle lamellae causes a rapid softening or necrosis of the tissues during humid weather. If the air is dry the collapsed tissues dry, crack or wrinkle, forming the characteristic cankers of the twigs and mummies of the fruit. Subsequently the pathogen in the mummies lives saprophytically, forming a dense mat of mycelium from which the apothecia develop the following season. The host relation is shown in Fig. 141.

The conidia are readily disseminated by wind, rain and insects while the ascospores depend mainly on the air currents. Still cloudy weather with frequent showers favors conidial formation, germination and infection. In addition such weather prevents pollination, which prolongs the susceptible flowering stage of the host. Alternating wet and dry periods cause the fruits to rupture and greatly increases infection of the mature fruits.

CONTROL.—The control of this pathogen involves sanitation and prevention of infection through spraying. From the life history studies already recorded, it should be obvious that destruction of cankers and mummied fruit is highly desirable. The sanitary measures are most effective when carried out just after the crop has been harvested. The infected twigs should be burned and the mummies buried two feet in the ground. The prevention of blossom and fruit infection through spraying must begin when the flowers are in bud and continue at intervals until the fruit is ripe. Since scab lesions and curculio wounds afford avenues of infection, the

time and type of application and of fungicide also must be effective for these pests. In some sections the growers prefer the liquid fungicides, others, the dusts. Lime sulphur and arsenate of lead mixtures are generally used on plums, and various forms of sulfur in suspension in water, or dry sulphur mixed with lime and lead arsenate, for peaches. Fruit going to market or held for home consumption should be stored at temperatures below 10° C. to prevent the further growth and development of the pathogen.

Lettuce Drop

Sclerotinia spp.

The lettuce drop and watery soft rot may be caused by any one of three different species of *Sclerotinia*, *S. sclerotiorum* (Lib.) Massee, *S. intermedia* Ramsey and *S. minor* Jagger. The significance of these organisms on lettuce did not become known in this country until 1900. The parasites were first known in Florida, North Carolina and Massachusetts, while at present they are known and destructive wherever lettuce is grown. The ravages of these pathogens do not stop with the losses to crops in the field and greenhouse, but extend to lettuce in transit, in the market and in storage. Different species of *Sclerotinia* are among the most destructive organisms known on vegetables en route to market. Despite control practices, heavy losses have been recorded from California, Arizona, New York, Texas and Virginia during the last two decades.

The organisms causing lettuce drop also may attack potatoes, cabbage, celery, carrots, tomatoes, parsley, cauliflower, pepper, spinach, onions, cucumbers and many other plants, their destructiveness ranging from a trace to sometimes the whole crop. It is said that in 1920 more than 50 per cent of the lettuce, celery and cabbage shipped to the markets was infected. Of 613 cars of lettuce inspected, 108 showed varying amounts of the watery soft rot. In 36 of these cars, from 75 to 100 per cent of the heads were infected.

Of the cars shipped from the eight leading lettuce producing states in 1920, 54 per cent of the heads were decayed.

SYMPTOMS.—A conspicuous symptom of the lettuce drop disease is the rapid wilting of the leaves brought about by necrosis of the stem, petioles and crown of the plant. These

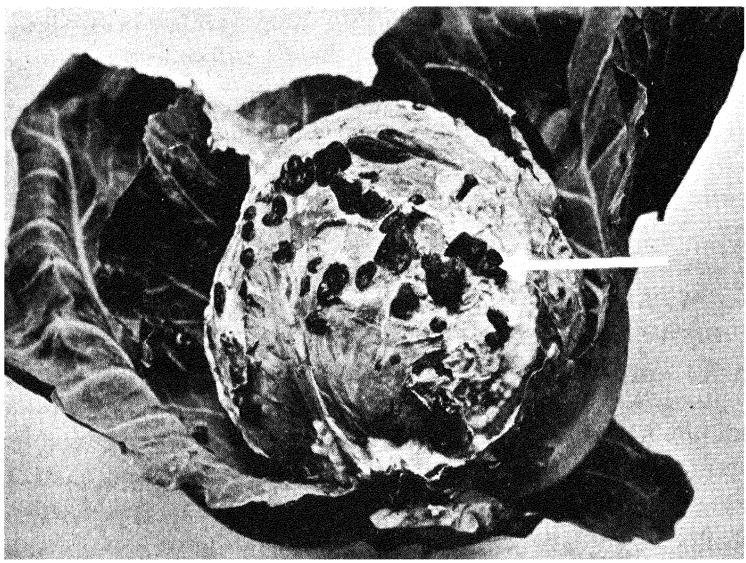


FIG. 142. Rot of cabbage produced by the lettuce drop organism, which produces whitish superficial mycelium and numerous sclerotia. (After J. G. Brown and Karl D. Butler.)

changes usually are coupled with signs of the disease, the profuse development of the mycelial and sclerotial stages of the pathogen over the infected tissues.

The organisms are destructive to seedlings as well as to mature plants. On the seedlings, damping-off develops at the surface of the ground. After the plant topples over on the soil, the pathogens produce a rapid necrosis of the flaccid leaves, which quickly dry and turn brown.

In the early stages of the attack on older plants, lesions appear on one or two of the lower leaves in contact with the ground, causing them to wilt. As the pathogen advances up into the crown, still other leaves similarly are affected.

Finally all the leaves of the plant are prostrate and the petioles water-soaked and discolored. These leaves have been described as resembling a "dull-green, wet, folded rag." As in the more rapid rot the head becomes, finally, a shapeless, watery, brown rotted mass having no odor.

The glassy, soft condition of the infected tissues, called watery soft rot, is due to the destruction of the middle

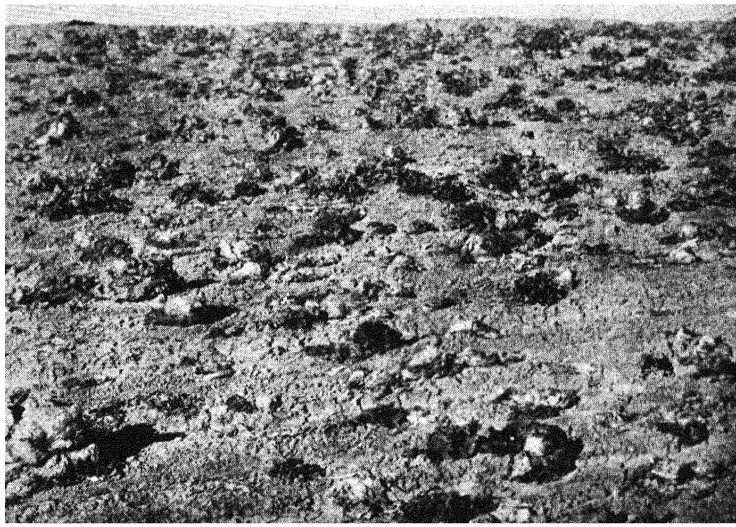


FIG. 143. A field of lettuce destroyed by *Sclerotinia spp.* (After J. G. Brown and Karl D. Butler.)

lamellae of the tissues by the pathogen. If the soil continues wet and the humidity high, the pathogen becomes evident as a white felt-like growth, especially on the upper surfaces of those leaves that are permeated by the mycelium. Dense white knots that appear in this mycelial weft develop into the black overwintering sclerotia. These sclerotia are irregular in shape and vary from a few millimeters to an inch in length. In Arizona where lettuce is grown as winter and spring crops, these sclerotia become especially abundant on lettuce

refuse in connection with the trimming at the packing sheds. In such places sclerotia may become so abundant that the ground appears as if covered with cinders.

ETIOLOGY.—The organisms causing lettuce drop, *Sclerotinia sclerotiorum*, *S. intermedia* and *S. minor*, have a host relation much like that of *S. fructicola*. (See Fig. 144.) The mycelia are white and flourish intercellularly within the host tissues as well as on the surface of living and dead plants. Under favorable conditions they often form a dense mat over the surface of infected tissues. They also are known to live saprophytically in the soil. In this way they differ from the brown rot organism already described that lives in the dead fruit and in the living plant.

Infection of a lettuce plant may be produced only by a saprophytically growing mycelium. When a spore of the fungus (ascospore) falls on a healthy leaf, it requires sufficient nutrient material to allow the production of a certain amount of saprophytic mycelium before it is capable of invading the living leaf tissues. Infection from mycelium growing in the soil or from a contiguous leaf occurs in the same manner. Once inside the leaf the mycelium spreads rapidly and produces the symptoms described above.

To bring about infection or to spread within the host tissues, the pathogen secretes enzymes that dissolve the middle lamellae and cell walls in advance of its hyphae. This process apparently is similar to that in brown rot and possibly accounts for the watery nature of the lettuce rot. The organisms may secrete toxic substances that kill the adjoining cells.

At different places on the aerial mycelium dense, tiny white (later salmon-colored) knots of hyphae are formed, which increase in size until they are often as large as peas. At this stage they become solid masses of mycelium with a black surface and a flesh-colored center. The sclerotia differ, therefore, from those of *S. fructicola* in that no host tissue is included with the fungous threads in the resting bodies. These sclerotial bodies frequently are produced in great abundance and fall to the soil where they serve to carry the organism

from one season to the next. Under conditions favorable for their growth and development, the sclerotia may send out hyphae from their surface or develop apothecia and ascospores similar to those already described for the brown rot organism. The asci in the perithecia are accompanied by paraphyses and contain eight unicellular ovoid spores. The eight spores are all forcibly discharged at the same time. When the ascospores are liberated they are scattered by the wind, and in that way come in contact with susceptible hosts.

The asexual stages of the *Sclerotinia* species attacking let-

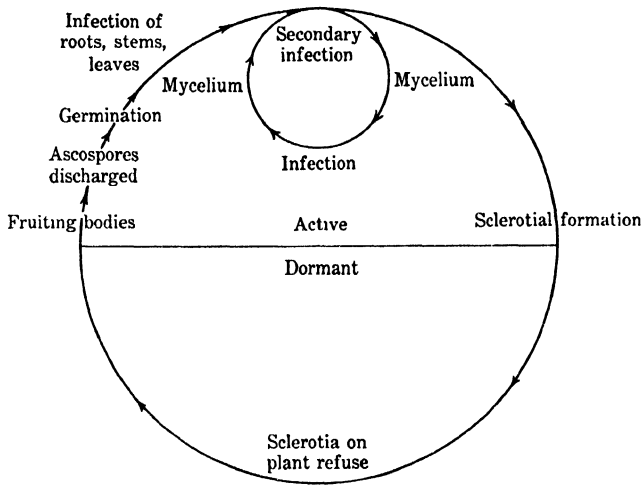


FIG. 144. A diagram of the host relation of *Sclerotinia spp.* These organisms survive the winter as sclerotia on plant refuse.

tuce are not so common and abundant as those of the brown rot organism. Very small globular conidia are produced in chains on short conidiophores under certain conditions. Their role in initiating infection, however, is not clearly understood and it may be that these spores are really spermatia and function as described in *Sclerotinia fructicola.*

In addition to the above methods, the organisms are spread by masses of mycelium breaking loose and floating in water or being disseminated with soil, and sclerotia similarly may be distributed. Although the sclerotia are produced in the seed

stalks and are liable to be included with the seed at threshing, no definite report of seed contamination by sclerotia has yet been seen.

The organisms are favored in their growth by wet soil and high humidity. Temperature is, however, of much greater influence on the causal agents. The optimum temperature for the organisms is between 15° and 21° C., while the optimum development of apothecia occurs at 18° to 22° C. In the southern lettuce growing sections the fungi are inactive during the hot summer season, passing this period in the form of sclerotia. The disease appears in any section when the minimum temperatures run between 10° and 15° C. for any considerable time.

CONTROL.—The control of lettuce drop is more easily accomplished under greenhouse conditions than in the field. When it is suspected that this pathogen exists in the soil, the soil should be subjected to some method of partial soil sterilization before the crop is planted. Where the organism makes its appearance on plants in the bed, these plants should be pulled immediately and the area drenched with copper sulphate solution, one pound dissolved in seven gallons of water. Much can be done to hold the lettuce drop in check by allowing as much ventilation as possible and growing the crop with a minimum of water. Where the greenhouse soil is infested and sterilization is not practical, it is advisable to grow in the rotation a less susceptible crop as tomatoes, cucumbers or peppers.

Under field conditions it is necessary to turn to rotation as a means of control, using less susceptible crops as small grains or other close growing crops the first year and after that tomatoes, potatoes, spinach or sweet corn. Any cultural practice that tends to keep the surface soil dry has an inhibiting effect on the lettuce drop organisms, as does deep plowing.

Infected refuse about the packing sheds should be destroyed rather than fed to cattle or made into manure. In addition the sclerotia around old refuse dumps should be collected and burned. Take care to avoid scattering infected plant parts over fields that may be used for lettuce production.

Roguing should be practiced in the field, and a similar separation of infected heads during packing and subsequently during shipping and storing will markedly lower the storage loss. The organisms may be limited in their spread to other heads in storage by lowering the temperature to 5° to 8° C.

Thielavia Root Rot of Tobacco

Thielavia basicola (B. and Br.) Zopf

Thielavia root rot was first reported in the United States on violet roots in 1891. Its chief economic host is tobacco, but it also occurs on violets, beans, ginseng, begonia, cucumber, nightshade, catalpa and clover, being parasitic on plants in more than 100 species in 20 families.

Good cigar tobacco land in the New England states is limited. Thus, farmers in this region practice continuous cropping and apply heavy quantities of fertilizer. In Kentucky, where the Burley variety is grown extensively for filler tobacco, growers practice a six- to ten-year rotation, chiefly because of the susceptibility of the Burley varieties to the root rot organism. In the New England section growers have been forced to select resistant varieties to maintain the industry, while in the Kentucky district they try to avoid root rot by long rotation and the use of resistant varieties.

Careful observations over a considerable part of the tobacco growing sections of the United States in recent years have shown that the damage from root rot annually runs into millions of dollars. The annual loss in Kentucky alone exceeds $2,000,000.

Symptoms.—The chief symptoms of black root rot of tobacco are observed on plants in the seedbed. When the very young seedlings are attacked, a damping-off similar to Pythium damping-off is produced. The organism may spread to the cotyledons and through the root system.

In older plants in the seedbed the lateral root tips are killed and blackened. The organism spreads up these young infected laterals into the main roots and causes a general

progressive rotting and blackening. Wounds at the origin of secondary roots also serve as a point of entrance. As these are killed, other laterals are produced above the lesion, which generally are attacked and destroyed later. This destruction appears to result in a stimulation of root growth

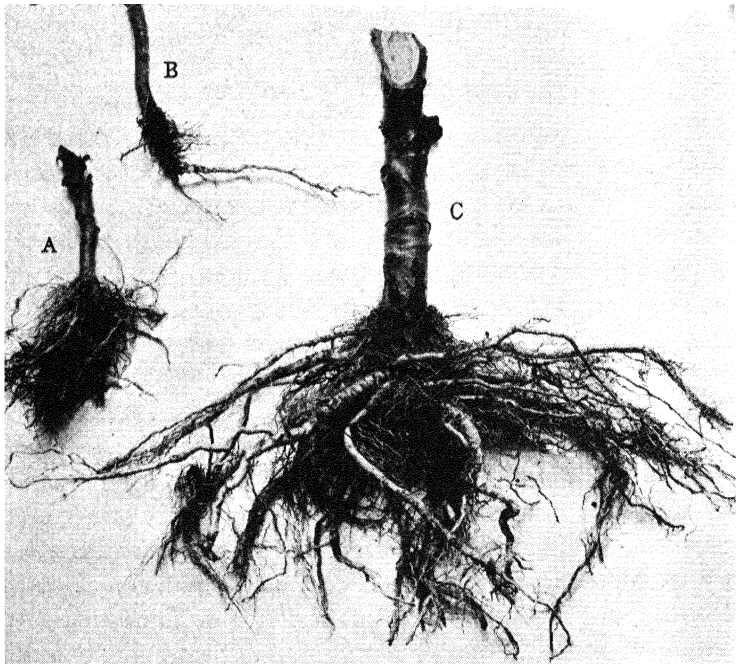


FIG. 145. Symptoms of Thielavia root rot on underground portions of tobacco: A and B, plants with roots nearly destroyed; C, plant with roots showing few black root lesions caused by Thielavia. (Courtesy of James Johnson.)

higher on the primary root, resulting in a bunching at the neck of the plant.

Symptoms of the disease in the field usually are confined to stunting, wilting and death. Infected seedlings or healthy seedlings that are set out in infested soil generally show marked symptoms. These plants are stunted, grow very little, become yellowed, wilt rapidly and generally die within two to three weeks. In light sandy soils some of these plants may recover sufficiently to develop during the hot weather

and produce a crop. It is seldom, however, that such recovery occurs in plants in a soil rich in humus. During a cool season the crop may have to be reset two to three times before a stand is obtained that will make harvesting profitable.

Although there are no macroscopic signs of the disease as such, the blackening of the infected roots might be in-

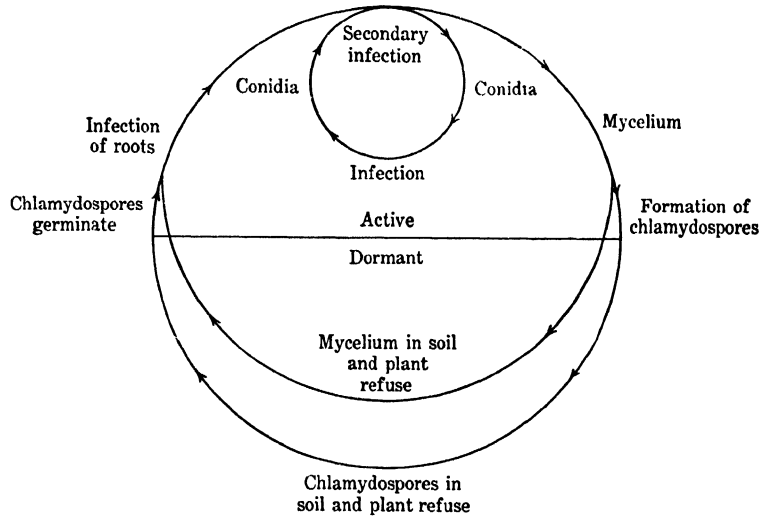


FIG. 146. A diagram of the host relation of *Thielavia basicola*. This organism survives the winter in the mycelial and chlamydospore stages.

cluded as signs. The blackening of the roots is due primarily to the production of numerous dark chlamydospores and perithecia within the cortex and stele of the infected roots.

ETIOLOGY.—Black root rot is caused by the fungus *Thielavia basicola* which mainly confines its attack to the roots of the plant. The mycelium typically is white turning to light brown when old. The organism is noted, however, for its ability to produce colonies not typical of the parent. It is possible, therefore, to obtain cultures of *Thielavia basicola* of nearly any color. On the host roots the mycelium bears three kinds of spores; the characteristic cylindrical conidia, which are produced in chains within terminally enlarged hyphae and are pushed out the ends of these conidiophores; the heavy-walled multicellular chlamydospores formed in

club-shaped clusters under the surface of the host, causing the blackening that is so characteristic of the disease; and, the ascospores, which are found free within the simple perithecia, since the asci disappear as soon as the contained

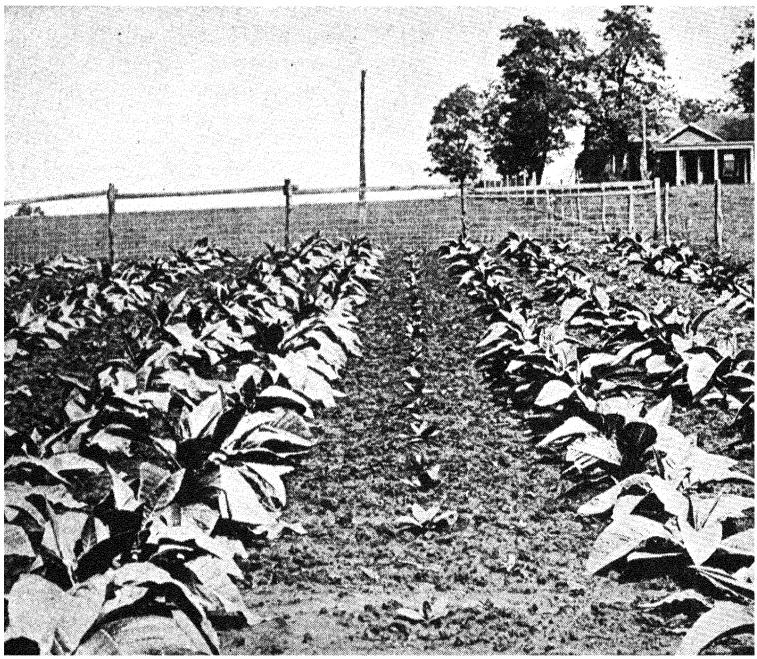


FIG. 147. Relative growth response of several resistant and susceptible strains of the tobacco variety, Burley, when planted in Thielavia-infested soil. (Courtesy of James Johnson.)

spores are mature. There is some doubt concerning the relationship of the perfect and imperfect stages. It has been suggested that the ascospore stage is not part of the life cycle of the root rot fungus, but that it is a parasite upon the mycelium bearing the conidia and chlamydospores. (See Fig. 146 for the host relation of the pathogen.)

Infection, penetration of the roots, does not occur easily in regions covered by an epidermis. Entrance is thus gained through wounds, at the origin of secondary roots, or at the tips of the root where there is still an uncutinized epidermal layer. The penetration process is unique in that a single

hypha rarely infects. The hyphae in contact with a cell seem to pause and mass themselves, secrete enzymes that reduce the suberized and lignified portions of the cell walls of the host to pectic substances and then enter the cell *en masse* after the swelling and softening of the pectic substances. This process is apparently repeated as each cell layer is encountered. The hyphae within the roots are intracellular and thus produce a rapid and complete death of the cells as they advance, mainly through the cortical tissues.

The fungus may live on dead organic matter in the soil in the absence of the tobacco plant or other hosts. It gradually dies out, however, although this may require from five to ten years or more. While the exact time required is not yet determined it may, perhaps, vary with the soil type and other conditions.

The organism spreads in much the same way as other organisms causing diseases of plants, although it is sometimes difficult to account for the first infected plants in a field. The more common means of spread are through the transference of infested soil, either by wind, water, farm animals or machinery, to non-infested soil; or by the transplanting of diseased plants from infested seedbeds into non-infested fields.

Slightly acid to alkaline soils favor the development of the pathogen. Soil moisture is not an important factor but soil temperature appears to have a predominating influence. The most favorable temperature range for the development of the disease is from 17° to 23° C. The fungus is checked in its pathogenic activities below 15° and above 26° C. At 32° C. the fungus does not infect the host, even in heavily infested soil. In a relatively warm season, with a soil temperature of 26° to 37° C., diseased plants may partly or wholly recover, while in cool seasons with soil temperatures of 15° to 24° C. the plants are severely injured. Often, however, when the early portion of the growing season is cool and the plants make little or no growth as compared to plants in neighboring non-infested soil, a week or two of very warm wet weather will start the crop into rapid recovery.

CONTROL.—Control of the Thielavia root rot has been achieved in various ways in different localities. In the Connecticut valley, where land is not easily available for rotation, resistant varieties have been developed. In the Burley district of Kentucky, control has been effected through long rotations. Recent trials have shown that the resistance to Thielavia root rot varies greatly among the standard types of tobacco. The White Burley, Oronoco, Payor and Maryland types are highly susceptible, while the Little Dutch and Connecticut Broad Leaf are resistant. From these resistant varieties a resistant strain of Burley has been developed. This new strain is of the leaf-drooping type as contrasted with the more commercially desirable stand-up type of Burley. This shortcoming of the new resistant strain is not enough to interfere with its general use on land that is infested with the Thielavia organism.

In those tobacco growing sections where the soil is free from Thielavia or in districts where the soil is already infested, where susceptible sorts are grown in preference to the resistant, seed treatment and seedbed sterilization need serious consideration in order that the organism may not be introduced into the field on transplanted seedlings. A mercuric chloride solution (1–500) is used as a seed disinfectant, while heat or chemicals are used for partial soil sterilization of the seedbeds. Recently dilute acetic acid has been used successfully for Thielavia root rot soil treatment. The possibility of carrying the pathogen from one farm or field to another on machinery, hoofs of horses and shoes of man should not be overlooked. The water supply of the seedbed should be well regulated, and all seedlings should be thoroughly inspected before being set out in non-infested soil.

ERGOT OF RYE

Claviceps purpurea (Fr.) Tul.

Ergot is a well-known disease of rye that is chiefly important because of the poisonous properties of the black sclerotia. It is this property that gives the disease its real

significance, since the actual reduction of the crop is never great, rarely exceeding five per cent. Flour made from infected rye is unfit for consumption and is liable to cause ergotism if it contains more than one-tenth of one per cent by weight of ergot sclerotia. With the improvement in milling processes, coupled with the increased use of wheat instead of rye bread, ergot poisoning has become less common in man. Ergot is important because of its use in medicine, where it finds extensive use in causing contraction of the blood-vascular system, preventing excessive bleeding.

As a plant disease, ergot of rye reduces the yield through replacement of the seed by sclerotia and by necrosis of the flowers. The estimated reduction in yield for the whole United States for the years from 1932 to 1934 inclusive was only 168,000 bushels, less than one-half of one per cent of the rye produced. It is only in case of a local epiphytotic that the loss in grain yield caused by ergot becomes an economic factor. The presence of sclerotia in grain requires special cleaning machinery, which may influence the price received by the grower.

Ergot also occurs on wheat, but it is usually less prevalent than on rye. It may, however, under certain conditions, become prevalent and destructive. In 1921 a severe epiphytotic of ergot occurred in the durum wheat growing sections of the northwest, where it was not uncommon to find fields with 50 per cent of the heads attacked. Wheat offered for sale showed 10 per cent ergot by weight. The Crosby Milling Company of Minneapolis reported that in 1922 their buyers were directed to purchase 20 cars of macaroni wheat, but they were able to find only eight that they considered safe for the manufacture of macaroni. Of these eight, seven had to be rejected at the mill because of ergot.

Symptoms.—The most conspicuous signs of ergot in the field are the presence of honey-dew oozing from certain of the flowers and later the large, slightly curved blue to black horny sclerotia. Blasting of the flowers as a result of necrosis of the very young pistil may also occur, but its occurrence is not so obvious. There develops in the flower a yellowish-

white mass of mycelium about the ovary, and there arises from the infected ovary a yellowish, sweetish and sticky honey-dew.

After the honey-dew has disappeared, the necrotic remains of the ovary are replaced with mycelium that becomes hard and horny, purple to black in color and several times the size of a normal grain of rye. This is the sclerotium, a long, curved body resembling in outline the normal kernels of the host upon which it develops. Often its surface is split or cracked, and at its tip it may have a small whitish brush.

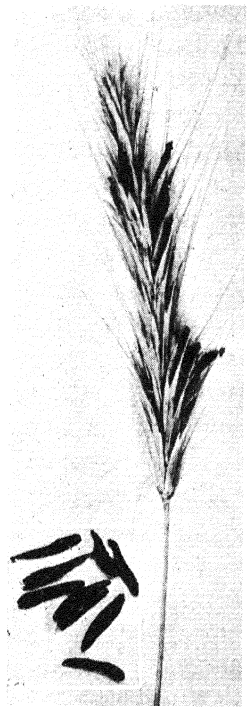


FIG. 148. Head of rye showing extrusion of ergot sclerotia; insert, ergot sclerotia.

The ergot fungus attacks only the ovary of the cereal spikelets. In many instances the ovary is destroyed in its initial stages of development, causing blasted flowers. It is held that frequently only about 10 per cent of the infections result in sclerotia, and the ergotized heads of rye merely become shortened or dwarfed.

ETIOLOGY.—The fungus that causes ergot, *Claviceps purpurea*, produces two spore stages in addition to the black sclerotia in its normal life history. The honey-dew, if examined microscopically, will show the presence of numerous oval, one-celled hyaline spores produced on the furrows and folds of the mycelial mass that replaced the ovary in the infected flower. These conidia are carried to other open flowers by insects, which are attracted by the honey-dew. The spores germinate on the stigma to produce infective hyphae, which grow down the outside of the ovary and penetrate at its base.

The horny mass of ergot mycelium that replaces the kernel

is made up of thick-walled resting hyphae and is known as a sclerotium. It passes the adverse period in the soil, and in the spring at about the time the flowers of the host open, the sclerotia develop the perfect stage. Sclerotia that are stored with the grain and sown in the spring may not always function.

Upon germination, the sclerotium produces several erect slender stalks about one-half inch long, each of which bears a small spherical head, the perithecial stroma. These heads are yellowish at first but become black after passing through shades of red. In the tissue of the head (stroma) the perithecia are formed as flask-shaped cavities opening to the surface. Within the perithecia are the asci containing the eight multiseptate thread-like ascospores, which are forcibly discharged into the air at maturity. The host relation of the pathogen is outlined in Fig. 149.

Infection of the host takes place only through the open flower. The flowering period is influenced by weather and other environmental conditions. Often not all the florets on a spike open at the same time, which extends the period of possible infection. Thin seeding with the possible attendance of excessive stooling brings some florets out earlier than others. Mixtures of early and late sorts also extend the opportunity for infection. It is only in the early stages of the development of the ovary that it is susceptible to infection. The ergot fungus is composed of at least five varieties, only two of which attack rye. The other three varieties are restricted largely to other cultivated or wild grasses.

Infection is dependent upon weather conditions that affect the flowering time of the host and the pathogen. Warm, dry weather, when rye is in flower, shortens the flowering period and is unfavorable to the development of ascospores and conidia of the fungus. Wet weather at this critical period in the life of the host lengthens the time the flowers are open and favors ascospore development. Such evidence as is available indicates that warm, wet weather is most favorable for the development of the disease in epiphytotic form.

The poisonous effect of the ergot sclerotia is due to an alkaloid, which produces convulsions and gangrene. The most important of these alkaloids has been variously named ergotinin, ergotoxin, cornutin and sphacelic acid. It has the properties of adrenalin, and the active principle is p-oxyphenylethylamin, $HO \cdot C_6H_4 \cdot CH_2 \cdot CH_2 \cdot NH_2$.

CONTROL.—In the control of ergot, it is imperative that susceptible grass hosts occurring in fence rows and along

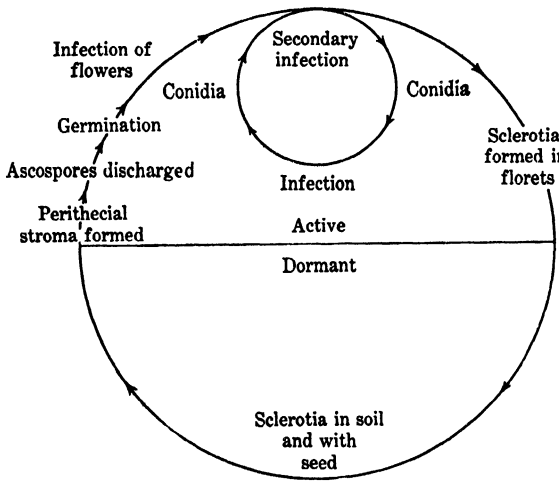


FIG. 149. A diagram of the host relation of *Claviceps purpurea*. This organism survives the winter as sclerotia in the soil or with the seed.

roadsides be destroyed before the plants blossom. Rotation should prove beneficial where a previous crop was infected. When possible, it is advisable to select seed free from ergot sclerotia. If this cannot be obtained, then it is well to remove the sclerotia by sedimentation. This process consists of placing the ergotized grain in a 30 to 32 per cent solution of common salt and thoroughly stirring the same. The ergot bodies, having a lower specific gravity than the grain, rise to the surface and may be skimmed off. Following such treatment the grain should be washed and dried before it is sown. A potassium chloride solution (32 to 37 per cent) also has been used for floating out the sclerotia. It is held that the potassium chloride is less injurious to the seed.

Mixed grains and those broadcasted rather than drilled tend to favor the ergot organism. The increased length of the blossoming period in the mixed grain and grain that is broadcast affords a longer period for infection. After a field has grown an ergot-infected crop, it should be plowed deeply to prevent the sclerotia from functioning. Meadow grasses in pastures where ergot is known to occur should be mowed before blossoming or intensively grazed so as to prevent the formation of sclerotia.

Alfalfa Leaf Spot

Pseudopeziza medicaginis (Lib.) Sacc.

Alfalfa is an important hay crop in many parts of the world. It is a native of Asia where it was grown before the Christian era. Alfalfa leaf spot has been known on this crop for a long time and seems to be world-wide in its distribution. The pathogen does not, as a rule, kill the plants suddenly, but tends to defoliate them, thus decreasing the hay value and weakening the plants.

The leaf spot organism probably is most destructive on new seedlings, which may become yellow and lose many of their leaves, predisposing the young stand to drought injury and winter killing. Since the alfalfa leaf spot organism seldom causes serious losses at one time, it has come to be regarded as one of the unavoidable diseases of the crop. In the central United States the second and third crops are often damaged more than the first.

Alfalfa is the host of a number of organisms that produce a local necrosis or leaf spot type of disease. The leaf spot caused by Pseudopeziza may be differentiated from the others by the small, circular, black spots with their irregular margin and the raised black disc in the center of the spot.

Symptoms.—On the leaves the spots vary from mere specks to necrotic areas half a centimeter in diameter. While visible on both surfaces, except when very young, the spots are most conspicuous on the upper surface. The spot is brown or black in color and irregularly circular in outline

with no definite margin. Usually, although not invariably, the spot is surrounded by a yellow margin, which is most evident between several adjacent spots. When the leaf is seriously affected, it turns yellow, dies and drops from the stem, and in severe cases the plant is entirely devoid of leaves, except for a few at the top. A single spot never involves the entire leaf, but several may cause it to turn yellow and fall.

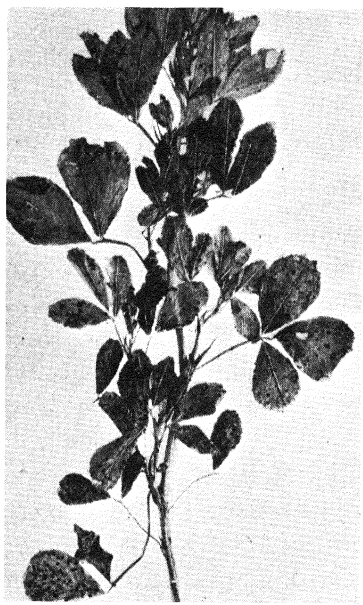


FIG. 150. Irregular brown spots on alfalfa leaves infected by *Pseudopeziza medicaginis*.

An examination of the older spots with a hand lens reveals a small, dark, shiny elevation, which may or may not be split at the top to form a minute pustule or crater-like opening. The torn epidermis forms the border of the pustule, which is the fruit body of the pathogen and is usually found on the upper side of the leaf. On the alfalfa stems the fungus produces necrotic areas similar to those on the leaves. The pedicels and calyces of the flowers also may be attacked, but apparently the seed escapes.

ETIOLOGY.—Alfalfa leaf spot is caused by *Pseudopeziza medicaginis*. Its sequence of development is shown diagrammatically in Fig. 151. As far as known this pathogen does not have a conidial stage, while the ascospore stage occurs throughout most of the growing season of the host. The mycelium spreads intercellularly or intracellularly in the tissues and induces local necrosis of the leaf and stem tissues.

In this dead tissue the pathogen forms a dense mat of mycelium called a stroma, on the surface of which

arises a single tiny brown pustule bearing a palisade of asci and paraphyses. The ascospores are forcefully discharged, as already described in the brown rot organism, and germinate immediately, sending a germ tube directly through the cuticle into the epidermal cells. Later, the palisade layer is invaded. The entrance of this pathogen into the host is not dependent on the presence of natural openings.

The ascospores seem to be rather resistant to cold and dry air, according to laboratory tests. It is probable that the

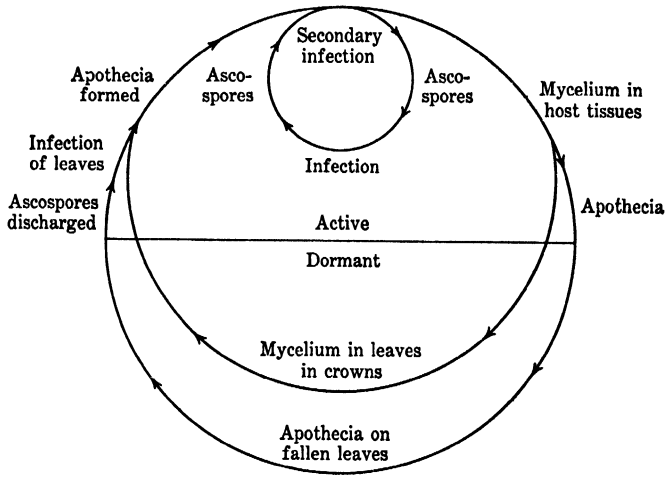


FIG. 151. A diagram of the host relation of *Pseudopeziza medicaginis*. This organism survives the winter in the apothecial stage on the fallen leaves or as mycelium in the crowns.

pathogen passes the winter in the mycelial and ascospore stages on the fallen leaves or on leaf refuse in alfalfa seed. It also is likely that the pathogen hibernates in the infected leaves on the young shoots in the crown of the host. There is little or no evidence that the pathogen lives in the seed, although it is not improbable that the sticky spores may adhere to the seed. When the pathogen is present the disease may develop at any time during the growing season regardless of weather. Cultural conditions that produce early shading of the ground probably create a favorable environment for

the germination of the spores of the pathogen and infection of the host.

CONTROL.—At present there are no effective control measures for the alfalfa leaf spot pathogen. Seed treatment and

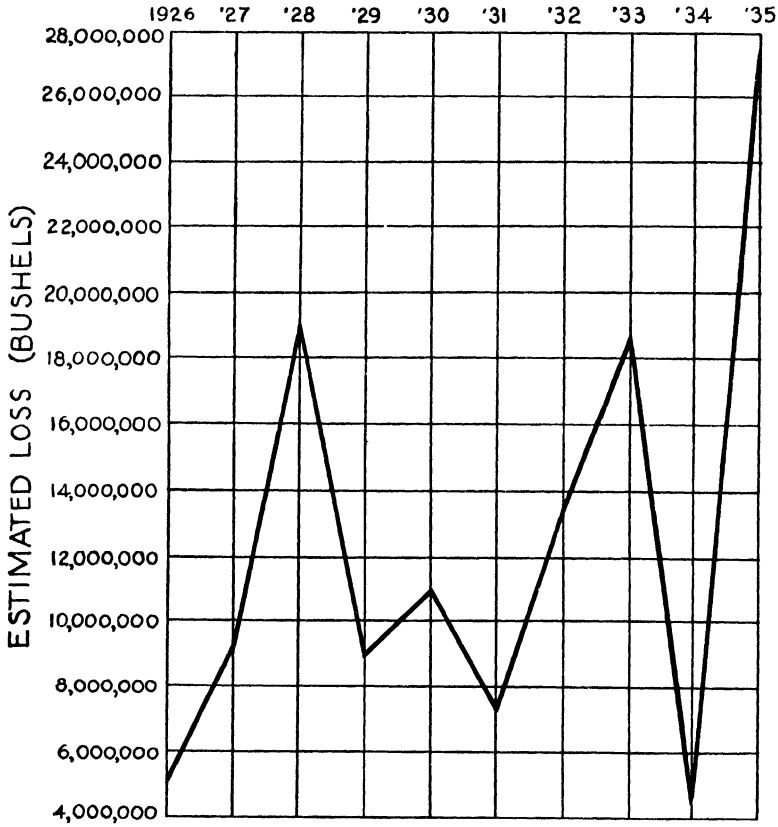


FIG. 152. Estimated loss of apples caused by the apple scab organism in the United States (reporting area) 1926 to 1935, inclusive. (After N. E. Stevens and Jessie I. Wood.)

rotation offer little hope because the organism is too universally distributed and because the spores are readily disseminated by the wind. No resistant strains are known. The only way that losses from this disease can be alleviated is to harvest the crop before the lower leaves are killed. Not

only does this maintain the hay value, but it also removes much of the inoculum from the field.

Apple Scab

Venturia inaequalis (Cooke) Winter

Apple scab has been known since early in the nineteenth century. Schweinitz, one of the first collectors of fungi in the United States, found it in New York and Pennsylvania. This is a common disease of the apple wherever grown, though the injury may vary considerably in different localities and especially during different seasons. The organism is probably most injurious in the Mississippi valley region and in the northeastern and northwestern United States. There are apparently no immune varieties, although there is great difference in susceptibility, which may be modified by seasonal variations. Many of the different species of apple are also hosts of the apple scab organism; but the scab on pears, cherries, etc., is caused by different species of *Venturia*.



Fig. 153. Dark, olivaceous, velvety colonies of *Venturia inaequalis* on apple leaf showing the fimbriate margins.

Symptoms.—The fungus commonly affects leaves, flowers, fruit and twigs. On the leaves, small circular brownish or gray spots develop on the surface, finally becoming olive green. The attacked tissues appear to swell and bulge upward;

finally death and necrosis of the tissues result. The mycelium forms dense mats just below the cuticular layer, from which it radiates in dendritic branching fashion. A stroma forms from which arise numerous olive-brown conidiophores bearing spores. The torn cuticle rolls back, forming a light border about the area involved by the scabbed portion of the leaf.

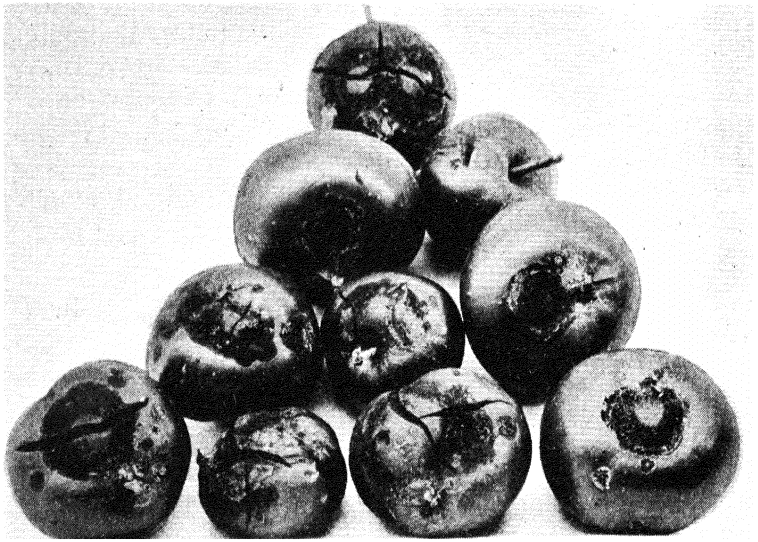


FIG. 154. Large fruit lesions caused by the apple scab organism. Note the cracked and velvety appearance of the lesions.

Under conditions particularly favorable for the growth of the pathogen, its activity may not be limited to a small spot. The brown mycelium may grow over the under surface of a considerable area of the leaf. In such cases the leaves are reduced in size, curled or killed quickly, depending upon the activity of the pathogen. When such spots are numerous, the leaf turns yellow and drops. It requires from 8 to 15 days for infection to become evident after the spore comes in contact with the under surface of the leaf.

Flower bud leaves frequently show infection first. This infection probably is initiated when the flower buds begin to show pink. Lesions similar to those described on the leaf

may develop on the sepals, petals, pedicels and young ovary. When such lesions develop on the pedicels the flowers are killed, resulting in a light set of fruit. This type of injury is sometimes wrongly attributed to frost or cold rain.

The fruit may be attacked throughout its whole period of development. When infection takes place very early, the fruit is either deformed or much reduced in size, and later infections stunt and disfigure the fruit. Where many infection centers are initiated on the apple, they may coalesce and form a solid crust of scab lesions. As on the leaf, the pathogen

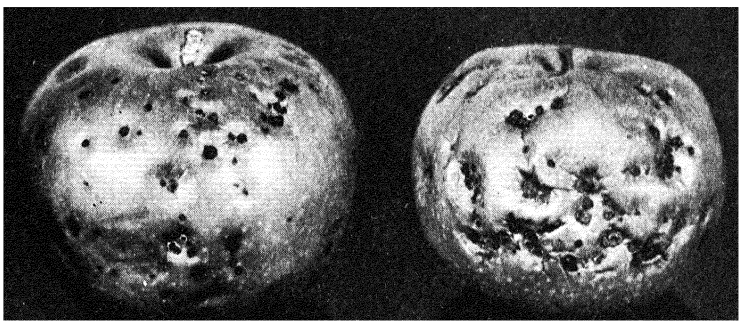


FIG. 155. Apple scab showing the dry rot which may develop adjoining pin-point lesions when the fruit is in storage. (Courtesy of H. H. Plagge.)

forms a dense stroma just below the cuticular layer from which a forest of olive-brown conidiophores bearing spores arises. The fruit also may become infected during the harvesting period and the lesions fail to manifest themselves until the fruit is in storage. In such cases, the lesions are small, pinpoint-like and darker brown, and the pathogen seldom fruits on these lesions.

Infection also may occur on the stem, but it is only the one-year-old wood that is susceptible. Light brown, oval to elongate cankerous areas develop in the bark. The pathogen seldom overwinters on these lesions. Therefore, this type of infection has little to do with initiating primary infection in the spring.

ETIOLOGY.—Apple scab is caused by *Venturia inaequalis*, a fungus that has two spore stages—a conidial stage and a per-

fect or ascospore stage. The host relation is shown diagrammatically in Fig. 156. The ascospores are two-celled and olive-brown. When these are ripe and discharged, they germinate immediately, sending a germ tube into the cuticle to the outer wall of the epidermal cells. Once in the cuticle the pathogen extends radially, raising the cuticular layer. Gradually the mycelium accumulates in a localized area, from which arise the short, olive-brown conidiophores bearing the

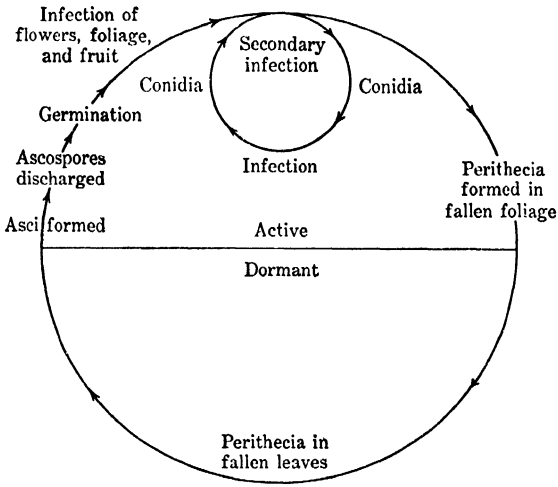


FIG. 156. A diagram of the host relation of *Venturia inaequalis*. The conidial stage is very effective in causing infection during the active stage of the host. The ascospores function in renewing infection in the spring.

one-celled conidia. In this way the ascospores initiate primary infection on the apple, giving rise to the conidial stage, which is responsible for secondary infection. The subcuticular mycelium produces toxic substances that cause the disintegration of the mesophyll cells immediately below it. It is only following this toxic action that the mycelium penetrates between the epidermal cells and into the palisade layer. The nearly pear-shaped conidia, after being disseminated by meteoric water, germinate by a germ tube, which penetrates the cuticle directly to produce a subcuticular mycelium similar to that of the ascosporic infection.

It requires from 8 to 15 days for the conidia to germinate,

infect the young leaves and produce another crop of spores. This cycle may be repeated many times during the summer season. When the infected leaves fall to the ground in the fall, the mycelium begins its saprophytic existence. In the stroma formed in the mesophyll of the dead leaf there is initiated in the fall the ascospore stage. The spherical, short-necked, dark-brown perithecia break through the

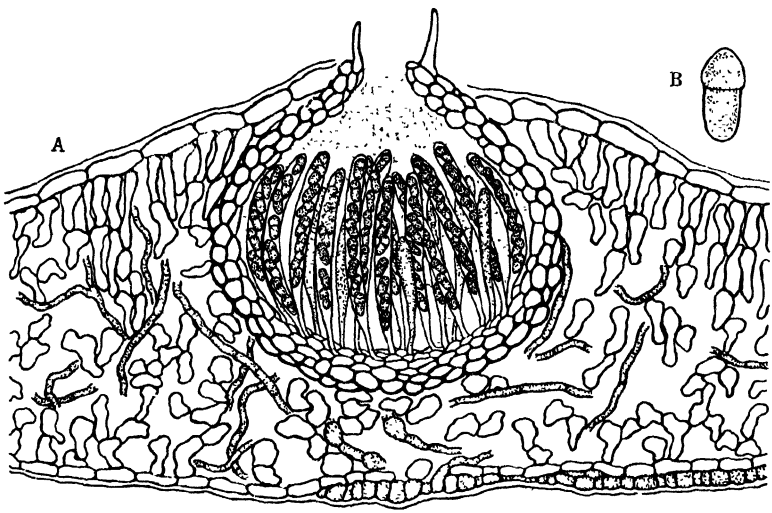


FIG. 157. A, cross section of perithecium of *Venturia inaequalis* in overwintered apple leaf; B, ascospore.

upper surface of the leaf. Asci do not form in the perithecia until early spring, and when the asci are ripe, they protrude through the ostiole or mouth of the neck, burst and forcefully discharge the ascospores. The perithecia mature at about the same time that the flowers show pink, although this may vary somewhat from one season to another. When the ascospores are discharged earlier, a serious epiphytotic of apple scab is more liable to develop.

The ascospores are not all discharged at once. The time of discharge varies in different sections, but as a rule they are discharged during April, May and June. The lower leaves of the tree show more primary infection than the upper.

The discharge of ascospores is influenced by moisture and temperature, a temperature range of 0.5° to 30° C. is favorable; while low temperatures, 10° to 12° C., combined with rain, are optimum.

CONTROL.—The control of apple scab involves sanitation and the application of a fungicide for the prevention of in-

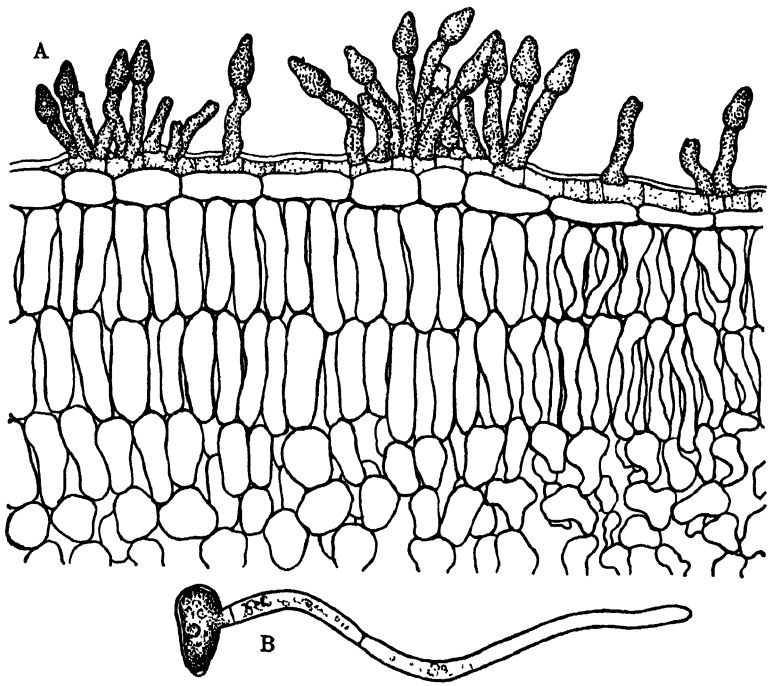


FIG. 158. A, cross section of young apple scab lesion. The subcuticular mycelium above living mesophyll produces conidia from the erect conidiophores, while conidiophores from mycelium overlying dead mesophyll cells seldom form conidia; B, germinating conidium.

fection. Cultural methods that prevent the pathogen from developing in the orchard are of first importance in combating apple scab. Dense foliage, as develops where trees are not properly pruned, favors dissemination of the scab organism. The infected leaves on the ground should either be raked up and burned or plowed under, thereby removing the source of primary infection. It is important, in locating an orchard,

to avoid planting on low ground, or in pockets where the circulation of air is poor. It is also important to plant, as far as practical, those sorts that are most resistant in a given district. Orchards with many varieties planted promiscuously aggravate the scab problem, and at the same time increase the expense and decrease the effectiveness of spraying.

Spraying is the most important means of controlling apple scab. In general, any adequate spray or dusting schedule is so timed as to prevent infection of the host by killing the spores before or during germination. For this purpose one may use either a wet or dry fungicide. To date the wet fungicides, namely, lime sulphur and Bordeaux mixture, have proved most effective; lime sulphur occupies first place since it causes less burning of the fruit and foliage during cooler weather than Bordeaux mixture. Success in spraying for this pathogen involves the protection of all exposed surfaces of the leaves and fruit. The fungicide must be on the foliage before the spores arrive. Dusting is gradually growing in popularity and appears to be effective when the epiphytotics are not too severe. Spray and dusting schedules vary in different states and countries because of the difference in orchard practices and the influence of climate and weather on the pathogen.

Powdery Mildew of the Grape

Uncinula necator (Schw.) Burr.

The powdery mildew of the grape probably is indigenous to the Orient. It first came into prominence in 1845 when it occurred on the grape in England. In 1847 it was found in France and during the next decade it spread throughout the grape growing districts of the Continent. Among plant pathogens its spread was without a parallel, and it seriously threatened the grape growing industry. The advent of this pathogen and the losses it occasioned were sufficient to arouse governments to appoint special commissions for its investigation. While the powdery mildew of the grape is world-wide in its distribution, it is probably most destructive in Europe.

Before remedial measures were worked out, it became so severe in some sections of France that many grape growers turned to other pursuits. In America it is most destructive in New York, California and Oregon, although it frequently becomes destructive in less intensive grape growing sections.

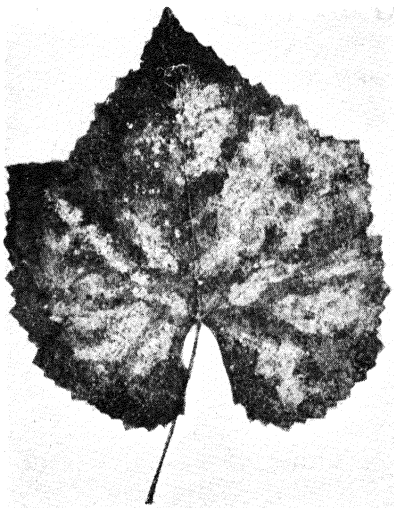


FIG. 159. Powdery mildew on a grape leaf showing the grayish mycelium and spores of the pathogen.

SYMPTOMS.—The fungus that causes the powdery mildew of grapes, *Uncinula necator*, attacks all above-ground parts, showing first as small whitish patches on the upper or lower surface of the leaf. As the organism spreads, the different centers of infection become discolored, giving the leaves a powdery-white appearance. The tissues of the leaves become puckered and discolored. Frequently the white surface is sprinkled with small black dots, the perithecia of the pathogen. In later stages the infected tissue turns brown and dies. On the young vine, the pathogen does not limit its activity to the leaves, but attacks the canes, causing them to become dwarfed and discolored. The flowers and fruits also may be attacked, causing the fruit either to drop prematurely or to become malformed and small. Badly infected vines become much stunted; the old foliage is shed and small new leaves are developed.

ETIOLOGY.—The powdery mildew of grape is caused by the fungus *Uncinula necator*, the host relation of which is shown diagrammatically in Fig. 160. The white patches that occur on the leaves in the spring are the result of growth of mycelia from the germ tubes of ascospores. This mycelium consists of a number of branching, sparsely septate hyphae,

which cover the surface of the leaf and form appressoria at the points of contact of the lobed, lateral, swollen hyphae with the cuticle. From the appressoria arise haustorial branches that penetrate the epidermal cells and become swollen balloon-like sacs, which absorb food directly from the protoplasm of the epidermal cells.

On this mycelium shortly after it starts its growth there arise numerous erect, slightly elongated conidiophores, consisting of a basal cell arising directly from one of the

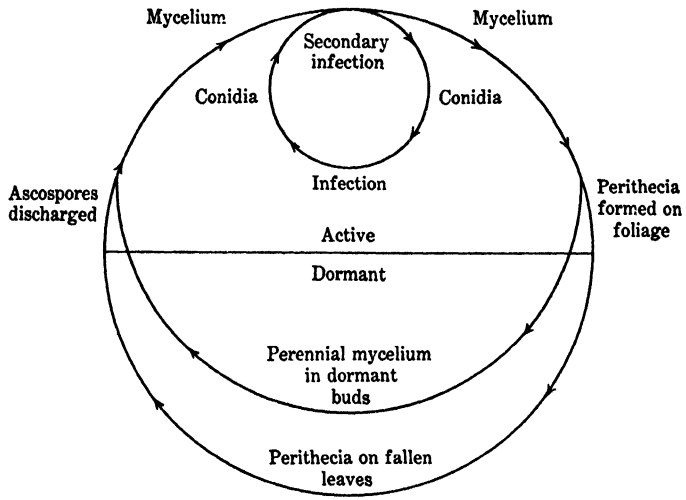


FIG. 160. A diagram of the host relation of *Uncinula necator*. This organism survives the winter in the ascospore stage in perithecia on fallen leaves or as perennial mycelium in dormant buds.

hyphal strands and a chain of immature conidia surmounted by one to five mature conidia. These barrel-shaped conidia are said to be able to overwinter although this is doubtful. The germination of the conidia is very similar to that of the ascospores, resulting directly in the establishment of a new mycelial colony.

Later in the season there appear on the mycelium small, yellowish, spherical structures that, on enlarging and turning black, produce the typical mature perithecia, which often are numerous and easily visible on the surface of a diseased

leaf. They are characterized by the determinate, terminally coiled appendages, the length of which is four to five times the diameter of the perithecium. Within each perithecium are formed, generally after a resting period, four to eight asci, each of which may have four to eight oval, hyaline, single-celled ascospores. The perithecia generally remain on the canes, leaves, or in the soil during unfavorable conditions and renew the organism on the susceptible hosts in the spring. The mycelium also lives over within the young canes and buds, which suffer from a late infection.

Uncinula necator, although not requiring drops of moisture for the germination of its spores, is favored by a moist atmosphere. The temperature conditions favorable for rapid development are 21° to 33° C., with a complete range of 10° to 38° C. A sudden drop to about 10° C. after a warm, moist period generally is considered as especially favorable to the formation of the overwintering stage, the perithecia.

CONTROL.—In combating the powdery mildew of grapes, the vineyard should be kept in a good state of tilth. Clean culture, coupled with pruning and training practices that facilitate rapid evaporation of moisture, tends to retard the development of the pathogen.

Dusting with sulphur is effective under most conditions, being most effective when applied during temperature ranges between 24° and 37° C. Below 24° C. the sulphur fails to volatilize rapidly enough to be sufficiently effective. Above 37° C. some burning is liable to result unless lime is mixed with it. Dusting is not practical when the vines are wet. The first application should be made when the young shoots are about six inches long; the second application must be made before blooming; and subsequent applications depend upon the severity of development of the pathogen.

There seems to be considerable difference in the susceptibility of different varieties. The European varieties are held to be more susceptible than the American. In sections where this disease is a limiting factor in grape growing, this point might well be taken into consideration, utilizing varieties recommended as resistant by the state pathologists.

Powdery Mildew of Barley

Erysiphe graminis var. *hordei* Marchal

This obligate parasite, known since 1815, is common on barley and many other grasses. Although the organism is seldom reported as becoming epiphytotic, it may produce a great deal of damage when favorable environmental conditions allow widespread infection early in the season.

It has been known since 1902 that the powdery mildew of grasses was composed of at least seven varieties, each limited to hosts in a certain genus, as wheat, barley, oats, rye, etc. The symptoms and etiology of all the seven varieties are similar. The variety on barley, known as *Erysiphe graminis hordei*, has been chosen for detailed study because of its general prevalence throughout the United States and the ease with which it develops in the greenhouse.

Symptoms.—The signs of this disease are much more evident and characteristic than the symptoms. Although under the conditions most favorable to infection the stems and glumes may be infected, the fungous attack is generally limited to the leaves. The pathogen grows in numerous colonies on the upper and lower surfaces of the leaves. These colonies appear first as small, more or less circular, white, felty masses of mycelium and spores. Since the pathogen grows largely on the surface, the flour-like mass may be scraped off, revealing below a nearly normal area of leaf tissue, which appears to have been only slightly injured. As the mycelium ages or undergoes adverse conditions, the hyphae and spores become ochre or brown and form a thick cushion-like growth. The leaves show a slight yellowing, followed by the appearance of a reddened zone around the colonies. The mildew causes the host to transpire more readily than the normal plant. Infected plants transpire 67 per cent more water from equal leaf areas than plants not infected. The comparative dry weight of healthy and diseased plants is 100 and 141 respectively. Infected plants become stunted through a reduction in the size and number of leaves, and delayed necrosis, and in

severe cases the host is defoliated and the crop seriously injured, as a result of the reduction of food reserves.



FIG. 161. Lesions and discoloration caused by older colonies of mildew on barley leaves.

Infection takes place when the young leaves are exposed to conidia under cool, moist conditions. The heavily infected leaves die after 14 to 20 days. These leaves dry up and the younger leaves are successively infected. This continued destruction of the food-making portion of the plant leads to a premature heading and the formation of small, shriveled grains. The leaves of these plants are thinner than normal, while the plants suffer a decrease in the total fresh and dry weights. In cases of severe infection the host evidences the damage in the form of yellowish lesions, which may unite to cause large areas of yellowing, followed by necrosis. The mycelium is stopped in its development with the death of the host cells. In nearly "immune" varieties the only evidence of mildew is the small "flecks" typical of rust infection on highly resistant hosts.

The infection on the glumes, at least, often is followed by secondary or-

ganisms, as *Cladosporium herbarum* Pers., which produces a blackening or other type of variation in the true symptoms or signs. Stem infections, if severe, often weaken the plant, resulting in lodging.

ETIOLOGY.—This disease is caused by the fungus *Erysiphe graminis hordei*. (See Fig. 162.) The profuse external my-

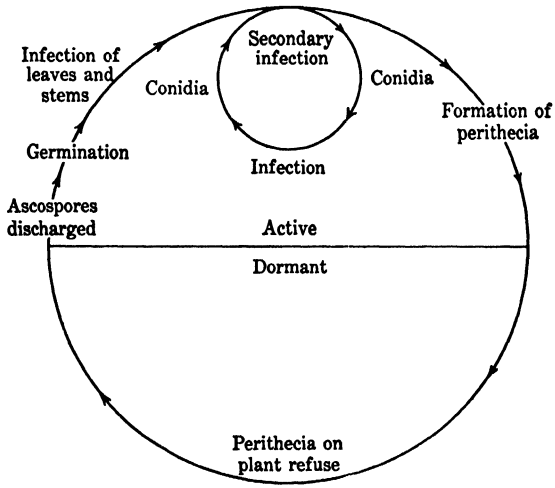


FIG. 162. A diagram of the host relation of *Erysiphe graminis* var. *hordei*. This organism survives the winter in the perithecia on plant refuse. The perithecia mature and discharge the asci in the spring.

celium produces haustoria from small, flat appressoria developed on the hyphae. The haustoria enter the epidermal cells of the host, where they produce characteristic finger-like projections extending in the longitudinal direction of the host cell.

The conidiophores are produced from the hyphal cells of the external mycelium. They consist of a swollen basal cell, a sporogenous cell, two to four developing conidia and a terminal barrel-shaped mature conidium, which, after being forcibly discharged, becomes elliptical to spindle-shaped. These conidia germinate by the production of several germ tubes, which vary in length, depending on environmental conditions, although they are seldom more than ten times the

diameter of the conidium. The conidia rapidly lose their viability after they are free from the conidiophores. A temperature of 12° C. appears to be the optimum for the conidial germination, while 21° C. is the optimum for the growth of the resultant mycelium. The germ tubes touch the

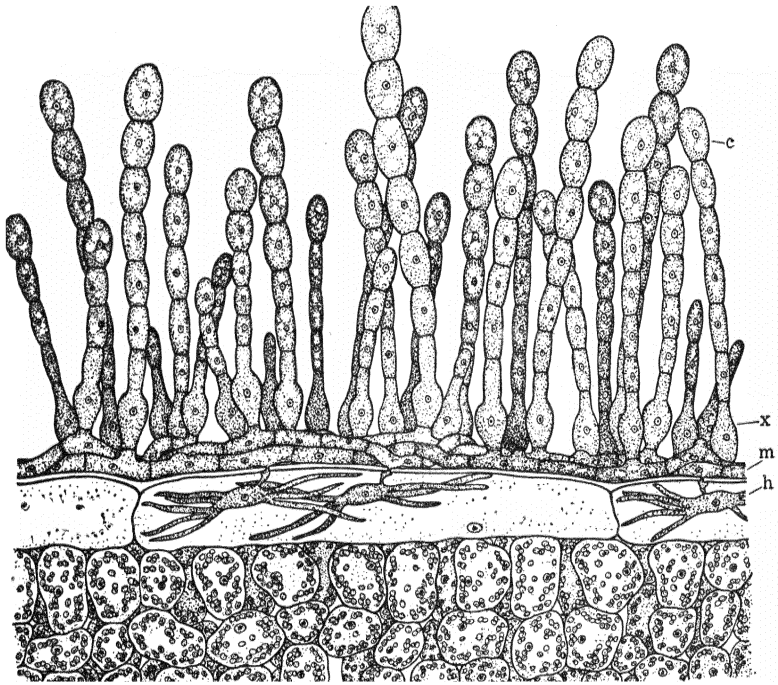


FIG. 163. Partial cross section of barley leaf supporting a colony of *Erysiphe graminis* var. *hordei;* hypha, m; haustorium, h; conidiophore, x; conidium, c.

cuticle where they form appressoria from which small penetration tubes enter the epidermal cells, without regard to natural openings. From these penetration tubes may be formed the first digitate haustoria, which may fill the lumen of the epidermal cells. From the germ tube, outside the host, arise the hyphae that form the external mycelium. This newly formed mycelium may form conidiophores and conidia within two to three days.

Perithecia, which are produced after the flowering of the

host, are sexually formed, spherical structures, with typically hyphal-like appendages, that usually are completely covered with the sterile setae. The ascospores, however, are not developed until a rest period has elapsed and even then they are not produced in great abundance. These sexual spores have been shown to develop when the perithecia are exposed to a moist atmosphere and alternating temperatures. When the ascospores are formed, each of the 8 to 25 asci within each perithecium produces four to eight elliptical ascospores. Germination of the ascospores is little known, except that generally the tubes never extend over twice the diameter of the spore. The ascospores die within 30 hours unless they come in contact with the epidermis of the host when appressoria, haustoria, mycelia, etc., are produced as in the case of conidial infection.

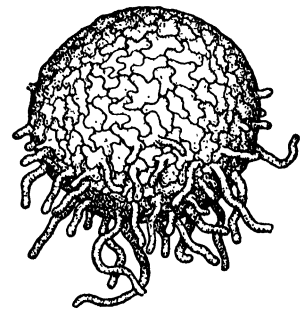


FIG. 164. Perithecium of *Erysiphe graminis* var. *hordei* showing wall structure and nature of appendages.

The species *Erysiphe graminis* exhibits a marked host specialization, having at least seven varieties as follows: *Erysiphe graminis hordei*, *E. graminis secalis*, *E. graminis tritici*, *E. graminis avenae*, *E. graminis bromi*, *E. graminis poae* and *E. graminis agropyri*. These varieties are constant as to host range unless the host plants are grown under abnormal conditions or wounded. Still further each variety may show constant variations in its pathogenicity permitting further segregation into races. Five are known to occur in the variety on barley.

CONTROL.—Since the powdery mildew of barley is favored by thick seeding and high humidity, these conditions should be avoided in cultural practices. Sulphur applied as a dust is an effective fungicide, but the value of the crop and the cost of treatment makes sulphur dusting impractical. There are differences in varietal susceptibility, which may be used to the grower's advantage in regions where the mildew becomes destructive.

Peach Leaf Curl

Taphrina deformans (Fckl.) Tul.

Peach leaf curl has been known in England since 1821. Its origin is not definitely known, but the organism may have found its way into Europe from central China, the native home of the peach. The pathogen probably was introduced into the United States in the early Eighties of the last century. It has become destructive wherever peaches are grown, and is limited to the peach and to forms derived from it, as the nectarine and peach almond. The peach is probably the natural host of the organism, which attacks orchard trees and nursery stock alike, and is very destructive under favorable conditions, frequently causing complete defoliation.

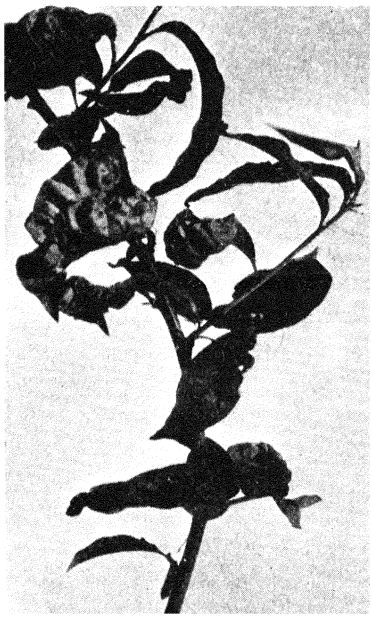


Fig. 165. Peach leaf curl showing blistered and curled areas covered by asci of the causal agent, *Taphrina deformans*.

Before remedial measures for the peach leaf curl organism were devised, peach orchards frequently suffered serious damage; e.g., in 1893 the damage in the United States was estimated at $3,000,000. Since remedial measures have been developed, the annual loss has decreased materially, although even now in some sections the losses are serious, usually because of faulty or neglected control practices.

Symptoms.—The disease shows first on the young leaves as they come out of the bud. The first evidence is an arching and reddening of affected spots in the young unfolded leaves as they begin to protrude from the scales. Lesions may be

confined to a part of the blade or the petiole, or the entire leaf may be involved. The portions are thick and become brittle and pale yellowish, often tinged with red; finally a silvery bloom covers the upper surfaces. This distortion of the leaves is due to the growth and action of the pathogen between the cells. Since the host cells, which are caused to enlarge and multiply, are located chiefly in the parenchyma tissues, a one-sided development of the leaf blade is produced.

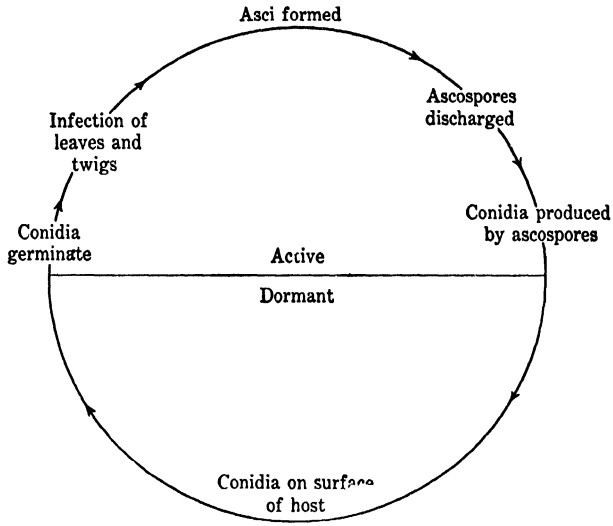


FIG 166. A diagram of the host relation of *Taphrina deformans*. This organism may survive the winter in the conidial stage on the surface of the host.

The chlorophyl in the cells of the infected area disappears, and the infected leaves finally die and drop from the tree in June or early July. The tree in turn develops a new set of leaves from the dormant buds. This abnormal development drains the strength of the tree, resulting in its failure to set fruit or to make other satisfactory growth.

The pathogen may attack the twigs, causing marked swellings and yellowing of the rapidly growing shoots. Although as a rule only the current year's wood is involved, the pathogen may grow down into the previous year's wood. This excessive growth is evidenced by swollen canker-like lesions

often accompanied by a gummy exudate. The flowers and young fruit also may be invaded by the pathogen, which causes them to drop early. The more mature fruit may be attacked, resulting in swollen, irregular, wrinkled areas, bright in color.

ETIOLOGY.—Peach leaf curl is caused by the fungus *Taphrina deformans*. Its host relation is shown diagrammatically in Fig. 166. The mycelium is of three types: (1) vegetative hyphae, occurring intercellularly in the parenchyma of the leaf, and consisting of cells of irregular size and shape; (2) distributive hyphae, developing just below the epidermis of infected parts, with long cells irregular in diameter which may run parallel between the host cells, and whose function is thought to be that of spreading the pathogen; and (3) fruiting hyphae, differing little from the vegetative hyphae except that they bear the stalk cells at the tip of which the asci are borne.

As the asci enlarge, the cuticle on the upper surface of the infected leaf is raised and separated from the epidermal wall by the crowded erect asci. This separation of the cuticle produces the silvery appearance of the infected leaf. When the cuticle ruptures it is curled back in a number of sections, which give the lesion a frayed edge. As each ascus matures, its contents become divided into eight spores, which are forcefully discharged upon the rupturing of the apical end of the ascus. In this stage of the development of the pathogen, the upper surface of the leaf takes on a glistening-white appearance.

The thin-walled ascospores in turn may produce conidia by a budding process or form a thick germ tube. Since no one has been able to induce infection with the ascospores, their method of functioning in initiating infection is not definitely known. The ascospores produce the conidia that persist on the twigs and leaf scales until the next spring when they enter the forming leaf, flower, fruit or twig by a germ tube that penetrates directly.

The development of the pathogen is dependent upon wet, cool weather, when the leaves are unfolding from the bud. A

high temperature and dry atmosphere check the development of *Taphrina deformans*. The conidia are known to be killed by short exposures at 30° C. but are rather tolerant of dry air. It is known that they will tolerate drying on a cover slip for 315 days.

CONTROL.—The control is readily effected by applying one of the following fungicides: Lime sulphur, Bordeaux mixture or miscible sulphur. The most practical spray is one application of lime sulphur, diluted one gallon in eight of water. The spray may be applied in the fall, winter or early spring. It also is beneficial for insect control. The important consideration is to make sure that it is applied before the buds begin to swell in the spring. One thorough application covering every bud is sufficient; late applications are less effective.

GRAPHIUM WILT OF ELM

Ceratostomella ulmi (Schwartz) Buisman

A serious new wilt of elm known as Graphium wilt was discovered in Holland, Belgium and France in 1918. This wilt also is known as the Dutch Elm Disease, because it was first described from Holland. The origin of the causal agent is not known, although various theories have been advanced. Since its discovery the causal agent has been reported in the United States, England and throughout Continental Europe as far east as Poland and Rumania. Although not definitely known to be in the Orient, the fact that resistance is found only in the Asiatic elms lends weight to the theory that the organism came from China. The pathogen occurs on all species of elm commonly planted and is apparently limited in its parasitism to species of *Ulmus* and the related genus, *Zelkova*. The damage is greatest to ornamental trees, which makes it very difficult to appraise the financial loss.

Graphium wilt was first recognized in the United States in Cincinnati and Cleveland in 1930 on a few trees. At present the majority of the infected trees occur within a 60-mile radius of New York City. A few isolated infected

trees have been found in Indiana, Virginia and Maryland. The disease does not seem to be general in this country, although 28,500 infected trees were located previous to 1939 in the eradication campaign now in effect against the Graphium wilt pathogen. In certain areas of Europe 90 per cent of the trees in a single locality have been removed.

SYMPTOMS.—The typical symptom of the disease is the rapid wilting and curling of leaves at the tips of twigs.



FIG. 167. The two trees at the right of the central group of elms have been killed by the Graphium wilt fungus. (Reproduced from U. S. D. A. Circ. No. 322.)

This early symptom usually appears from the first of July through August. The wilt may be gradual and be accompanied by yellowing and finally browning of the leaves. On the other hand, the wilting may be so rapid that the leaves curl and dry while still green. Accompanying the wilting is a browning and marked curling of the tips of the twigs, and the ends of the twigs may droop.

Shortly after the wilting and drying of leaves there may result an early defoliation of an entire branch or the whole

tree. In a small tree all the leaves may fall in a week to ten days. The leaves near the tips of the twigs may, however, remain hanging. The entire tree may be so affected that it dies quickly, or the infection may be limited to one branch and the tree may survive for several years. In the latter

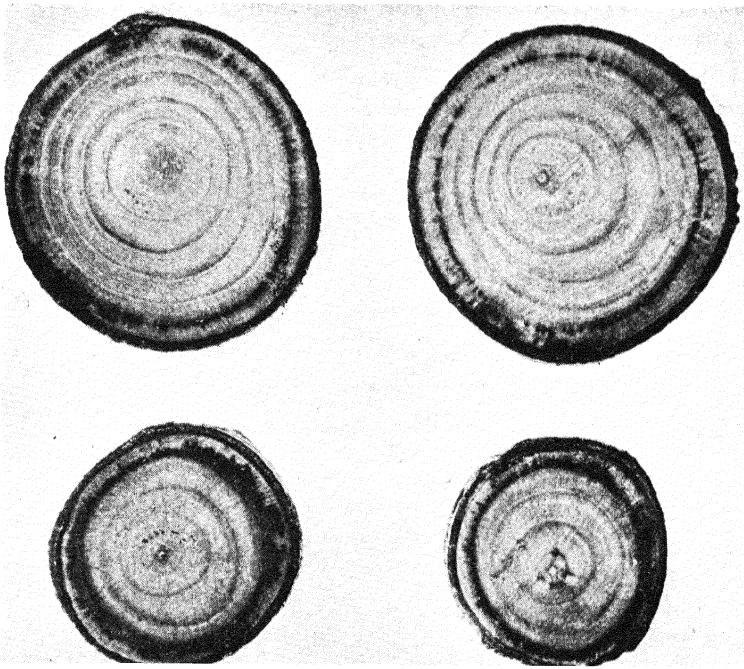


FIG. 168. Discoloration of the sapwood caused by the Graphium wilt organism in cross sections of branches of the American elm.

case the new leaves that come out in the spring following infection will be small, curled, yellowish and delayed.

These symptoms are those of nearly any vascular or limb disease of the elm. Similarly the internal appearance of a limb suffering from the Graphium wilt is much the same as any other wilt of elms. The limbs or trunks, when observed in an oblique section, show a brownish ring or spots in the xylem. In a longitudinal section of the wood these discolored areas show as long brownish streaks. If the bark is

peeled off an infected tree the cambium shows long, large brownish stripes where the fungus has destroyed the cambial cells.

Since the symptoms, internal and external, are so common in other diseases, actual cultural investigation is necessary to definitely establish the cause of the disease. The wilts of elm caused by *Verticillium* spp. and *Cephalosporium* spp. exhibit nearly identical symptoms.

Etiology.—The organism that causes Graphium wilt is *Ceratostomella ulmi*, and the prevailing imperfect stage is

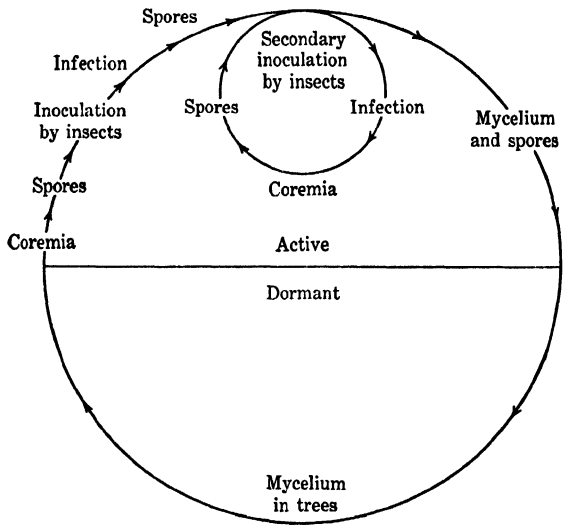


Fig. 169. A diagram of the host relation of *Ceratostomella ulmi*. The organism passes the winter as mycelium within the host.

known as *Graphium ulmi* Schwartz. The pathogen lives chiefly in the wood and produces its fruiting structures only on wet portions, freshly cut ends of logs, fresh stumps, under loose bark or in insect tunnels. The mycelium is normally white, tufted and in its early growth ringed, later becoming striated. The formation of conidia produces a slimy yellowish appearance. Variation in the pathogen is common and brown pathogenic strains have developed in pure culture and have been isolated from naturally infected trees.

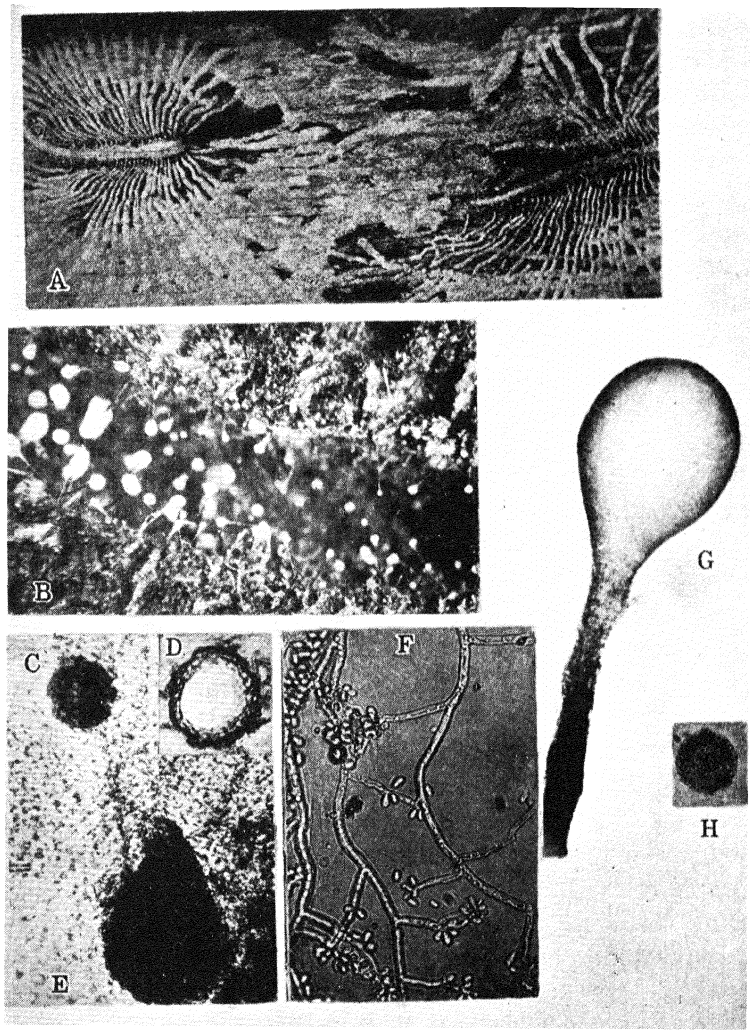


FIG. 170. A, characteristic vertical tunnels in elm wood just beneath the bark caused by *Scolytus multistriatus;* B, coremia of *Ceratostomella ulmi* growing in insect tunnels; C, sclerotium in culture of *C. ulmi;* D, cross section of sclerotium; E, immature perithecium in uncrossed culture; F, mycelium producing Cephalosporium spores; G, mass of viscid spores at tip of coremium; H, cross section of stem of coremium. (After G. P. Clinton and Florence A. McCormick.)

In nature the intracellular mycelium produces short, black, stalked coremia having enlarged, glistening, yellowish heads. The stalk may be one millimeter in height producing the minute egg-shaped to pear-shaped conidia in masses that make up the glistening heads. These conidia may bud before forming pathogenic mycelia and in culture form a yeast-like growth. In culture another asexual stage is formed that seldom occurs in the field. These asexual spores are produced in clusters at the tip of short side branches. These conidia as those produced in coremia are elliptical, more or less flattened lengthwise, and usually enlarge slightly after discharge.

In addition the fungus is known to form a small sclerotia-like body, which is found sparingly in culture and in infected wood. The manner of formation and function of these structures is unknown, although they may be undeveloped perithecia.

Ceratostomella ulmi is a heterothallic fungus, and the perithecia are rarely found in nature. The perithecium has a black spherical base surmounted by a long black neck, which is crowned by a few short colorless hairs. The asci inside the bulbous base are oblong and contain eight single-celled spores, which are shaped as an orange segment. The asci disintegrate early leaving the ascospores free to be forced out through the neck in a mass. The ascospores germinate, forming a short mycelium that very early forms conidia.

Infection by the various spore forms occurs only through wounds in enfeebled trees and it is much more likely to succeed during the active growing season. The spores may be carried to the wounds by wind or by insects. The chief insects concerned are the elm bark beetles (*Scolytus multistriatus* Marsh., *S. scolytus* Fab. and *Hylurgopinus rufipes* Eich.), which tunnel under the bark of weakened trees and lay their eggs. The organism, carried in by the adult beetle, grows into the wood from the larval tunnels, fruits in the channels and coats the emerging beetle with conidia. The insects also are known to ingest the conidia and eliminate the viable spores in the excrement. The young beetles feed in the crotch of young

succulent branches of healthy trees and while feeding inoculate the succulent tissue with viable spores. The fungus has been shown to be transmitted to some extent by other species of bark beetles, borers, mites, the buffalo tree hoppers and bark weevils. Evidence of overwintering within the insect vectors is somewhat doubtful. The underground transmission of the pathogen from tree to tree through root grafts also has been demonstrated.

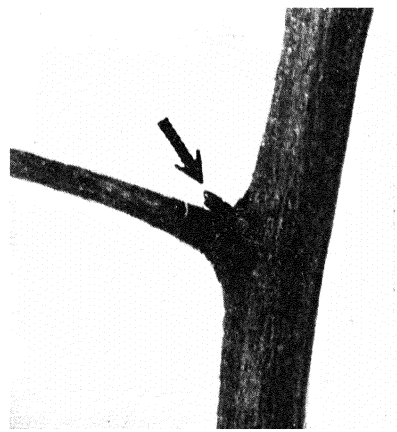


FIG. 171. Elm bark beetle feeding in crotch of healthy American elm branch. (Reproduced from U. S. D. A. Circ. No. 322.)

Within the xylem the fungus travels radially and tangentially through the pits from vessel to vessel. The longitudinal spread occurs by growth of the mycelium in the vessels and by conidia carried up the vessels in the sap. Spores of the fungus have been shown to move upward 30 feet in three hours and 24 feet downward in two days. The Ceratostomella mycelium growing in the vessels causes a plugging of the water-conducting elements by stimulating the formation of tyloses and gum in the vessels. This causes not only a stoppage of water flow but a discoloration of the tissues. There also is evidence that a lethal substance is produced in advance of the discoloration.

The tests conducted on varietal reactions have indicated that all the more desirable elm species are susceptible and that resistance is shown only by Asiatic species. The most resistant forms are *Ulmus pumila* and its variety *pinnatoramosa*, which, however, are not desirable for commercial or ornamental purposes. A new selection of *U. foliacea*, known as No. 24, has shown resistance as well as desirable qualities and is ready for commercial use in infested areas. The nature of resistance in these varieties seems to be physio-

logical, possibly a resistance to the action of the toxin. It has been shown, at least, that the mycelium spreads as rapidly in the resistant trees as in the susceptible ones.

CONTROL.—The control of the wilt pathogen through the development and utilization of resistant varieties such as *U. foliacea*, No. 24, is a possibility.

At the present time the United States Department of Agriculture has in force a rigid application of quarantine and eradication against the pathogen. The infested area in the United States is the only one that lends itself to this type of control. A quarantine has been placed on all foreign elm lumber, ties, burls, etc., and a domestic quarantine on the movement of elm wood out of the infested area.

In the eradication measures all diseased trees are cut and burned, and the stumps are thoroughly disinfected after the removal of the bark, which is burned. For disinfection purposes copper sulphate and creosote usually are used. The stumps are liable to harbor beetles unless peeled, and the infected wood of stumps will harbor the fungus for at least two years. It also is good practice to remove the weakened trees in the infested area, to lessen the chance for infection and to lower the beetle population by removal of their breeding grounds. In areas where the removal of trees is not practical, they are killed by chemicals such as creosote, coppersulphate, etc., that are detrimental to the beetles.

Chapter Twelve

DISEASES CAUSED BY FUNGI IMPERFECTI

THE Fungi Imperfecti constitute a group of organisms in which the complete life histories are not known. Usually only one conidial stage is present; however, two or more asexual stages may occur. Fortunately most of the species of this group are not parasites. Many of them are saprophytes occurring on dead, decaying matter found on or in the soil, where they play, therefore, an important role in hastening decomposition of organic matter, making it again available as plant food. It should not be understood that the Fungi Imperfecti constitute a natural group like the Phycomycetes, Ascomycetes or Basidiomycetes. It is rather an arbitrary class established to include many fungi, probably having no close relationship to one another. It is probable, however, that many of the fungi of this group are merely conidial stages of certain Ascomycetes. Some smaller number also may be found to be conidial stages of the other two groups, the Phycomycetes and Basidiomycetes.

The parasitic members of this group cause a wide variety of symptoms and injuries to herbaceous and woody plants. The imperfects, however, induce more local and general necrosis (rots of fruits and vegetables and spot diseases on foliage) than any other group of symptoms. Some few are very destructive vascular parasites, e.g., the cabbage yellows organism, the flax wilt organism, tomato wilt organism, cotton wilt organism, watermelon wilt organism, etc. Most of the imperfects attack the aerial parts of plants, as the stems and twigs of trees and shrubs, but some also are known to attack the underground parts. Few are known to be obligate parasites, and many are dependent on wounds as avenues of

infection. Others remain alive throughout the year in perennial parts of their hosts.

Diplodia Dry Rot of Corn

Diplodia zeae (Schw.) Lév.

Diplodia zeae is the most destructive of the several fungi that cause dry rot of the ear and blight of the seedling of the corn plant. Although *Diplodia zeae* has been known since 1822, its pathogenicity on ears and stalks of corn was not demonstrated until 1908. More recently it has been shown to be parasitic upon the corn plant in all stages of its growth, causing seedling blight, crown rot, necrosis of the roots and leaf sheaths and dry rot of the ear.

The disease appears throughout the United States except for the more arid western states and has been reported from Europe, Africa, New Zealand and Australia. The reported loss for the state of Iowa in 1921 was as follows: (1) four per cent of the ears, or 17,767,000 bushels, were discarded in the field at harvest time; (2) 11 per cent, or 141,499 bushels, of the seed corn was infected; (3) the field stands were reduced 25 per cent by seedling blight; and (4) ten per cent reduction in field stands from crown and stalk infections.

The estimated loss caused by ear rot in Iowa was five per cent in 1924, two per cent in 1925 and six per cent in 1926. The estimate from Illinois was four and one-half per cent, or 5,621,000 bushels, in 1907, and 2,000,000 bushels in 1907 of which 90 per cent was caused by *Diplodia zeae*. The total loss from *Diplodia zeae* (ear rot, seedling blight and reduction in growth) in this state has been conservatively placed at 10 per cent. The loss for the United States in 1922 was 40,000,000 bushels and for 1923 it was 65,000,000 bushels.

Symptoms.—Diplodia-infected seed may appear lighter yellow than normal with white streaks running from the tip halfway to the crown and with a distinct brown discoloration and shrunken appearance of the tip. The dirty-gray mycelium develops on the tip of severely infected kernels. Less severely infected seed, however, cannot be detected by macroscopic

examination, and the only positive test for infection is to germinate the seed, since the mycelium may frequently enter the seed tip without visible injury.

The symptoms on the seedling appear as sunken, darkened lesions on the mesocotyl, at the origin of the secondary

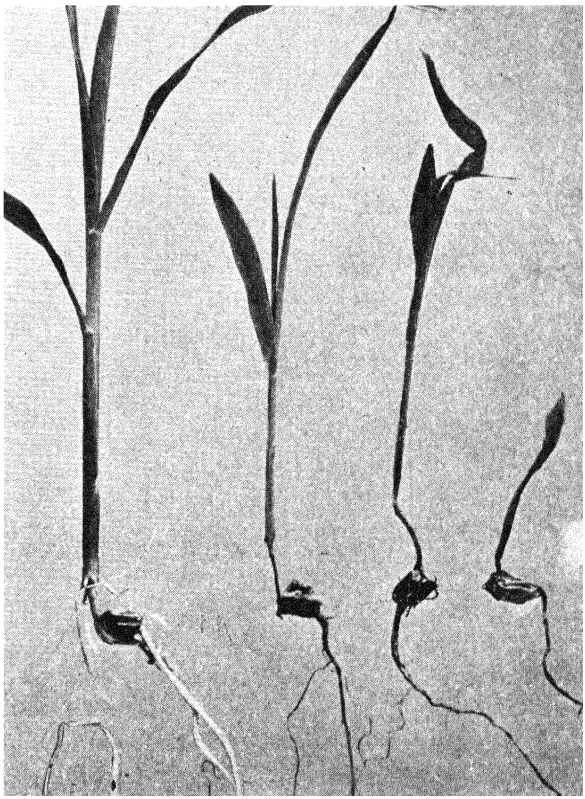


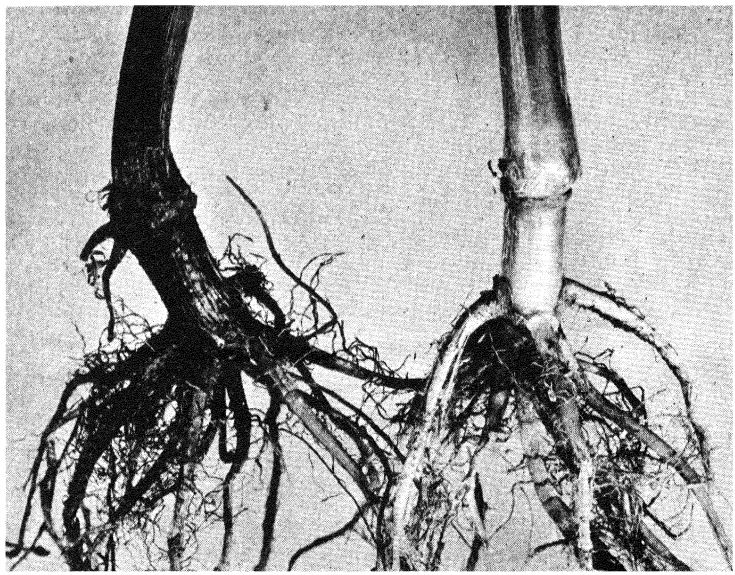
FIG. 172. Seedling blight of corn caused by *Diplodia zeae*. Healthy seedling at left and different stages of seedling blight to the right.

roots. These lesions spread and gradually girdle the mesocotyl, and the seedling dies unless adventitious roots develop from the crown to supply the water needed.

The girdling of the mesocotyl before the roots are established is dependent upon the degree of seed infection and the conditions under which the seed is germinated. The meso-

cotyl also may become infected by the pathogen that has overwintered on infected plant refuse in the field.

Plants with infected mesocotyls, that do not die in the seedling stage, develop restricted dark-brown lesions on the base of the crown among the roots. Such plants do not



A B

FIG. 173. Crown rot of corn caused by *Diplodia zeae:* A, infected plant showing pycnidia of causal agent in discolored internode; B, healthy plant.

develop normally and mature from one to three weeks early. As the plant dies, the mycelium spreads throughout the crown and up the prop roots, causing the lower internodes to become dark straw-brown in color and covered with pycnidia of the fungus.

The fungus may invade the stalk at the node, from spores falling back of the loosened leaf sheaths. The resulting mycelium may cause a brown discoloration and general weakening of the stalk and leaf sheath. On the leaf sheaths the fungus produces red or purple areas of varying size and shape, in which the center area is frequently bleached.

The mycelium in the stalk usually spreads more than one internode, but in the shank of the ear it grows out into the cob and finally into the kernels. *Diplodia zeae* is most evident on the leaf sheath and stalk when pycnidia emerge as minute white, evenly distributed dots that later turn black.

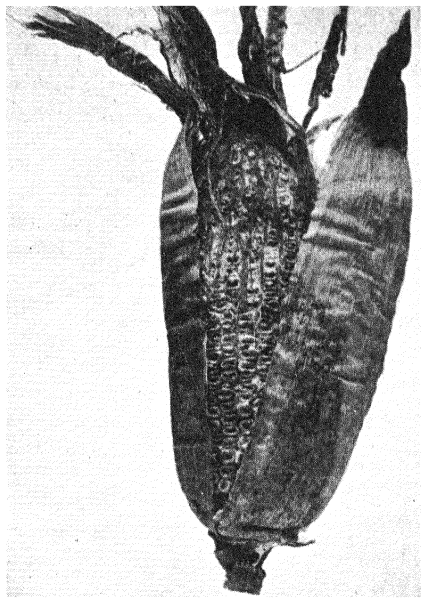


FIG. 174. A corn ear in which *Diplodia zeae* has completely filled the spaces in and around the kernels and the husks of the rotted ear.

Ears may be invaded from either the shank or the silk end. The degree of injury varies greatly, depending on the age of the ear at the time of infection. At one extreme is ear infection in the milk stage when a dense mat of white or dirty-gray mycelium with embedded black pycnidia partially replaces the ear and firmly attaches the husk to it. At the other extreme is the late infection when the broken, slightly shredded shank attachment and light-brown discoloration of the cob may be the only indication of the disease. The black fruiting bodies may be found inside a badly infected cob when broken, and the white mycelium frequently is found around the base of the kernels.

ETIOLOGY.—The fungus causing the dry rot of corn, *Diplodia zeae*, has been known as a parasite since 1908. (See Fig. 175.) The mycelium, as is typical of the Fungi Imperfecti, produces only one type of spore. These spores are produced in the black, nearly spherical ostiolate structures, pycnidia, found in most infected portions. The dark-brown, cylindrical, usually curved, one-septate pycnidiospores are

produced in great abundance within the pycnidia, and are extruded in long, slender black tendrils. These spores under conditions favorable for germination produce the mycelium, which causes the secondary infection of the host. At this time infection takes place back of the leaf sheath and husks.

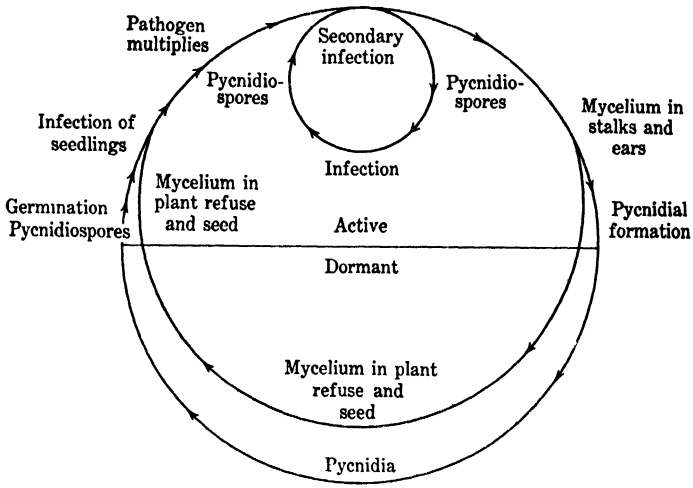


FIG. 175. A diagram of the host relation of *Diplodia zeae*. This organism may survive the winter in the mycelial or pycnidiospore stages.

The leaf sheaths become loosened at flowering and the pollen and spores fall into this cavity, where ideal conditions for germination of the spores and infection of the tissues prevail.

Infection of the seedling results from the mycelium inside the kernel, which begins to grow under moisture and temperature conditions favorable to the germination of the seed. The mycelium proceeds under the pericarp, which is elevated by the emerging seedling, to the junction of the mesocotyl and the seed. At this point it becomes established in the natural wounds at the point of emergence of the secondary roots. The destruction of the cells produces the typical brownish lesion on the mesocotyl.

It has been shown recently that the pathogen generates a toxic substance that inhibits its own growth. If Diplodia-infected seed is soaked for 12 to 16 hours in a water solution

of the inhibitor and placed to germinate at 15° to 18° C., the pathogen is retarded and the host escapes. On the other hand, if the infected seed is soaked for a longer period, the host may also be injured.

Moisture and temperature have marked effects on infection of the host and the development of the pathogen. In pure culture the pathogen grows most rapidly at 30° C. although it will grow at any temperature between 10° and 35° C. The germination of the pycnidiospores is greatest at 33° C. These favorable temperatures exist in late summer when the ears and stalk are susceptible to infection. In the infections in the seedling stages or on the crown of the plant, the moisture conditions of the soil mainly determine the growth of the fungus, the best growth occurring at the optimum moisture conditions for the growth of the host. If this is accompanied by a low temperature, the growth of the corn plant is delayed, and the fungus produces a greater destruction of the tissues and hence more damage to the plant. In the aerial infections occurring late in summer, the temperature always is favorable for growth of fungus and host. The extruding of pycnidiospores, their germination and the development of the mycelium depend on the presence of abundant moisture. The growth of the fungus continues in infected ears until the moisture content drops to 21 per cent.

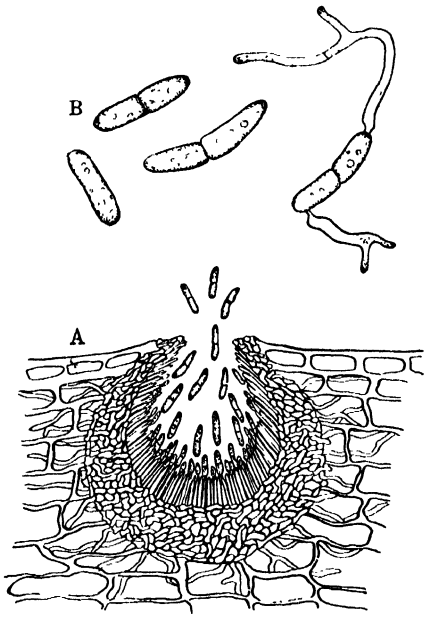


FIG. 176 *Diplodia zeae:* A, cross section of pycnidium; B, pycnidiospores and germ tubes. (After L. W. Durrell.)

CONTROL.—Although there is no known method or practice

that will absolutely control *Diplodia zeae* at all stages of the host, the losses may be materially decreased if careful attention is given to seed selection and storage, seed testing, seed treatment and crop rotation.

Many infected ears may be eliminated before storing by careful hand selection of the seed corn. Full, well-dented ears on unbroken, healthy shanks of green standing stalks should be selected well before frost. The selected ears should be harvested early to prevent spread of the mycelium. Any ear with the cob showing a brown or ragged shank attachment should be discarded. The good ears should be stored immediately in a cool dry place to prevent the spread of the pathogen that escaped notice.

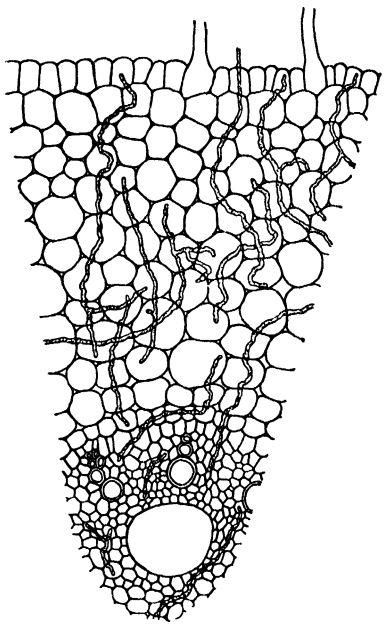


FIG. 177. Partial cross section of root hair region of corn root showing intracellular mycelium of *Diplodia zeae*.

Since much of this seed-borne infection is not apparent on the seed, the germinator test is the only satisfactory method of diagnosis. Much of the seed testing is done efficiently in standardized seed laboratories where covered germinator trays are used. After trying several different kinds of seed corn germinators, however, the modified rag-doll type has been found the most suitable for ordinary farm practice.

Since absolutely disease-free seed cannot be obtained at all times and since Diplodia invades the seedling from the soil, seed treatment is recommended for all seed to be planted. The shelled seed is thoroughly coated with an organic mercury dust by rotating in a mixer.

If corn is grown consecutively for a number of years on a piece of ground, the population of this pathogen increases and, under favorable weather conditions, causes heavy losses. A long system of rotation should greatly reduce this infestation. A five-year rotation, arranged so that corn is not grown in two successive years, is to be encouraged.

Phymatotrichum Root Rot

Phymatotrichum omnivorum (Shear) Duggar

Phymatotrichum omnivorum was first observed and has been most fully investigated as causing a root rot of cotton known by various names as cotton root rot, Texas root rot, Ozonium root rot and Phymatotrichum root rot. The early observers attributed the root decay to drought, alkali, weather conditions, etc. In 1888, however, the causal agent was shown to be a fungus. Phymatotrichum root rot is caused by a soil-

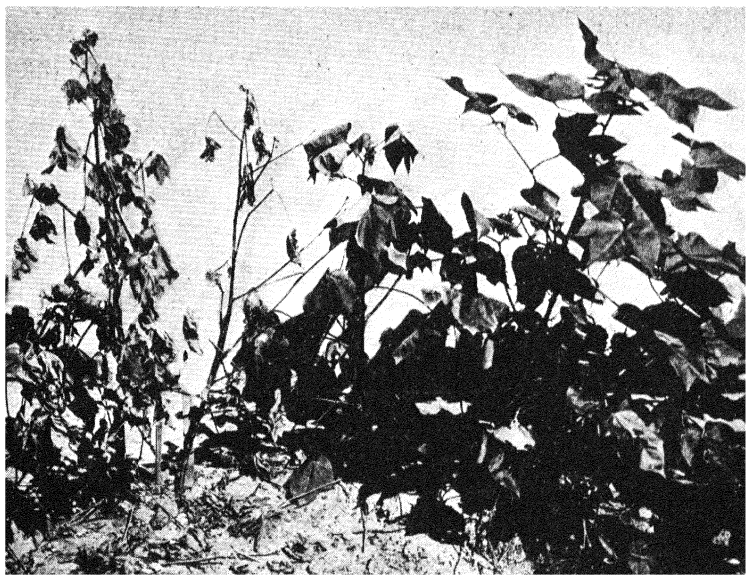


Fig. 178. Effects of *Phymatotrichum omnivorum* on cotton. At left two plants killed by the root rot fungus, in the center a recently wilted plant, and at right a healthy plant. (After J. J. Taubenhaus and W. N. Ezekiel.)

inhabiting pathogen widely distributed in southwestern North America, which causes serious losses on a wide range of host plants; for example, in 1919 the loss of cotton in Texas alone was estimated at ten per cent, and in 1937 the aggregate loss on all crops in seven states was $150,000,000.

Although the fungus is most destructive on cotton, okra, alfalfa and other crops, it has been shown to be pathogenic to

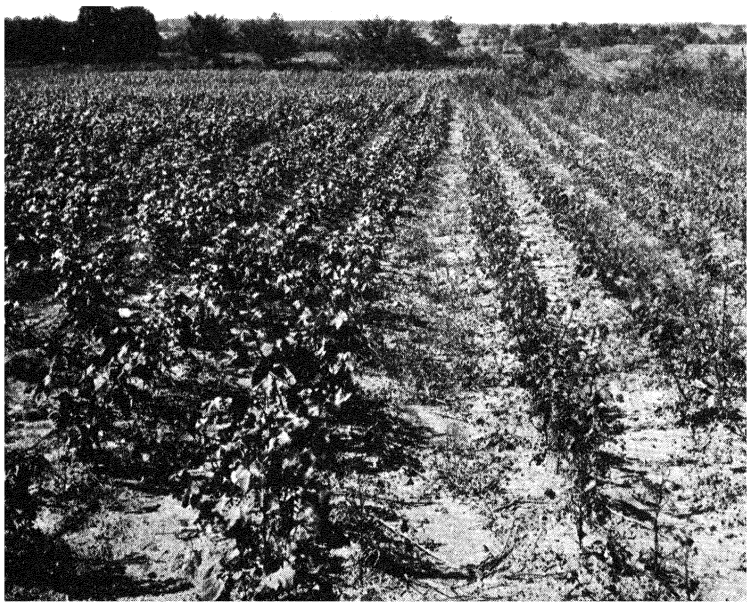


FIG. 179. A plot of cotton plants injured following inoculation with the cotton root rot organism and adjoining healthy plants. (Courtesy of DIV. of Pl. Path. and Phys., Texas Ag. Exp. Station.)

plants in more than 2,000 species. These plants are mainly in the Dicotyledonae and Gymnospermae, while no Monocotyledonae have been found susceptible to the organism. The causal agent has been definitely reported as occurring only in the southwestern part of North America, being most important in Texas, New Mexico, Arizona and Mexico. The organism is believed to be indigenous in that section.

SYMPTOMS.—The first evidence of the disease is a yellowing or bronzing of the upper leaves and it is seldom noted except by an experienced observer. This yellowing frequently occurs when the plants are six to eight inches high. A few days after the plant becomes yellow there is a slight wilting of the upper leaves and the next day a sudden collapse of the entire foliage. The initial wilting may not be permanent, but recovery will occur only a few times. After the permanent wilting the leaves rapidly become brown and dry, indicating the characteristic sudden death of the plants.

The presence of the brown dried leaves on brown to blackened stems occurring over circular areas in the field is typical of the disease. These areas may increase in diameter at the rate of 30 feet in one season. The dying plants usually occur in more or less circular areas that increase for five to eight years, after which normal plants may develop over most of the previously infested area.

The underground portions of plants that have just wilted are found to be well decayed and covered with the brownish rhizomorphs and sclerotia of the pathogen. The presence of the fungus three to four feet beyond the region in which plants are dying is indicated by the decay of the roots and the presence of rhizomorphs and sclerotia. Following the wilting and death of the aerial portions, the roots undergo a rapid, soft wet rot and even the rhizomorphs decay in about three weeks. The lint and seed produced on plants that die before the bolls mature are of lower grade and hence even a late infection decreases the crop returns.

After the middle of June, snow-white cottony mycelial mats 2 to 12 inches in diameter may form in wet or moist shaded soil. These mycelial mats quickly turn tan or buff and become powdery as they sporulate. A rain or heavy irrigation favors the formation of spore mats. The presence of pockets of sclerotia in the soil may indicate where roots were destroyed by the pathogen.

ETIOLOGY.—The vegetative body of the root rot pathogen, *Phymatotrichum omnivorum*, is made up of fuzzy, tan to brownish threads or strands, which branch frequently and form single

interconnecting hyphae. These hyphae arise from the numerous small, multicellular hyphae, which surround the large central hypha of the strand. The branches of the strands as well as the appendages, which produce the fuzzy appearance, are characteristic in their right angled branching and production of straight, acicular side branches. These

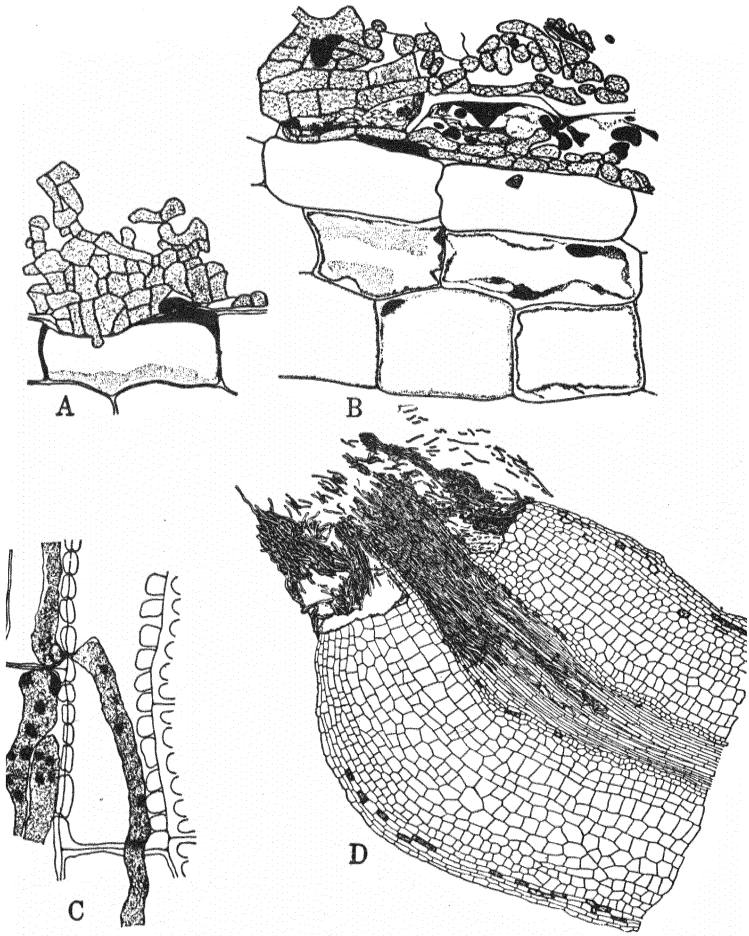


FIG. 180. A, pseudoparenchymatous hyphal mass in process of entering cortical cell; B, effect of pseudoparenchymatous hyphal mass in penetrating host, epidermis has been destroyed, outer cortex is occupied, and collapse of the inner cortical cells is apparent; C, hyphae in tracheids, note hyphae in the walls; D, destruction of the tip and penetration into the young root by a hyphal mass. (After G. M. Watkins.)

fuzzy strands seem to be rather short lived and their function in the life history is not definitely known. There also are produced smooth, dark-brown strands of much the same internal structure, which are long lived and serve as an overwintering stage of the pathogen (see host relation in Fig. 181.) These are probably true rhizomorphs.

Intermixed with the fuzzy strands often are found small, whitish, woolly mycelial knots. These sclerotia-like bodies often are abundant, but like the fuzzy strands seem to have no known function. In the fall, however, there are formed

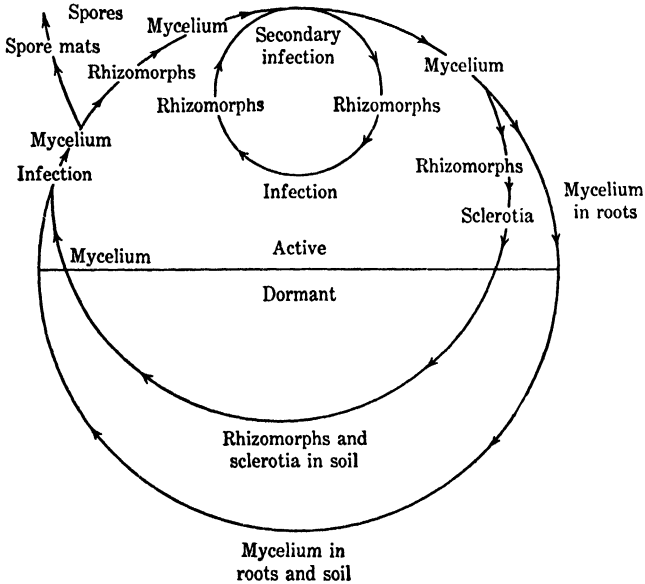


FIG. 181. A diagram of the host relation of *Phymatotrichum omnivorum*. This organism overwinters as rhizomorphs, sclerotia or mycelium in roots or soil.

abundant small, light to dark-brown, variously shaped sclerotia. These sclerotia may germinate producing the white hyphal strands when placed at 24° C. for 24 hours with sufficient moisture. The sclerotia also may survive in the soil for three to five years without losing their viability. They seem to be formed in groups in pockets anywhere in the top eight feet of soil, although they are most frequent at 6 to 24 inches.

During the rains, which usually occur in the summer or early fall, the mycelial strands grow to the surface of the soil and form, overnight, a cottony white mycelial mat. These mats, usually formed in shady places, may be 2 to 12 inches in diameter, a quarter of an inch thick. They turn to a powdery buff-colored, spore-forming body in a few hours. The white cottony strands of the mat develop swollen tips and side branches on which tiny spores are borne. The spores, which are responsible for the change of the mats to a powdery buff-colored condition, have never been germinated. The spore mats are ephemeral and disappear in two to three days.

As might be suspected from the numerous hosts of the pathogen, it is not exacting in its metabolism and grows on nearly all forms of carbohydrate and most nitrogenous substances occurring in natural materials. It seems, however, that ammonia is not only used with difficulty by the organism, but may become toxic in concentrations of 200 to 300 parts per million. Sclerotia and rhizomorphs seldom are formed in media containing ammonium nitrogen. In natural conditions the fungus grows in a circular area in the soil and on all plants within its area for three to eight years. Suddenly the fungus appears to die out in several places or all over the spot. Reinfection apparently may take place, and there is no explanation for the sudden disappearance of the pathogen.

Infection of the host plant may take place on any portion of the root system, but is most frequent on the root at depths of 6 to 24 inches. Penetration follows the nearly complete enveloping of the root by a fungous mantle. The hyphal strands send out the characteristic branches at right angles, and penetration is direct or occurs through lenticels, or wounds such as those at the origin of secondary roots. In young roots and in the cortex, phloem, cambium, etc., of old roots the pathogen penetrates either as massed hyphal strands or as an individual hypha. The hyphae form enzymes or poisons that cause the death of cells and the swelling and discoloration of cell walls three to six cells in advance of the hyphae. The hyphae are intercellular until the enzymatic action causes a break in a cell wall; thereafter the cell cavity becomes filled

with fungal hyphae. In old roots the cutinized walls of the cork cells seem to markedly slow the diffusion and action of the toxins or enzymes to such an extent that penetration appears to be mechanical. There usually is an incubation



FIG. 182. Spore mats of *Phymatotrichum omnivorum* around the base of a cotton plant killed by the fungus. (Courtesy of the Div. of Pl. Path. and Phys., Texas Ag. Exp. Station.)

period of 12 days before the appearance of the disease within the plant. Death usually results not earlier than 25 days after infection.

The spread of the organism seems to occur by the smooth strands or rhizomorphs that grow through the soil at about two to eight feet per month. The dissemination from field to field occurs through transport of infected soil, diseased

nursery plants or plant debris. Experiments on insect dissemination have so far yielded only negative results.

The pathogen is inactive from the latter part of October until late in May. This inactive period is passed as mycelium in the living roots of plants, in plant debris and as sclerotia or rhizomorphs in the soil. Since the mycelium survives in roots anywhere in the top eight feet of the soil, plowing is of no value.

The pathogen survives adverse conditions in all types of soils, but it is most active in heavy soils having a high moisture content. With this high moisture content there apparently must be some air, as flooding reduces the population of the organism in the soil. The organism survives in soils with a pH range of 5.0 to 9.0 with the percentage of infection increasing on the alkaline side. The optimum temperature for the disease processes is about 24° C.

CONTROL.—Crop rotation is the most practical control measure. Immune crops, chiefly monocotyledonous plants, are grown four of the five years in the rotation. The immunity of monocotyledonous plants to the attacks of the pathogen is said to be due to the presence of toxic materials in these plants. The toxic material responsible seems to be ether soluble, and a water extract of either monocots or dicots usually is toxic. The use of resistant varieties offers little help, since, with the exception of cotton, little advance has been made in their production. Even in the cotton varieties resistance is not of a sufficiently high degree for the plant to survive in heavily infested soil. Periodic flooding has been practiced in some cases and yielded fair control. Cotton, however, is rather susceptible to flood injury and this treatment cannot be used while a crop is on the land. Fertilizers high in sulphur and low in nitrogen are said to be of value. The reduction of the pH of the soil by the sulphur below the region favoring fungal activity is one explanation for the benefit from the treatment. Barriers of soil saturated with sulphuric acid or copper sulphate have promise of being an additional method of prohibiting the spread of the pathogen in a field.

In small nursery or seedbed plots, control may be affected by the use of fungus-free soil and deep, permanent, impermeable barriers. Quarantine may be used to prevent the introduction of the organism into non-infested areas. Partial soil sterilization by formaldehyde, steam, organic mercurials, chloropicrin or carbon disulphide are recommended under certain conditions. Ammonium sulphate also is recommended on the basis of the toxicity of ammonia to the fungus. A one to two per cent solution of ammonia is said to kill sclerotia, rhizomorphs and surface mycelium on infected roots. The killing of mycelium within the roots requires a higher concentration of ammonia. It has been shown that applying heavy applications of manure or other organic matter decreases the ravages of the pathogen. The growth of *Phymatotrichum* in the soil has been shown to be slowed or even stopped by the presence in the same soil of actively growing species of the genera *Aspergillus*, *Penicillium* and *Fusarium*, and by *Trichoderma lignorum*. Whether this will be of use as a means of biological control of the root rot organism is not certain.

Cercospora Leaf Spot of the Beet

Cercospora beticola Sacc.

This disease, known since 1873, is most serious on sugar beets, although the garden beet, Swiss chard, manglewurzel and other plants are susceptible to this pathogen. The disease is prevalent wherever beets are grown, especially throughout America and Europe. It seldom is serious in the dry regions where beets are grown through a summer season low in rainfall. In some sections of this country losses of 50 per cent may result from defoliation and reduction in sugar percentage and total tonnage.

Symptoms.—The chief symptoms of this disease are the small brownish spots with reddish-purple borders that develop on the leaf. These spots soon change to ashen-gray color with a brown border as a result of the production of the conidia of the fungus over the infected central portion. The spots, seldom more than 0.8 centimeter each in diameter, may cover nearly the entire surface of the older leaves. As a result of a

heavy infection, the leaf, apparently poisoned, turns yellow, shrivels and dies. The infected leaves subsequently may be seen lying on the ground around the plant as shriveled, dried masses, dotted with conspicuous necrotic spots.

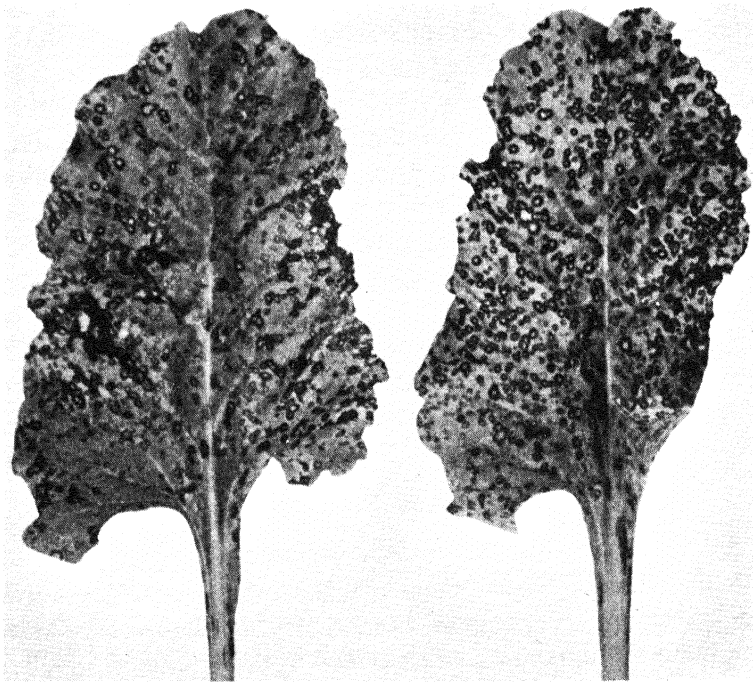


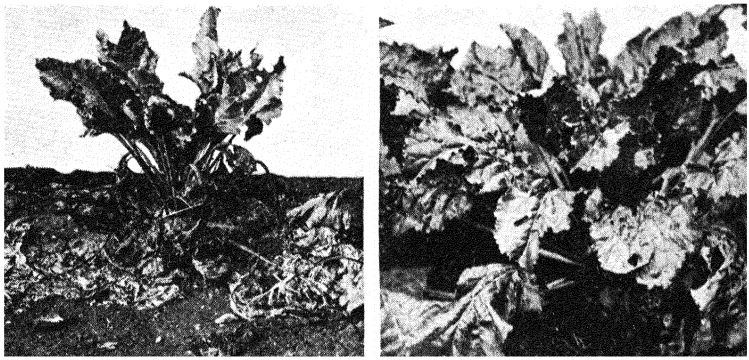
FIG. 183. Cercospora leaf spot of sugar beet. The two leaves show numerous well-developed lesions.

If conditions remain favorable for the initiation of the disease, the pathogen spreads to the next older leaves. In this way the pathogen advances from the older to younger leaves. At the same time the plant is producing new leaves to replace those destroyed by the fungus. The successive production and destruction of leaves leads to an elongated and roughened crown. This condition has aptly been called "pine-apple disease." The lesions may be found on petioles, roots, flower stalks, the flower and the seed ball.

The loss is not so much the result of the decrease of leaf

surface as the depletion of the sugar stored in the root. New leaves that are formed to replace those destroyed by the pathogen develop at the expense of the sugar reserves.

ETIOLOGY.—The fungus that causes the disease is *Cercospora beticola*. The host relation of this fungus is very simple since it is only known to produce one type of spore, a long, slender,



A B

FIG. 184. A, partial defoliation of sugar beet as a result of severe infection by *Cercospora beticola;* B, a healthy plant.

multi-septate conidium (see Fig. 185). It is these conidia on the surface of leaf lesions that impart the ashen-gray color referred to above. The fungus infects the leaves by sending germ tubes from the conidia through the stomata, producing the mycelia within the leaf. The gnarled, olivaceous mycelium grows intercellularly within the leaf and produces the necrotic spot. After some time the fungus grows out and produces the conidia on the upper surface of the leaf. These conidia are spread by splashing rain, insects, man and wind, and are capable of immediate germination. Favorable conditions for germination of the spores are high temperature and high humidity. The optimum temperature for spore production is 29° C., with a range of 15° to 37° C. These same conditions may stimulate the opening of the stomata, which afford the germ tube its only port of entry. The humidity must be high, and the stomata are said to remain open if the humidity is above 60 per cent.

The overwintering of the fungus is chiefly as mycelium on infected plant refuse in the field. The pathogen also may survive on susceptible weeds and sporulate in the spring. It also has been found in the soil as a free-living organism.

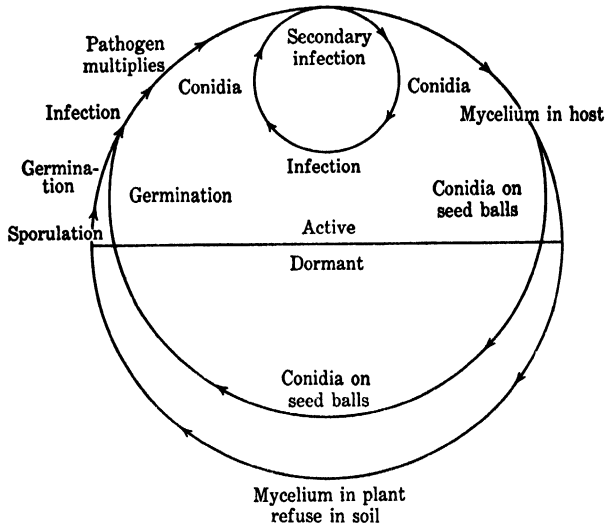


FIG. 185. A diagram of the host relation of *Cercospora beticola*. This organism survives the winter either in plant refuse or as conidia on seed balls.

Further inoculum is furnished from infected seed or from "stecklings" (small beets saved for seed) that were infected before storage but had not developed recognizable symptoms. It is said that root infections of "stecklings" are particularly hard to recognize and eliminate.

CONTROL.—The control of the pathogen lies chiefly in correct methods of culture. Since the primary source of infection is from the mycelium overwintering in the field, the infected portions should either be removed from the field or plowed under deeply. Plowing allows quick decay of the substrate of the fungus and prevents its spring sporulation. Susceptible weeds must, of course, be eliminated. Since high humidity is the main factor in regulating infection, wide spacing of the beets, allowing free circulation of the air around the plants, is essential. Blocking or checking the beets at

20-inch intervals in 20-inch rows has been found successful in the middle west. Checking not only reduces infection and retards the pathogen, but also permits the use of machinery in planting and cultivation, lowering the cost of production.

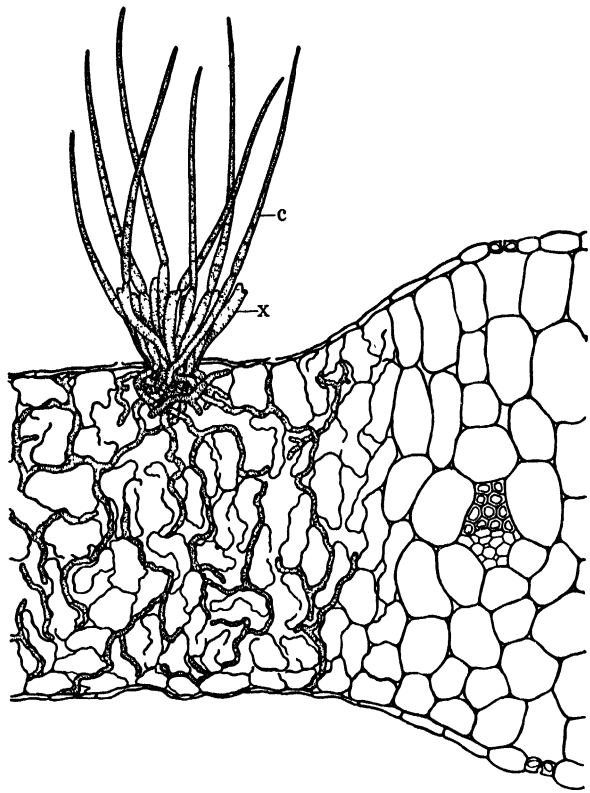


FIG. 186. Cross section of sugar beet leaf infected with *Cercospora beticola:* conidium, c, on conidiophore, x, from the mycelium which is limited to the necrotic area of the leaf.

The seed should be clean and, if infected, should be treated with a mercury dust. The same applies to treating diseased stecklings. Spraying has been recommended, but, besides being only partly successful, it is many times too expensive to be utilized in practice. Leaf spot resistant beets are being developed and should be available shortly.

Watermelon Wilt

Fusarium bulbigenum Cke. and Mass. var. *niveum* (E.F.S.) Woll.

The watermelon probably is native to Africa, having been carried to Asia and Europe and finally to America. In this country it is grown extensively in the southern half of the United States and to a lesser extent in the northern half.

The watermelon wilt organism seems to be generally distributed wherever melons are grown, in this country, Africa, Japan and Australia. Just how long the watermelon wilt pathogen has been in this country is not known. It was first described from the state of South Carolina in 1894.

The watermelon, *Citrullus vulgaris* Schrad., comprises many varieties, some of which are edible and others not. All edible varieties are susceptible to the wilt organism, while many of the unedible ones, as the African stock citron, are rather resistant. Dr. W. A. Orton obtained the resistant sort known as the Conqueror by crossing the African stock citron on an edible variety. This was the first time any one ever had developed a plant resistant to a fusarial parasite by hybridization. Orton's discovery on this occasion initiated a line of research that has yielded very large savings to agriculture through the development of resistant varieties, not only in watermelons, but also cotton, cow-peas, flax, cabbage, tomatoes and many other crops attacked by different wilt-producing organisms.

The loss caused by watermelon wilt is difficult to estimate, since serious losses have been sustained through forced changes in cropping practices besides actual decrease in yield. By 1924 the disease had virtually eliminated watermelon growing in the sand land region of the southeastern part of Iowa. The acreage in this district had been reduced from 8,000 to less than 850, while the annual carload shipments had dropped from 3,000 to 50. Actually, many sand land farms have been abandoned, and the land is lying idle.

In the Leesburg watermelon section of Florida, the soil is so generally infested that new land is sought every year by the

larger growers. New land is available only by clearing virgin land, which becomes an item of considerable expense. Further, the new fields often are farther from shipping points and highways, which increases the labor cost. Many similar instances might be cited from practically every watermelon section where wilt has become established. It is, of course,

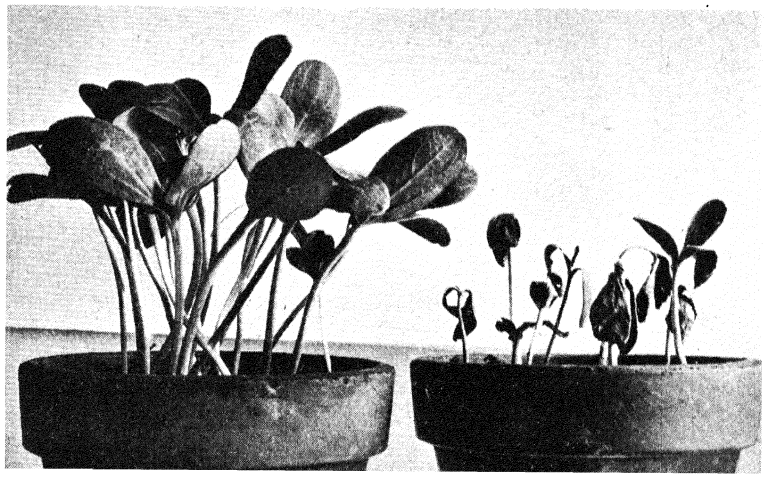


FIG. 187. The watermelon seedlings at the right show symptoms associated with early infection by the wilt organism. Healthy seedlings at the left.

clear that such serious disturbance to a crop decreases the grower's income and in many cases leads to financial distress because he is forced to grow other crops or migrate to other pursuits.

SYMPTOMS.—The watermelon wilt organism may produce four more or less distinct types of symptoms on seedlings. These are, in the order of their appearance, seedling necrosis, stunting, damping-off and wilting. Seedling necrosis prevents the emergence of the seedling from the soil, killing the plant soon after the seed germinates. Stunting is commonly observed when plants are about one week old. The cotyledons are smaller than normal and the plants are not so large nor so vigorous as those grown in disease-free soil. Damping-off causes the seedlings to fall over, necrotic areas occur on the

hypocotyl and below the soil line. Where wilting occurs, the cotyledons lose their turgidity, and droop until their tips touch the hypocotyl, finally wither and the plant dies.

Older plants attacked by the watermelon wilt organism lose the turgidity of their stems and leaves and wilt, in the course of two or three days, or they may linger for two or three weeks



FIG. 188. Wilted watermelon vine infected by *Fusarium bulbigenum* var. *niveum*.

before they finally die. Often several days elapse between the earliest symptoms and ultimate death of the plant. Such an infected plant may wilt during the heat of the day and recover overnight. This process may be repeated for three or four days before the plant dies. More often, however, plants die within 12 hours after showing the first evidence of infection. Often only one or two runners of a plant may wilt, the others remaining normal and healthy.

In many infected plants little or no wilting of the foliage develops. Instead, the leaves turn yellow, especially those near the crown, the internodes are shortened, the flowers are smaller and more numerous, and the fruits, if any set, are

dwarfed. Such plants may live until frost, but most of them die earlier.

In addition to these aboveground symptoms, various root symptoms may be observed if the plants are removed from the soil. In the seedling wilt stage the roots appear normal and generally show no discolored root lesions. Plants in the two true leaf stage may show complete decay of the lower portion of the root system, accompanied by a superficial branching and bunching of new small roots on the upper portion of the main root. In the more mature stage of the host, when wilting occurs, the larger roots frequently show localized lesions, and many small secondary roots are decayed.

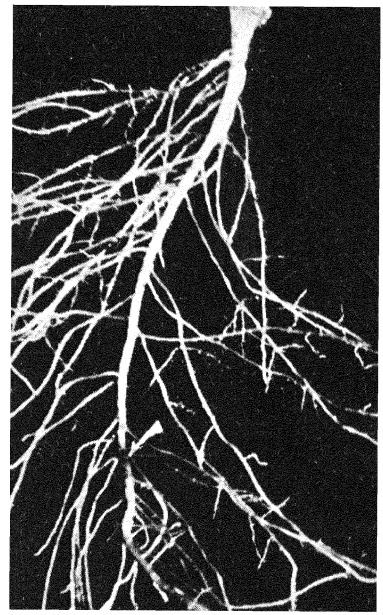


FIG. 189. Lesion on primary root of watermelon caused by the wilt fungus that entered through a lateral root.

ETIOLOGY.—The fungus responsible for this disease, *Fusarium bulbigenum* var. *niveum*, produces three spore stages, all of which are asexual and may be produced at the same time. The large, sickle-shaped, hyaline septate spores are termed macroconidia, and the small, single-celled, straight and hyaline spores, microconidia. The chlamydospores are dark brown, thick-walled, multicellular spores which develop in the hyphae or on short side branches. No sexual stage is known. The fungus grows rapidly and has many strains designated by the ability or lack of the ability to produce pigmentation, etc. The host relation is shown in Fig. 190.

The fungus is capable of overwintering on dead plant material in the field or as a free-living organism in the soil. The fungus usually infects the host by the hyphae penetrating

through the root cap, epidermal tissue, through the epidermal cells of the root back of the root cap in the region of elongation and maturation or through wounds marking the origin of secondary roots. The mycelium, on penetrating, progresses intracellularly to the stele where it enters the xylem tubes

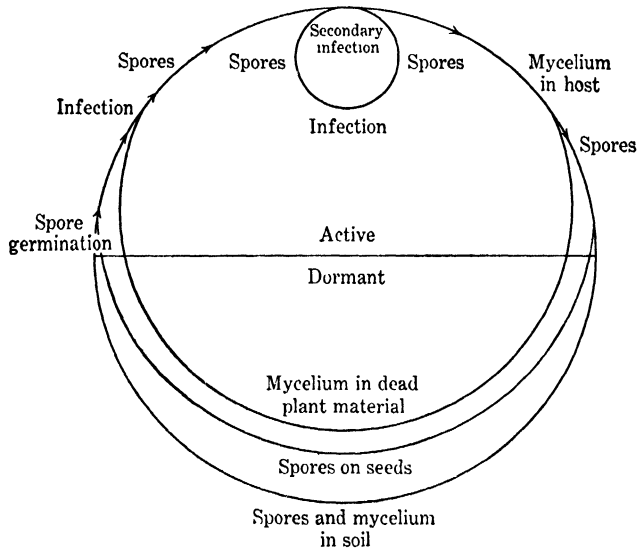


FIG. 190. A diagram of the host relation of *Fusarium bulbigenum* var. *niveum*.

in which the pathogen may spread into the aboveground stem.

In mature plants infection of the roots may take place many times, finally causing foliage symptoms. The vessels become filled with mycelium, a heavy gum and numerous large tyloses. Gummy products and tyloses in the xylem of the diseased plants seem to be produced by the living cells of the host, injured by toxic, metabolic or enzymatic products of the wilt pathogen. Older resistant plants, past the seedling stage, may survive the invasion of the parasite.

CONTROL.—The control of watermelon wilt is largely a problem of prevention. Once a field becomes infested little can be done to eradicate the fungus and many years must elapse before watermelons can again be profitably grown.

The fungus is able to live on the organic matter in the soil even in the absence of watermelons. Since the fungus is carried on the seed, it is important that seed to be planted

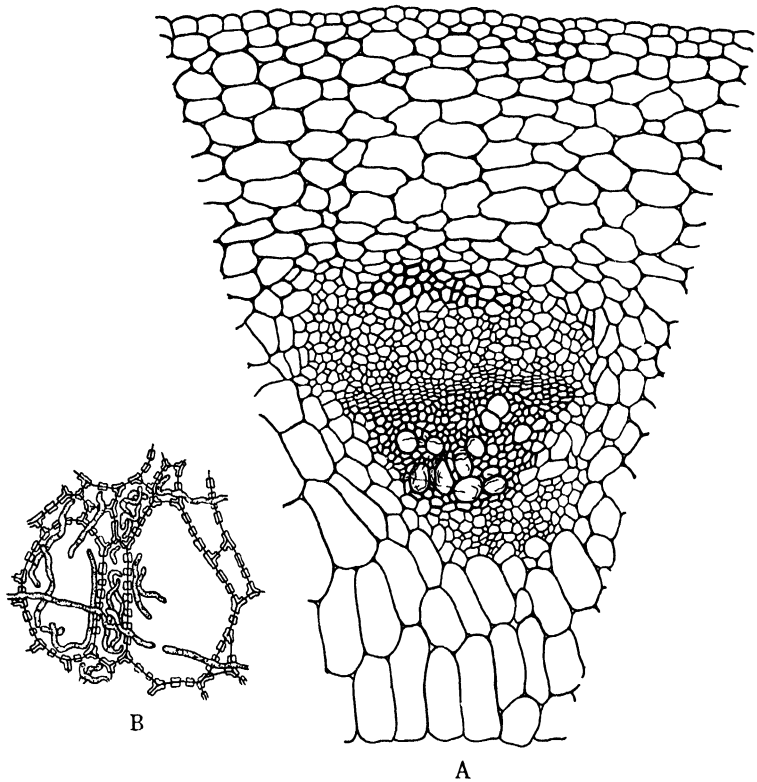


FIG. 191. Cross section of a vascular bundle of a watermelon stem infected with the wilt pathogen: A, fungus confined to the xylem, B, intratracheal position of hyphae.

on non-infested soil should be treated with a mercury dust.

The use of resistant varieties is the most efficient, as well as the most economical method of controlling the watermelon wilt organism. The following varieties are known to be resistant and of high commercial quality: Pride of Muscatine, Kleckley Sweet No. 6, Iowa Belle, Leesburg and Wilt-Resistant Thermond Grey. Since the seed of these varieties may carry the organism, they should also be treated to prevent infesting clean ground.

Cotton Wilt

Fusarium vasinfectum Atk.

Cotton wilt, although known for some time previously, was first described in 1892. At that time it was reported under the name of "frenching" and since then it has been called "black root" and other names that indicate the various symptoms.

The disease is important in all states of the cotton belt from Virginia to New Mexico and has been reported from India and Egypt. In many respects it is the most important disease of cotton. In 1914 it was estimated that the cotton wilt organism and the cow-pea root knot organism caused an annual loss of $10,000,000. The loss is not alone that of reduced yields, but also that of lowered land values, increased cost of production because of lowered yields and the necessity of using infested land for growing crops less profitable than cotton.

In 1935, more than 30 years after Orton introduced the variety Dillon, the first example of the development of a resistant host strain by selection, the losses still amounted to four or five per cent annually. In specific cases where non-resistant varieties are used, the losses may range from 75 to 90 per cent. In most cases where susceptible varieties are grown, the loss is 25 per cent or more.

Symptoms.—The chief symptoms of the disease are the premature wilting and death of the plant and the discoloration of the vascular system of the root and stem. Although these symptoms are most obvious when the plants are 8 to 12 inches high, the plant may be attacked at any stage of development. This fungus also causes a seedling rot and early wilt typical of most of the fusarial pathogens.

Symptoms may appear as sudden wilting and dying or they may follow more slowly, showing as a yellowing of the edges of the leaves and portions between the veins. In the case of the latter symptom plants are dwarfed, although one branch may develop normally to give a one-sided appearance. Accompanying all these symptoms may be found a discclora-

tion of the vascular bundles at the base of the stem. This discoloration often extends up into the leaf petioles and even into the seeds. The brown or black discoloration of the bundles is frequently the only symptom distinguishing wilt



FIG. 192. The reactions of resistant and susceptible cotton varieties when planted on soil heavily infested with *Fusarium vasinfectum*. (Reproduced from U. S. D. A. Farmer's Bul. 1745.)

from the physiologic disease known as cotton "rust." The dark discoloration of the vascular bundles may appear before any outward symptom is apparent.

If the established plants that wilt are dug, the tap roots may be found shortened and blackened, especially around the few much-shortened laterals. As a result of this apparent accumulation of infection, the leaves may be found to wilt one or two at a time and then are shed, which leads to the barren stalk symptom. A light attack allowing the plant to mature, will effect early ripening of the bolls. The only sign of the disease commonly found does not develop until

after the plant is dead, when a pinkish coating caused by the fruiting of the causal agent develops on the stems.

ETIOLOGY.—The fungus causing this disease, *Fusarium vasinfectum*, is another of those organisms capable of living either on decaying plant parts or as a free agent in the soil. As a result, infection may occur through root tips, wounds



FIG. 193. Cotton plant showing stunting and partial defoliation caused by *Fusarium vasinfectum*. (Reproduced from U. S. D. A. Farmer's Bul. 1745.)

from cultivation, insect injuries or natural injuries at the origin of secondary roots, or the fungus may penetrate healthy roots. (See Fig. 194.)

Earlier reports of the disease attributed the wilting to the plugging of the vascular elements by the pathogen. Recently,

however, it has been shown that the mycelium does not become luxuriant enough to plug the vessels and that toxic materials are produced by the fungus that are capable of causing wilting of the host. The fungus also is said to spread in the vessels by the production of one of its spore forms, the small unicellular microconidia. The other spore forms are produced with the microconidia on the outside of the dead stems. When produced in large masses, these spores produce the pink crusts on dead material.

The macroconidia are large, crescentic spores, which are

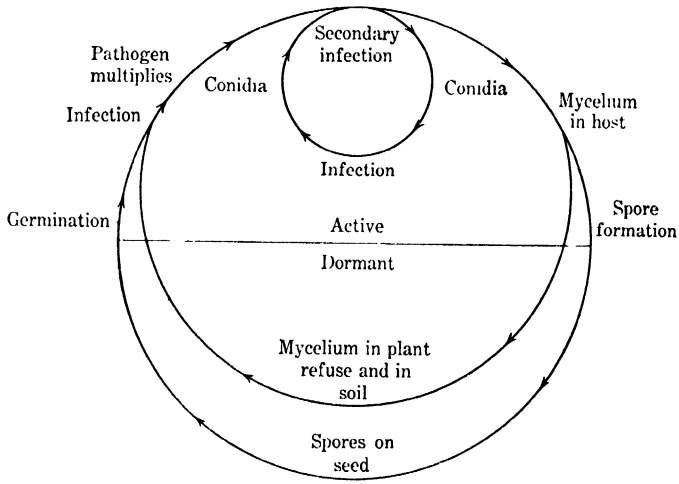


FIG. 194. A diagram of the host relation of *Fusarium vasinfectum*. This organism survives the winter in the soil and on the seed.

generally septate. In addition there often are formed spherical, thick-walled chlamydospores either at the ends or in the middle of the hyphae. The fungus is said to be disseminated by the carrying of these spores on implements, animals, drainage water, wind, etc. There seems to be no evidence of natural seed infection, although it has been obtained artificially.

The fungus seems to grow best in well-aerated soils that are low in nutrients. The disease is most prevalent in plants growing in light sandy soils, which probably is explained by the fact that the maintenance of proper nutrient and

water relations of the soil produces vigorous plants that in part resist the pathogen. The greatest amount of disease appears at soil temperatures of 28° to 30° C., while there is little or no disease above 36° or below 18° C. The percentage of infected plants is highest in soil at 50 to 60 per cent water capacity content and under an alkaline reaction. Certain Egyptian strains of the pathogen are known to be more virulent in acid soils.

CONTROL.—Since the fungus produces such a large number of easily disseminated spores and lives for several years in the soil, crop rotation is of little value as a control method. The crop rotation using nematode-resistant plants does help, however, by lowering the root injuries caused by nematodes, which give the fungus a point of entry. The fungus commonly occurs in soils deficient in nutrients and it is known that the use of fertilizers having a high potassium content is a good control measure. The use of 600 pounds per acre of a 6–6–6 or 6–8–12 (NPK) fertilizer is suggested.

Greatest relief from the infestation of the cotton soil is through the use of varieties of cotton that show a high degree of resistance, as Dixie Triumph and Dixie 14 of the short staple cottons, and Superseven, Miller and Arkansas Rowden 40 of the longer staples. The use of resistant varieties accompanied by the use of potash fertilizers appears to be the best control practice.

Chapter Thirteen

DISEASES CAUSED BY BASIDIOMYCETES

MANY of our most destructive plant pathogens are Basidiomycetes, or stalk fungi, e.g., the smuts, rusts and fleshy fungi. Just as the ascus characterized the sac fungi, so a club-shaped, single- or several-celled stalk structure (basidium) bearing usually four single-celled spores, characterizes the Basidiomycetes. Although the usual number of spores is four, it is not uncommon in some species to have more and in other species less. The mycelium is usually many-celled and binucleate, although here again exceptions are common.

As a group the Basidiomycetes are more familiar to the layman than any of the other groups studied. The smuts and rusts are well known on our cereal crops, and the fleshy fungi (mushrooms and bracket fungi), because of their prevalence and size, are familiar objects on decaying organic matter in the soil, on fallen logs, stumps, lumber and living trees. These three groups of the stalk fungi will be discussed in greater detail in the following pages.

SMUTS

The smut fungi usually are considered the most primitive Basidiomycetes, having a rather simple structure and life cycle. They are all parasitic in habit, living chiefly on herbaceous flowering plants. The grasses are their most common hosts, although they are known to attack plants of some 35 families of spermatophytes and several species have been described on ferns. The mycelium of the smuts is septate, branched, mostly intercellular and practically limited to the interior of the host. The mycelial stage of the smut fungi is heterothallic, and may, therefore, be either haploid or binucle-

ate, depending upon whether the number of nuclei has doubled through cellular fusion, which may occur in two stages. First there is a cytoplasmic fusion of two haploid protoplasts, which produces a binucleate mycelium. Later there occurs in this binucleate mycelium a nuclear fusion, which results in the uninucleate diploid condition. At maturity the fertile hyphae become abundantly septate, the outer walls gelatinize and a heavy wall is laid down about the rounded cells, the chlamydospores. They are usually dark colored and may occur singly, in pairs or in spore balls. The nuclear fusion occurs in the chlamydospore. These spores may germinate immediately or may serve as resting spores. Upon germination, however, each spore puts out a short, thick tube, the promycelium, on which small, single-celled, thin-walled haploid spores are produced, known as sporidia. Like the mycelium the sporidia may be of different sexes. In some cases the sporidia fuse before entering the host, while in other cases the sporidia form haploid mycelia in which fusions take place, forming binucleate mycelia after penetrating the host. It is the binucleate mycelium that actively and markedly parasitizes the host and not the haploid.

Certain smuts attack their host plants at flowering time, infecting the developing ovary and seed, and become dormant as the seed matures. When the seed is sown in the spring, the mycelium of the parasite follows along in the growing point of the developing plant. In certain other smuts the spores are scattered by the wind at harvest time and lodge on the surface of the seed. The spores germinate when the seed starts to sprout, and the mycelium enters the seedling before it emerges from the ground. Since the mycelium then grows in the growing point of the host, it may be present in the young flower bud. In still another group, the spores are liberated into the air, soil or manure and are not associated with the seed but may attack the plant at any stage of its development when susceptible host tissue is encountered under favorable environmental conditions. This association dictates the control measures and permits the following grouping of some cereal smuts:

GROUP A. Non-association of the pathogen with seed.
Corn smut (*Ustilago zeae*).
Head smut (*Sorosporium reilianum*) of sorghum.
Onion smut (*Urocystis cepulae*).

GROUP B. Direct-association of the pathogen with seed.

Class I. On the surface of the seed as spores.
Loose smut (*Ustilago avenae*) of oats.
Covered smut (*Ustilago levis*) of oats.
Stinking smut (*Tilletia levis*) and (*Tilletia tritici*) of wheat.
Flag smut (*Urocystis tritici*) of wheat.
Stem smut (*Urocystis occulta*) of rye.
Covered smut (*Ustilago hordei*) of barley.
Kernel smut (*Sphacelotheca cruenta*) and (*Sphacelotheca sorghi*) of sorghum.

Class II. In the seed as mycelium.
Brown loose smut (*Ustilago nuda*) of barley.
Loose smut (*Ustilago tritici*) of wheat.
Smut (*Ustilago striaeformis*) of timothy.
Smut (*Ustilago striaeformis*) of red top.
Covered smut [1] (*Ustilago levis*) of oats.
Loose smut [1] (*Ustilago avenae*) of oats.

Group A cannot be controlled by seed treatment, because infection does not result from seed-borne spores or mycelium. Group B, class I, may be controlled by surface disinfection in most cases, since the parasite is on the surface of the seed, and the spores may cause infection of the seedling.

In control, when the mycelium is internal, as in class II, advantage is taken of the difference between the thermal death point of the pathogen and the embryo in the seed. If the mycelium is confined to the superficial parts of the seed, however, surface disinfection is effective.

CORN SMUT

Ustilago zeae (Beckm.) Unger

Corn smut is perhaps the best known and most widely recognized disease of the maize plant. The pathogen has been the subject of investigation since the classic studies of

[1] The pathogens causing loose and covered smut of oats also may remain as spores on the surface of the seed and cause infection when the seed germinates.

Brefeld in 1883 in which he first worked out the method of infection and life history of the corn smut fungus.

The yield reduction attributed to corn smut is variable, depending upon the amount of infection and the environment. It varies with the number of galls, e.g., one gall reduces the yield about 25 per cent and two, 50 per cent. The yield reduction in 1928 for the United States was greater than average (3.5 to 5 per cent) from Ohio westward to Kansas and in the dry farming regions of Colorado, California and Arizona. Estimated reductions in 1928 ranged from 15 per cent in California to 0.3 per cent in Maryland, while the estimated reduction for the whole United States was 4 per cent. The prevalence of corn smut in 1929 was considered about average, with an unusual amount of ear infection reported from Connecticut, Pennsylvania, Ohio and Iowa. In Mississippi and Arkansas corn smut was especially prevalent in some of the overflowed river valleys where corn was grown as a late crop. In the same year, infection ranged from 5 per cent in Ohio, Nebraska and Iowa to a trace in states west of the Rocky Mountains, while the estimated reduction in yield for the United States was 3.2 per cent. In 1930 reported infection varied from 5 per cent in West Virginia, Iowa and Nebraska to a trace in the western states, causing an estimated reduction of 2 per cent. Data presented for the year 1933 indicated corn smut as unusually prevalent in Massachusetts and some of the Great Lake states, the greatest damage occurring in sweet corn. The damage ranged from 15 per cent in sweet corn in Michigan to a trace in some of the less important corn growing regions. The estimated reduction in field corn for the United States in 1933 was approximately 3.3 per cent or 70,800,000 bushels and in 1934 about 4.6 per cent, amounting to 55,526,000 bushels.

Symptoms.—Corn smut symptoms appear on the various aerial parts of the corn plant, anywhere from the tip of the tassel to the ground line. They include chlorophyl changes, as blotch and striping, the formation of anthocyan pigment and hyperplasia and necrosis of infected organs.

One of the earliest symptoms to appear, and one most frequently overlooked, is yellowing of the leaves resulting from a reduction in the amount of chlorophyl in localized infected areas. Leaf infection often does not induce smut galls because of the maturation of the tissues, but remains throughout the life of the plant as yellowish irregular areas or streaks. Yellowing may occur at any point from the tip to the base of the leaf blade, while the infected areas may range in size from minute streaks between the fibrovascular bundles to elongated streaks extending practically the entire length of the leaf. Infected areas also may occur in irregular shapes, which do not seem to be limited by the vascular bundles and, as streaking, may be limited to minute spots or in some cases may include the entire leaf. Such symptoms are easily detected on young plants but become increasingly difficult to identify as the corn plant matures, and in some cases the changes of the affected area may entirely disappear.

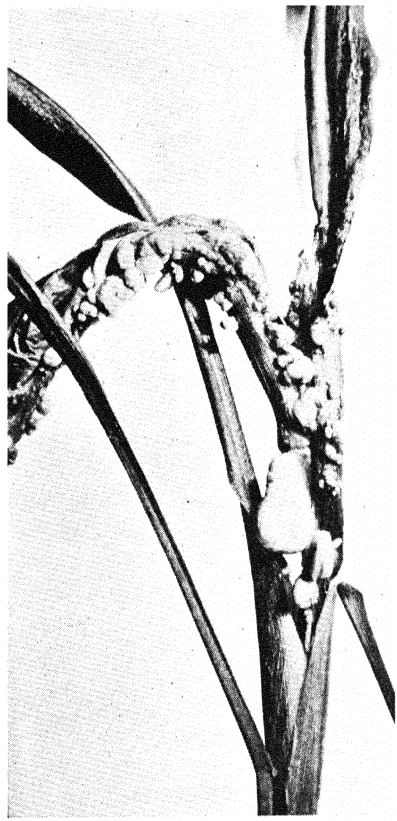


FIG. 195. Foliar smut galls on maize.

Infected areas may appear on the leaves of corn plants as reddish or purple irregular blotches or streaks. Like yellowing, the discolored areas may appear anywhere on the leaf blade and vary in size from minute spots to larger regions covering most of the leaf. The red or purple areas may appear in solid spots or they may completely encircle yellowed

areas or regions of gall production. When portions of a leaf bearing these discolored areas are examined under the microscope, it is seen that many of the epidermal cells contain a red coloring matter, anthocyan, in solution in the cell sap, and occasionally this red substance separates out in crystals.

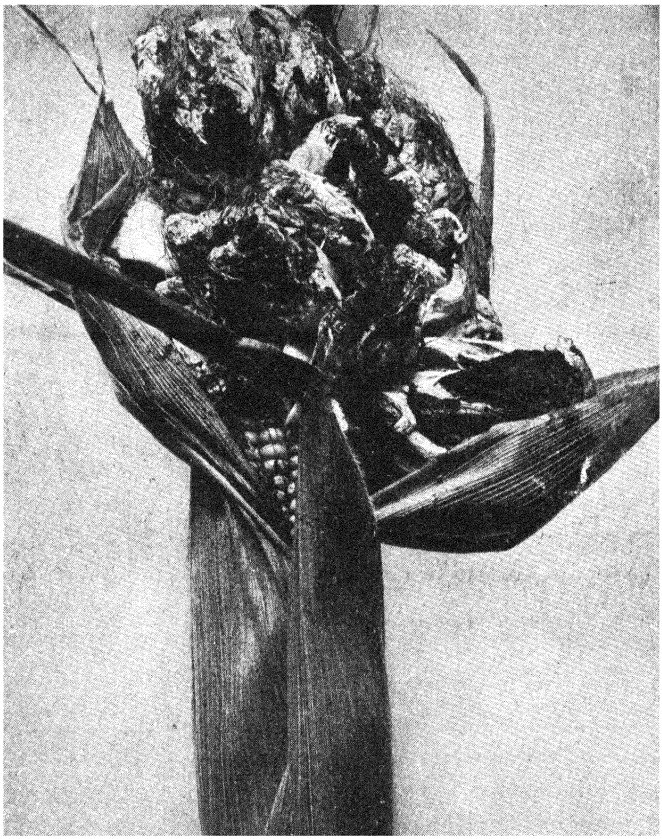


FIG. 196. Smut galls on single kernels on the tip of an ear.

The symptom most easily recognized is the formation of galls caused by the hypertrophy of the infected tissues. Smut galls may vary in size from minute pustules on the leaves to several inches in diameter on the stalks or ears. They are at first of a whitish-gray color because of a covering

membrane of host tissues. Later the galls turn black with the development of the enclosed smut mass, and the covering membrane dries and ruptures, exposing the dry powdery mass of black spores.

Smut galls may appear at almost any place where meristematic tissue occurs. They are common on or near the midrib of the leaves, at the junction of the leaf sheath and blade or at the nodal buds on the stems. Individual flowers of the ear, groups of flowers or the entire ear may be involved. In the staminate inflorescence the individual organs may be converted into smut galls. Many smut galls occurring at the nodes below the ear are commonly overlooked. They are formed in the tissue of the axillary buds and consist of masses of spores varying in size from those barely recognizable with the unaided eye to elongated flattened galls several inches in length. Often when the pressure of the galls is not sufficient to rupture the leaf sheath, the galls may encircle the stalk inside the sheath. Such smut galls are completely enclosed and hidden by the leaf sheath and may be detected only by stripping the leaves from the host.

Etiology.—The corn smut disease is caused by a fungus known as *Ustilago zeae*. Its host relation is shown diagrammatically in Fig. 197. The mycelium is many-celled, much-branched, largely binucleate and intercellular. The chlamydospores, which are liberated by the breaking of the membrane enclosing the galls, germinate forming a promycelium, which in turn produces sporidia laterally and terminally. The reduction division takes place when the chlamydospores germinate and form the promycelium. The sporidia are haploid. Since the smut organism is heterothallic, the two sexes are separated in the process of reduction division previous to sporidial production, and prevail in approximately equal numbers.

Early in the growing season, the chlamydospores and sporidia are blown about by the wind. Some of them may fall into the spiral whorl and come to rest upon the growing point or very young leaves of the corn plant, where they may germinate and cause infection. The corn plant is only

susceptible to infection as long as new tissues are being formed. This usually ceases when the plant is about 18 inches tall. Subsequently, the plant is no longer liable to infection unless it is stimulated to form new tissues through injury.

If hyphae of opposite sexes fuse with one another, nuclear migration from a cell of one to that of another takes place.

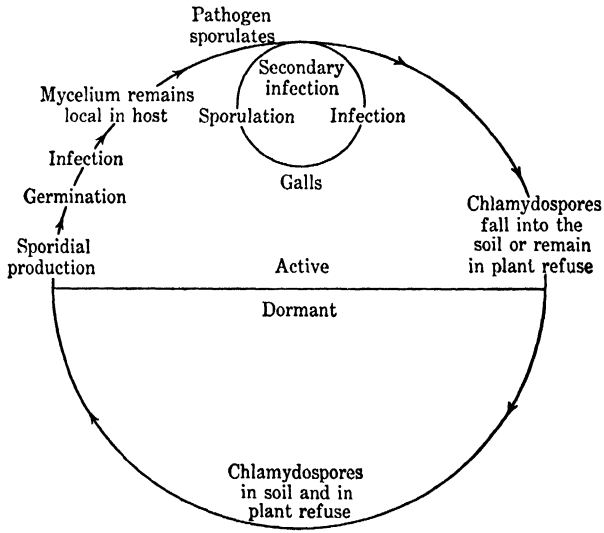


FIG. 197. A diagram of the host relation of *Ustilago zeae*. The organism survives the winter in the chlamydospore stage on plant refuse and in the soil.

From this binucleate cell arises a new hypha, known as the fusion hypha, which develops more vigorously than the uninucleate mycelium. Uninucleate mycelia probably never develop sufficiently to form galls or chlamydospores.

When the host approaches maturity and the galls start to dry out, the smut mycelium segments. Each segment becomes spherical in shape and takes on a thick, slightly roughened dark wall. When the galls dry the great mass of spores appears as a brown dust. These chlamydospores may germinate at once or remain dormant through unfavorable periods of summer drought or winter cold.

In the corn smut organism there are several races that

develop more vigorously on certain varieties of corn than on others. Just how many of these different races exist under natural conditions in the open is not known, but that there are several is perfectly clear. Such being the case, a variety of corn may seem to be more resistant when grown in one locality than when grown in another.

The corn smut organism flourishes best at relatively high temperatures. The optimum temperature of chlamydospore germination lies between 26° and 32° C. with a maximum at 36° to 38° C. and a minimum at about 8° C. The same temperature range applies to the germination of the sporidia. Chlamydospore germination is favored in nutrients, as manure decoctions, soil filtrates or dilute sugar solutions. Sporidial production is most profuse where the promycelia extend above the liquid medium. The sporidia are thin-walled and not very resistant to atmospheric conditions that prevail in the open. Most sporidia are killed by alternate freezing and thawing.

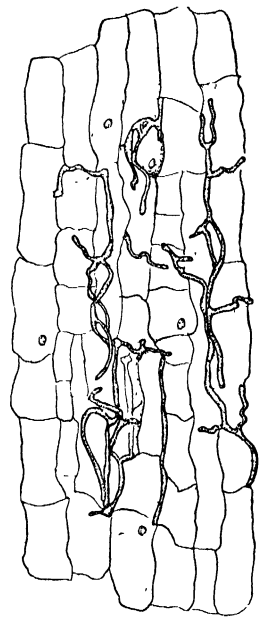


FIG. 198. Smut mycelium in the tissues of a leaf (husk) of an apparently disease-free axillary bud. (After G. N. Davis.)

Moisture in the form of rain and dew has little effect on infection, providing there is ample for the normal development of the host. The susceptible host tissues are deeply seated in the young corn plant where moisture is adequate for infection.

CONTROL.—The type of infection in corn smut precludes the possibility of seed treatment. The rate of planting seems to influence the development of smut galls. Two to three plants per hill show less smut than either a larger or smaller number. Where corn is grown extensively, no control measures are known that will markedly decrease the amount of smut. This is because of the proximity of fields, presence of

smut spores in barnyard manure, and the wind-blown chlamydospores and sporidia. Some reduction in the amount of smut may be effected through crop rotation, however, avoiding injury to the plants in cultivating, in regulating the stand to three plants per hill (in Iowa) and in using as far as possible varieties, strains or hybrids known to possess some degree of resistance. Where smut is abundant in a field, the inoculum may be effectively and profitably destroyed by cutting the crop and making silage; the fermentation process kills the spores.

Oat Smuts

Ustilago avenae (Pers.) Jens. and *Ustilago levis* (K. and S.) Magn.

The oat smuts occur wherever oats are grown. There are two smuts that attack oats, known as loose smut and covered smut. The pathogens may infect the plant in either the seedling or flower stage and become systemic, ordinarily sporulating in some or all of the floral organs. As a rule the oat smuts do not destroy a high percentage of the crop. An epiphytotic may develop, however, causing serious reductions in yield. In South Carolina in 1927 it is estimated that there was 3.5 per cent reduction in yield, while head counts showed 90 per cent in many fields. Previous to 1906 it was estimated that the average loss from such smuts in this country was about $20,000,000. The loss in the United States was estimated at 91,000,000 bushels in 1917. In more recent years the yield reduction has been considerably ameliorated by seed treatment and the development of smut-resistant oat varieties. The average reduction during the ten-year period from 1925 to 1934 in the United States was 3.9 per cent with a maximum reduction of 62,776,000 bushels in 1928.

There are at least seven species of oats, namely: *Avena sativa* L., *A. byzantina* C. Koch, *A. orientalis* Schreb., *A. nuda* L., *A. strigosa* Schreb., *A. abysinica* Hochst. and *A. fatua* L. The cultivated oat varieties in the United States belong to the first four species. The number of varieties of oats in existence

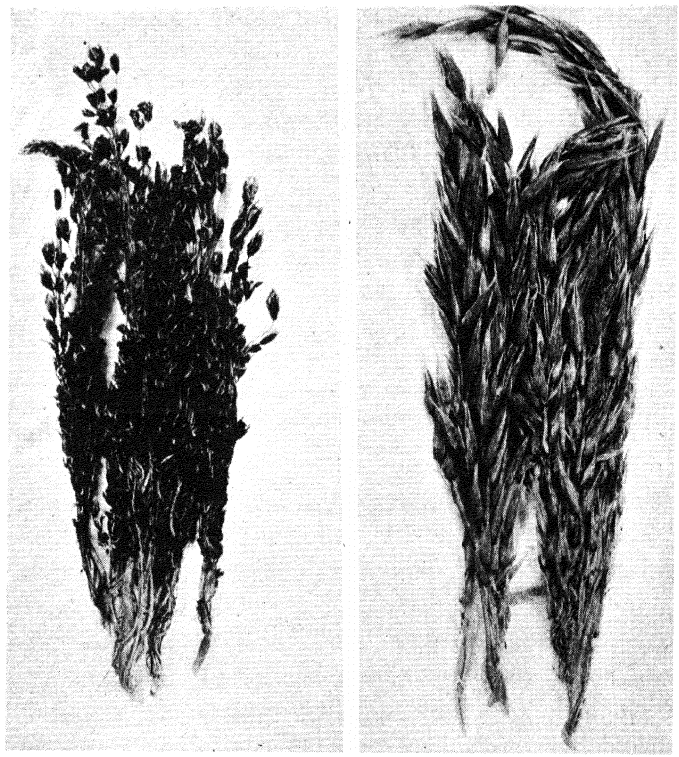
is large, probably about 500. The above-named species vary markedly in resistance and susceptibility. Most of the varieties are susceptible, although some are either moderately or highly resistant.

SYMPTOMS.—In general, loose smut transforms the entire panicle into a dark, dusty powder, while in covered smut only the grains or kernels are destroyed. In the former disease the glumes are destroyed, while with the latter they are uninjured. In both diseases the organism enters the ovary from the growing point when the flower is formed. The intercellular mycelium spreads generally in the floral parts and maturing seed, resulting in complete necrosis of the infected organs. At this stage most of the mycelium segments; each cell rounds up and takes on a heavy, smooth or echinulate brown cell wall, forming finally a dry powdery mass of soot-colored spores. The spores may shatter out, leaving only the naked branches of the panicle. In covered smut only the kernel and inner set of glumes are destroyed. This is not, however, a constant reaction of the parasitic relationship.

ETIOLOGY.—The host relations of these two organisms are so much alike that they may be discussed together. (See Fig. 200.) The organisms that cause the oat smuts are *Ustilago avenae* and *U. levis*. These two species produce much-branched, hyaline, septate, binucleate mycelia and two types of spores, binucleate chlamydospores and uninucleate sporidia.

The black, dust-like particles are the diploid chlamydospores, which are oval to globose, from five to eleven microns in diameter. In *Ustilago levis* the surface of the spore is smooth, while in *U. avenae* the spore wall is usually spiny. When the chlamydospores are allowed to mature, they may germinate at once or may remain dormant under favorable conditions for several years. When placed in water or a nutrient solution the spores produce a one- to three-septate tube or promycelium on which are borne several crops each of four small, long to oval, hyaline, thin-walled sporidia. Reduction division takes place in the

development of the promycelium. The sporidia are haploid and heterothallic. These haploid sporidia are unable to cause infection of the oat plant until fusion to produce a



X Z

FIG. 199. Oat smut: X, panicles of oats showing loose smut caused by *Ustilago avenae;* Z, panicles showing covered smut caused by *U. levis*.

binucleate sporidium has occurred and an infection hypha developed. This comes about when two sporidia of opposite sex come to lie within about five microns of each other, through the development of a hypha from one of the sporidia uniting another of the opposite sex. The nucleus of the second sporidium migrates into the hypha connecting the two sporidia and establishes the binucleate condition. From this initial binucleate hypha develops a branch, also binu-

cleate, known as the fusion hypha, which parasitizes the host and reproduces the chlamydospore stage.

Oat smut infection may take place in either the seedling or flower stage. In the case of seedling infection, the viable spores lodged on the surface germinate under favorable

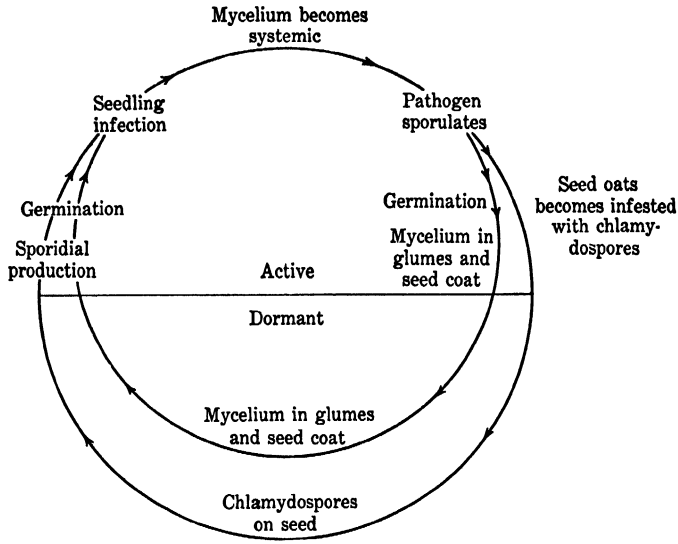


FIG. 200. A diagram of the host relation of either *Ustilago avenae* or *U. levis*. The organism survives the winter in the chlamydospore stage on the seed or as mycelium in the glumes and seed coat.

conditions, producing an infection hypha capable of penetrating the young oat seedling. The pathogen establishes itself in the formative tissue of the growing point of the host. Flower infection occurs after spores fall into the flower, germinating and developing an infection hypha, which penetrates the young formative tissue of the flower. When the seed matures, the mycelium assumes a resting condition in the glumes or pericarp and remains dormant until the seed is placed under favorable growing conditions when it again becomes active and spreads into the formative tissue of the developing seedling. In each type of infection, the pathogen becomes systemic in the host.

In *Ustilago avenae* spore germination may take place between

4° and 34° C., minimum 4° to 5° C., optimum 15° to 25° C. and maximum 31° to 34° C. Soil moisture also influences germination of the chlamydospores. It is greatest at 30 per cent soil moisture-holding capacity and least at 80 per cent. For oats, the optimum temperature for seed germination is 18° to 20° C. Thus, there is approximate coincidence for the pathogen and host insofar as growth conditions are concerned. However, since infection is limited to embryonic tissues, conditions that promote rapid growth of the host permit the least infection. Investigations have shown that high temperatures and soil moisture conditions will reduce the amount of smut, while low soil moistures coupled with low temperatures, favor smut infections. It has been found that the highest infection on *Avena nuda* occurs at 25° C. with a low soil moisture. A neutral to slightly acid soil reaction is most favorable for infection.

There are many races of the two species of smut fungi causing oat smuts. These races vary in the extent that they attack different varieties of oats. The resistance of a particular variety of oats in a given locality naturally is influenced by the races that are common in the locality. Races may be introduced from one region to another or may be brought into existence by hybridization between existing races.

CONTROL.—The oat smuts may be effectively controlled through seed treatment at a very nominal cost, less than four cents a bushel. This control may be effected by either wet or dry fungicides. The wet method of treatment, using dilute formaldehyde, has largely been replaced by the dry methods involving formaldehyde and mercury dusts.

In the wet method, the grain is soaked or sprinkled with dilute formaldehyde solutions (one pint in 30 gallons of water) for 30 minutes, covered with a canvas for two hours and spread out to dry on a clean floor or canvas. Thirty gallons of solution will treat from 50 to 70 bushels of oats. A more concentrated solution of formaldehyde (one pint in ten gallons of water) may be sprayed on the grain.

The dry fungicides are applied by machines that shake or stir the grain, coating it with the dust. It requires from

one-half to two ounces (depending upon the product) of the dusts to treat one bushel of grain. The dust treatments are proving more practical than the wet treatments.

The time of seeding influences the damage caused by oat smuts. In the middle west, early seeding, causing slow germination, facilitates infection and increases the amount of smut in the crop. Oats should not be seeded until the temperature and soil conditions are favorable for rapid germination and growth.

The amount of smut can be reduced by selecting resistant varieties. Among the varieties of *Avena sativa*, Marion, Boone, Hancock, Markton, Black Mesdag, Brunker and Carleton seem to be highly resistant. Formerly all varieties of *A. fatua* and *A. nuda* were considered highly susceptible, although selections of *A. nuda* are now available that are highly resistant.

Onion Smut

Urocystis cepulae Frost

Onion smut is one of the most important diseases in the northern onion growing regions of the United States. The disease was first reported from the Connecticut valley in 1869. The pathogen has since spread into most of the large onion sections including New York, New Jersey, Ohio, Wisconsin, Illinois, Iowa, Oregon, etc. Although the soils of the southern states have had equal opportunity to become infested with the pathogen, the disease is not known in that territory because the soil temperatures are unfavorable for its development. Until the formaldehyde drip method was discovered, onion smut caused commercial growers heavy losses. Many growers turned to other crops rather than suffer the yield reductions. Even since the discovery of an effective remedy, the losses in stand may equal 30 to 40 per cent when treatment has been omitted or imperfectly done. Such stand reductions influence the yield. Onion smut occurs not only on the cultivated onion, but also on many wild species in the genus *Allium*. In Europe onion smut also

occurs on leeks, shallots, chives and the Welch onion. Almost all varieties of the common onion are susceptible.

SYMPTOMS.—Onion smut is chiefly a seedling trouble, appearing in the crop soon after the seedlings emerge. Light yellow spots and streaks form in the cotyledon, which enlarge and turn dark. Later the epidermis breaks, exposing the black powdery mass of fungous spores. Many of the

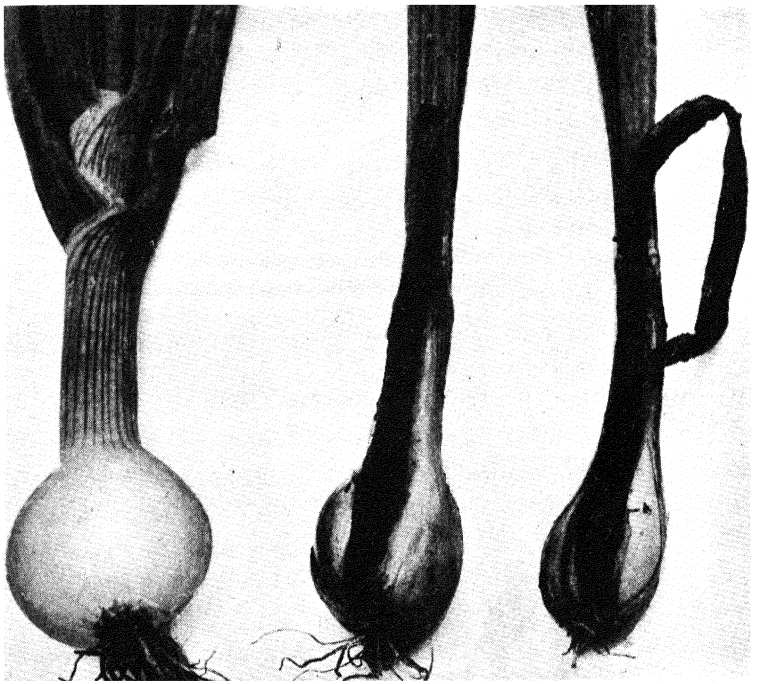


FIG. 201. The two onions at the right show smut pustules and lesions produced by *Urocystis cepulae*.

plants attacked in the seedling stage are killed. Some of those that survive may be distorted and stunted; few recover and usually the stand is seriously decreased. When infection takes place at or near the junction of the root and stem the pathogen may find its way into the growing point and the first true leaf. In such cases the parasite continues to spread to the new leaves as they develop. The infected leaves, like the cotyledons, become distorted, enlarged and dis-

colored because of the developing smut sori, and finally the epidermis of the leaves ruptures exposing the black powdery mass of chlamydospores. When the bulb scales become infected the sori become large and contain large masses of spores. Infected plants in all stages of development may wilt down, dry up and disappear, and few infected bulbs ever attain marketable size.

ETIOLOGY.—Onion smut is caused by *Urocystis cepulae*, one of the soil-inhabiting basidiomycetous fungi that attacks its host only in the seedling stage. The spores of the pathogen consist of spherical cellular masses called spore balls. Only the center cell of each ball is fertile and capable of germination; the others are sterile. Germination begins in three to six days after the spores are brought under favorable conditions. The optimum temperature for germination of the spores ranges between 13° and 22° C., and at 25° C. a marked reduction in germination results. A short hemispherical promycelium develops bearing a whorl of branches. These branches may continue to grow as mycelia indefinitely without forming sporidia. The hyphae become septate and new branches may arise just below each septum. The ultimate effect is a mat of hyphae so dense as to obscure the spore ball. The host relation of the pathogen is illustrated in Fig. 202.

The onion smut organism is able to live and grow in the soil, utilizing the organic matter present, for a period of twelve years. The temperature range for the growth of the mycelium is the same as for spore germination. The saprophytic mycelium in the soil may penetrate seedling onions at any point between the tip of the cotyledon and the root joint. The onion is susceptible only in the early seedling stages (12 to 20 days). The mycelium of the pathogen penetrates the cuticle and enters the epidermal cells, and once inside the host cells the hyphae branch out and grow through the cell into the intercellular spaces next to the epidermis of the cotyledon. In these initial stages of invasion, the parasite seems to have little effect on the host cell protoplasts. When the host tissues have become extensively invaded, the pathogen proceeds to sporulate and the infected cells collapse.

The mycelium becomes binucleate only after it enters the host. The saprophytic mycelium is always uninucleate. Doubling of the nuclei always prevails previous to sorus formation. Just how the diploid condition comes about is not definitely known.

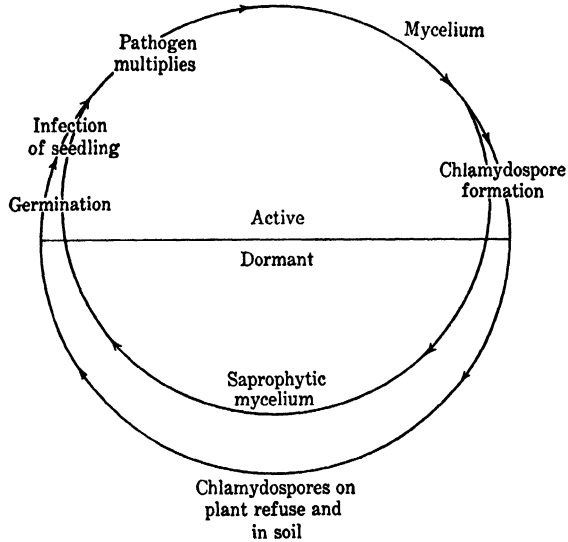


FIG. 202. A diagram of the host relation of *Urocystis cepulae*. This organism survives the winter in the chlamydospore stage on plant refuse and in the soil.

Soil temperatures of 10° to 25° C. favor infection, but little infection occurs at 27° C. and none at all at 29° C. Soil moisture seems to exercise less influence on the pathogen than on the host. Infection takes place regardless of the soil moisture content. The fungus is chiefly a soil organism and is spread by farm implements, feet of man and animals, surface water and dust. The pathogen may also be carried on sets, and its spores have been found on the onion seed. Sets do not become infected when planted in infested soil.

CONTROL.—There are three avenues open to the grower whose land has become infested with the onion smut organism. He may practice (1) a long rotation, 10 to 15 years, (2) plant onion sets instead of seed or (3) treat the soil

at seeding time. The first two methods are not very practical for most growers, while the third method is easily applied and effective where seed is sown in infested soil. It is worthy of emphasis that in all other smut control measures the fungicide is applied to the seed, but onion smut control involves primarily the destruction of the saprophytic myce-

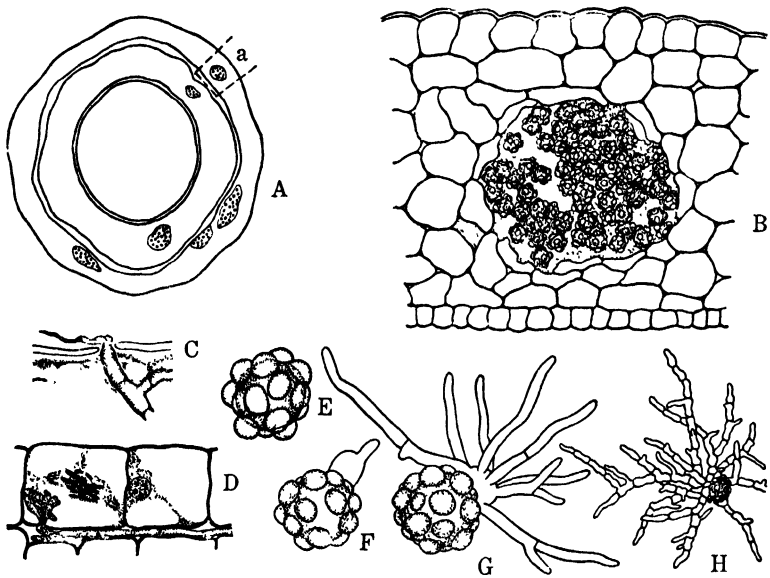


FIG. 203. *Urocystis cepulae:* A, cross section showing smut galls in young onion leaves; B, an enlargement of pustule shown at a; C, Penetration of epidermal cell; D, intercellular hyphae with haustorium in disintegrating cell; E, chlamydospore; F, G and H, germination of chlamydospore. (C redrawn from R. I. Evans and D, G & H redrawn from P. J. Anderson.)

lium and spores in the immediate vicinity of the seed and seedling. The seed and soil may be treated with formaldehyde, which is dripped into the furrow with the seed at planting time, using one pint in 16 gallons of water, applied at the rate of 200 gallons per acre or one gallon to each 185 feet of row. When seed is sown for sets, the flow should be increased proportionately to the rate of seeding. Onion refuse should not be returned to the soil. Warehouse wastes, especially, may carry the fungus.

Stinking Smut or Bunt of Wheat

Tilletia levis Kühn and *Tilletia tritici* (Bjerk.) Wint.

Stinking smut or bunt is world-wide in distribution. It has been known for more than 2,000 years and is a serious menace to the chief bread crop of the human race. The chief losses from bunt comprise decreases in yield and dockage in price of smutted grain caused by the fish-like odor imparted by the smut spores, and the necessity of scouring the smutty grain preliminary to milling. Another loss may result from explosions and fire of threshing machines. The large number of spores in the air at this time forms a highly inflammable mixture, which explodes when an electric current is passed through it.

The yield reductions in the United States are variable from year to year. The reduction in 1931 was 12,749,000 bushels; in 1932, 12,613,000 bushels; in 1933, 8,762,000 bushels, and in 1934, 5,382,000 bushels. In the different wheat growing states the yield reductions may range from 1 to 20 per cent depending upon seasonal variations. Previous to 1900 bunt threatened to become a limiting factor in wheat production in western Canada.

In addition to yield reductions, losses result from price dockage because of the smut in the wheat. Of the 17,973 cars of Montana wheat shipped to market in 1928, 12.3 per cent, or 9,463,620 bushels, were smutted. At a dockage of four cents a bushel the loss would total $378,545. In 1932 4,300 cars of Minnesota wheat were received at the Minneapolis terminal, and 23.2 per cent graded smutty. Until ways were devised for the prevention of explosion and fires resulting from the abundance of smut spores in the threshing process, many machines and much grain were burned up, amounting to a loss of thousands of dollars annually.

There are two different fungi that cause the bunt disease. *Tilletia levis* is more common in this country than *T. tritici*. The first named species, however, is said to be less common west of the Rocky Mountains in the famous wheat growing

section of the Palouse valley of Oregon and Washington. In Canada *T. tritici* is more common than *T. levis*.

SYMPTOMS.—The bunt organisms transform the wheat kernel into a slightly enlarged mass of smut balls, leaving the glumes entirely free. In the field three symptoms are usually evident in that the glumes are set at a slightly wider angle, the heads do not nod and the diseased culms are not as tall as the healthy ones. Sometimes the smut will show through the glumes and the spike will appear darker than normal. Another distinguishing characteristic is the odor, which has been likened to herring brine and is due to the presence of trimethylamine. Where the two smuts occur in the same field it is held that they may be distinguished by the difference in height of the head at the time the smut matures. Thus, *Tilletia tritici* may cause "low" bunt, while *T. levis* may cause "high" bunt. It also has been shown that the former has a more western distribution.



FIG. 204. Bunt or stinking smut of wheat. A healthy head at the left.

ETIOLOGY.—The causal organisms of bunt are *Tilletia levis* and *T. tritici*. They are alike in most of their characteristics, perhaps the chief point of difference being that the spores of the former are smooth, of the latter, finely reticulate. These spores are spherical, 18 to 24 microns in diameter and formed inside the seed coat of the infected "berry." They are scattered by the breaking of the smut balls at harvesting and threshing time. They may adhere to the

threshed grain or blow into the air and finally descend on the soil in a so-called "smut shower" in the semi-arid northwest. In the humid regions the smut spores in the soil are of little importance, since they germinate immediately and the sporidia are short-lived. In the dry regions, the same rain that favors the germination of the seed in the fall also favors the germination of the fungous spores. In this way the spores in the soil are often the more important source of infection.

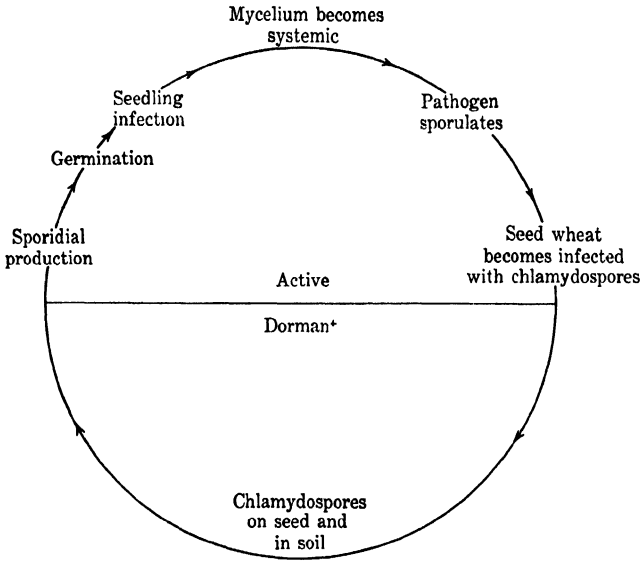


FIG. 205. A diagram of the host relation of either *Tilletia levis* or *T. tritici*. The organism survives the winter in the chlamydospore stage on the seed or in the soil.

The spores germinate at temperatures of 0.5° to 29° C., optimum 18° to 20° C., to form a short promycelium and a crown of sickle-shaped sporidia, which fuse in pairs and produce a secondary mycelium, bearing secondary sporidia, which are forcibly discharged. The secondary sporidia produce the infection threads, which may enter any portion of the coleoptile of the wheat seedling. The mycelium penetrates to the growing point where it persists throughout the vegetative stage. Ultimately the pathogen grows just below the meristematic tissues of the growing point until flowering when it

enters the ovaries and converts the spikelets into a smutty mass. The host relation of the bunt organisms is shown diagrammatically in Fig. 205.

Recent investigations show that infection takes place below 20° C. and that the optimum lies between 5° and 10° C. Moisture is also an important factor within certain limits, the higher the moisture content of the soil, the greater the amount of infection up to 22 per cent of the water-holding capacity of the soil. The spores lose their viability within 30 days in a soil with a moisture content of more than 22 per cent. The amount of infection in plants is directly correlated with the depth of planting in a given soil. The number of spores present on the seed also determines the amount of smut that will result.

Although a number of races have been described for each species, recent investigations indicate that eleven races of *T. tritici* and eight of *T. levis* can be distinguished when tested under identical conditions on varieties of winter and spring wheats. Temperature appears to affect the virulence of the pathogens and the resistance of the host.

CONTROL.—Control measures for bunt vary more or less with the geographical location. In the humid regions of the east and middle west, formaldehyde, copper carbonate and the mercury dusts are used. Before dipping in formaldehyde solution the grain should be thoroughly fanned, and during the treatment the seed should be stirred and the floating smutted kernels skimmed off. In the drier parts of the west, formaldehyde has been shown to injure the seed if it is sown in a dry soil. It also fails to prevent infection resulting from infested soil.

In view of the fact that treatments with dust fungicides have been discovered that are as effective as the wet treatments and more easily applied, dusting has become largely universal. Dusting the seed with copper carbonate, three ounces to the bushel, has been found effective in preventing infection not only from spores on the seed, but also from spores in the soil. In humid regions, a tendency for the dust to cake and bind the machinery has made it undesirable,

especially with certain types of seeders. Recent work has shown that the ethyl mercury salts mixed with an inert dry filler give excellent control. The dust is applied at the rate of one-half ounce of dust per bushel of wheat.

Varieties differ widely in resistance. The hard red winter wheats are the most resistant and the soft wheats most susceptible, although one variety of the latter, Florence, is highly resistant and several others show more than average resistance. The most promising of these are Martin and three strains of White Odessa. The hard red spring and soft red winter varieties are intermediate in susceptibility. Another variety known as Banner Berkeley is also said to be very resistant. The club wheats are very susceptible as a class; while durum and Polish wheats as well as emmer and spelt are, in general, more resistant than the common wheats. Resistance to bunt seems to be of two types: First, resistance to infection; and second, resistance after infection has occurred, as shown by the partial smutting of the spike.

Loose Smut of Wheat
Ustilago tritici (Pers.) Jens.

This smut is different from those previously studied in that the young embryo becomes infected in the early formative stages in the flower. Flower infection in this smut has been known less than 40 years. Previously many different explanations of how infection took place were current.

Loose smut of wheat seems to occur wherever wheat is grown. The annual loss in the United States is estimated at 10,000,000 bushels. It appears to be most prevalent and destructive in the more humid regions, and less severe in the more arid sections such as the northwest. The wide range in varietal resistance, however, may influence the prevalence of the smut in a given section. Certain varieties of wheat are highly resistant, but most sorts are susceptible. The only other hosts known other than species of *Triticum* are rye and barley. Its occurrence on these hosts, however, is of little economic importance.

Symptoms.—Loose smut of wheat as the loose smut of

oats described above destroys all the floral organs. The black smut masses are covered, before they dry out, by a delicate membrane, which is finally broken before the head emerges from the leaf sheath. The spore mass disintegrates and is soon distributed by the wind, leaving only the bare rachis of the spike. The smutted heads appear just at the flowering time of the healthy heads and afford a striking contrast between the naked diseased and dark-green healthy spikes. It sometimes happens that not all the spikelets of a head are destroyed. In such cases it is usually those near the tip of the head that escape. In rare cases the smut may develop on the leaves and leaf sheaths. Generally all the heads in a stool are infected, but sometimes a head may escape.



FIG. 206. A, head of healthy wheat; B and C, heads largely destroyed by the loose smut pathogen.

As the crop matures the smutted plants become less conspicuous, since the black smut masses have been disseminated and the infected plants are usually shorter than the healthy ones. This smut is not easily confused with bunt, which attacks only the interior of the kernel leaving the glumes intact. Loose smut of wheat is, however, very similar to loose smut of barley and loose smut of oats, although these three loose smuts are caused by different organisms.

ETIOLOGY.—The causal organism of the loose smut disease of wheat is *Ustilago tritici*, which differs from the other smuts studied chiefly in its method of attacking the plants. See its cycle of development in Fig. 207. The spores of the loose smut organism are spherical in shape and delicately echinulate. These may germinate as soon as mature, sending out a promycelium that serves as an infection hypha. Few if any sporidia are ever formed.

Infection of wheat by the loose smut organism is of the floral type; that is, the smut spore is carried by the wind to

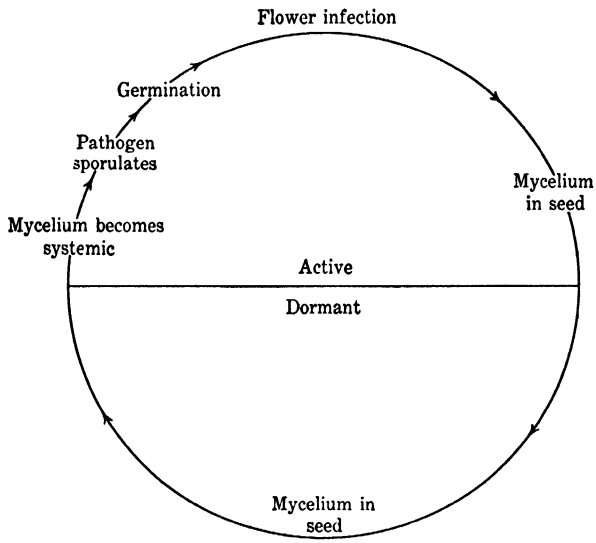


FIG. 207. A diagram of the host relation of *Ustilago tritici*. This organism survives the winter in the seed as mycelium.

the open flower of a similar host plant where, if it alights on the stigma, it germinates, and without the intervention of sporidia, penetrates the ovary. The germ tube of the chlamydospore penetrates the stigma as a germ tube of a pollen grain. Within the ovary the organism finds its way to the growing point of the embryo. There the hyphae remain dormant until the seed germinates, when the pathogen keeps pace with the development of the seedling and growing plant until the head is formed. The pathogen then invades all

the young spikelets and causes necrosis of the floral parts. The infected plants appear perfectly normal throughout the growing season except in size, and the infected and healthy seeds appear alike. The advancing mycelium in the growing plant lives intercellularly without causing any apparent injury to the adjoining cells. Likewise, the mycelium of the pathogen causes little injury to the maturing seed.

The loose smut pathogen is favored by humid weather conditions during flowering and is inhibited when the conditions are arid. Morphological characteristics of the flower seem to have no effect on the amount of infection.

CONTROL.—The only method of controlling the loose smut organism, once the seed has become infected, is to kill the dormant mycelium without injuring the seed. Soaking the seed in cold water for five hours followed by steeping in hot water for ten minutes at 54° C. will practically eliminate this smut. It is well to dip in cold water immediately after removing from the hot water. Despite all precautions, however, some injury to germination usually results, and therefore it is usually advisable not to treat all the wheat to be sown, but to treat enough for a seed plot. This should be isolated so as to prevent reinfection. The formaldehyde or dust treatments will not control loose smut. If the grain is sown wet, allowance in rate of seeding should be made for the swollen condition.

The fact that seed treated by the hot water method may be injured, leading to decreased stands and yields, suggests the desirability of turning to disease-free seed if at all possible, or using resistant varieties or strains, if such exist, that are adapted to local farm conditions. Some of the varieties that have been reported as resistant are Barska, Blackhull, Forward, Fulcaster, Hussar, Leap, Preston, Purplestraw, Ridit, Russian, Sol, Trumbull and Wyandotte.

RUSTS

Rust of cereals has been known since antiquity. It is recorded that the Romans held a festival on April 25 of each

year and offered sacrifices to the gods to protect their fields from rust. For a long time the cause of rust was unknown and many theories were advanced to explain the rusting of the grain crops. It was not known as a fungous disease until the latter part of the eighteenth century, when this fact was established by Persoon in 1797. A complete understanding of the complicated life history of the stem rust fungus, however, was not known until de Bary proved that the black stem rust organism was heteroecious in 1865.

A relationship between the aecidium on barberry and stem rust was suspected long before it was substantiated experimentally by de Bary. In 1818 Schoeller, a Danish school master, produced infection on grain with aecidiospores from the barberry bush, but he did not succeed in convincing his associates. That the majority of farmers were convinced of a definite relationship is obvious from legislation enacted not only in Europe, but also in the United States more than a century before the complete life history of the stem rust organism was known.

A law was passed at Rouen, France, providing for the eradication of the barberry in 1660. The state of Connecticut in 1726 allowed each town to forbid the growing of barberry within its limits. Another state, Rhode Island, passed a similar law in 1766, and the state of Massachusetts in 1755 passed a law providing that all barberries must be eradicated within five years. The province of Schaumburg-Lippe in Germany passed a law in 1805 providing for eradication of barberry. Bremen in 1815 enacted legislation providing that all barberries within 165 meters of grain fields be eradicated. All this was done despite the fact that it was not until 1865 that the exact relation of the rust on barberry and that on grains was known. This simply means that the barberry must have increased the amount of rust, or such laws never would have been passed.

After the relation of the barberry to the rust was known, further laws were passed. Prussia in 1880 provided for barberry eradication. France in 1888 permitted the local administrative district to proscribe barberries within their

limits. Denmark in 1903 enacted a law for the gradual extermination of all barberries. In 1917, the state of North Dakota and the province of Manitoba passed barberry eradication laws. In 1918 and 1919 all the states in the upper Mississippi valley enacted laws requiring barberry eradication.

Nature of the Rusts

The rusts are closely related to the smuts just discussed. The promycelia of the smuts and the rusts may have two points in common; namely, (1) cellular stalk bearing sporidia and (2) the reduction division in the nucleus that passes from the spore into the young promycelium.

Obligate parasitism is developed to a most remarkable degree in this group; no rust has been successfully cultured except on the living host. More than 200 species are heteroecious and about 1,800 autoecious.

More than 2,000 species of rusts are known, and they occur chiefly on the flowering plants; some few are known on ferns. Their mycelia are many-celled, binucleate, branched and provided with haustoria. The mycelium may be local or systemic, frequently inducing hypertrophy of the tissues and deformation of the host as galls or witches' brooms. The rust mycelium may develop extensively within the tissues without killing the cells at once or directly interfering with their function. The pathogen ultimately exhausts the cells and causes a breaking and splitting of the tissues. Some rusts have five different types of spores.

Spore Forms of the Rusts.—Many of the rusts are polymorphic, giving rise to two or more types of asexual spores from the same mycelium. The structures in which the different spores are borne have different names, as follows: spermogonium (pycnium), aecidium (aecium), uredosorus (uredium) and teleutosorus (telium). The spores that develop in these structures are known by the following names:

Spermatium or Pycniospore.—The spermatia are the small, one-celled hyaline spores produced in large numbers in the flask-shaped spermogonia. The latter are the brown fruiting structures in the upper surface of the leaves and are in con-

nection with the aecidial initials on the lower surface. The spermatia function as gametes in the formation of the mature aecidium.

Aecidiospore or Aeciospore.—The aecidiospores are borne in a cup-shaped sorus, called an aecidium, usually in the lower half of the leaf. These aecidiospores are one-celled, yellow, wind-disseminated, and germinate immediately, giving rise to the mycelium, which bears either the uredospore or teleutospore stages. Except in a few rusts they never give rise to another generation of aecidiospores.

Uredospore, Urediniospore or Urediospore.—These are the true summer spores which give the parasite a wide distribution through wind dissemination and immediate germination. The mycelium that bears uredospores may produce one or several crops. The uredospores produce mycelia, which may repeat the uredospore generation in six to ten days, or give rise to the teleutospore stage. The uredospores are hyaline to dark brown, spherical or ovoid, possess a thin, usually delicately echinulate wall and are borne singly on short stalks.

Teleutospore or Teliospore.—Teleutospores are usually the winter spore stage. They are thick-walled, dark colored and in most cases two or more celled, borne in sori and may remain fixed to the host during their existence. When these spores germinate they produce a four-celled promycelium on which are borne sporidia or basidiospores.

Sporidium or Basidiospore.—The sporidium is a thin-walled, single-celled, hyaline spore that is explosively discharged from the protuberance of the promycelial cell on which it is borne. The explosive discharge places the spores in the air currents, which expedites their distribution. On germinating they give rise to the haploid mycelium that produces the spermogonia and aecidial initials.

There are several spore terminologies in the rust literature; the two most generally used are cited above. The first is more prevalent in the literature than the second, and the first term in each case is used in this text.

Field Characteristics of the Cereal Rusts.—The cereal

rusts often are confused with one another because in some cases a single cereal (as barley) may serve as a host for two rusts at the same time. Each rust, however, has certain gross distinguishing characteristics, signs and symptoms that may serve as an aid in diagnosis. These are enumerated below

Stem Rust (*Puccinia graminis* Pers.) of Wheat, Rye, Barley and Oats.

1. Uredospores in linear brick red pustules.
2. Teleutospores in black linear open pustules.
3. Chiefly on the stems and leaf sheaths.
4. Alternate host, common barberry (*Berberis vulgaris* L.).
5. Overwinters in the mycelial or teleutospore stages.
6. General throughout the grain-growing regions of the world.

Crown Rust (*Puccinia coronata* Corda) of Oats.

1. Uredospores in round orange-yellow pustules.
2. Teleutospores in black covered round pustules.
3. Chiefly on the leaves and leaf sheaths.
4. Alternate host, European buckthorn (*Rhamnus cathartica* L.) and lance-leaved buckthorn (*R. lanceolata* Pursh.).
5. Overwinters in the mycelial or teleutospore stage.
6. General in Europe and America.

Leaf Rust (*Puccinia rubigo-vera* (DC.) Wint. var. *tritici* (Eriks. and Henn.) Carl.) of Wheat.

1. Uredospores in round, light brown pustules.
2. Teleutospores in black covered pustules.
3. Chiefly on leaves, leaf sheaths, sometimes glumes and awns.
4. Alternate host Meadow Rue, species of *Thalictrum*.
5. Overwinters in mycelial stage in wheat plant.
6. General in Europe and America.

Stripe Rust (*Puccinia glumarum* Eriks.) of Wheat.

1. Uredospores linear long yellow pustules.
2. Teleutospores in black covered pustules.
3. Chiefly on the leaves and stems.
4. Alternate host unknown.
5. Overwinters in mycelial stage in wheat plant.
6. General in the Old World and northwestern United States.

Leaf Rust (*Puccinia rubigo-vera* (DC.) Wint. var. *secalis* (Eriks.) Carl.) of Rye.

1. Uredospores in round brown pustules.
2. Teleutospores in black covered pustules.
3. On leaves.
4. Alternate host Alkanet (*Lycopsis arvensis* L.).

5. Overwinters in mycelial stage in rye plant.
6. General in the temperate parts of the world.

Leaf Rust (*Puccinia anomala* Rostr.) of Barley.

1. Uredospores in round brown pustules.
2. Teleutospores in black covered pustules.
3. On leaves.
4. Alternate host, Star of Bethlehem (*Ornithogalum umbellatum* L.).
5. Overwinters in mycelial stage in barley plant.
6. General in North America and Eurasia.

Stem Rust of Wheat

Puccinia graminis Pers.

Stem rust of wheat and other small grains is a disease that has been known at least since the time of Pliny, the Roman philosopher. It was not until 1797, however, that it was definitely shown that this disease was caused by a fungus, and it was 1865 before the relation of the aecidial stage on the barberry and the two stages on cereals was definitely proved. It is one of the most destructive diseases known to plant pathology; each year it is responsible for heavy losses, not only in America, but in the other important grain growing sections of the world.

The pathogen is probably most destructive in the upper Mississippi valley of North America, although it is known to cause serious losses in European countries. In the upper Mississippi valley it has caused, during the last 35 years, several very serious epiphytotics; e.g., in 1904, much of the grain in the upper Mississippi valley was so seriously damaged that the growers had difficulty in marketing their crops. The yield in many fields dropped to four bushels per acre. In Minnesota and the two Dakotas the yield reduction was estimated at 23,000,000 bushels. In 1916 the reduction in yield in the United States was more than 180,000,000 bushels, and in Canada, 100,000,000 bushels. Between 1915 and 1935 it is estimated that the reduction on wheat was 644,061,000 bushels; oats, 420,491,000 bushels; barley, 73,177,000 bushels. The most serious epiphytotic of stem rust in recent years

occurred in 1935 when reductions in yield were estimated at 121,882,000 bushels of wheat, 14,007,000 bushels of oats and 26,414,000 bushels of barley. The stem rust organism is more destructive in the spring wheat area of the United States than in the winter wheat districts.

SYMPTOMS.—Stem rust shows itself on the plant, first as a red rust, and second as a black rust. Within a short time, 48 hours after infection of the cereal host if conditions are favorable, a light-yellow spot will occur about the point of entry. In about four to six days there develops at this point a small pustule, which consists of the uredospore stage of the organism, breaking through from the inside of the leaf. As a result, the torn epidermis forms a border for the red pustule, made up of uredospores. The pustules or sori usually are linear. They may occur either on the stems, leaves or head of the plant, and are formed most commonly, however, on the leaf sheath and stem. Surrounding the sorus there is usually a narrow chlorotic border.



FIG. 208. Severe infection of *Puccinia graminis* on culms and leaf sheaths of wheat. (Courtesy of the Bur. Ent. and Pl. Quar., U. S. D. A.)

The teleutospores are borne on the same mycelium as the uredospores. When the host tissue has been completely depleted, either by the rust organism or by translocation incident to maturing the grain, the mycelium in the tissue produces this second type of spore. These, as in the case of the uredospores, break through the

epidermis, which makes a frayed and ragged border. It is, of course, the mycelium inside the tissues of the open wounds caused by the uredospore and the teleutospore pustules that damages the wheat plant. The mycelium ramifies between the cells absorbing the food that normally should go to filling the grain. The ruptured epidermis over the sori permits abnormal loss of moisture that interferes with the normal flow of water and plant nutrients in the stem. As a result,



FIG. 209. Shriveled wheat kernels taken from plants injured by the black stem rust organism (left) as compared with plump kernels (right) from healthy plants. (Courtesy of the Bur. Ent. and Pl. Quar., U. S. D. A.)

badly infected plants bear only partly filled grain that shrivels upon drying.

The alternate stage on the common barberry is much less destructive than that on wheat. Here the first symptom is a small etiolated spot from one to six millimeters in diameter that later enlarges and takes on a yellow color. The action of the mycelium frequently causes the leaf tissue to become distorted, bulging either upward or downward, and later,

small, black pinhead points, the spermogonia, appear on the upper surface of the yellow spot. On the lower surface of the leaves are formed the aecidial cups, which are surrounded by a thin layer of cells that may extend out a little from the surface of the leaf. Inside the cups are formed large numbers of yellow spores.

ETIOLOGY.—The fungus causing stem rust of cereals is known as *Puccinia graminis*. The life cycle of the stem rust organism begins with the germination of the teleutospores in the spring, sometime in April or May, in the latitude of Iowa. The two cells of the teleutospores each produce four-celled promycelia on which are borne the small and hyaline sporidia. These are forcibly discharged from the promycelium up into the air currents, which may carry them to the leaves of the common barberry. (See Fig. 211.)



FIG. 210. Aecidial stage of *Puccinia graminis* on common barberry.

In a favorable environment, these sporidia germinate, penetrate the cuticle and epidermal cells and form a mass of mycelium above the palisade layer just below the upper epidermis. From this mass of cells, certain hyphae make their way between the palisade cells down into the spongy parenchyma where the aecidial initials develop. From the mycelium just under the upper epidermis a spermogonium develops, finally discharging haploid spermatia. While the spermatia

are being formed and discharged, the hyphae from the base of the spermogonium form the aecidial initials. From these initials receptive hyphae make their way to the surface through the spermogonia, stomatal openings and epidermis. When a spermatium of opposite sex falls upon these receptive hyphae, its nucleus migrates into the terminal cell of the receptive hypha. This cell, which has been made binucleate, continues the development of the aecidial initials, leading ultimately to the development of large numbers of stalk cells from which numerous aecidiospores are produced in chains. About the periphery of each aecidium there develops a layer of peridial cells, which ultimately ruptures the lower epidermis and extends out from the leaf. The aecidiospores are forcefully discharged upon maturity into air currents where they may be carried to susceptible hosts, as any of the cereals and several wild grasses.

In the presence of moisture and suitable temperature the aecidiospores germinate, producing a hypha that penetrates through the stomata into the tissues of the wheat plant. Once inside the stem tissue, the mycelium spreads very rapidly, forming later the pustule containing the uredospore stage. The uredospores are borne singly on short stalks, are brick red in color, slightly oval in shape and spiny. This stage of the stem rust fungus is well suited for wind distribution. The uredospore stage functions in the very rapid development of secondary infection on adjoining wheat plants.

From the mycelium of these secondary infections and from the mycelium forming the uredospores, the second spore stage on grasses, the teleutospore, is formed. This is borne in long, linear sori. Each two-celled black spore is produced on a separate stalk. The teleutospores function in carrying the rust fungus over from fall to spring when they reinitiate the cycle by the production of sporidia.

The uredospores are resistant to adverse conditions, but cannot survive from one crop year to the next in the upper Mississippi valley. They have been found to live over in the southern part of the Mississippi valley to a limited extent.

They may, however, function in initiating scattered infections or an epiphytotic when blown from the southern sections into more northern wheat growing regions of the Mississippi valley. Early local infection must come from the teleutospore stage and subsequent infection of the barberry leading to

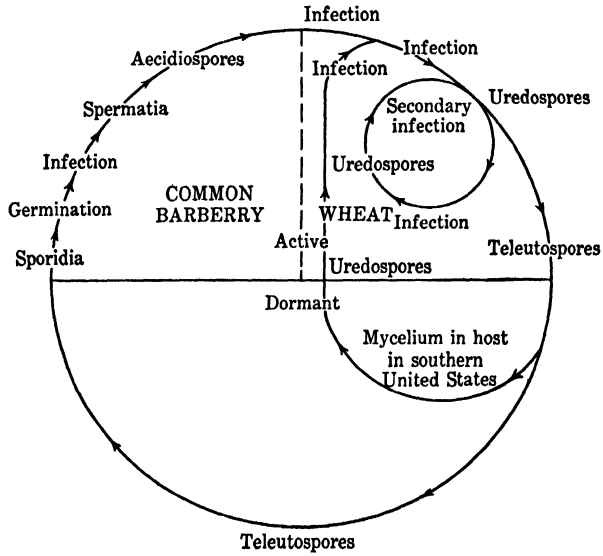


FIG. 211. A diagram of the host relation of *Puccinia graminis*. The pathogen survives the winter in the teleutospore stage, and as mycelium in the cereal host in the southern part of the United States.

the development of the aecidiospore stage. The aecidiospores are not so well adapted for wind dissemination, and as a result they cannot function over so large an area. As a consequence, infection centers frequently are formed on wheat, other cereals and susceptible grasses near infected barberries. These local centers serve to initiate extensive secondary infection through the uredospore stage. The wheat plant as well as other cereal and wild grass hosts is susceptible throughout its whole life history.

Even though the organism is present on the susceptible host, certain environmental conditions must prevail before infection can take place. There must be a thin film of moisture on the surface of the leaf and the temperature must range between 5° and 25° C. The most favorable temperature for the germi-

nation of the uredospores is about 18° C. This combination of moisture and temperature must prevail long enough to permit the hypha of the germinated spore to gain entrance into the leaf or stem. The subsequent growth of the mycelium also is favored by a high humidity and comparatively high temperature, 20° to 30° C.; thus it is that stem rust flourishes during wet, foggy weather. In the same way, these required conditions explain why stem rust is so prevalent where grain has lodged or is growing in low, wet places.

There are at least eight varieties of the stem rust organism, but only three infect cereals. The other five occur on certain wild and cultivated grasses. These varieties are: *Puccinia graminis tritici* Eriks. and Henn. on wheat, barley, and many wild grasses; *P. graminis avenae* Eriks. and Henn. on oats and some wild grasses; *P. graminis phleipratensis* (Eriks. and Henn.) S. & P. on timothy and *Festuca* spp.; *P. graminis secalis* Eriks. and Henn. on rye, barley and many wild grasses; *P. graminis agrostidis* Eriks. on species of *Agrostis; P. graminis poae* Eriks. and Henn. on species of *Poa; P. graminis airae* Eriks. and Henn. on species of *Aira; P. graminis bromi* Eriks. on species of *Bromus*.

In the varieties occurring on wheat and oats there exists a still further specialization. This specialization is shown by the presence in a variety of a number of different races; although not all these races exist in the same section of the country, more than 150 races of the variety *P. graminis tritici* occur in different parts of the United States and the Old World. In some cases two or more races may exist in the same field, in fact, even on the same plant. This condition makes it difficult to develop strains of wheat that are resistant to stem rust over a considerable section of the country or in different countries.

CONTROL.—In the control of stem rust, three methods of procedure present themselves: (1) the eradication of its alternate hosts, species of susceptible barberry; (2) utilization of resistant varieties, and (3) the modification of cultural practice so as to make environmental conditions as unfavorable as possible for the growth and development of the rust

fungus. Sulphur dust is commonly used to control black stem rust of wheat in certain parts of California.

The first method, that of eradication, is probably the most practical for the grain growing region of central United States. Legal action directed toward the destruction of the common barberry has already led to general eradication in Europe and is making its influence felt in this country. There are different methods of killing the common barberry, the most practical being with salt. It requires about ten pounds of salt piled on the crown of a barberry to kill it. The Japanese barberry (*Berberis thunbergii* DC.) is immune and may be substituted where the common barberry needs to be replaced. *Berberis mentarensis* Ames, a new hybrid, is held to be very resistant.

There is apparently no variety or strain of wheat that is highly resistant to all the races. Kanred, Mindum, Arnautka, Thatcher, Hope and others have proved resistant to some races, and two strains of emmer have proved resistant to all the races known at the present time. To utilize resistance in the control of stem rust, it probably will be necessary to select and build up a strain for each given section of the country.

Crown Rust

Puccinia coronata Corda

Crown rust is a leaf rust of oats, which occurs generally in the oat growing regions of the world. It takes its common name from the crown-like appearance of the terminal cell of the teleutospore. It occurs on all varieties of oats, more than 80 species of the wild grasses and at least 16 species of buckthorn (*Rhamnus*) in the United States. The organism is more destructive in the southern oat growing districts than in the northern. It is estimated that in 1927 crown rust caused the loss of 56,978,000 bushels of oats. Under favorable conditions, crown rust may cause a 50 per cent reduction in the yield of susceptible sorts. In the upper Mississippi valley the whole crop is sometimes destroyed in the vicinity of susceptible buckthorn bushes.

It is significant to note that crown rust of oats was the

second rust to be determined as heteroecious in 1866 by de Bary. He showed that crown rust would develop its aecidium on buckthorn (*Rhamnus frangula* L.), but it remained for Nielson to complete the evidence in 1877. Nielson took aecidiospores from European buckthorn (*Rhamnus cathartica* L.) and produced infection on rye grass (*Lolium perenne* L.) and with the uredospores transferred the rust to oats.

SYMPTOMS.—This rust is most common on the leaf blades and leaf sheaths. It is rare on the stems and panicles of the oat plant. The oval or slightly linear, orange-yellow pustules are the most conspicuous sign of this disease. After the uredospores and aecidiospores have germinated and their germ tubes have entered the host through the stomata, a small, light-yellow spot about one millimeter in diameter becomes evident in three to eight days. In the center of this spot an orange-yellow pustule develops. After the uredospores mature, the epidermis of the leaf is ruptured and the spores are disseminated by the wind. When crown rust occurs in epiphytotic form it is not uncommon for the grain harvester to be coated with a layer of orange-yellow rust spores. If the weather is calm and the humidity low, a cloud of spores will follow the machine.



FIG. 212. Uredospore pustules of crown rust on leaves and leaf sheaths of oats.

Necrosis results from the invasion of the pathogen, leading ultimately to the formation of numerous local dead areas. If the pustules are numerous, the areas invaded by the mycelium in the host may coalesce and a cluster of pustules develops.

Following the orange-yellow pustules there develops from the same mycelium a number of black teleutospore pustules at the periphery of the infected area of the leaf. The teleutospore pustules as a rule do not rupture the epidermis, but remain covered.

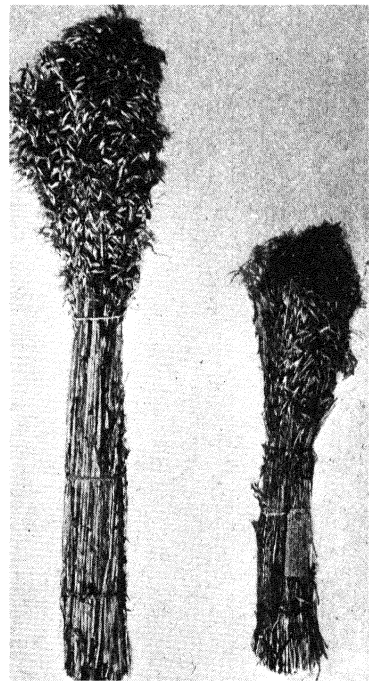


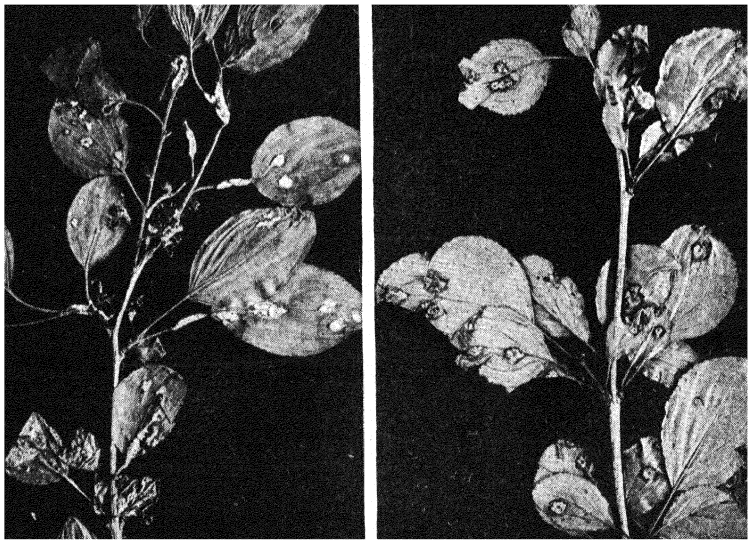
FIG. 213. Yields of oats from similar plots of healthy and crown rust infected plants.

On the alternate host, the buckthorn (species of *Rhamnus*), orange-colored flecks may result from the sporidial infection of the leaves, young twigs and fruits. On the leaves the orange-colored flecks spotted with very small dark-brown dots first appear on the upper surface, while on the lower surface the epidermis finally is ruptured and a cluster of yellow cups extrudes from the leaf. When the spore stages on the upper and lower surfaces of the leaf are mature, necrosis of the infected tissues results. On the young twigs and green fruits the two spore stages may occur side by side. Where the rust fungus attacks twigs and fruits, considerable hypertrophy and distortion frequently follow.

ETIOLOGY.—Crown rust is caused by the fungus *Puccinia coronata*. The host relation is shown in Fig. 216. The parasite consists of much-branched mycelia bearing five spore stages, two of which occur on oats and certain other species of grasses, two on several species of buckthorn, and one develops from the mature teleutospores in the spring.

There are two parasitic spore stages on the gramineous host, the uredospore and teleutospore stages. The former are able to

reinfect the susceptible grasses, while the latter give rise to the sporidia, which can infect only species of buckthorn. These spore stages develop from binucleate or diploid mycelium living intercellularly in the host. The uredospores are slightly spiny, spherical, single-celled and orange-yellow in



A B

FIG. 214. A, the aecidial stage showing cluster cups of *Puccinia coronata* on European buckthorn; B, late stage showing the remains of the aecidia and the local necrotic lesions.

color. They are developed singly from a short stalk cell in the base of the uredosorus. The black, two-celled teleutospores are each borne on a separate stalk. The end cell of the teleutospores bears a circle of prongs suggesting a crown.

Under favorable conditions the black teleutospores germinate in the spring, forming a four-celled stalk on which are borne four single-celled hyaline spores, known as sporidia. In the formation of these sporidia, reduction division takes place, hence the sporidia and likewise the mycelium produced by them are haploid. In addition, the sporidia may be of opposite sex, or heterothallic. If conditions are favorable, the sporidia may be carried by the wind to the buckthorn,

where they germinate, sending out a germ tube that penetrates through the epidermal cells of the host. Once below the epidermal layer, the mycelium spreads laterally between the epidermis and above the palisade cells. In a few days



FIG. 215. Field of oats showing damage caused by the crown rust organism which spread from the buckthorn hedge in the background.

masses of hyphae develop the spermogonial initials. As these structures develop they become spherical with necks breaking through to the surface of the leaf. In these structures large numbers of tiny spores are produced that are discharged through the openings in the necks of the spermogonia. These spores are known as spermatia and are lodged in a sweetish liquid that attracts insects.

From about the base of the spermogonium, long, slender hyphae grow up through the epidermis in and around the spermogonia, or out through the stomata to the surface of the leaf. If a spermatium chances to lodge on the tip of one of

these receptive hyphae, extending to the surface of the leaf the nucleus of the spermatium migrates into the receptive hypha. This leads to the doubling of the number of nuclei, which results in the binucleate condition, such as prevails in the mycelium producing the uredospore and teleutospore stages.

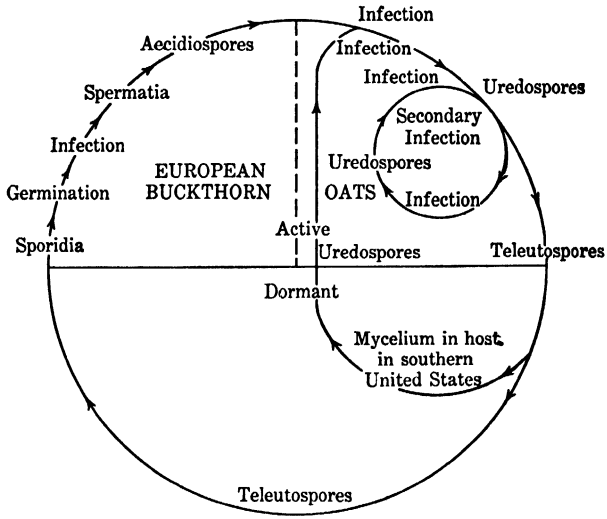


FIG. 216. A diagram of the host relation of *Puccinia coronata*. The pathogen survives the winter in the teleutospore stage and as mycelium in the host in the southern part of the United States.

This doubling of the number results only from the fusion of opposite sexes and unless this binucleate condition is established, the aecidial stage does not develop.

Before the binucleate mycelial stage has been established, the mycelium grows down between the palisade cells into the spongy parenchyma and forms aecidial initials near the lower surface of the leaf, stem or fruit. From the doubling of the nuclei in some of this mycelium as a result of the action of the spermatia, there develops in these initials a large number of short stalk cells on which are formed chains of aecidiospores. The aecidiospores are single-celled, smooth surfaced and bright yellow. When mature they are forcefully discharged from the aecidial cups and may be picked up by the air currents and carried some distance. Some of them may

finally lodge upon leaves of susceptible gramineous hosts. Under favorable conditions of moisture and temperature, the aecidiospores germinate, sending out germ tubes, which enter the stomata of the leaf. In the leaf each germ tube gives rise

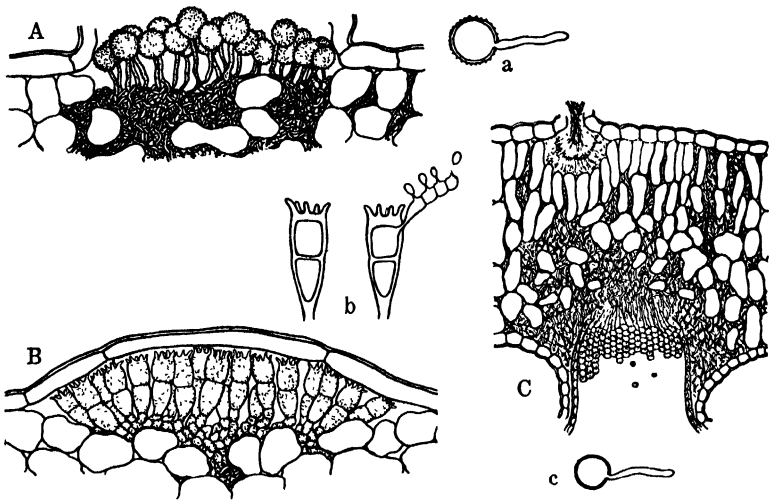


FIG. 217. Spore stages of *Puccinia coronata:* A, a cross section of an oat leaf showing a uredospore sorus; a germinating uredospore, a; B, a section of a teleutospore sorus; a germinating teleutospore with sporidia, b; C, a cross section of a European buckthorn leaf showing the aecidium on the lower surface and spermogonium on the upper; a germinating aecidiospore at c.

to an intercellular mycelium, which later bears the uredospores.

The spread of crown rust in the northern oat growing regions may start from the buckthorn as shown in Fig. 218. The aecidiospores may initiate infection on oats in the immediate vicinity of the buckthorn and later spread by successive uredospore generations over comparatively large areas before rust infection becomes general in a given district as shown in Fig. 219. The table on the following page shows the rate of spread of crown rust and the area involved from a buckthorn hedge near Hinton, Iowa, in 1921.

The oat crop in the vicinity of Hinton, Iowa, was a failure in 1921 and it is probably safe to say that this local epiphytotic spread early rust infection over much of northwest Iowa and

northward into South Dakota and Minnesota. In the southern oat growing sections the mycelium may live over the winter in the oat plant and in the spring produce uredospores that function in spreading the rust. In still other cases, the uredospores may be blown considerable distances

Period	*Source of Infection*	*Area*	
		Acres	Square Miles
May 22 to 28	Aecidospore	2,980	5
May 29 to June 4	Uredospore	21,120	33
June 5 to 7	"	80,640	126
June 8 to 10	"	407,040	636

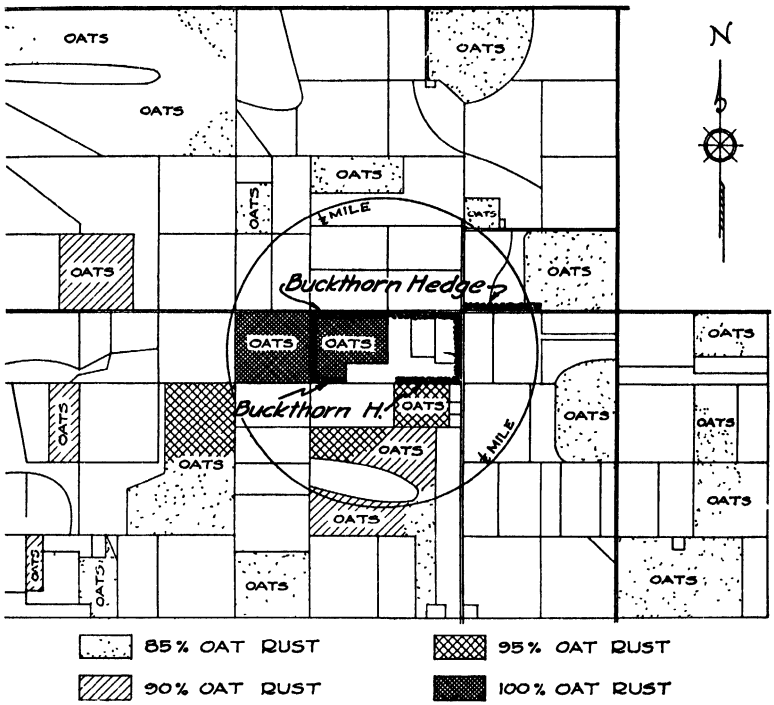
from the south northward, and give rise to crown rust infection.

The crown rust pathogen is greatly dependent upon weather conditions for the production of an epiphytotic. Its spores require a film of water for germination and infection. The spores must actually be on a film of water to obtain good germination; a relative humidity of 99 per cent results in only 17 per cent germination. The mycelial development in the host is most rapid at summer temperatures when the humidity is high. The optimum temperature for uredospore germination and development of the resulting germ tube is 17° to 22° C. High humidity conditions are favored by dense stands, dews or rains.

Biological specialization also exists in the crown rust organism; e.g., there are at least four varieties of crown rust in the upper Mississippi valley. These take their names from their most congenial host and are designated as follows: *Puccinia coronata avenae* Eriks. on species of *Avena; Puccinia coronata calamagrostis* Eriks. on reed grass, *Calamagrostis canadensis* (Michx.) Beauv.; *Puccinia coronata festucae* Eriks. on meadow fescue, *Festuca elatior* L.; *Puccinia coronata holci* Kleb. on velvet grass, *Holcus lanatus* L.

These varieties are distinguished from one another by the

degree of infection produced on different species of gramineous and rhamnaceous hosts. For instance, *Puccinia coronata avenae* develops best on species of *Avena* and European buckthorn, *Rhamnus cathartica*, and the lance-leaved buckthorn, *R. lanceolata*, but it also develops on reed grass and *Rhamnus*


OATS
OATS
OATS

selection of varieties and eradication of susceptible alternate hosts. Oats should be grown on well-drained soil not too thickly seeded. The time of planting should be as early as consistent with the best yields. In some sections crown rust can largely be controlled by using an early-maturing variety. Such sorts may mature before an epiphytotic has opportunity

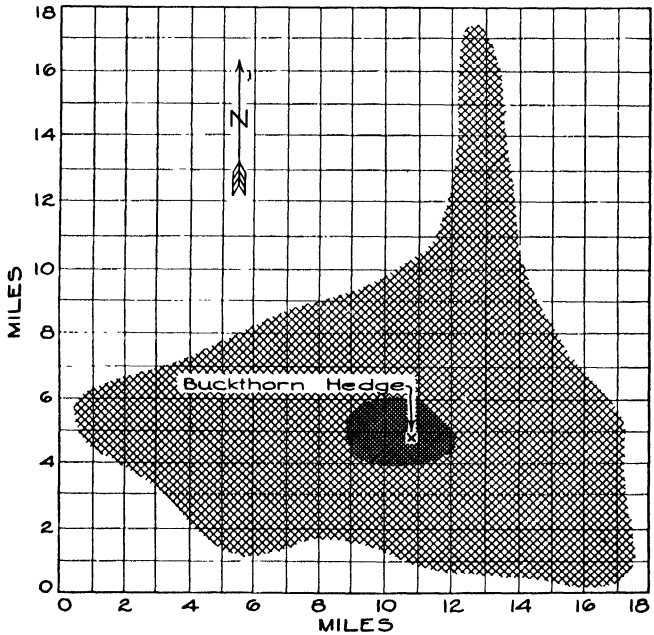


FIG. 219. The spread of crown rust from a buckthorn hedge near Hinton, Iowa, in 1921. The rust was most severe nearest the hedge where the oat crop was destroyed. By successive uredospore generations the rust spread over the cross-hatched area by June 7. Rust infection from this local epiphytotic extended northwest 53 miles by June 10. (After S. M. Dietz.)

to develop. For example, the upper Mississippi valley, the variety Richland and others may escape serious rust damage. Certain varieties are known to be more or less resistant to crown rust, as Rainbow, Schoolmam, Red Rustproof and Green Russian varieties; while other varieties, as Victoria, Bond, Alber, Marion, Hancock and Boone are very resistant and high yielding.

Where the crown rust pathogen spreads from buckthorns,

these should be eradicated, thus preventing local epiphytotics. Crown rust of oats may be controlled by dusting with sulphur, but this method is too expensive to warrant its general use.

Asparagus Rust

Puccinia asparagi DC.

Asparagus rust is a good example of an autoecious rust that causes severe damage to a crop plant. It was first reported in 1805, in Europe where it caused only slight damage until recently, when it has caused an annual loss estimated at $2,500,000 in Germany.

The organism was first epiphytotic in the United States in the eastern asparagus growing sections in 1896. It spread rapidly through the entire United States and was reported from Iowa and Texas in 1900, reaching California two years later. The early epiphytotics are said to have wiped out certain eastern asparagus growing districts; i.e., fields at Concord, Massachusetts, valued at $100,000, were destroyed in 1906. On Long Island the yield was reduced two-thirds. The early damage resulted in the establishment of breeding projects, which developed the highly resistant Washington varieties now serving as the main method of control in this country. Although the organism does not cause much damage in hosts other than the cultivated asparagus, it may attack the onion.

Symptoms.—The first symptoms of asparagus rust can be found on the stems and smaller branches of uncut plants in late April and May. At this time inconspicuous light-yellow, oval spots develop. Within these lesions the early signs of the disease, the brownish spermogonia and the concentric rings of yellowish aecidial cups develop. The aecidial cups soon liberate the yellowish aecidiospores that are responsible for the secondary spread of the organism. These two stages, occurring together in the small stem lesions, seldom are noticed by growers.

The most characteristic evidence of the disease is noted after the plants have grown up following the cutting season. At this time the needles and smaller branches become covered

with the reddish or rust-colored uredosori. This stage increases until the tops become completely covered and may appear red because of the numerous uredosori. The destructive development of the organism in the tops makes them turn yellow and die prematurely. About this time the organism produces the teleutospore or black spore stage. The yellowed stems become covered with the black teleutosori and the field becomes a mass of bare, blackened stalks. The remains of the three previous spore stages often may be found, on close examination, in the center of the blackened sori. Often before complete destruction of the stems, the color changes to dull gray, resulting from the growth of secondary organisms which parasitize the rust fungus



FIG 220. Asparagus plant bearing teleutosori of *Puccinia asparagi*

ETIOLOGY. — Asparagus rust is caused by the full-cycled, autoecious rust, *Puccinia asparagi*. (See Fig. 221.) The early spring weather that starts the asparagus plants also favors the production of the four-celled promycelium by the teleutospores, which have overwintered on the plant refuse in the field. The promycelia each produce four small, hyaline oval sporidia that cause the primary infection on the uncut stalks in the fields. The sporidial germ tubes enter the asparagus stems and produce a ramifying, uninucleate, intercellular mycelium. This mycelium with its numerous

haustoria grows through a localized area of cortical cells and produces, just under the epidermis, a stromatic layer. From this stroma are produced the small, flask-shaped, yellowish spermogonia that later turn brown. In these spermogonia are produced the spermatia. These spermatia apparently function as sex cells and are responsible for the initiation of the binucleate mycelium and the stimulus that results in the production, in concentric rings, of the yellowish aecidia.

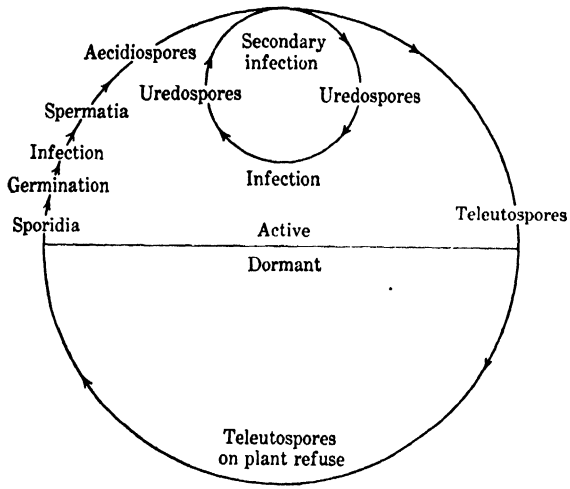


FIG. 221 A diagram of the host relation of *Puccinia asparagi* The pathogen survives the winter in the teleutospore stage on plant refuse. This organism is autoecious and polymorphic.

The aecidia are formed by the differentiation of a subepidermal mass of binucleate mycelium into the sterile cells of the peridium, surrounding the chains of spherical yellow aecidiospores. The enlargement of this structure ruptures the epidermis, the peridium opens, and becomes cup-shaped. With the rupture of the peridium, the aecidiospores mature and are rattled loose into the wind, which disseminates them. The aecidiospores germinate to form an infection tube, which by entering the stomata of other asparagus plants, produces the third stage of the rust—the uredospores.

From the mycelium of the aecidiospore infection tube, or from the mycelium around the edge of the aecidium, a

stromatic layer under the epidermis produces the large, single-celled, bright-red, spiny uredospores. These globular uredospores are formed singly on stalks arising from the stromatic layer, and their growth ruptures the epidermis of the host, producing the red coloration of the plants. These uredospores are freed and on germination infect other asparagus plants. This is the third infection cycle during the season. The uredospores are said to be able to cause infection and to produce new uredospores within 12 days. The rupture of the epidermis by the uredosorus causes an increased drain on the plant in exposing unprotected host tissues to desiccation. This water loss, in addition to the consumption of food materials, results in the yellowing and "needle" dropping that characterize the disease.

When cool weather sets in or when the weather becomes dry and the remains of the host plant begin to fail as a source of food, the fourth or teleutospore stage is formed. From the edge of the numerous uredosori are produced singly on long stalks the elliptical, two-celled, black teleutospores. These spores may cling to the host over winter. In the spring they germinate to produce the promycelia and sporidia that initiate the new infection.

The damage caused by rust is worse on plants growing in dry soil than in wet, although water is necessary for germination and infection. In fields full of weeds or otherwise protected from the drying winds, the dew does not dry quickly and conditions favor the rapid spread and development of the pathogen. Such conditions, together with many soil and other factors that weaken the host, may favor the development of a destructive epiphytotic.

CONTROL.—The establishment of such epiphytotics in the first part of this century caused the growers to seek resistant varieties of asparagus. Since the early resistant strains, Palmetto, Argenteuil, etc., were undesirable commercially, cooperative breeding projects were instituted and the varieties Martha Washington and Mary Washington were produced. These varieties have shown high resistance to the rust in this country and have in addition to resistance, the desirable

qualities necessary for commercial foodstuffs. Recent trials in Germany have shown that these varieties are susceptible to the rust in that country.

The main control measure that has permitted the continuation of asparagus culture despite the rust has been the use of the highly resistant varieties Martha Washington and Mary Washington. In addition to the use of resistant varieties the following practices are advised to prevent rust injury. The most important of these is the application of finely divided sulphur twice during the season, three weeks after cutting stops, and again a month later. The sulphur is applied at the rate of 25 to 30 pounds to the acre. This practice must be supplemented by clean culture and the destruction of inoculum by cutting out the wild hosts and destroying infected plant refuse in the fall. Where possible the rows should be well spaced, about ten feet, and should run with the prevailing winds so as to facilitate rapid drying of the plants.

Cedar-Apple Rust

Gymnosporangium juniperi-virginianae Schw.

Cedar-apple rust is native to this country and generally is distributed over the eastern and midwestern parts of the United States. It seems that this disease has been gradually becoming more destructive, probably because of the intensive development of the orchard industry. Cedar-apple rust is destructive in that it causes early defoliation and necrotic areas on the apple, resulting in malformed fruit. Where the trees are defoliated early, they go into the winter in a weak condition and are liable to suffer from winter injury. Where the apples and cedars grow in close proximity the loss is very great. In Virginia the loss is said to range from $2,000 to $3,000 per orchard per year. In one county in Virginia the loss was said to be $75,000 in one year. If the cedars are sufficiently prevalent and conditions are favorable, the crop may be ruined. This condition occurred in 1928 in Harrison County, Iowa.

As a rule, much less damage is done to the cedar, although the galls may become so numerous that the tree is materially injured. If the galls become too numerous, the lower branches are bent to the ground as a result of the added weight of water absorbed by the gelatinous horns extruding from the galls. Where young cedar trees stand close to an older orchard, they may be killed by the rust.

The cedar-apple rust fungus attacks almost all varieties of cultivated apples, but not all these varieties are equally susceptible. The varieties Wealthy and Jonathan are two of the most susceptible, and the Delicious is one of the most resistant—at least in the middle west. The flowering crabs are probably more susceptible than most susceptible cultivated apple varieties.

There are two other rusts that occur on the apple and the red cedar in some parts of the United States. These are the hawthorn rust, *Gymnosporangium globosum* Farl., and quince rust, *Gymnosporangium germinale* Kern.

Symptoms.—On the apple, the cedar-apple rust fungus attacks leaves, twigs and fruits. The disease appears first as a yellow blotch on the upper surface of the leaf in the center of which are small brown dots, the spermogonia, visible to the naked eye. The mycelium of the fungus is intercellular with haustoria extending into the host cells. In some unknown way this mycelium stimulates the spongy mesophyll cells to excessive division and enlargement. As a result the leaf becomes considerably thickened in the discolored, infected region. Later in the season yellow, cup-like aecidia develop under the lower epidermis of the hypertrophied region. These cup-like structures break through the epidermis and free large numbers of yellow spores. When the points of infection are numerous, the leaf tissue is killed early in some varieties, and the cup-shaped structures on the lower surface of the leaf fail to develop. In such instances the leaves almost always fall early in the season, interfering materially with the normal development of the apple tree. When the fungus has matured its aecidia on the lower surface complete necrosis of the invaded tissue follows.

On susceptible varieties it is common to find lesions on the young twigs, like those on the leaves, for instance, cup-shaped structures usually are fully as large as those on the

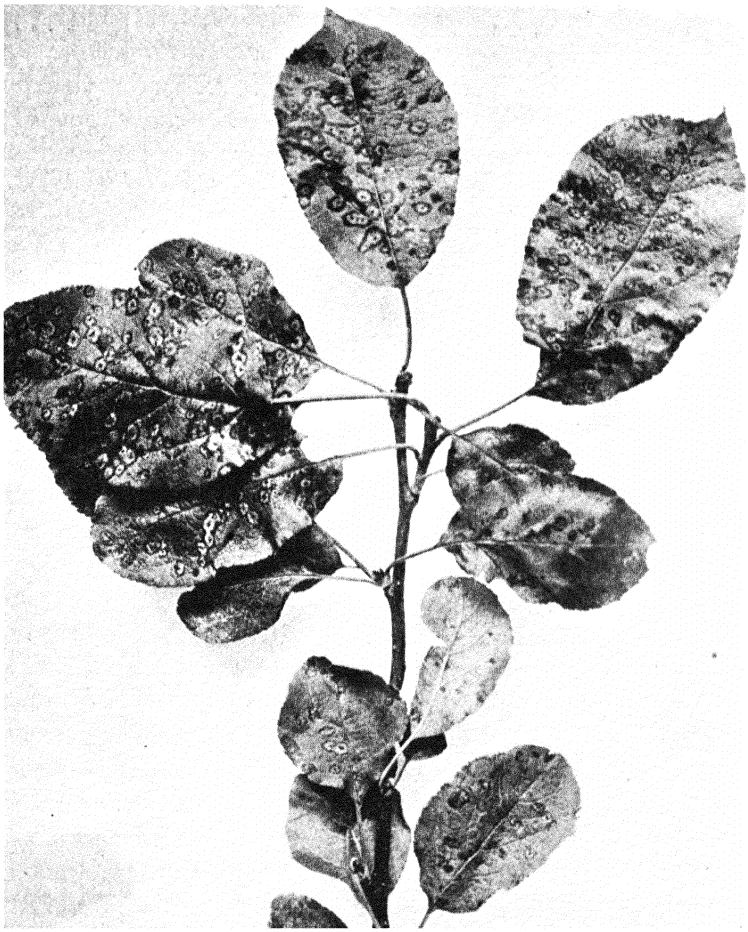


FIG. 222. Cedar-apple rust on apple leaves showing spermogonial and aecidial pustules.

leaves. The mycelium causes the same effect as on the leaves, except that in very susceptible sorts, the swelling of the buds is excessive and results in forcing. Where the twig infections occur, girdling often results and the twigs are killed; a condition that is common on flowering crabs, in

fact, so common that it is impractical to grow flowering crabs near red cedars.

This pathogen also attacks the green fruit, causing yellow blotches as those produced on the leaves, in which the fruit is often much deformed. The fruit, like the twig, is completely invaded by the mycelium and marked swelling often results. The spermogonia and aecidia occur side by side as on the twigs. The infection of the fruit, however, is less common than that of the foliage because of the seasonal development of the pathogen.

On the red cedar this pathogen has an effect very different from that on the apple, e.g., instead of producing yellow hypertrophied areas, it causes the formation of swellings or galls on the young leaves and twigs. These galls vary in size from that of a pea seed to that of a small walnut. The surface of the galls is pitted, and in the center of each pit occurs a brown raised area. The color of the gall is the same as that of the stem of the host. When these galls are fully matured, deep-yellow gelatinous horns extrude from the brown spots in the pits. These may be so numerous on the gall as to give it the appearance of a light-brown flower. This sign on the cedar is only manifested during May and June when the weather is damp and cool, but when the weather is dry these gelatinous protuberances become dry, brown and much less conspicuous.

Etiology.—The fungus that causes the cedar-apple rust disease on the apple and cedar is known as *Gymnosporangium juniperi-virginianae*. This organism is a rust that has its aecidiospore stage on the apple and its teleutospore stage on the red cedar. It is unlike any of the rusts studied so far in that it does not have a uredospore stage. (See Fig. 224).

Two spore forms are produced on the apple. On the leaves the spermatia develop on the upper surface in small, flask-like structures appearing to the unaided eye first as yellow and later as brown dots. The yellow spots appear from six to ten days after infection and the spermogonia after 18 to 24 days. The spermatia are produced within and freed from the spermogonia as in the other rusts and on

coming in contact with the receptive hyphae discharge their nuclei into them. This results in the doubling of nuclei, which provides the stimulus for the production of aecidia. The spermatia are sexual spores. On the lower surface



FIG. 223. Gymnosporangium gall on red cedar showing the extruded horns.

the aecidiospores occur in the cup-shaped pustules about 60 to 70 days after infection. The cup is made up of a wall of cells that often grows out from the surface of the leaf about a quarter of an inch. As this layer of cells approaches maturity it spreads and curves outward in a stellate manner. When these cups are numerous and in close proximity, they give rather a conspicuous appearance to the surface. The aecidiospores are yellow and are borne in chains inside the cup. The movement of the cup walls causes the spores to be discharged forcibly.

These aecidiospores are then carried by the wind to the red cedar. In the presence of moisture and after a certain rest period, these spores put out a germ tube that probably

enters the upper surface of the leaves or the twigs. Once inside the tissue, it has the same stimulating effect on the cells of the cedar that it had on the apple. The cedar may become infected in August and September but there are no visible signs of infection until the following July, when a

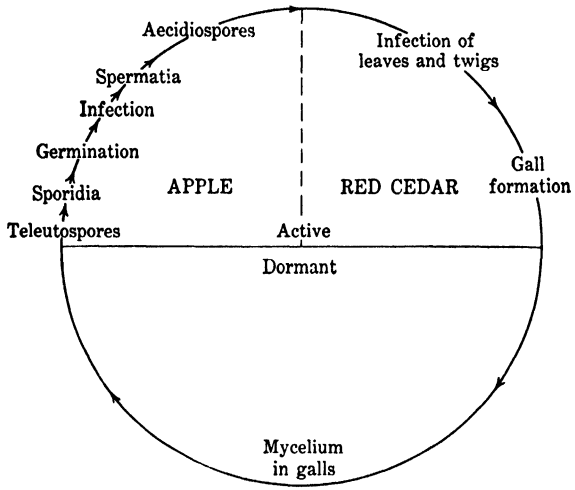


FIG. 224. A diagram of the host relation of *Gymnosporangium juniperi-virginianae*. The pathogen requires one whole and part of two other growing seasons to complete its development. It survives the winter as mycelium in the red cedar.

small gall is evident, which grows very rapidly until frost. The next spring the teleutospores in the sori develop, breaking through the cortex of the gall and producing the yellow spore horns. The discharge occurs in May and June. The sporidia are forcibly discharged from the promycelia in the spore horns into the air currents, which may carry them back to the apple or some other susceptible host. These sporidia are hyaline, thin walled, and they germinate by a thin germ tube that enters the apple leaf and produces the spermatial and aecidial stages on the apple. It requires, therefore, two years for this fungus to complete its life history.

This rust, probably more than any other we have studied, is very dependent upon moist, cool weather for its normal development. If the weather is dry and hot in May, the spore horns from the cedar galls are inhibited. Likewise,

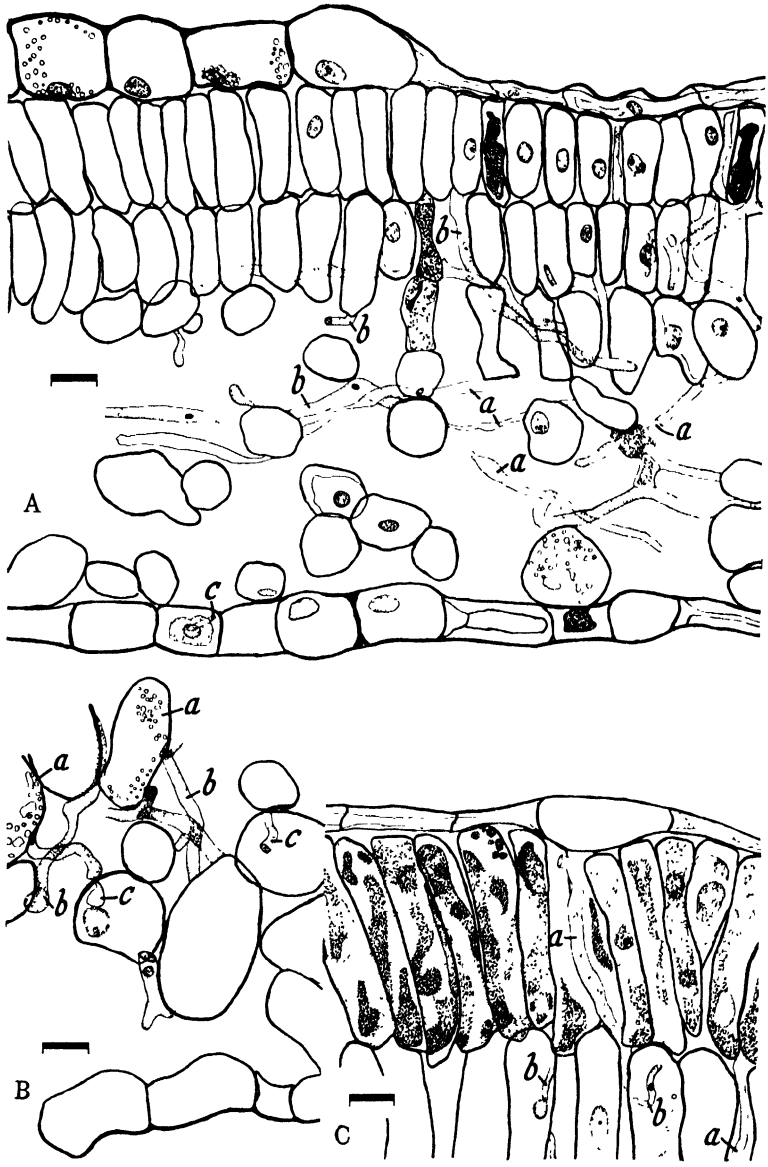


FIG 225. Pathological anatomy of cedar-apple rust on apple leaves. A, transverse section showing empty hyphae, *a*, active hyphae, *b*, and an haustorium in epidermal cell, *c*; B, section of spongy mesophyll showing mycelium, *b*, haustoria, *c*, and slightly injured host cells, *a*, C, section of fleck on resistant leaves Primary infective hyphae, *a*, between dead host cells and haustoria, *b*, in injured cells. (After G. L. McNew.)

the sporidia, if they are developed, are easily destroyed by dry air. The amount of rust that develops also is dependent on the age of the leaves when the spores are discharged. It is only the very young leaves of the apple that are susceptible. It is not uncommon to find a certain whorl of infected leaves on young apple trees growing in a nursery, while the older whorls are quite free. Recently it has been shown that at least four and probably more races of the cedar-apple rust fungus exist.

CONTROL.—The control of cedar-apple rust is most readily effected by separating the apple and the red cedar by a distance sufficient to prevent the sporidia and aecidiospores from bridging the gap. It is held that if the cedars are destroyed within a radius of from one to two miles of an apple orchard, comparatively little damage will result to the apples. This method of control is applicable only where commercial orcharding is intensively practiced.

Spraying has not proved generally practical because the teleutospores germinate over such a long period in the spring and the apple tree forms young foliage very rapidly during that time of the year. Where the varieties grown are comparatively resistant and the amount of inoculum is not too abundant, satisfactory results may be obtained by spraying. The use of colloidal sulphur has given the most promising results when used frequently. Under farm orchard conditions where the red cedar is needed as a shelter belt, it often is problematical whether the cedar should be destroyed or the apple. If a new orchard is being set out, careful consideration should be given to the selection of varieties that are adapted to the region and are resistant to cedar-apple rust.

WHITE PINE BLISTER RUST

Cronartium ribicola F. de Waldh.

The white pine blister rust pathogen is held to be native in the Baltic provinces of the U. S. S. R. where it was first found on species of *Ribes* as early as 1854. In 1861 it was collected on pine in Finland, and gradually it spread south-

ward into central Europe and later to North America. The rust had become generally distributed in Europe by 1883 and probably was in this country as early as 1898 although the first authentic collection was not made until 1906 at Geneva, New York. In 1915 it was found on white pines in New England. At present white pine blister rust is general throughout the eastern United States and Canada, in the white pine area of the Great Lakes region and in the northwest, comprising Washington, Oregon and Idaho and the contiguous Canadian province of British Columbia. Blister rust also occurs in Japan and central China.

Only the five-needle pines and species of *Ribes* serve as hosts of the rust pathogen. Those pines having their leaves in fascicles of two or three are immune. The native host of the rust is held to be *Pinus cembra* L., Swiss stone pine, which is somewhat more resistant than *P. monticola* D. Don., *P. flexilis* James and *P. albicaulis*, Engelm.

The amount and extent of pine infection largely depends upon the kind of currant and gooseberry bushes, their number, size and exposure to the wind. Thus, the rust from three cultivated black currants in a single year infected 68 per cent of the pines in an adjacent eight-year-old plantation, but the damage was restricted to trees within 125 feet of the bushes. At another point, five cultivated black currants have supplied sufficient inoculum in a period of 15 years to kill or commercially destroy 47 per cent of the pines within 300 feet; but at a distance of 800 feet, fewer than ten per cent of the trees were diseased. Rust from a single wild gooseberry bush, three and one-half feet high and fairly well screened by surrounding forest growth, infected every white pine (25 trees) within a 20-foot radius. No evidence of infection from this bush could be found at a distance greater than 50 feet. In contrast with this, rust from an isolated wild gooseberry bush seven feet high, growing in the center of a young pine plantation and fully exposed to the wind, caused infection at a distance of 500 feet. Infections occurred over a period of four years in all cases.

At present, the white pine stand on 20;000,000 acres in

the United States is in danger of serious damage from the blister rust fungus. The potential white pine area when planted is doubtless equally as great as the present area of white pine stands. The coming of a destructive plant disease on a forest tree occupying so large an area is indeed a material catastrophe. The seriousness of the introduction of the blister rust fungus into America affects not only the United States, but also Canada's extensive white pine forests in the east and west. Although *Cronartium ribicola* has been known in central Europe for more than 50 years, the losses have not been so great as in America, because of their better-established system of forest production. Even in Europe the blister rust fungus has become so serious a pest that extensive propagation of the white pine is largely discontinued.

Ribes hosts suffer comparatively little injury from the rust, although in Canada it is reported that the rust defoliates currants. Wide variation in the susceptibility of species and varieties of *Ribes* prevails. The blister rust fungus is known to occur on at least 26 species of *Ribes*, 13 of which comprise the native cultivated species of currants and gooseberries. The currants, as a whole, are more susceptible than the gooseberries, and more of the varieties of black currants (*Ribes nigrum* L.) are susceptible than the varieties of red currants (*R. vulgare* Lam.). Fortunately, in this country, the black currant has not been as extensively planted as species of red currants and gooseberries. Almost all of the cultivated varieties of gooseberries (*R. grossularia* L.) are resistant. Among the native species of currants, *R. bracteosum* Dougl. and *R. petiolare* Dougl. are the most common alternate hosts in the northwest.

Symptoms.—The rust fungus attacks the leaves, twigs, branches and trunks of the five-needle pines. The pathogen enters the leaves by direct penetration, and once inside the leaves it spreads, causing yellow spots, which become brown following the necrosis of the invaded tissues. In other cases the mycelium may grow downward, following the bundles into the base of the leaf fascicles and the bark of the twigs or

limbs of the tree. The first noticeable symptom on the bark is a golden-yellow to yellowish-red streak. The rate and extent of spread varies with the vigor of the tree. If the tree makes a weak growth, the invaded tissues turn brown and die and the leaf fascicle in part or as a whole is killed; a conspicuous early symptom of infection



FIG. 226. Aecidial cushions extruded from cankerous area of white pine infected with *Cronartium ribicola*. (Reproduced from U. S. D. A. Dept. Circ. 226.)

When the tree is growing actively the infected tissues swell, causing a spindle-shaped enlargement of the stem on which the pathogen sporulates. The spermogonial stage develops usually from six to nine months after infection. The spermogonia appear as small blisters that exude a sweet, yellowish, sticky fluid containing the minute, pear-shaped spermatia. The next year the fungus produces blister-like, orange pustules, one-eighth to one-half inch across. With the breaking of the covering from these irregular or elongate pustules, a fine yellow powder, the aecidiospores, is sifted out and blown about by the wind. After the spores have disappeared, round depressions remain in the dead bark. The spreading of the mycelium and sporulation continues, leading to large dead areas that may girdle the stems. In cankers that do not girdle the stem, the mycelium may spread and produce new crops of spores each year. Exuding from these cankers, resin streams down the limb or trunk, forming long white streaks. This condition is very striking and is quickly perceived when searching for trunk cankers.

Small pine trees three to four years old may be killed in five years, and trees five to ten feet high are killed, or at least commercially destroyed, in five to ten years. Trees 10 to 20 feet high frequently are destroyed in eight to ten years after infection, although a longer period may elapse. Trees more than 20 feet high may suffer seriously in 12 to 15 years, depending on the number of branches infected and on the number of tips of long or short branches attacked by the pathogen. Occasionally, large trees may be killed in a few years by abundant short twig infections. It is known that a tree 40 feet high may have 260 twig infections in a single branch, and 36,000 separate infections may occur in one tree. When the leaves produced by adventitious buds on the trunk become infected the pathogen may reach the trunk of the tree in a comparatively short time and cause a cankerous condition that will promote the death of the tree.

The infection on currants and gooseberries appears first as small, yellow, slightly raised spots on the lower surface of the leaves. On these yellowish areas uredosori develop,

followed later by teleutosori. Necrosis of the tissues invaded by the pathogen follows sporulation, while under certain other conditions the leaf tissues are killed before the pathogen fruits. The injury to the currants and gooseberries is trifling compared with that on the susceptible pines.

ETIOLOGY.—The cause of white pine blister rust is the fungus *Cronartium ribicola*, a heteroecious rust that has two of its spore forms on species of *Ribes* and two on the pines, as shown in Fig. 227. It requires at least two years and often longer for this pathogen to complete its life history. Infection of the pine usually takes place in July. The mycelium, as typical of rusts, is intercellular with many haustoria extending into the adjoining cells. The intercellular mycelium seems to push the adjoining cells apart, which explains the presence of the characteristic swelling of infected tissues. In newly infected pines the spermogonia usually appear one growing season in advance of the aecidia. The very small, light-orange, rounded spermogonia usually break through the bark in April and May. The spermogonia contain many sporophores on which are developed the very small pear-shaped spores. These are held in a honey-colored sweet-tasting liquid exuded from the blister-like surface of the crevices in the periderm of the host. When the liquid has dried, only the scars of the spermogonial stage remain. The aecidia covered by their light-colored peridia, come to the surface through cracks in the bark. When mature they burst, liberating great quantities of yellow, rather thick-walled aecidiospores. In older infections the two stages may occur more nearly at the same time, because the mycelium becomes perennial and fresh crops of each type of spore may be produced annually. The aecidiospores may be disseminated by the wind, and if they fall on a susceptible Ribes host, they germinate and send germ tubes through stomata or wounds on the lower surface of the leaves.

The aecidiospores may retain their viability for some time under favorable conditions, and initiate new infections within a radius of more than 100 miles from their source. On Ribes the hyphae produced from the aecidial infection tube may

give rise to yellow hemispherical uredospore pustules in ten days. This spore stage will reinfect currants or gooseberries in the same growing season. They may remain viable over winter and initiate new infections on gooseberries the following season. Later in the growing season the teleuto-

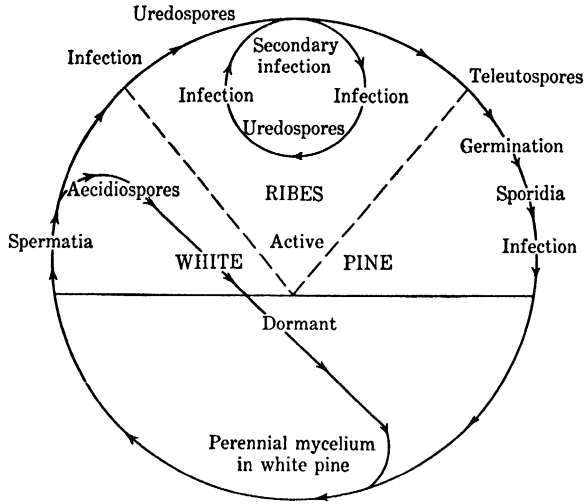


FIG. 227. A diagram of the host relation of *Cronartium ribicola*. This pathogen survives the winter in the teleutospore stage and as perennial mycelium in the infected white pine.

spore columns develop from the same mycelium that produced the uredosori. These curved, hair-like brownish columns consist of vertical rows of teleutospores. This stage may become so abundant as to impart a felt-like appearance to the lower leaf surface. These teleutospores germinate and produce sporidia, which function in bringing about infection of the pine needles. The sporidia are thin-walled, short-lived spores that are not blown far from the source of production. Few infections of pines take place beyond 300 yards from the gooseberry plant. The germ tube produced by the sporidium enters the pine leaf, and once the pathogen is inside the parenchymatous tissue, it may spread downward into the base of the leaf fascicle and into the bark of the twig. After a lapse of two to four years, the spermogonial and aecidial stages develop again.

CONTROL.—Forest tree diseases are difficult to control. In central Europe very few white pines are grown because of the rust. They are substituting other species in new plantings. This might be done in this country if no other effective control practice can be found. The great importance of the five-needle pines and their adaptability to much of our forest area, however, has led to the adoption of other control measures. The control of this pathogen is being attempted by foreign and domestic quarantines and by the eradication of the susceptible species of *Ribes* in and near the five-needle pines in the United States. The importation of susceptible pine seedlings and *Ribes* species is restricted through federal quarantines and the movement of the two host plants in the United States through regional and state quarantines.

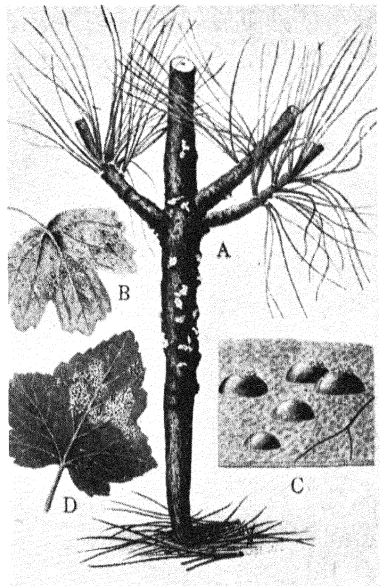


FIG. 228. A, white pine showing spermogonia and aecidia of *Cronartium ribicola* and hypertrophic area; B, uredosori of *C. ribicola* on Ribes leaf, C, enlargement of B; D, uredospores and teleutospores on a leaf of Ribes. (Reproduced from U. S. D. A. Farmer's Bul. 742.)

The shipment of five-needle pines from Europe and Asia has been prohibited since 1913. In 1917 similar restrictions were placed on species of *Ribes* originating in Eurasia. To prevent the spread of the blister rust from the eastern to the western part of the United States, a federal quarantine was established in 1917 prohibiting shipment of white pines or Ribes westward across the line following the western boundaries of Minnesota, Iowa, Missouri, Arkansas and Louisiana. Many western states supplement this transcontinental federal restriction by enacting state quarantines against the move-

ment of pines and Ribes. The Canadian government also is using quarantine restrictions as control measures.

Eradication of the native and cultivated species of *Ribes* has been practiced in New England for a period of years and has proved effective at a comparatively low cost. In five years the cost of removing 19,224,118 wild currants and



FIG. 229. Eradication of ribes for control of white pine blister rust. (Reproduced by permission of Division of Blister Rust Control, Bur. Pl. Ind., U. S. D. A.)

gooseberries from 1,504,945 acres of forest and pasture averaged 31.8 cents per acre. It is believed this will protect the white pine stand for five to ten years and in many cases for even a longer time. The eradication of the alternate hosts is done by scouts who go through the forest uprooting and destroying the Ribes. Such eradication measures have resulted in the destruction of 97.2 per cent of the bushes in a single area surveyed. This reduction of the Ribes materially reduces the amount of rust in the pines. During the ten-year period from 1918 to 1928 more than 6,000,000 acres of white

pine land have been covered by scouting parties to destroy the Ribes.

More recently, as stated earlier, the blister rust has invaded the pine districts of northwestern United States. The rough, mountainous condition of the area involved, coupled with a greater number of Ribes, makes the eradication more difficult and costly, but no more effective means of control has been found. Hand uprooting of Ribes is supplemented with the use of herbicides and fire. Indications are that Ribes eradication will prove as effective, although more costly, in the northwest as it has in the eastern part of the United States.

Recently a red currant known as the Viking has been found that is very resistant to the white pine blister rust fungus. It is a hybrid of a currant (*Ribes petraeum* Wulf.) native in the mountainous district of Norway.

WOOD AND ROOT ROTS

Palisade fungi, represented by the pore and gill fungi, are the highest group of the stalk fungi or Basidiomycetes. Most of them are saprophytic, and live on the dead organic matter present in the soil or in logs, stumps, lumber, etc. In each case the woody tissues are converted into crumbling masses known as punk; such fungi simply cause timber rots. The house fungus and white butt rot fungus are common examples. Others in this group are capable of attacking the living tissues of the tree through wounds and may be thought of as wood parasites; e.g., the Armillaria root rot and white streaked sap rot are such organisms. A few are parasitic, occurring not only on woody plants, but also on many different herbaceous plants.

Many of the diseases caused by the palisade fungi are so highly destructive that it is difficult to comprehend the extent of their destruction. It is known that fungi that attack wood are the most important agencies of wood destruction. Millions of cubic feet of standing timber, lumber and poles are destroyed annually by these fungi. Their damage to wood is greater than the combined destructiveness

of fire, wear, insects and weathering. In lumber products, it is easier to visualize the destructiveness of wood rotting fungi through specific familiar examples.

It is well known that the farming industry is our largest user of wood. The farmers use 40 per cent of all wood cut in the forests of the United States either for building material or for fuel. This industry uses less treated lumber than any other industry. Eight per cent, or nearly 2,000,000 cubic feet of our forests, annually is used for fencing material. Some further idea of the depreciation is apparent when we realize that 900,000,000 new posts are set every year. The great majority of this wood is returning only a portion of its maximum service, and therefore constitutes waste and much loss to the consumer. The losses caused by these palisade fungi to lumber in buildings, bridges, railroad ties and wharves are equally great.

The railroads are the second largest users of wood and the most extensive conservers of our forests through prevention of wood rots. The Chicago, Milwaukee and St. Paul railroad, studying the length of life of treated ties, found their replacements over a 20-year period have been 12 and 90 per cent respectively of the treated and untreated ties, or an actual saving through treatment of more than 2,000,000 ties per year.

In the case of the Atchison, Topeka and Sante Fe railroad with 17,709 miles of roadbed, for the 27 years, 1898 to 1924 inclusive, the tie replacements per mile were about 336 in 1898 and 115 in 1924 when using treated ties. The saving in board feet of lumber during the 27 years is so large that it is hardly conceivable. A treated tie lasts about three times as long as one not treated. Control of lumber rot through treatment in ties alone in 1924 saved 125,000,000 ties. Knowing that the average forest yield is about 60 ties per acre, about 2,100,000 acres were saved by tie treatment alone.

This destructiveness of the palisade fungi has another significance even greater than the actual loss of the lumber. It is the hastening of the depletion of our forests. It may

help to understand the significance of our uncontrolled wood-rotting problem when it is known that originally there were about 822,000,000 acres of forest; today there are only 98,000,000 acres of virgin forest with about the same amount of second growth forest and twice this acreage only suitable for cordwood. In other words, there remains today less than 400,000,000 acres of timberland. About two-fifths of the original forest are left and timber is being cut more than four times as fast as it grows; e.g., more than 4,500,000 acres are cut each year. In addition, 7,500,000 acres are burned over, constituting a loss of one-half billion dollars annually. This loss combined with the acreage cut over amounts to 12,000,000 acres denuded annually. Only about 36,000 acres are being reforested each year. If the ravages of the palisade fungi could be stopped, much of the losses now experienced would be avoided and the forests conserved much longer.

EFFECT ON WOOD.—Wherever fungi attack wood in any form, the fungus feeds on the wood and disintegrates the cells and tissues. The cellulose walls of the wood are changed into food through the action of enzymes or digestive ferments. These may act on the lignin and cellulose of the cell walls, and the wood may become discolored and spongy. In some instances the fungi liberate products that stain the walls of the wood. In other cases black lines border the advance of the mycelium. Diseases of the bark and trunk interfere with the passage of food normally moving down in elaborated form from the leaves to the roots. The fungi that attack the sapwood often cause discoloration and destruction of the cells and in this way interfere with the upward passage of water and the utilization of the stored reserves in the sapwood. In such instances the tree suffers from lack of water, causing a part or the whole of the tree to take on a yellow, stunted appearance, and later it may die. Where the fungi invade the heartwood, destroying some or all of the wood, the wind and storm resistance of the tree is endangered and the wood made worthless for lumber. Heart rots seldom produce symptoms that can be detected on the foliage or decrease the rate of growth. They enter

the heartwood through wounds of all kinds (fire, frost, lightning and insects). A few minor rot-producing organisms are said to be able to enter the heartwood through the unbroken bark.

When the palisade fungi attack the roots, the effects are much the same as when the bark and sapwood of the trunk and branches are parasitized. The tree takes on an unhealthy appearance, stops growing and finally dies. If the heartwood of the larger roots is invaded, the tree finally dies and falls.

The parasitic palisade fungi may be segregated into three major groups: (1) parasites of herbaceous plants, (2) parasites of roots of herbaceous and woody plants and (3) parasites or saprophytes causing wood rots.

Black Scurf of Potatoes

Corticium vagum B. & C.

This disease is general in its distribution, and it probably occurs wherever potatoes are grown. Its destructiveness varies with soil conditions and environment. Before the perfect stage of the causal agent was discovered, the imperfect stage was known as *Rhizoctonia solani* Kühn. This name has become so well-fixed in our literature that the black scurf disease of potatoes is often referred to as being caused by *Rhizoctonia solani*. Although the black scurf disease is best-known on potatoes, it is found on many other cultivated and wild hosts. The organism has been described on more than 75 host plants.

Symptoms.—The most conspicuous sign of the black scurf disease is the presence of the shiny-black sclerotia on the surface of the potato tubers. The shiny-black color is most pronounced when the tubers are wet. The laity often think of these sclerotia as particles of dirt adhering to the surface of the tuber. These sclerotia are really masses of mycelium formed by the fungus after the tubers have started to develop in the soil. They are superficial and can be removed with a little effort.

This fungus causes dark-brown lesions on the stems, stolons

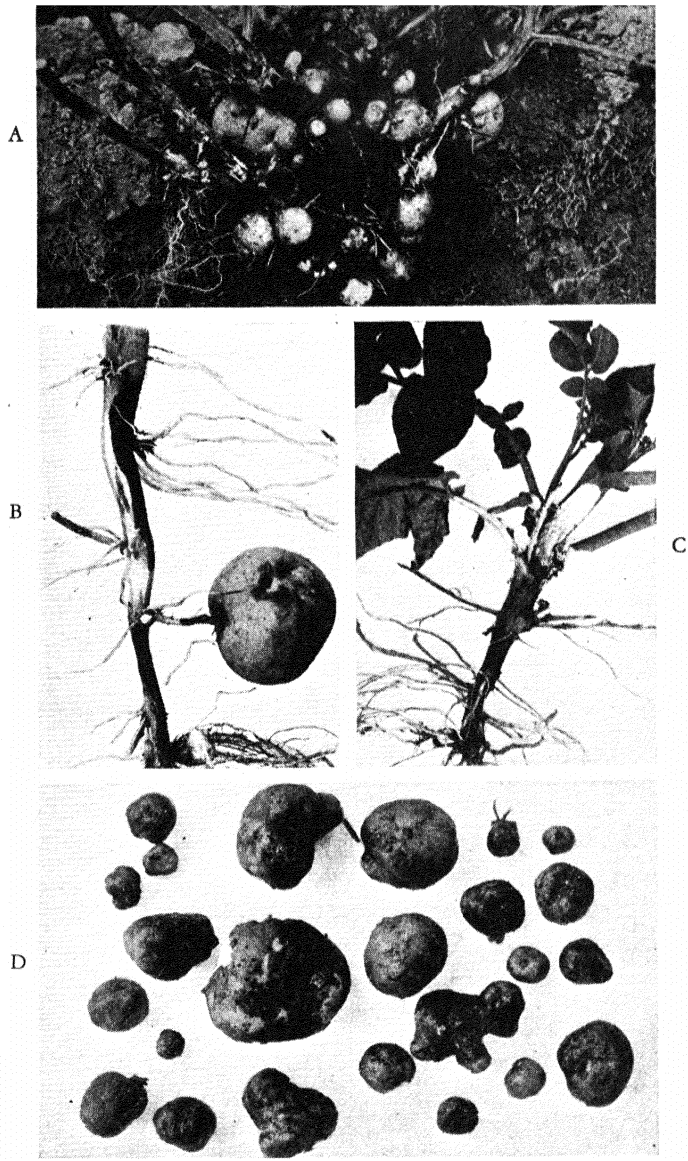


FIG. 230. A, lesions on stems and roots of a potato plant infected with *Corticium vagum;* B, black scurf lesions on potato stem, roots and tubers; C, fruiting stage of *C. vagum* on the upper portion of the potato stem; D, black scurf lesions and sclerotia on potato tubers.

and roots of the potato plant. These may vary in size from little pin-point spots to areas several centimeters in length. In some instances the lesions entirely girdle the stems and stolons. The mycelium invades chiefly the cortex and seldom attacks the more woody portions of the underground parts.

Another characteristic symptom of the black scurf disease is the formation of tuber-like swellings in the axils of the leaves and on the stem at or just below the surface of the soil. These swellings or tuber-like bodies are a result of the lesions on the under part of the stem, which prevent the translocation of the assimilated food down into the tubers. Enlarged buds, or small tubers, may vary from one-half centimeter to four or five centimeters in diameter. Their color may be either green or white, depending upon whether they are aboveground or below.

The foliage of plants producing "little potatoes" is often a rich green, especially in the early stages of the formation of the little tubers. Later, they may become etiolated. The internodes are frequently shortened, the whole plant taking on a stocky, low, bushy appearance. As a rule plants showing these symptoms bear no marketable potatoes.

The black scurf fungus is one that occurs in destructive form on many crops, notably onions, beets, lettuce, celery, beans, tomatoes, sugar beets, etc. On these crops it may cause a variety of symptoms, ranging from root lesions to fruit rots. Its chief destructiveness in the case of the crops listed, however, is much the same as that on the potato. It is mostly the underground parts of the plants that are injured. The symptoms manifested on the aboveground parts of the plants are secondary, resulting from the activities of the pathogen on the underground roots and stems.

ETIOLOGY.—*Corticium vagum* is chiefly a soil-inhabiting fungus. The mycelium in the imperfect or Rhizoctonia stage is brown and is often evident on the surface of the tubers in long, rather thick strands. The mycelium of the perfect stage is grayish-white and does not form the coarse strands. On the tuber the mycelium knots itself together into dense,

shiny-black masses, the sclerotia, which are actually resting organs. When placed under favorable growing conditions, the mycelial threads may grow out from the surface. No asexual spores are produced. (See host relation in Fig. 231.)

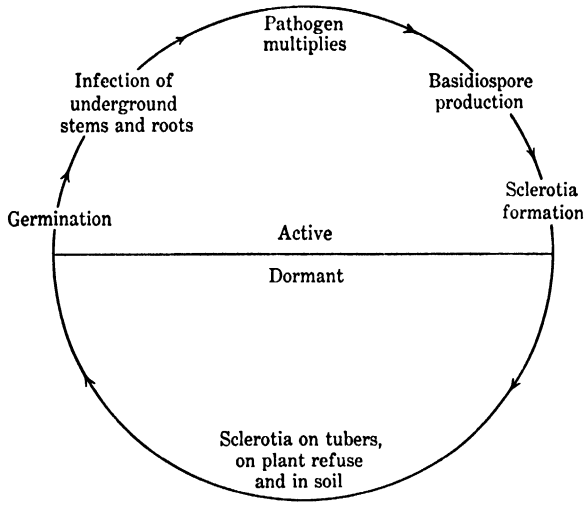


FIG. 231. A diagram of the host relation of *Corticium vagum*. This organism survives the winter as sclerotia on tubers, on plant refuse and in the soil.

Under favorable conditions of moisture and temperature, the mycelium may grow up the stems of plants, forming a rather compact, dense mat on the surface of which are borne club-shaped basidia bearing basidiospores. This stage of the fungus is very dependent on moisture conditions. The basidiospores are forcefully discharged and fall to the soil where they germinate immediately.

Corticium vagum is most destructive to the sprouts of the germinating potatoes when the soil temperature is between 9° and 18° C. After the temperature increases above 18° C. the organism becomes less destructive to the underground parts of the plant.

It is held that *Corticium vagum* is favored by an acid soil. Soils that are inclined to be wet and heavy are probably more favorable than those that are light and sandy. High

soil temperatures, above 25° C., in the fall seem to inhibit the development of sclerotia on the tubers.

CONTROL.—Control of the black scurf disease is not simple in view of the fact that it is not only a soil-inhabiting fungus, but also one that forms sclerotial bodies that are very resistant to adverse growing conditions. For potatoes probably the most effective control measure consists of securing clean seed and growing the crop in a long rotation, at least not oftener than every three years. The other crops in the rotation should be plants that are not very susceptible—the cereals are probably such crops. Where infected seed is used, sprout and stem lesions may be avoided by delaying planting until the soil temperature is high—about 18° C. Seed treatment also tends to prevent injury to the sprouts. Unless the seed is strikingly free from Rhizoctonia, either in the mycelial or sclerotial stages, it should be treated.

MUSHROOM ROOT ROT

Armillaria mellea (Vahl.) Quel.

The common name of the fungus *Armillaria mellea* is honey mushroom. It is called this because the color of the sporophore resembles that of honey. *Armillaria mellea* is a wound parasite, but it may live saprophytically on decaying organic matter such as rotting logs and stumps, as well as parasitically on the roots and crowns of trees in the forest and orchard. It also is known to attack small fruits and herbaceous plants in the northwestern part of the United States.

The mushroom root rot fungus is rather generally distributed in the Old and the New Worlds, but it seems to be more prevalent in this country west of the Rocky Mountains. In Europe it is considered a very serious pathogen on coniferous trees. In this country in the state of California, it is reported as destructive in citrus orchards where the trees have been planted on newly cleared ground, especially where oaks preceded the citrus trees.

SYMPTOMS.—The first evidence of the mushroom root rot is the sickly appearance of the tree caused by the light-yellow

foliage and poor growth. In general, a tree never recovers but continues to become worse, and finally the leaves are dropped prematurely. If the dirt is removed from the roots of infected trees, necrotic areas of various sizes will be found. The mycelium of the parasite enters the roots through wounds caused by some other agency, and finally necrosis of the tissues takes place. The result of the fungus action in the cortex and wood is a serious interference with the function of the roots and the translocation of food materials from the leaves downward.



FIG. 232. Bark removed from peach root to show mycelial mat of *Armillaria mellea* in the cambial regions. (After Harold E. Thomas.)

On the surface of the roots, long, black, shoestring-like strands are evident. These are made up of compact mycelium and are known as rhizomorphs. They push their way through the soil and in that way serve as a means of spreading the organism. In the fall of the year after the mushroom root rot fungus has become well-established, if there is abundant moisture, it produces sporophores in great abundance about the crown of the plant. The sporophores are honey-colored, large and have a sticky surface.

ETIOLOGY.—*Armillaria mellea* is a typical agaricaceous fungus. (See host relation diagram in Fig. 236.) Its honey-

colored sporophores occur in clumps about stumps and bases of trees. Each sporophore is made up of a stipe, a convex pileus and the annulus. The annulus is present although it is rudimentary and disappears quickly. On the gills are the short, stalk-like cells, characteristic of the Basidiomycetes, on which are borne the basidiospores. These basidiospores are forcibly discharged, and when they fall on the ground they germinate and, if possible, invade organic matter. In this organic matter the mycelium may spread considerable distances in rhizomorphic strands, which may vary in length from a few inches to several feet. These are made up of loose mycelia covered with a brown cortex. The end of each rhizomorph is covered with loose mycelium extending out from a gelatinous covering. When the rhizomorphs enter the roots of trees they flatten out, invade the cambial area and cause the separation of the bark and wood. In such cases they become fan-shaped. Under favorable conditions the rhizomorphs make their way into the wood through the medullary rays as flat, fan-shaped, mycelial masses.

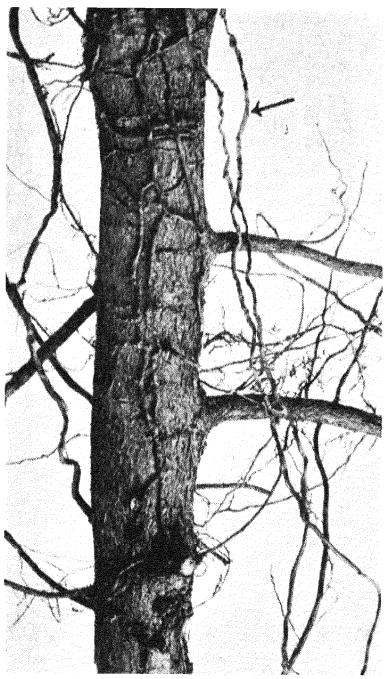


FIG. 233. Rhizomorphs of *Armillaria mellea* on and adjacent to roots of *Pyrus ussuriensis*. (After Harold E. Thomas.)

CONTROL.—Once the mycelium of the *Armillaria mellea* has entered the wood, it is difficult to save the tree. If the invasion of the tissue is discovered early enough, it is possible to cut out the infected portion, surface disinfect and dress the wound. It is good practice to avoid planting newly



FIG. 234. Sporophores of *Armillaria mellea* growing on a cherry stump. (Courtesy of Donald Cation.)

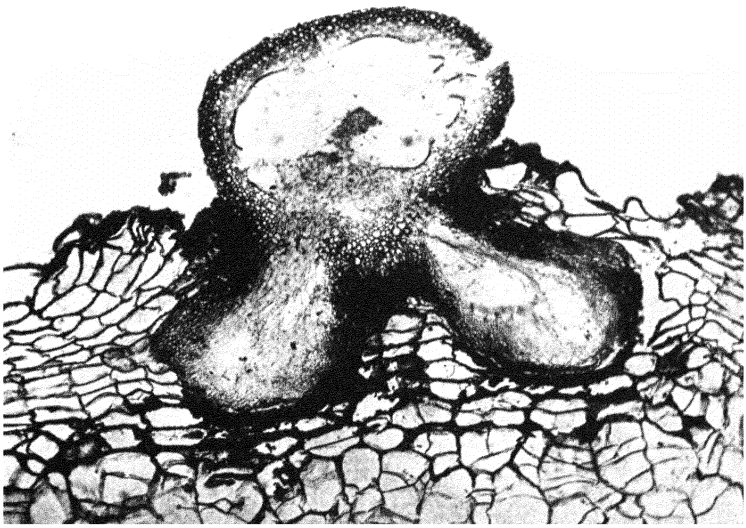


FIG. 235. Section of dahlia tuber showing rhizomorph with two branches growing into the host tissue. (After Harold E. Thomas.)

cleared land to fruit trees until it has been cropped for three or more years. In Europe it is considered unwise to follow deciduous trees with conifers. Where single trees are infected in an orchard, it is sometimes possible to confine the parasite by digging a ditch around the tree about two feet deep and two and one-half feet wide and throwing the dirt inward.

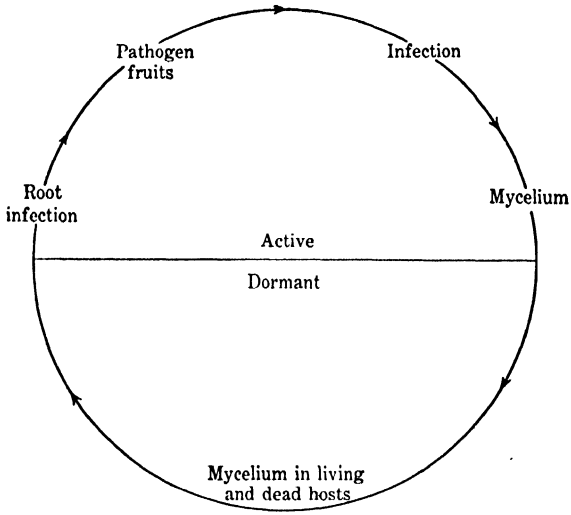


FIG. 236. A diagram of the host relation of *Armillaria mellea* This organism survives the winter as mycelium in living and dead hosts

Where the disease occurs in an orchard and the infected trees are removed, it is well to work the ground two or three years before resetting with other trees.

WHITE HEART ROT

Fomes igniarius (L.) Gill

The common name of the organism causing white heart rot is false tinder fungus. It attacks chiefly deciduous trees, as aspen, oak, apple, butternut, walnut, willow, maple, birch, beech and cottonwood, and is widely distributed, occurring in every continent throughout the range of its different host plants. The pathogen causes enormous losses in hardwood forests through the decay of the trunks of the trees. There

are, however, more than 20 different species of *Fomes* that cause wood decay on either coniferous or deciduous, hard or soft woods. *Fomes igniarius* is particularly destructive on aspen, a short-lived tree yielding low-grade forest products as pulp, excelsior, posts and low-grade lumber. In Utah

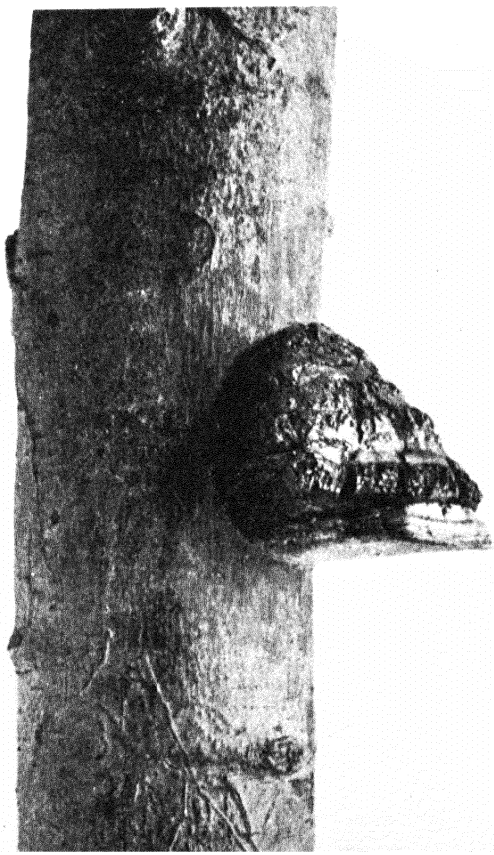


FIG. 237. Fruiting body, or conk, of *Fomes igniarius* on aspen.

there is an average gross cull of aspens of over 18 per cent for all age classes from 30 to 170 years. In Minnesota the total rot of the merchantable volume of aspens of different ages is reported as follows: 70-year trees, 31.2 per cent; 60-year trees, 27.5 per cent; 50-year trees, 23 per cent; 30-year

trees, 14.8 per cent. When only the late stage of the heart rot is considered the loss to the merchantable tree value is less, ranging from 9.1 per cent in 70-year trees to 0.7 per cent in 30-year trees. It is almost impossible to find healthy aspens of any appreciable age in New England, Colorado and New Mexico. The beech and the sweet and yellow birch

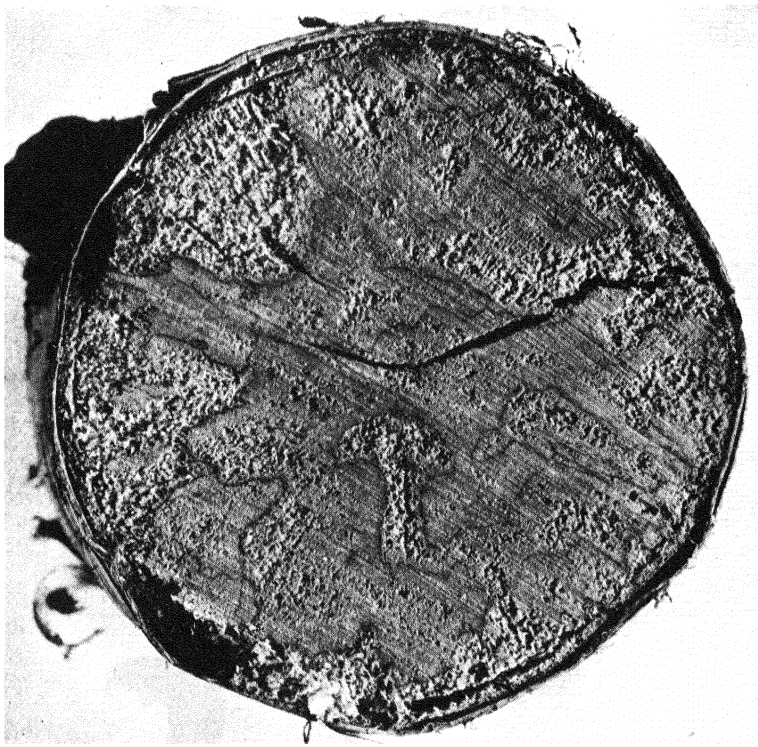


FIG. 238. White rot and marginal discoloration of rotted area of birch infected with *Fomes igniarius*.

are severely injured in New England, and apples, plums, pears and oaks may be seriously damaged in Europe.

SYMPTOMS.—The white heart rot fungus attacks chiefly the heartwood of trunks of deciduous trees. It is unknown in the roots and fails to develop in cut lumber or structural timber. The heart rot symptoms are most apparent in cross sections of the trunk as irregularly shaped yellowish-white

areas bounded by yellowish-green to black bands. The bands, which may vary in color in different hosts, mark the invasion zone. The discolored band marks the area of greatest activity of the advancing pathogen. Under certain conditions the fungus may invade and kill the sapwood and bark of living trees. The decayed wood within the discolored band is light, soft, whitish and uniform in texture, having fine, irregularly concentric black lines. The area of decay is irregular because certain annual rings may be invaded at one point but not at another. The destructive action of the pathogen may extend comparatively long distances up and down the trunk, frequently from 2 to 20 feet, but the tree seldom becomes hollow. The organism is also a common scavenger on dead trees and culled logs in the forest.

The organism that causes white heart rot develops large perennial hoof-shaped sporophores known as conks on dead and dying trees. The conks may vary in size and shape on different hosts, but in general the sporophores are hard, woody and rough above with a smooth brownish palisade of tubes on the lower surface.

ETIOLOGY.—The causal agent of white heart rot is a fungus usually known as *Fomes igniarius*. On the surface of the infected trunk or log the fungus forms conspicuous hoof-shaped, woody, grayish-black sporophores or conks. The upper surface of older conks is cracked, furrowed and ridged. The tissues of the conks comprise woody layers alternating with remnants of successive sporiferous layers made up of pores filled with white punky material. It is sometimes possible to determine accurately the age of conks by counting the remnants of the sporiferous layers. Some conks may have 50 and 80 layers representing as many years. One conk may indicate a linear spread in the heartwood of 15 feet, but in the aspen the conks follow more closely the progress of decay in the heartwood.

The fungus shows wide variability producing sporophores that differ in shape, appearance, pore and hyphal diameter on different trees. The fungus seems to be heterothallic with haploid lines of different sex groups. The above-

mentioned variability has led to the subdivision of *Fomes igniarius* into several species or groups based on sporophore characters on different hosts. The sporophores are perennial, producing annually a new brown fruiting layer on the lower surface of the conk. The fruiting surface consists of a palisade of tiny tubes or pores lined on the inner surface with numerous basidia bearing brown spores. These sporidia are forcefully ejected from the basidia when mature and may be carried

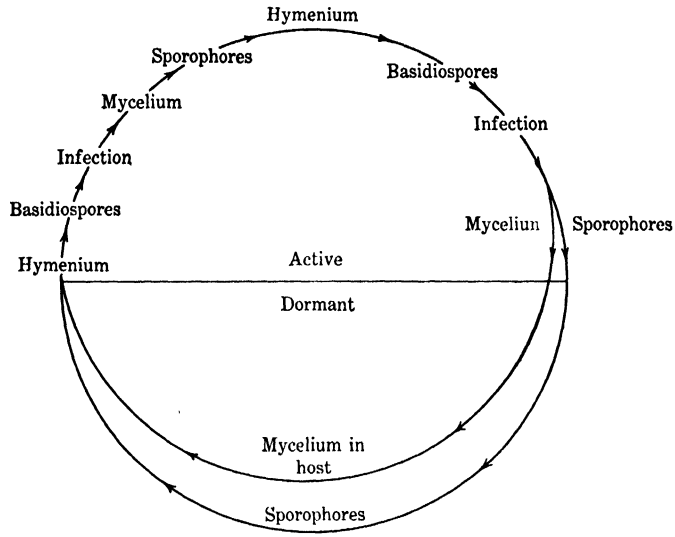


FIG. 239. A diagram of the host relation of *Fomes igniarius*. This organism has perennial fruiting bodies and usually requires more than one year to complete its life cycle.

great distances in air currents. When these spores fall on a wound (stub of a branch) they germinate and penetrate the heartwood. Infection is most likely to occur on the butternut tree at 15 to 20 years, aspen 20 to 25 years, yellow birch 20 to 25 years, beech 20 to 35 years and silver maple 35 years.

The penetrating hyphae may grow through the cell walls or move through natural openings in the walls. The pathogen produces enzymes that destroy the pigment and disorganize the lignin and cellulose of the cell walls. The mycelium may be of two sizes, the older mycelium in the decayed wood is

large and brown in color, while the young mycelium in the invading zone is small, much-branched and hyaline.

CONTROL.—The control of forest tree diseases offers many difficulties not inherent in cultivated annual crops. Trees in a forest, because of the low value do not permit such costly practices as spraying, dusting and wound dressing with fungicides.

The pathologist and forester must turn to the utilization of preventive measures such as promotion of tree vigor through timely thinning, site selection, lowering the rotation period and protection against wounds. The proper practice to follow depends largely on the value of the tree product. In the aspen, a tree of low commercial value, the most practical control measure is that of shortening the rotation and preventing wounds through fire and other agencies.

Chapter Fourteen

DISEASES CAUSED BY SEED PLANTS

THERE are about 1,700 species of parasitic seed plants belonging to 12 distantly related families of the Dicotyledonae, distributed throughout the world. None of the Monocotyledonae or Gymnospermae are known to be parasitic. The mistletoe (Loranthaceae) and figwort (Scrophulariaceae) families contain the majority of the parasitic species; e.g., in the mistletoe family there are said to be 850 species distributed in 21 genera and in the figwort family 350 species in 11 different genera. Parasitic plants are scattered in ten other families, none of which contains as many representatives as the mistletoe or figwort families.

There are about 250 parasitic flowering plants known to occur in the United States. The most important and well-known of these are 54 species of dodder and 40 species of mistletoe. Other less important representatives are species of cancer root (*Conopholis*), eyebright (*Euphrasia*), cowwheat (*Melampyrum*), red rattle (*Pedicularis*), yellow rattle (*Rhinanthus*), ratany (*Krameria*), etc.

The parasitism of the seed plants may be expressed as an attack either on the roots of the host plant or on the aerial parts, or in a few cases the parasite may attack the stem and roots. The parasite in the majority of the species is only partially dependent on the host. In a few cases, however, the parasite is completely dependent on the host for its development, similar to the obligate parasites in the fungi.

ROOT PARASITES.—Among the parasitic seed plants that attack the roots of their hosts, the least dependence on the host attachment for mature development is shown in species of Sandalwood and *Comandra*, which are mainly tropical

in their distribution. In these forms, which are capable of carrying on photosynthesis and of independent growth, the attachment to the host roots allows the parasite to obtain only water and mineral elements. In the eyebrights and cowwheats, which are general in the tropical and temperate countries, and the red rattles and yellow rattles of Europe and the Americas, the parasites are dependent on the attachment to the roots of their hosts, mainly grasses, for their maturation. In the absence of this host root attachment, the parasites are able to produce only a feeble growth. If they flourish and develop normally, the parasites are attached by their sucker-like specialized roots to the host. The disc-shaped attachment sends haustorial plugs into the conducting tissues of the host, where the parasite obtains water, mineral elements and food materials. The parasites are green and carry on photosynthesis, but depend on some food or other material synthesized by the host for their development. Some of the root parasites have poorly developed leaves and only a small amount of food is formed. Here again the parasitic roots, in contact with all the conducting elements of the host, obtain food and minerals.

In the root parasites that depend completely on the host connection in order to flourish, all of the plant food is absorbed through the attachments to the host. In the case of broomrape (*Orobanche*), which is found in North America and western Europe, the seed, when it germinates, produces a filamentous root-like structure with no differentiation into stem and leaves. This filament penetrates to the roots of a host plant, as clover, hemp, ivy, tobacco and many others, where it forms a flask-shaped attachment organ. From this attachment there are produced conical plugs, which penetrate into the vascular system of the host. The leaves of the parasite are reduced to mere scales on the flowering stalk.

The parasitic flowering plants that show the greatest dependence on their hosts are species of the families Rafflesiaceae and Hydnoraceae, which are confined to the tropics or south temperate zone. In these plants the vegetative body is reduced to filaments, much resembling the hyphae of fungi,

which ramify throughout the roots of the host, extending in some cases into the stems. The parasite resembles the bracket fungi in that its vegetative body remains entirely submerged within the host until it produces the enormous solitary flower.



FIG. 240. Four plants of broomrape growing on roots of tomato. (After G. L. Stout.)

STEM AND LEAF PARASITES.—In the parasitic seed plants that are located on the aerial portions of the host the simple condition of partial dependence on the host is best seen in the leafy mistletoes, the *Phoradendron* of North America or *Viscum* of Europe. Here the sticky seed of the parasite on being wiped off a bird's beak after the bird has eaten the enclosing fleshy ovary wall, remains on the limb of the tree and germinates. The root on making contact with the limb of the tree forms a disc-shaped sucker from which the haustoria-like roots penetrate into and grow with the cambium of the host. The opposite, leathery, green leaves produce practically all the food, the water and minerals being ab-

sorbed from the host plant. In the related dwarf mistletoe on the conifers of North America there apparently is absorption of food also from the host as evidenced by the injury and the fact that the leaves are not only small and underdeveloped, but also deficient in chlorophyl.

Finally there are the aerial parasitic forms which may be completely dependent on their hosts for a food supply but are distinguished by their twining habit. This type is represented by the species of *Cassytha* on the leaves and twigs of tropical plants and by the species of dodder (*Cuscuta*). In the dodders the twining parasite obtains its food, minerals and water through suckers extending into the stem of the host. The parasite may lack chlorophyl and may be limited to one species.

Dodder of Clover and Alfalfa

Cuscuta arvensis Beyrich, *C. epithymum* Muir. *C. planiflora* Ten., *C. indecora* Choisy

These parasites are known by a number of names, as love vine, hell-bind, strangleweed, hairweed, devil's-hair and many others. There are about 100 species of dodders that are nearly universal in their distribution, and are abundant throughout the tropic and temperate zones. Besides being found on some crop plants, they are frequently abundant on wild plants.

When the dodder is encountered in the field, it usually is considered as a twining weed, and few people know that it is one of the most important parasitic flowering plants. The region of its greatest destructiveness is in Europe, especially in southern Europe. In the southern soviets of the U.S.S.R. *Cuscuta epilinum* Weihe is reported to reduce the yield of flax by half. The loss from dodder in this country, as in England, is not generally considered of great importance. This probably is accounted for in that of 54 species in North America, only seven are on plants of economic importance. Only three crops, clover, alfalfa and flax, are subject to much damage from this parasite. The damage, however, is often underestimated by the growers.

SYMPTOMS.—The presence of dodder in a field may be suspected when a group of plants is seen to be pulled together and drawn to the ground as if by wires. The yellow- or orange-colored, thread-like stems of the plant, bearing the small colorless leaf-scales, radiate from the point of initial contact and draw all plants toward this point. The parasite winds around the host, often binding the leaves to the stem, and may extend from one plant to another and by the coiling of its stems draw them together. This bunching of the host plants, with the evidence of the entwining yellow parasite with its numerous clusters of white or pink flowers, is very characteristic. At certain points of contact, the host may be stimulated to gall formation.

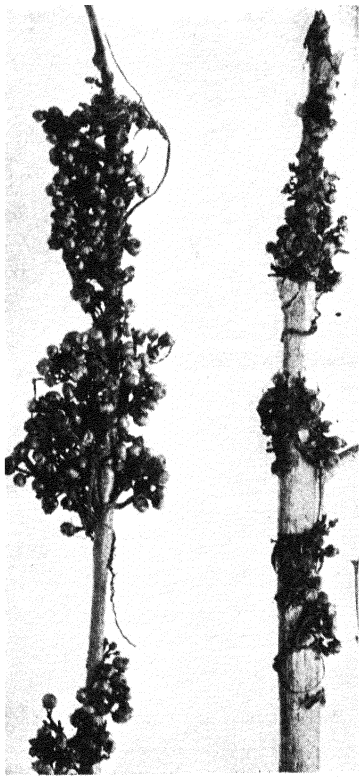


FIG. 241. Dodder on sumac showing the mature fruit.

The area of the field infested seldom becomes more than ten feet in diameter in the first year, although the spread during the second and third years is much more rapid, and may cover large tracts. The parasite, besides greatly interfering in the haying operations, saps the host plant of food, minerals and water and greatly reduces its yield and food value.

The parasite may be detected easily when contaminating clover and alfalfa seed. The small oval to tetragonal, rough-coated, dark-colored seeds are generally easily distinguished from the smooth, shining, lighter-colored alfalfa seeds.

ETIOLOGY.—The common dodders *Cuscuta arvensis*, *C. indecora*, *C. epithymum* and *C. planiflora* belong to the family Cuscutaceae. The twining wire-like, yellow or orange stem bears at intervals the small scale leaves, which, like the stem, are devoid of chlorophyl. After the first of June the stems are almost concealed by the large clusters of white or pink flowers. The stamens are inserted upon the throat of the gamopetalous corolla and alternate with its segments. The biloculate ovary, bearing two distinct styles, forms a capsule in which the seeds mature. An average plant is said to produce about 3,000 of the black rough-coated seeds per year. The plant is

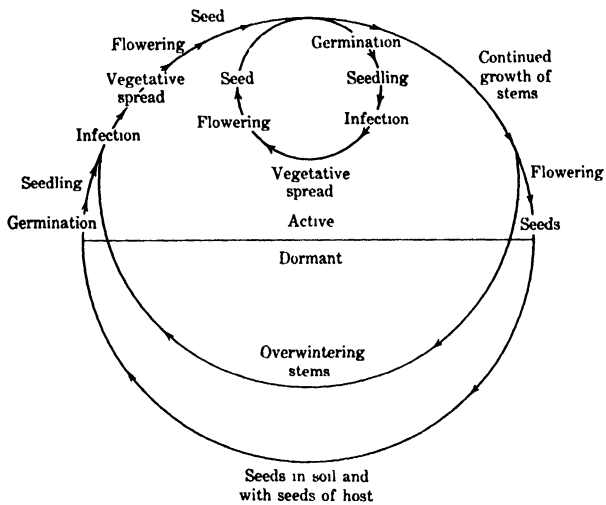


FIG. 242. A diagram of the host relation of dodder The plant normally overwinters as seed in the soil or in farm seeds. It may survive also in vegetative portions of perennial hosts.

unique in that cut plants seem to have enough stored food in the stem portion bearing the flowers to mature seed if they are nearly half-formed at the time of cutting. It is further claimed that seeds approaching maturity will germinate under favorable conditions without finishing the process of maturation. The seed, germinating in the spring, produces a small wire-like seedling, with rudimentary roots, which dies in a short time unless the circling stem tip finds a host plant. If a host is encountered, the seedling curls around

it two to three times and haustoria are produced at points of contact.

The haustoria that attach the parasite to its host and also function in the absorption of food and water are root-like, adventitious outgrowths produced as a result of contact stimuli. The haustorium, by a combined process of pressure and dissolution of the cell wall material of the host, forces its way into the conducting tissue of the host. The central cells push into the pith cells; the cells outside these come in contact with vessels of the host and become differentiated as tracheids connecting the xylem of the parasite with that of the host; similarly the next outer layers become differentiated as connecting phloem strands; the outside layers come in contact with the cortex. Therefore, the parasite may obtain its food, water and nutrients directly from the translocation streams of the host. It is probably the result of the action of the haustorium that stimulates the gall formation.

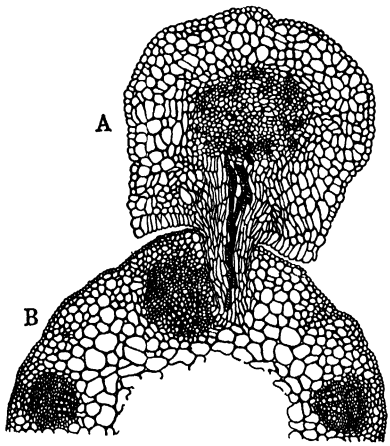


FIG. 243. A, cross section of dodder stem; B, stem of parasitized clover plant. The haustorium is in contact with a fibrovascular bundle of the host.

The dissemination of the parasite is said to be by spread in clover and alfalfa seed, in hay, in manure, by stock or by implements. The prevalence of the dodder on clover, alfalfa, flax, etc., may possibly be attributed to the very close agreement in size of the seeds of the host and parasite making the separation difficult. The parasite overwinters mainly as seed, although the stems of the parasite are said to be able to grow a second season if not detached from the host.

CONTROL.—It is important to prevent the introduction of dodder seed in clover and alfalfa seed and to avoid infested

manure and hay. Seed stocks containing dodder may be cleaned. In some species of dodder, however, the seeds are so nearly the same size as the host seed that cleaning is not readily possible on the farm. In such cases the seed may be sent to commercial seed-cleaning centers where better equipment is available. In some states clover and alfalfa seed contaminated with dodder cannot be sold.

Infection in the field should be eradicated by cutting, allowing the plants to dry, sprinkling with kerosene and burning in the field. This kills the seeds and stems and prevents further growth or spread of the parasite. If the area contaminated is large and infection light, a five-year crop rotation without susceptible crops is advisable.

Mistletoe

Phoradendron and *Arceuthobium*

Although mistletoe is known by nearly every one for its decorative and sentimental uses at Christmas, few know it as a dangerous parasite of trees. The parasites are found in

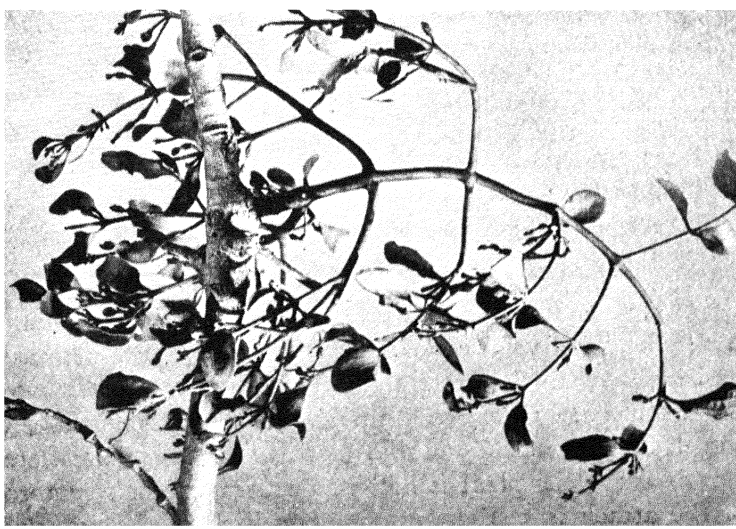


Fig. 244. A mistletoe growing on a limb of a cottonwood tree in New Mexico. (Courtesy of J. N. Martin.)

Europe, Australia and North and South America. The distribution in the western hemisphere extends from Oregon, southern Colorado, Indiana and New Jersey, south into the northern portion of South America. Species of *Phoradendron* and *Arceuthobium* are found on deciduous and coniferous trees. The damage produced is only of importance in western United States where the parasite seriously endangers the trees in the arid and mountainous regions. The chief damage produced by the parasite is in the death of young trees, a decrease in the yearly increment of growth and through the opening of the host to the attacks by fungi.

The species found in the western hemisphere are only partial parasites although the family contains parasitic and free-living members. The American species are really "water parasites." There are some 28 species of *Phoradendron* and five species of *Arceuthobium* in the United States. The latter are confined to North America, while the 240 species of *Phoradendron* are spread over the Americas and certain islands of the Pacific.

The species commonly found in this country are:

Phoradendron flavescens Nutt.
P. villosum Nutt.
P. californicum Nutt.
P. libocedri Howell
P. juniperinum Engelm.
Arceuthobium campylopodium (Engelm.) Piper
A. americanum (Nutt.) Kuntze
A. douglasii (Engelm.) Kuntze
A. pusillum PK.

SYMPTOMS.—The obvious sign of the disease is, of course, the presence of the bushy, dark-green growth of the parasite on the branches of the host. These are especially evident in the winter when the green mistletoe is very conspicuous on the deciduous hosts. In the case of the dwarf mistletoe, *Arceuthobium*, the plant is inconspicuous. The first symptom of the attack is a slight fusiform swelling of the branch of the host at the point of infection. After the growth of the parasite, the branch beyond the infection dies and is easily

broken, leaving the parasite terminating the branch. Large limbs and trunks may be deformed, and the reaction of some hosts is the production of burls or witches' brooms.

Infection also is evident in the delayed growth in the spring



FIG. 245. Dwarf mistletoe on a young pine branch. (Courtesy of J. N. Martin.)

and a marked stunting. The host, however, is rarely killed the first season unless the attack occurs on seedlings or young trees. With the possible exception of some species of *Arceuthobium*, the parasites apparently are much more abundant in small or isolated plantings than in forests.

ETIOLOGY.—The American mistletoes, species of the genera *Phoradendron* and *Arceuthobium*, belong, with the European mistletoes, *Viscum* and *Loranthus*, in the family Loranthaceae. There are a large number of species in each of the American genera. Each genus has species that attack coniferous and deciduous trees.

The parasites are seed plants and their distribution is dependent on the dissemination of the seeds. The fruits are berries that contain a single seed within a sticky mesocarp.

These berries are much sought after by birds, which wipe the sticky seed off their beaks on the limbs and distribute the seeds in this way.

Under favorable conditions the seed germinates by produc-

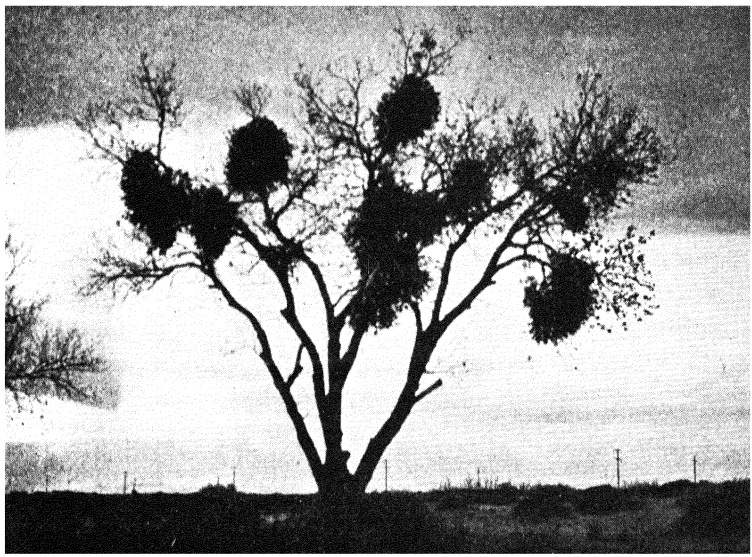


FIG. 246. A cottonwood parasitized by mistletoe. (Courtesy of J. N Martin.)

ing a short hypocotyl and radicle. This latter structure on coming in contact with the host bark forms a flattened, circular appressorium. From the appressorium a small projection, or haustorium, grows into the host and makes contact with the xylem. The water supply and nutrients of the parasite are gained through this projection. The growth of the parasite is slow and by the end of the second year it still has only one pair of leaves. The parasite continues growth and the leaves and stems being well supplied with chlorophyl produce all the food for the plant. There is no connection with the host phloem such as would allow the exchange of food.

The mistletoes have short internodes, and opposite leaves that are petiolate in *Phoradendron* and scale-like in *Arceu-*

thobium. They are dioecious and have inconspicuous apetalous flowers. The sepals are distinct and persistent in the fruit, and the stamens are two-celled and sessile at the base of the sepals. The one-celled, one-ovuled ovary produces a white or yellow berry, with a characteristic sticky mesocarp around the single seed.

The haustorium that is produced by the growth of the

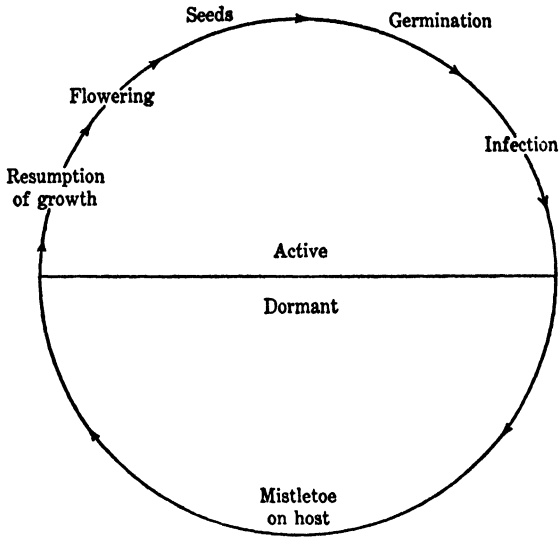


FIG. 247. A diagram of the host relation of mistletoe. The plant may overwinter on the living host.

appressorium generally surrounds the infected twig. If the shoots are detached from this appressorium, the latter is capable of producing adventitious buds and re-establishing the parasite.

The phenomenon of biological specialization, already discussed in the fungi, has been found in *Viscum album*. There is as yet insufficient work on the American species to indicate whether this condition is present in *Phoradendron* and *Arceuthobium*.

The 28 species of *Phoradendron* in the United States have as their dominant hosts the oaks, poplars, willows or ashes, depending on the region of the country. The most prevalent

species, *Phoradendron flavescens*, occurs on at least 38 different hosts. The five species of *Arceuthobium*, which are limited to the conifers mainly, are probably most serious on yellow pine and Douglas fir, although nearly all species of conifers may be attacked.

CONTROL.—The dissemination of seeds must be prevented and existing infections destroyed. The stems of the parasite may be removed with a hook. Since the haustorium is capable of producing new sprouts, however, it must be removed also. A simpler method, if infections are not too numerous, is to prune out all infected limbs. When the infection is general in the tree or on large limbs, however, the removal of the food-producing portions would probably be more detrimental to the tree than the presence of the parasite. The removal in the latter case might be done with a gouge or chisel, being sure to remove all of the suckers and haustoria. All large wounds should be filled with tar and then painted with carbolineum or asphalt paint. If the latter is used, the wound should be covered with burlap after the painting. In lumbering tracts all trees that are infected should be removed as a preventive measure, whether or not they are useful for lumber.

Chapter Fifteen

DISEASES CAUSED BY PLANT PARASITIC NEMATODES

NEMATODES are animals commonly called round worms, eelworms or "nemas." They inhabit tropical, semitropical and temperate regions and occur in all types of habitats. Many are free-living, flourishing under diverse conditions, while others are parasitic in animals and plants. Some of the plant parasitic nematodes have a wide host range, attacking plants that belong to the monocotyledons, dicotyledons, gymnosperms and ferns, while other species are, of course, much more restricted and specialized in their host range.

The nematodes that are responsible for the most destructive diseases of plants are obligate parasites, although a few semi-parasitic and saprophytic eelworms may cause minor diseases. Parasitic nematodes bore into plants and live on the liberated cell contents or draw nutrient substances out of the cells. The eelworms may attack any part of the plant including the roots, stems, leaves, flowers and fruit. Nematodes usually invade parenchymatous tissues and stimulate hyperplasia and hypertrophy, which result in enlargement and distortion of the invaded tissues. In other cases the worms produce an immediate mottling or yellowing frequently accompanied by dwarfing. Necrosis of the invaded tissues may follow the above pathological effects or may be the immediate results of nematode activity. Vascular and woody tissues are seldom invaded, although they may be twisted and misplaced through the pressure of the distorted adjoining parenchymatous tissues.

The nematodes are classified in the phylum Nemathel-

minthes. The body, as is typical of the phylum, is elongate and worm-like, tapering to pointed ends. The worms take in food through the anterior, centrally located mouth, which is surrounded by lips, papillae, setae and glands that are thought to aid in feeding. In many cases a barbed, spear-like stylet in the pharynx is capable of being inserted into the surrounding host cells for the purpose of absorbing food. From the buccal cavity the food is passed through a complex, muscular oesophagus with its associated glands into the simple intestine where the food is absorbed by the animal, and the waste is passed out. The eel-worms have simple nervous and excretory systems but no respiratory or circulatory systems.

The sexes are usually separate and the female may lay eggs or give birth to living young. The entire body is covered by a three-layered wall consisting of an outer, impermeable, resistant, usually transparent cuticle; an inner cellular, muscular layer, and a median non-cellular, multinucleate layer. The muscular body makes possible the characteristic undulating motions and the rotating movement of the head in feeding.

The genera of plant parasitic species are separated on the basis of body form, female worm-like or swollen and sac-like, location and nature of sex organs, structure of the tail regions, nature and number of oesophageal glands and structure of the feeding stylets. The more important plant pathogenic species are placed in three genera—*Anguillulina*, *Heterodera* and *Aphelenchoides*.

Root Knot

Heterodera marioni (Cornu) Goodey

Root knot has been known in the United States since 1805 and has long been considered a serious disease of many crop plants in the tropic, subtropic and temperate zones and of greenhouse plants everywhere. In the United States it is particularly destructive to tobacco, cotton, melons, cowpeas and many other crops in the southern states. The host range

of the nematode is very extensive; over 1,115 species were listed as susceptible in 1937. The root knot nematode is an obligate parasite living in the roots of plants and is capable of distinguishing its hosts by a chemical sense organ.

SYMPTOMS.—*Heterodera marioni* induces three types of injury to the host plant: First, by the food removed, second, by the distortion of root tissues, and third, by preparing the host for invasion by other pathogens. The first type of injury is direct but very nominal; the second and third are indirect but may result in serious injury or death to the host.

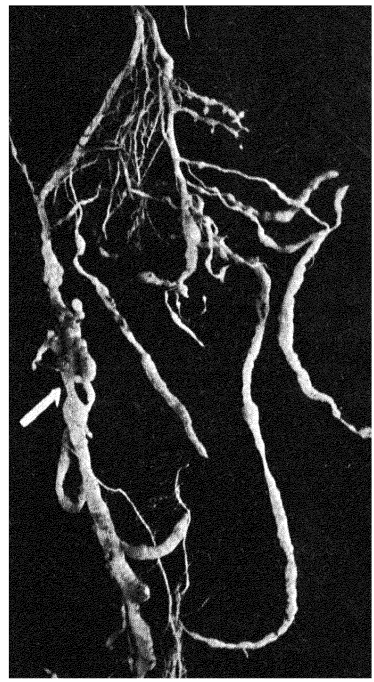


FIG. 248. Root knot nematode infection of tomato roots.

Nematode knots on the tomato roots vary in size and form according to the number and position of the female nematodes present in the tissues. The knots chiefly comprising cortical cells may range in size from one-sixteenth of an inch to three inches in diameter. If such knots are sectioned and examined with a hand-lens, numerous opalescent, pearlish bodies, the female nematodes, are evident. In some of these females there may occur a mass of eggs containing nematode embryos in all stages of development. In cross sections of infected roots, dark specks occur in the cortex marking the location of the pathogen. These dark areas contain nematodes in all stages of development ranging from the undifferentiated granular egg to a pear-shaped female nematode. Knots in late stages of necrosis may contain countless eelworms.

On the monocotyledonous plants, the knots are spindle-shaped, and on dicotyledonous plants they are tubercle-like. In Irish potatoes the tubers may become rough and warted showing more injury than the roots or stems. Similar symptoms occur in carrots and turnips. Tubers and infected roots may show brown necrotic spots just below the skin where young larvae or enlarged females are located. Knots, if carefully dissected or broken open, may show the female cysts as pearly white rounded or pyriform bodies visible to the naked eye.

ETIOLOGY.—The root knot nematode is a small hyaline worm scarcely visible to the naked eye, 12 to 15μ in diameter, 500μ long and pointed at both ends. In the larval stage the male and female are quite alike, but as the female matures, just before her egg-laying period, her body becomes pear-shaped and visible in the tissues as a minute pearly granule. Each female may lay 300 to 600 eggs that may hatch without being fertilized in two to three days in warm soil. The male remains approximately the same size throughout its life. The larva, which hatches from the egg, usually infects the host, and maturation occurs within the plant. The young larvae may find their way into the soil or into the roots between the epidermal and parenchymatous cells of the root tips. Finally the head of the eelworm penetrates the central cylinder where "giant" cells are formed about the anterior end of the worm. The worm derives its nourishment from these "giant" cells. In the roots, nematodes may begin development immediately, while those set free in the soil without susceptible vegetation undergo little or no change and die within two years.

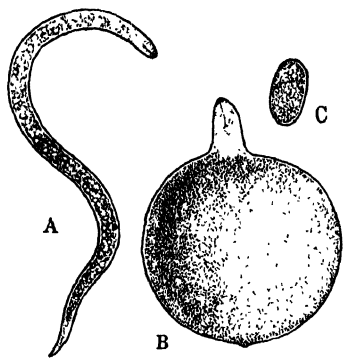


FIG. 249. Stages of root knot nematode: A, larva (140X); B, pear-shaped female (40X); C, egg stage (80X).

The nematode passes the winter in the roots of perennial plants. In the south where the growing season is long, 8 to 12 generations may develop each year, while farther north the number of generations of the organism is more limited. The eelworm flourishes in light soils where there is a medium amount of moisture, 40 per cent of the water-holding capacity. Heavy clay soils and water-saturated soils retard the development of the worms, especially at temperatures of 15° to 21° C. At low temperatures, 10° to 11° C., the parasite is compara-

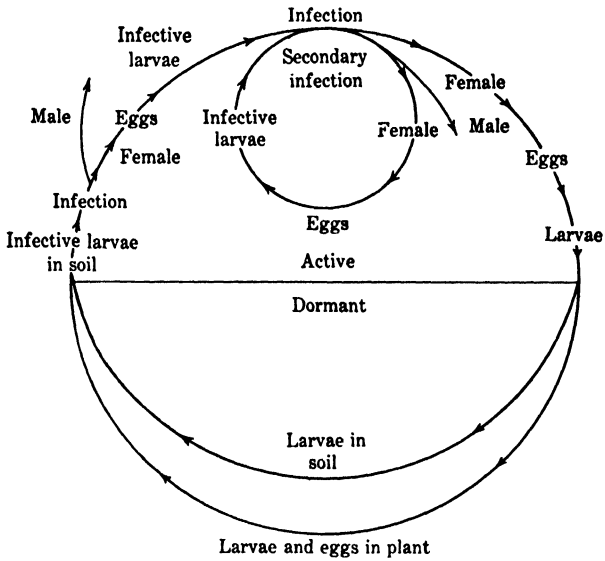


FIG. 250. A diagram of the host relation of *Heterodera marioni*.

tively inactive, but at higher temperatures, 16° to 30° C., the eelworm forms galls as long as root growth continues. The most favorable temperature for the growth and development of the tomato root knot nematode is 24° to 30° C. The spread of the nematode may readily occur from one section, state or country to another in nursery stock, transplants, root cuttings, bulbs, corms, tubers, etc. The introduction of the root knot nematode to Long Island, Oregon, Washington and Idaho probably took place in shipments of seed potatoes. Wherever plants of any kind are moved from an infested region to another, the nematode may be included in

the plants or soil. Irrigation water, wind and farm implements may also move the nematode from one place to another, but probably not as extensively as plants. Seeds of plants, unless contaminated superficially, are not carriers.

CONTROL.—Where the nematode infestation is in greenhouse soil, it is much easier to combat the pathogen than when it occurs in field soils. Under greenhouse conditions all infected plants should be removed and destroyed rather than thrown on the compost heap where conditions are favorable for the multiplication of the parasite. The infested soil may be removed from beds or benches and the framework cleansed with a hot disinfectant. It is more practical in many instances, however, to disinfect the soil in the benches than to remove it. This may be done by treating the soil with either steam or chemicals.

Where the infestation is in field soil, summer fallowing for two seasons, the use of resistant crops and varieties, prompt destruction of all susceptible weeds and chemical treatment of the soil have been utilized individually and in combination to combat the pathogen. These practices are not always equally effective in different sections or in different seasons. Applications of 300 pounds of sodium cyanide and 450 pounds of ammonium sulphate per acre have proven effective against the eelworm attacking pineapples. On soils used for truck crops in Florida, 600 and 900 pounds of these salts, respectively, are applied per acre. There is wide variation in the resistance of different crops; i.e., most varieties of cowpeas and cotton are susceptible, but the iron cowpea and American Upland cotton have proven resistant in certain sections. Sorghum, velvet bean, beggarweed and *Crotalaria* spp. are very resistant and serve to reduce the nematode population when used in the rotation.

THE STEM NEMATODE

Anguillulina dipsaci (Kühn) Gerv. and v. Ben.

The stem nematode was first described over 80 years ago as causing a disease of the flowers of teasel, a garden flowering

plant native in Europe. This eelworm attacks the aboveground parts of the plant, a characteristic not evidenced by the root knot disease described in the preceding pages. The injury caused by the pathogen is best-known on cultivated crops, as alfalfa, oats, hyacinth, narcissus, potatoes, onions, clover, etc. The nematode is known to attack over 200 different host plants distributed in 42 families. Recently it has been shown that in the hyacinth and narcissus there are different strains not reciprocally infective.

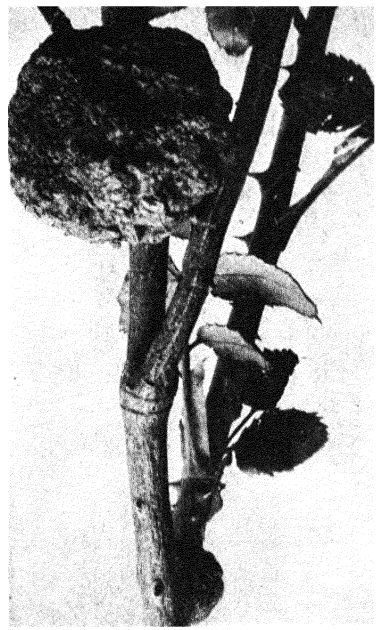


FIG. 251. Nematode gall on rose stem.

SYMPTOMS.—All of the aboveground portions of dicotyledonous plants may show localized areas of deformation resulting in separate or confluent galls. The gall formation depends largely upon the number of nematodes that attack the plant. In seedlings the galls or swellings are usually on the hypocotyl. In monocotyledons, bulbs and cereals, the galls form chiefly at the base of the leaves that subsequently undergo complete necrosis. Regardless of the class to which the plant belongs, an attack is accompanied by stunting, twisting and rolling of the leaves and stems, together with a general unthrifty and unhealthy appearance. In cross sections of infected bulbs, dark discolored rings may appear in the vascular tissue. Such infected bulbs also may show yellow streaks or blotches on the leaves.

In alfalfa, infected stems may be swollen, brown in color and brittle, and new buds arising from the crown are often enlarged, spongy and yellowish in color. The symptoms on

alfalfa are most conspicuous in the springtime before the first cutting.

In potatoes the disease manifests itself chiefly on the tubers where the flesh is brownish just beneath the skin that is frequently shrunken and cracked. Necrosis of the flesh of the tuber may become extensive, depending upon the number of eelworms that attack the tuber.

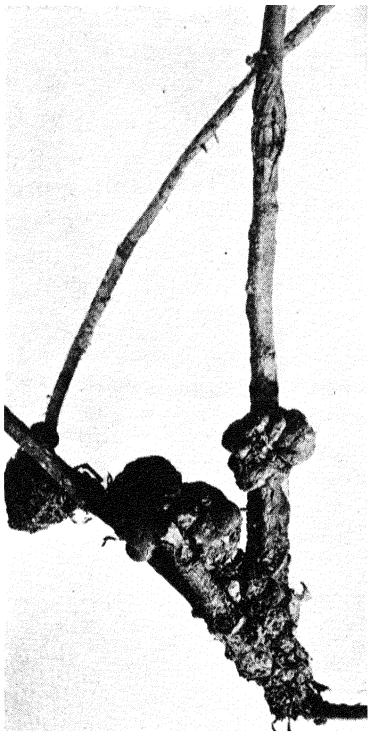


FIG. 252. Numerous galls on the stems and crown of a rose plant killed by nematodes.

ETIOLOGY.—The stem nematode is a small eelworm about one-twentieth of an inch long known as *Anguillulina dipsaci*. The organism multiplies by means of eggs—200 may be laid by a single female nematode. The newly hatched young or larvae are the same shape as the adults but are only one-tenth as large.

The parasite may overwinter in galls or swellings on leaves, buds and stems of many different host plants. The eggs are laid in clover plants at any time throughout the year, and the time from the egg to sexual maturity varies from 24 to 30 days, depending upon the temperature. The parasite may be disseminated by the active wandering of the infective pre-adult larvae in the soil, and its presence there permits it to be moved from place to place by water or other agencies, such as farm implements. Dissemination also may occur by means of infested seeds, as the eelworms are known to exist in the seeds of teasal, onions, oats and seed heads of many

The parasite attacks cortical and parenchymatous tissues rather than vascular, and it is an intercellular parasite that destroys the middle lamella. The loosening of the cells from one another, coupled with the presence of toxic secretions by the parasite, causes the cells to become spherical and enlarged. Following these changes in the cells the intercellular spaces

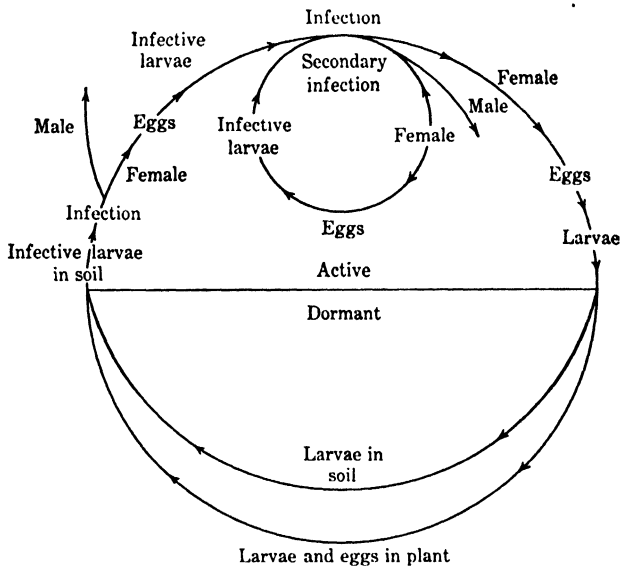


FIG. 253. A diagram of the host relation of *Anguillulina dipsaci*.

enlarge giving a glistening appearance to the tissues. When many cells are affected this way in a localized area, a gall results.

The nematodes soon die in moist soil, and they are unable to infect healthy bulbs after the lapse of one year. The larvae and eggs withstand −19° C. for 45 minutes if slowly restored to normal temperature, but adult forms are not so resistant. High temperatures of 43° to 44° C. for three hours kill the eelworms in hyacinth bulbs.

CONTROL.—In many crops that are infected with the stem nematode, no satisfactory control measures have been worked out. Where the pathogen becomes seriously destructive the infested crop should be replaced by non-susceptible crops.

Where resistant varieties are known, these, of course, may replace the susceptible ones. Narcissus and hyacinth bulbs, if not too badly infected, can be treated by soaking in hot water for three hours at 43° to 44° C. The bulbs should only be treated when they are fully dormant, and after treatment they should be cooled gradually.

Chapter Sixteen

DISEASES CAUSED BY NON-PARASITIC AGENTS

THE numerous plant diseases, which are not caused by parasitic organisms are summarily grouped together as diseases of non-parasitic origin. These diseases may be characterized as the sum of the deviations of the vital functions beyond the latitude of health, which are marked by characteristic symptoms.

In animals, especially in man, a wide range of abnormalities are induced by unfavorable environment, deficient or excessive foods or water, and poisonous substances. It is generally known that certain foods will inevitably cause indigestion in small children and sometimes in adults; while certain mineral deficiencies may cause anemia, excess minerals may lead to poisoning and overeating to excessive weight, and a long chain of other well-known afflictions. In all such cases, the individuals are sick or diseased, and obviously no parasitic organism is concerned. All living things including plants respond in a similar way. Excessive rainfall may lead to waterlogging of the soil which, in turn, interferes with aeration, causing a yellowing of the growing plant. Too little water may lead to underdevelopment—a dwarfed and stunted plant. Many plants grow tall and spindly in hothouses having inadequate light. Small quantities of boron may disturb the vital functions, causing yellowing and dwarfing in many different kinds of plants. High temperatures may cause a scorch of the foliage and cankers of the limbs and trunks of apple trees. Low temperatures on the other hand may injure or kill cambium cells, cortical tissues, and storage organs, as roots, fruits and tubers, to the extent that local or general necrosis develops.

There are many diseases of non-parasitic origin; some are of little importance economically, while others constitute the chief limiting factors in the production of a crop. The use of late varieties of corn in the northern range of the corn belt is impractical because of frost injury to the maturing crop. Likewise when killing frosts occur too early in the fall, much injury results to the corn crop in the central and southern range of the corn belt. In either case, the total loss may amount to millions of dollars. Sunscalding of potatoes at digging time in hot weather may cause serious damage to the crop in transit. Bitter pit and scald of apples, internal decline of lemons, blossom-end rot of tomatoes, heat canker of flax, black heart of potatoes, lodging of grain, injury by poisonous gases, chemicals, fungicides and insecticides are only a few of the diseases of non-parasitic origin that make heavy inroads on the yields and value of many crops. A discussion of a few of the diseases mentioned above will serve to illustrate the effect of certain non-parasitic agents.

Bitter Pit of Apples

Bitter pit was probably first described in Germany in 1869 where it was called stippen. It was not until 20 years later that this disease was described in this country.

Bitter pit occurs in all the important apple growing regions of the world, but seems to be most prevalent and severe in Australia and America. In this country it occurs on almost all varieties of apples, but is commercially important only on such varieties as Baldwin, Northern Spy, Grimes Golden, Stayman Winesap, Delicious, Rhode Island Greening, Tompkins King, Yellow Newton, York Imperial, Ben Davis, Rome Beauty, Winter Banana, Arkansas and Gravenstein.

Symptoms.—This is a disease of the fruit that develops chiefly in storage, rarely in the orchard. There develop on the fruit discolored areas of the epidermis, called pits, which are concave depressions in the surface caused by the shrivelling or shrinking of the tissue beneath the epidermis. The pits often are numerous and may be confined to one side of the fruit or extend completely around the calyx end of the apple.

The pits vary in size from two to six millimeters in diameter but may coalesce and form large depressions. Usually the pits are roundish in outline, having a regular or irregular margin. The color of the pits on the green, yellow or red surface of an apple is some blend of brown with the above-named colors, unless necrosis is sufficiently extensive to include the epidermal and hypodermal cells. On some fruits there may be no external symptoms whatever, and it is only

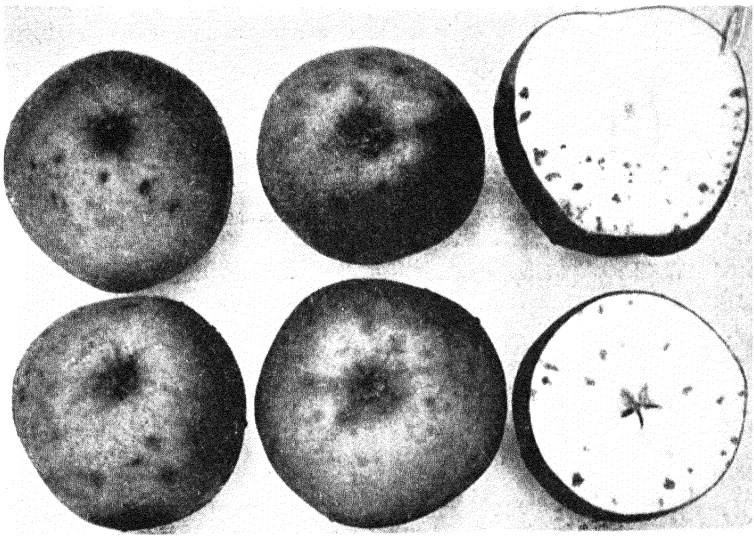


FIG. 254. External and internal discoloration and necrosis of bitter pit of apple. (After H. H. Plagge.)

when the apple is cut that lesions are discovered. It has been shown by means of the X-ray that the disease always develops internally before there is any indication of it externally. Usually, however, there are external indications of the disease, and where a transverse section is made, internal necrotic areas may be observed throughout the cortex, even reaching into the boundary of the ovary wall. The brownish elongated necrotic lesions usually extend along a vascular bundle into the cortex of the apple from the depression in the skin. The lesions at first are a pale brown, gradually turning to a

darker brown. The discolored tissues are generally dry and of a loose and spongy texture. The collapsed cells usually contain many starch grains, and their brown disorganized contents afford a marked contrast to the healthy tissue, which contains little or no starch in this stage of development of the fruit.

ETIOLOGY.—Bitter pit develops in apples picked while in a rapidly ripening yet immature condition, and is held to be largely determined by orchard-growing conditions. Many explanations have been offered as to the cause of the death and discoloration of cells in localized areas in the storage tissue of the apple. Students of this disease in this country are inclined to hold that pitting is due to a killing of the immature tissues of the rapidly growing fruit, or of fruit in storage, by excessive transpiration, inducing osmotic action between the starch-filled cells and those in which the starch has been converted to sugar. The cells in the starchy areas are killed by excessive desiccation. Sections of such pitted areas, when examined with the microscope, show darkened bands about clumps of starch grains and honeycomb-like cavities strikingly unlike the adjoining, regular, turgid, healthy, starch-free cells.

Bitter pit is more prevalent on fruit from young trees, especially if the crop is light, than on fruit from older trees. Large apples are more susceptible than small ones. Bitter pit is increased by heavy irrigation and heavy rainfall, especially when these occur late in the growing season. Heavy nitrogen fertilizer also favors the development of the disease. Bitter pit is temporarily retarded by storage at 0° C., but may develop rapidly on immature fruit when removed from cold storage.

CONTROL.—In the control of bitter pit, consideration must be given to promoting uniform growth of the fruit throughout the season. Marked fluctuations of moisture, temperature and fertility should be avoided in all orchard practices. The harvested fruit should be stored at 0° C.

The fruit should not be picked until it has reached sufficient maturity to continue its normal ripening process in storage.

This may vary with different varieties and season, and can only be most accurately determined by the iodine starch test. The apples should be harvested before all the starch in the fleshy tissues of the equatorial parts (a cross section through the middle of the apple) has been converted into sugar.

Apple Scald

Apple scald was first described in this country about 40 years ago, before artificial cold storage had assumed an important place in the apple industry. With the rapid development of cold storage, apples for the large markets found their way into large storage plants. In these plants it was possible to keep the fruit until late winter without any apparent diseases developing. When apples were removed from storage and placed on the market, however, scald frequently developed rapidly and resulted in heavy losses to the retailer and the consumer. It is reported that more than 15 per cent of the boxed apples and about 32 per cent of the barreled crop scald annually. Such scalded apples usually must be sold at 10 to 40 per cent discount because of the marred appearance, impaired flavor, reduced quality and increased susceptibility to fruit rot organisms.

Symptoms.—Apple scald starts in cold storage, and the earliest visible symptoms are superficial, brown, discolored spots limited to the outer cell layers of the "skin." The green side of the fruit is more susceptible than the more highly colored one. When apples are removed from storage, the light-brown spots increase in size, and necrosis of the "skin" and flesh below follows. In later stages the discolored areas may comprise a large part of the fruit's surface. The flesh underneath the brown dead "skin" becomes discolored, mealy and tasteless. The extent of the broken-down tissues may vary from one-quarter to one-half inch below the surface. In this condition, the skin is easily broken in handling, which may afford a ready entrance for fruit rot organisms. Scalded fruit becomes practically unmarketable.

Etiology.—Scald is caused by poisonous gaseous substances given off in the process of respiration of the stored fruit. It

has been established that volatile gases accumulate in the surface of the apple and induce necrosis of the tissues in localized areas. The products of respiration, which are injurious, are held to be certain volatile esters. When the fruit is packed in containers and held for storage where there is little circula-

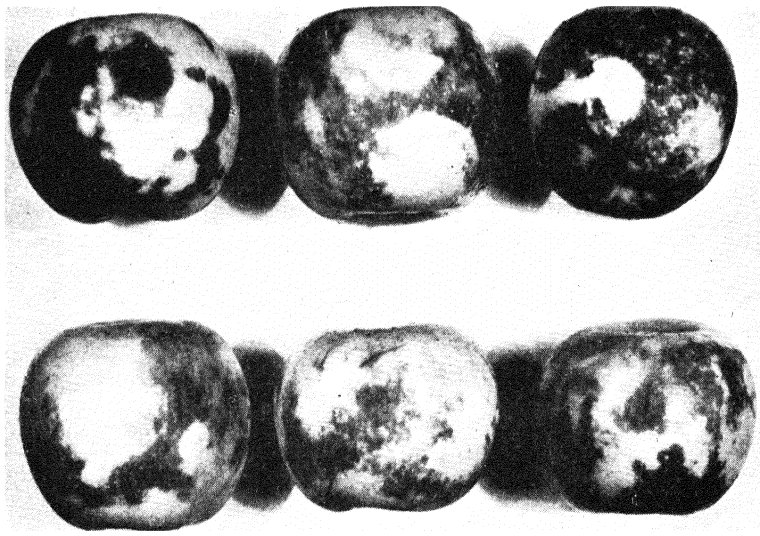


FIG. 255. Apple scald on Grimes Golden following removal from cold storage. (After H. H. Plagge, T. J. Maney, and B. S. Pickett.)

tion of air, the poisonous gases accumulate to such an extent that the cells are injured or killed.

Scald develops most rapidly when the fruit is removed from cold storage. Frequently a trace of scald may develop into severe scald in a few days after the fruit is placed on the market. Fruit apparently free when taken from storage may show a brownish-tinted appearance within 12 hours at ordinary room temperature, and within 24 hours marked scald symptoms appear. Within a few days such scalded apples may become practically unmarketable.

CONTROL.—Previous to 1917, scald was one of the most serious diseases of stored apples, but since that time control measures have been worked out that make it possible to avoid

serious losses. The prevention of scald is based on the removal from the fruit of the gaseous by-products of respiration through proper aeration in storage. It is not necessary to supply new, outside air, but merely to provide circulation of the existing air. It is desirable, therefore, to pack fruit in slat boxes and baskets that permit better aeration than tight barrels. The fruit must be harvested at the correct maturity, and immediately placed in cold storage at 0° C. Another method of removing the volatile gases from the surface of the fruit is to wrap it in oiled paper. The oiled paper used for making the wrappers should contain 15 to 20 per cent by weight of a special light, odorless and tasteless mineral oil. Placing the oiled paper in the containers with the apples also may decrease the amount of scald. One-half pound of shredded oiled paper should be used to each bushel of fruit or one and one-half pounds per barrel. The paper must be distributed through the pack, so that the oil may absorb the volatile esters. Aeration and the use of oiled wrappers are not effective in preventing scald when the fruit is overripe, nor are they effective when the apples are held in the packing house at high temperatures for several days after harvest.

Poorly colored apples scald worse than highly colored ones. In the same way the green or yellow side of an unevenly colored apple is said to scald worse than the dark-colored side, and early picked fruit is more subject to scald than that properly matured. Varieties vary widely in resistance to scald. Some are nearly immune, others very susceptible. The more resistant sorts are Jonathan, Delicious, Wealthy, McIntosh, Northwestern Greening, King David and Fameuse. The susceptible varieties include York Imperial, Arkansas, Stayman Winesap, Grimes Golden, Ben Davis, Rhode Island Greening and Baldwin.

Boron Deficiency

Toward the end of the nineteenth century it was announced that beets weakened by a severe drouth or as a result of high fertilization were very susceptible to parasitic invasion by *Phoma betae* Frank. The organism was said to produce a

typical heart or dry rot of the beet root. Later it was found that healthy beets would not become infected when inoculated with *P. betae* and that during the early stages of the rot, no fungus was present in the beet. Consequently, it was suggested that the rot was the result of a physiological disorder followed by the parasitic action of the fungus.

It has been only within the past decade that workers in Germany, noticing the similarity of symptoms produced in many plants suffering from a boron deficiency to those of the heart rot and dry rot of beets, have shown conclusively

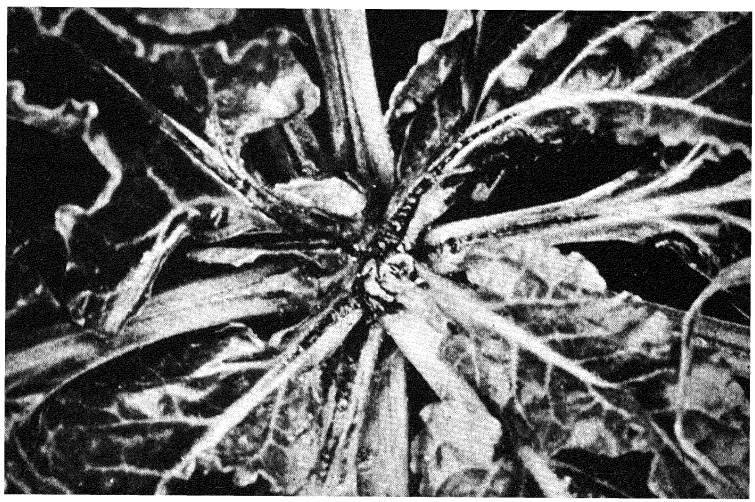


FIG. 256. Boron injury in sugar beet showing the ladder-like discoloration on the petioles. (After R. L. Cook.)

that the disorder is caused by the lack of boron. This announcement was soon confirmed by a number of workers in the United States and in France. It also was shown that numerous other plants, tobacco, legumes, etc., suffer from a similar derangement of the growth regions as a result of boron deficiencies.

The reduction caused by the disease has been severe in many cases. In forage beets growing in alkaline soils the total of infected beets may reach 90 per cent. The damage is evidenced in a decrease in yield that may amount to 25 to 50 per cent,

and in a lowering of the sugar content by two to six per cent.

SYMPTOMS.—The first evidence of boron deficiency is a yellowing, distortion and inhibition of growth of the young leaves and growing points of the infected plants. The disease generally appears during or shortly after the middle of the growing season. The young growing points become blackened, and disintegration follows shortly. The young expanding leaves first become yellow, and some puckering of the leaf blade may occur prior to the blackening of the base of

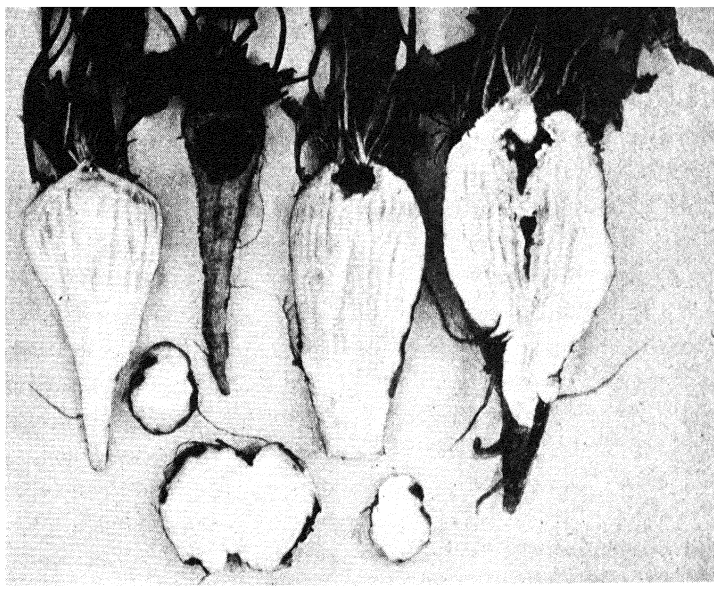


FIG. 257. Boron injury of sugar beet roots showing necrosis and discoloration. (After R. L. Cook.)

the blade and the petiole. The blackening extends up the midrib and out the veins into the leaf. At the same time the leaves begin to wither and blacken, while the petiole becomes stiff and brittle and the upper part of the petiole and midrib curl downward. The leaf seems to fold in at the edges and roll down from the tip. The blackened tissues on the upper surface of the brittle petiole become separated by the curling and produce a ladder-like design.

The blackening of the tissues extends from the surface

layers of the petioles into the vascular bundles and may be traced down through the crown and into the root. In the crown the presence of the meristematic tissues, which are particularly affected by a lack of boron, results in a general blackening of the tissues. The blackening in the roots is confined to the vascular bundles in connection with the diseased portions of the leaves and crown and the surrounding ground tissue. The discoloration seems to extend from the center to the edge of the bundles and is usually more pronounced around the outer vascular bundles.

ETIOLOGY.—With the use of chemically pure materials in the production of artificial fertilizers, the prevalence of heart rot and dry rot has been on the increase. This is explained by the fact that the amount of boron necessary to prevent, or cure the disease, is usually contained as an impurity in the natural fertilizer materials but is lacking in the artificially prepared fertilizers. Cultural experiments carried on in liquid and soil cultures indicate that from seven to ten parts per million of boron are sufficient to prevent the occurrence of the disease. The same quantity of boron is sufficient whether added in the acid form, boric acid, or alkaline form, sodium borate. Since the disease may be produced in acid or alkaline soils by boron deficiencies, and cured by addition of boron in an acid or alkaline form, it would appear to be a true deficiency disease.

Two explanations have been offered for the apparent increased severity of the disorder under alkaline conditions. It has been said that the chalk of the alkaline soils reacts with the boron, present probably in the form of borates, to render it unavailable to the plants. A further explanation is that the more vigorous development of the foliage of the beets, which occurs under alkaline soil conditions, increases the demand for boron on the part of the plant and hence accentuates the disease.

The action of the boron is not known except from the tissues that are affected. The destruction of the meristematic tissues and young growing portions results in a lessened leaf area and there results a decrease in total yield and in sugar

content. The dry weight of beets from soil to which boron has been added may reach five times that of the beets in boron-deficient soil. The use of larger amounts of boron, up to 40 parts per million, does not result in toxicity to the beets.

Field experiments in the use of boron-containing and boron-deficient fertilizers have definitely established the responsibility of the element. The disease has been shown to be more evident on a sandy soil with a porous subsoil. The deficiency may be corrected by boron addition, and the effect may extend over into the second year. The sugar beets are not so susceptible as the forage beets, although the former do suffer under extreme lack of boron.

CONTROL.—The disease, which develops from a deficiency, is obviously controlled by adding boron to the soil. The control is best when the boron is added with the fertilizer, preferably mixed with it, or added at planting time. Addition of five pounds of boric acid per acre will reduce the incidence of disease from 75 to 25 per cent. For complete control in fields where the plants are severely affected, 10 to 15 pounds per acre are necessary. Under additions of this amount of boron, a beneficial residual effect is noticed on beets the second year, although there is no evidence of toxicity to other crops. Applications as high as 20 pounds per acre can be made without injury to the beets.

The boron usually is applied in the form of boric acid, although sodium borate is equally effective. When using sodium borate, the quantity used must be multiplied by 1.6, i.e., 16 to 24 pounds per acre. The broadcasting of the borate or boric acid at thinning time, sterilization of the seed, or treatment of seed with boric acid will not prevent heart rot or dry rot.

GRAFT KNOT OF APPLE

Graft knots occur at the union on piece-root grafted apple trees as localized enlargements. These swellings also are known as wound overgrowths and callus knots. This type of injury at the union of piece-root grafted trees has probably occurred since the first grafted trees were grown. It is only

during the present decade, however, that graft knots have been clearly differentiated from the galls produced on young apple trees by *Pseudomonas tumefaciens*.

Young nursery apple trees having graft knots generally produce inferior trees when set in the orchard. The graft knot interferes with the normal functioning of the stem. The water flow through graft-knot-bearing trees has been shown to be reduced from 30 to 84 per cent. Frequently many trees develop graft knots in the nursery. In field surveys made during 1925, 1926 and 1927, when 55,668 trees were examined, the percentage of graft-knot-bearing trees was 16.8, 36.1 and 45.4 per cent, respectively. In the grafted trees, 61.3 per cent of the graft knots occurred on the tip of the scion lip, and the balance at other points on the union. When trees with well-developed graft knots are planted in orchards, they either die after a few years or develop into inferior trees.

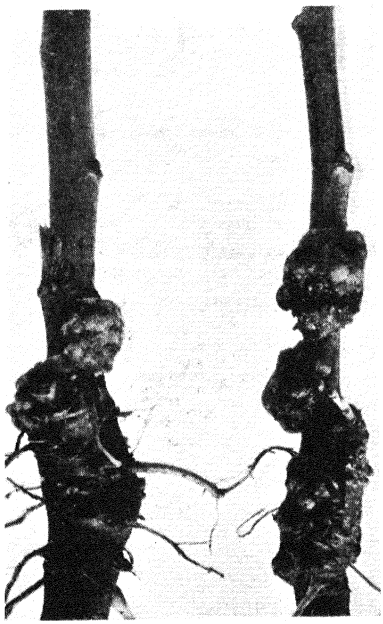


FIG. 258. Callus knots developing at and above the graft union on young apple trees.

SYMPTOMS.—In the early stages of graft knot formation, the tip of the scion lip shows a white and frosty appearance as callus tissue develops at the cut surface. This new tissue increases in volume under favorable growing conditions of moisture and temperature until a marked swelling or overgrowth is apparent to the casual observer. Gradually a phellogen is differentiated and a cork layer formed over a hemispherical knot. It may vary in size from one millimeter to one centimeter through the greatest diameter. From this time on, the knot, which may

be smooth or rough on the surface, grows chiefly through internal cambial activity. The interior commonly is very hard because of the irregularly arranged woody elements.

ETIOLOGY.—Graft knots occur at imperfect unions of grafted nursery apple trees. Enlargements develop about the lower tip of the scion at a point where cambial connection with the root has failed to be established or at any other point of the union. If partial continuity is established between the root and scion, the knot will usually expand more rapidly on one side than the other. If continuity between the stock and scion fails entirely, the knot ceases growth through the partial or complete death of the young tree.



FIG. 259. A large callus knot has formed at the tip of the misfitting scion lip.

It is the cells of the tip of the scion lip that produce the greatest amount of cell proliferation; probably because the food, as it descends in the scion to the cut, makes a slow lateral movement along the obtuse angle to the lower tip where it becomes concentrated and contributes to the development of callus. Graft knots may form on poorly fitted grafts or poorly bound unions or may result from wounds caused by tools in cultivation.

With increasing age of the tree having a graft knot, the knot may increase in size, a phellogen develops, irregular woody structures form and the cambium that produces the usual tissues is differentiated from the callus. Sometimes root primordia may develop. On older trees, knots may be subject to bacterial and fungous attacks developing later into a collar rot and subsequent death of the tree. Experiments have

shown that knotted trees have a marked reduction in the water movement through the union. When this condition prevails, the young trees become stunted, weak and unfit for transplanting to the orchard.

CONTROL.—It has been found that improved grafting and cultural practices are effective in reducing the amount of graft knot. Important factors of control are: (a) avoiding the use of scions of larger diameter than the stocks to which they are fitted, (b) proper fitting of the lower scion lip at the union, (c) tight wrapping with a material that forms a sufficiently strong bandage about the union and maintains its strength throughout the critical period of callusing, but rots before girdling can take place, (d) prevention of excessive callus development in storage and (e) avoiding injuries in cultivation. It recently has been shown that well-made wedge grafts tend to decrease the amount of graft knot and that a medicated adhesive wrapper facilitates the union of stock and scion in double-tongue and wedge-graft trees, thus materially preventing graft knot development.

GLOSSARY

ACCRETION. The formation of external additions or enlargements.

ACTIVE RESISTANCE. Resistance produced by cells as a result of their stimulation by the causal agent.

ACQUIRED RESISTANCE. Resistance gained during the life of the host.

ALBINISM. The white appearance of plant parts resulting from the failure of chlorophyl production.

ANTHERIDIUM. The male gamete-producing organ.

ANTIBODY. Substances produced in the body in response to the parenterical introduction of an antigenic material. (Zinsser)

ANTIGEN. Any substance that, when introduced parenterally into the animal tissues, stimulates the production of an antibody, and, when mixed with that antibody, reacts with it in some observable way. (Topley)

ANTISEPTIC. A substance that prevents, retards or destroys putrefactive organisms.

APOTHECIUM. A saucer-shaped ascus-containing fruiting body.

ASCUS. A sac-like, sporogenous cell in which nuclear fusion and reduction division precede the endogenous, free-cell formation of spores.

ATROPHY. The reduction in size of an organ by disturbed metabolism.

ATTENUATION. Lessening of the virulence of a pathogen.

AUTOECIOUS. The need of only one host for completing a life cycle.

BACTERIUM. A rod-shaped bacterial cell.

BASIDIOSPORE. A spore formed exogenously on a basidium.

BASIDIUM. A sporogenous cell in which nuclear fusion and reduction division precede the exogenous formation of spores (usually four).

BLIGHT. The rapid discoloration and death of the tissues over certain portions of plants. This term may be coupled with the name of the host part affected, leading to such common names of diseases as twig blight, blossom blight, cane blight, tip blight, etc.

BURN. The condition in which the cells of the host become reddish or dark brown and collapse.

CANKER. A dead area on a stem surrounded by living cortical tissues.

CARRIER. A plant that carries a virus or other infective agent without showing symptoms.

CHLAMYDOSPORE. A thick-walled spore formed by the modification of a hypha.

CHLOROSIS. The yellowish-white or gray condition of plant parts resulting from the destruction of the chlorophyl.

CILIUM. A hair-like vibratory filament attached to a cell.

CLEISTOTHECIUM. A closed, thick-walled ascus-containing structure.

CONIDIUM. An exogenously produced asexual spore.

CONIDIOPHORE. The hyphal branch that bears the conidium.

CONTAGIOUS. Spreading from one to another.

CORTEX. All tissues situated outside the stele (pericycle) and enclosed by the epidermis.

CORTICAL NECROSIS. Delayed necrosis resulting primarily through the activities of the causal agent in the parenchymatous tissues.

CURL. The distortion, fluting and puffing of a leaf resulting from the unequal development of its two sides.

DEGENERATION. A gradual disappearance, or catabolic modification of normal structures of the cells.

DIPLOID. The 2n number of chromosomes.

DIRECT NECROSIS. The rapid and complete death or disintegration of the tissues as the causal agent advances in the host.

DISEASE. The sum of the deviations of the vital functions beyond the latitude of health.

DISINFECTANT. Any agent for destroying the causal agent of disease.

DWARFING. The underdevelopment of any organ of a plant. For example, curly dwarf, leaf roll, spindle tuber and little peach are names designating particular forms of dwarfing.

ECTOPLASM. The outer limiting layer of protoplasm.

ECTOTROPHIC. Mycorrhizal association in which the fungus has its hyphae outside or between the cells.

ENDOGENOUS. Produced inside.

ENDOBIOTIC. Living within another organism.

ENDOPHYTIC. Living within another plant.

ENDOSPORES. Spores produced within the mother cell.

ENDOTROPHIC. Mycorrhizal association in which the fungus has its hyphae in the cells.

EPIBIOTIC. Living upon or outside another organism.

EPIDEMIC. The widespread and destructive development of a disease on many people in a community or communities.

EPIPHYTE. A plant which lives upon another plant deriving little or no nourishment from it.

EPIPHYTOTIC. The widespread and destructive development of a disease on many plants in a community or communities.

ETIOLATED. Yellowed as a result of insufficient light.

ETIOLOGY. The description of the cause of disease.

EXCRESENCE. An outgrowth of abnormal character from the surface of a diseased organ.

EXOGENOUS. Produced outside.

EXUDATIONS. Liquids discharged from the protoplasts.

FACULTATIVE PARASITE. An organism that is ordinarily saprophytic but under proper conditions may be parasitic.

FACULTATIVE SAPROPHYTE. An organism that is ordinarily parasitic but under proper conditions may be saprophytic.

FASCIATED. Formed by common growth of several buds that fuse laterally while under pressure in their formative stage.

FLAGELLUM. A long, delicate, contractile filament protruding from certain cells.

FLAGGING. The loss of turgor and the drooping of plant parts, usually following a water deficit.

FUNGICIDE. An agent that inhibits or kills fungi.

FUNGUS. A single or many-celled, naked or covered, irregular or filamentous plant body devoid of chlorophyl, usually with a chitinous cell wall.

GALL. An overgrowth induced by an infectious entity that stimulates the localized formation of imperfectly vascularized masses of abnormal cells.

GAMETANGIUM. A gamete-producing cell.

GAMETE. Sex cell formed from a mother cell in a characteristic manner.

GENERAL NECROSIS. The rapid and complete death or disintegration of the tissues as the causal agent advances in the host.

GIANT CELLS. Large, usually multinucleate cells formed by abnormal cell fusions or failure of proper cell wall formation following growth and nuclear division.

GRAM-NEGATIVE. A negative reaction to the standard Gram's stain.

GRAM-POSITIVE. A positive reaction to the standard Gram's stain.

HAIRY ROOT. The development of large numbers of small roots on a limited area.

HAPLOID. The chromosome number n of the gametophytic generation or phase.

HAUSTORIUM. A modified mycelial branch that grows into a cell, makes intimate contact with the protoplast and absorbs food.

HEALING. The process by which a wound is closed or protected by a new growth without replacing the lost parts.

HETEROECIOUS. Requiring two or more unrelated hosts for completing the life cycle.

HETEROECISM. The development of different spore stages of the life cycle of a parasite on two unrelated hosts.

HETEROTHALLIC. Producing fusing gametes on separate and distinct mycelia.

HOMOTHALLIC. Producing fusing gametes on the same mycelium.

HOST. The plant on or in which the parasite is living and obtaining its food.

HYPERPLASIA. The abnormal increase in the number of cells without their enlargement.

HYPERTROPHY. The abnormal enlargement of cells.

HYPHA. A single filament of a fungous mycelium.

HYPOPLASIA. The underdevelopment of cells, tissues or organs.

IMMUNITY. A zero relationship between a plant and a causal agent.

INDIRECT NECROSIS. The delayed death of the tissues that follows the advance of the pathogen in the host.

INDUCED RESISTANCE. The increase of resistance produced in an organism in response to the introduction of antigenic materials.

INFECTION. The invasion, multiplication and pathogenic action of a causal agent.

INTERCELLULAR. Between the cells.

INTRACELLULAR. Within the cells.

INTUMESCENCE. An enlargement formed by the distension of tissues through the elongation of groups of cells.

KNOT. An imperfectly vascularized knob-like overgrowth on roots or stems.

LESION. A local injury or morbid structural change.

LOCAL NECROSIS. The death or disintegration of cells and tissues in a localized area of an organ.

LYSIN. An antibody which on reacting with the antigen in the presence of a complement produces lysis.

LYSIS. The dissolution of the antigenic body by the action of the anti-body with complement.

MALADY. A multiplicity of diseased individuals.

METAPLASIA. The overdevelopment of the cell other than increase in the size.

MICRON. A millionth of a meter (a thousandth of a millimeter).

MONOTRICHOUS. Having only one flagellum.

MOSAIC. A disarrangement of the chlorophyl content of tissues causing the green and yellow areas to form variegated patterns.

MUMMIFICATION. The drying down and shriveling of fruits and other plant parts.

MYCORRHIZA. A structure consisting of the intimate association of a root and fungus.

NECROSIS. The death or disintegration of cells and tissues.

OOGONIUM. The female organ of the sexual stage of the thallophytes which forms one or more eggs internally.

OOSPORE. A fertilized egg that has developed into a resting spore.

PARASITE. An organism that lives within or upon another living organism from which it derives nourishment and in which it may cause various degrees of injury.

PARASITISM. The phenomenon of the growth of one organism, the parasite, at the expense of another, the host.

PASSIVE RESISTANCE. Resistance resulting from qualities in the host prior to the attack by the causal agent.

PATHOGEN. An entity capable of producing disease.

PATHOGENICITY. The capacity of an entity for producing a disease.

PECTINASE. The enzyme that breaks down pectic substances to simple carbohydrates.

PERITHECIUM. A round to flask-shaped, ostiolate, thick-walled spore case containing asci.

PERITRICHIATE OR PERITRICHOUS. Having flagella all over the periphery of the cell.

PLASMODIUM. A naked, multinucleate, vegetative body capable of amoeboid motion.

POLYMORPHISM. The possession of several asexual spore stages in the life cycle of an organism.

PRECIPITIN. The substance in immune serum thought to cause the precipitation of the antigenic material when the latter is in solution.

PUSTULE. A local elevation of the epidermis that may rupture to expose the causal agent.

PYCNIDIUM. A variously shaped cavity containing conidia.

RACE. A strain of a pathogen characterized by the limitation of its host range to certain species and varieties.

REGENERATION. The process by which lost parts of cells, tissues or organs are replaced.

REPRESSIVE. Retarding development.

RESTORATION. Returning a property to its original condition.

RESISTANCE. The sum of the qualities of the host and causal agent that retard the activities of the causal agent.

RETROGRESSION. See hypoplasia.

RHIZOID. Intracellular thallus branch that absorbs food and provides anchorage.

RHIZOMORPH. An aggregation of hyphae into a cord-like strand.

ROT. State of decomposition and putrefaction.

SAPROPHYTE. An organism that derives its nourishment from dead organic matter.

SCAB. The abnormal thickening of the outer layer or layers of tissues resulting from local irritation.

SCLEROTIUM. A small, compact, hardened mass of hyphae that may bear fruiting bodies.

SIGN. The manifestation of disease by the presence of structures of the causal agent.

SORUS. A compact aggregation of spores and/or sporophores growing out to the surface of the host.

SPORANGIOPHORE. A sporangium-bearing hypha.

SPORANGIUM. Sporogenous cell that forms the asexual spores endogenously.

SPORE. A one- to several-celled body that becomes free and may develop into one or more plants.

SPOROGENOUS. Capable of forming spores.

STAGHEAD. Dying of a tree from the top downward.

STROMA. A compacted mass of hyphae that may produce sexual fruiting bodies.

SYMPTOMS. Expressions of disease in plants.

SUSCEPTIBILITY. The sum of the qualities of a plant and causal agent that allows the development of the causal agent.

THALLUS. The undifferentiated vegetative body of the lower plants.

TOLERANCE. Ability of the plant to endure the development of the parasite without showing marked symptoms of disease.

TYLOSIS. A bladder-like intrusion of the protoplasm from a parenchymatous cell through a pit into the lumen of a xylem cell.

VASCULAR BUNDLE. The conducting elements together with the cambium.

VASCULAR NECROSIS. Necrosis that is induced primarily through the activities of a pathogen in the vascular elements of the host.

VARIETY. One or more races of a pathogen that are characterized by the limitation of their host range to a certain genus or genera.

VECTOR. An agent that may transmit a pathogen.

VIRULIFEROUS. Capable of transmitting a virus.

WILT. The loss of turgescence of plant tissues, usually as a result of inadequate water supply.

WITCHES'-BROOM. A broom-like overgrowth produced by the dense clustering of branches.

YELLOWING. The yellow color of plant parts resulting from the excessive proportion of yellow pigments produced by the underdevelopment or partial destruction of the green pigments.

ZOOSPORE. Naked motile spore.

ZYGOSPORE. A resting spore produced by the fusion of equal gametes.

A LIST OF BOOKS DEALING WITH SOME PHASE OF PLANT PATHOLOGY [1]

ANDERSON, O. G., AND ROTH, F. C. *Insecticides and Fungicides Spraying and Dusting Equipment*, John Wiley and Sons, Inc., New York, 1923.

ARBOISE DE JUBAINVILLE, A., AND VESQUE, JULIEN. *Les Maladies des Plantes Cultivées*, J. Rothschild, Paris, 1878, 328 pp.

ARTHUR, J. C. *The Plant Rusts (Uredinales)*, John Wiley and Sons, New York, 1929, 446 pp.

BEL, JULES. *Les Maladies de la Vigne*, Bailliére et Fils, Paris, 1890, 306 pp.

BENNETT, F. T. *Outlines of Fungi and Plant Diseases*, Macmillian and Co., London, 1924, 254 pp.

BOUCART, E. *Insecticides, Fungicides and Weedkillers*, (translated by Donald Grant), Scott, Greenwood and Son, London, 1913, 431 pp.

BROOKS, F. T. *Plant Diseases*, Oxford University Press, London, 1928, 386 pp.

BUNTING, R. H. AND DADE, H. A. *Gold Coast Plant Diseases*, Dunstable and Watford, London, 1924, 124 pp.

CHUPP, CHARLES. *Manual of Vegetable-Garden Diseases*, The Macmillan Co., New York, 1925, 647 pp.

COOK, MELVILLE T. *The Diseases of Tropical Plants*, Macmillan and Co., London, 1913, 317 pp.

CUNNINGHAM, G. H. *Fungous Diseases of Fruit Trees in New Zealand and Their Remedial Treatment*, Brett Printing and Publishing Co., Auckland, New Zealand, 1925.

DALE, HENRY H. *Viruses and Heterogenesis, An Old Problem in a New Form*, Huxley Memorial Lecture, Macmillan and Co., London, 1935.

[1] As far as possible these books might be made available to the student for reference reading.

DE BARY, A. *Comparative Morphology and Biology of the Fungi, Mycetozoa and Bacteria*, (translated by H. E. F. Garnsey, revised by I. B. Balfour), Clarendon Press, Oxford, 1887, 525 pp.

DELACROIX, GEORGES. *Maladies des Plantes*, Challamel, Paris, 1911, 595 pp.

DUGGAR, B. M. *Fungous Diseases of Plants*, Ginn and Co., New York, 1909, 508 pp.

ELLIOTT, C. *Manual of Bacterial Plant Pathogens*, Williams and Wilkins Co., Baltimore, 1930, 349 pp.

ERIKSSON, J. *Fungous Diseases of Plants in Agriculture, Horticulture and Forestry*, (translated by W. Goodwin), Baillière, Tindall and Cox, London, 1930, 526 pp.

FAWCETT, HOWARD S. *Citrus Diseases and Their Control*, McGraw-Hill Book Co., New York, 1936, 656 pp.

FISCHER, E., AND GÄUMANN, E. A. *Biologie der Pflanzenbewohnenden parasitischen Pilze*, Gustav Fischer, Jena, 1929.

FITZPATRICK, HARRY MORTON. *The Lower Fungi, Phycomycetes*, McGraw-Hill Book Co., New York, 1930, 331 pp.

FRANK, A. B. *Pilzparasitären Krankheiten der Pflanzen*, Paul Parey, Berlin, 1896, 574 pp.

FREEMAN, E. M. *Minnesota Plant Diseases*, The Pioneer Press, St. Paul, 1905, 432 pp.

FRYER, J. C. F., AND BROOKS, F. T. *Insect and Fungus Pests of the Farm*, Ernest Benn, Ltd., London, 1928.

GÄUMANN, E. A. *Comparative Morphology of Fungi*, (translated and revised by C. W. Dodge), McGraw-Hill Book Co., New York, 1928, 701 pp.

GRÆBNER, PAUL. *Lehrbuch der nichtparasitären Pflanzenkrankheiten*, Paul Parey, Berlin, 1920, 333 pp.

GRIFFITHS, A. B. *Diseases of Crops and Their Remedies*, Geo. Bell and Sons, London, 1890, 174 pp.

HARSHBERGER, J. W. *A Textbook of Mycology and Plant Pathology*, P. Blakiston's Son and Co., Philadelphia, 1917, 779 pp.

HARTIG, ROBERT. *Wichtige Krankheiten der Waldbäume*, Julius Springer, Berlin, 1874, 127 pp.

HARTIG, ROBERT. *Lehrbuch der Baumkrankheiten*, Julius Springer, Berlin, 1882, 198 pp.

HARTIG, ROBERT. *Textbook of the Diseases of Trees*, (translation by W. Somerville), The Macmillan Co., New York, 1894, 331 pp.

HECK, CARL ROBERT. *Der Weisstannenkrebs*, Julius Springer, Berlin, 1894, 163 pp.

HEALD, F. D. *Manual of Plant Diseases*, Second Edition, McGraw-Hill Book Co., New York, 1933, 953 pp.

HEALD, F. D. *Introduction to Plant Pathology*, McGraw-Hill Book Co., New York, 1937, 579 pp.

HESLER, L. R., AND WHETZEL, H. H. *Manual of Fruit Diseases*, The Macmillan Co., New York, 1917, 462 pp.

HILEY, W. E. *The Fungal Diseases of the Common Larch*, Clarendon Press, Oxford, 1919, 204 pp.

HOLLRUNG, MAX. *Handbuch der Chemischen Mittel Gegen Pflanzenkrankheiten*, Paul Parey, Berlin, 1898, 178 pp.

HOLLRUNG, MAX. *Die Mittel zur Bekämpfung der Pflanzenkrankheiten*, Paul Parey, Berlin, 1923, 406 pp.

HUBERT, E. E. *An Outline of Forest Pathology*, John Wiley and Sons, New York, 1931, 543 pp.

HUNT, G. M., AND GARRATT, G. A. *Wood Preservation*, McGraw-Hill, New York, 1938

HYDE, ROSCOE R., AND GARDNER, RAYMOND E. *Laboratory Outline in Filterable Viruses*, The Macmillan Co., New York, 1937.

KIRCHNER, OSCAR. *Die Krankheiten und Beschädigungen Unserer Landwirtschaftlichen Kulturpflanzen*, Ungen Ulmer, Stuttgart, 1890, 637 pp.

KLEBAHN, H. *Grundzüge der Allgemeinen Phytopathologie*, Gebrüder Borntraeger, Berlin, 1912, 147 pp.

KÜSTER, ERNST. *Pathologische Pflanzenanatomie*, 3 Aufl., Gustav Fischer, Jena, 1925, 558 pp.

LODEMAN, E. G. *The Spraying of Plants*, The Macmillan Co., New York, 1899.

LOVERDO, JEAN. *Les Maladies Cryptogamiques des Céréales*, J. B. Bailliére et Fils, Paris, 1892, 312 pp.

LÜSTNER, GUSTAV. *Krankheiten und Feinde der Zierpflanzen im Garten, Park und Gewächshaus*, Ungen Ulmer, Stuttgart, 1933, 266 pp.

McCUBBIN, W. A. *Fungi and Human Affairs*, World Book Co., Yonkers-on-Hudson, N. Y., 1924, 111 pp.

MARTIN, HUBERT. *The Scientific Principles of Plant Protection*, E. Arnold and Co., London, 1928, 316 pp.

MARTIN, HUBERT. *The Scientific Principles of Plant Protection, with Special Reference to Chemical Control*, E. Arnold & Co., London, 1936, 379 pp.

MASON, A. FREEMAN. *Spraying, Dusting and Fumigating of Plants*, The Macmillan Co., New York, 1928.

MASSEE, GEORGE. *Diseases of Cultivated Plants and Trees*, The Macmillan Co., New York, 1910, 602 pp.

MORSTATT, H. *Einführung in die Pflanzenpathologie*, Gebrüder Borntraeger, Berlin, 1923, 159 pp.

NEGER, F. W. *Die Krankheiten Unserer Waldbäume*, Ferdinand Enke, Stuttgart, 1919, 286 pp.

OWENS, CHARLES ELMER. *Principles of Plant Pathology*, John Wiley and Sons, New York, 1928, 629 pp.

RANKIN, W. HOWARD. *Manual of Tree Diseases*, The Macmillan Co., New York, 1918, 398 pp.

RIVERS, THOMAS M. *Filterable Viruses*, Williams and Wilkins Co., Baltimore, 1928.

SCRIBNER, F. LAMSON. *Fungus Diseases of the Grape and Other Plants and Their Treatment*, J. T. Lovett Co., Little Silver, New Jersey, 1890, 134 pp.

SMITH, E. F. *Bacteria in Relation to Plant Diseases*, Vol. I, 1905; Vol. II, 1911; Vol. III, 1914, Carnegie Institute, Washington, D. C.

SMITH, E. F. *An Introduction to Bacterial Diseases of Plants*, W. B. Saunders Co., Philadelphia, 1920, 688 pp.

SMITH, KENNETH M. *Recent Advances in the Study of Plant Viruses*, P. Blakiston's Son & Co., Philadelphia, 1934.

SMITH, KENNETH M. *A Textbook of Plant Virus Diseases*, J. G. A. Churchill Ltd., London, 1937.

SMITH, W. G. *Diseases of Field and Garden Crops*, Macmillan and Co., London, 1884, 353 pp.

SORAUER, PAUL K. M. *Handbuch der Pflanzenkrankheiten*, 6 Aufl. Bd. I., Paul Parey, Berlin, 1934; 5 Aufl. Bd. 2–5, Paul Parey, Berlin, 1926–32.

SORAUER, PAUL K. M. *Manual of Plant Diseases*, Vol. I, Non-Parasitic Diseases, Third Edition, (translated by Frances Dorrance), Record Press, Wilkes-Barre, Pa., 1922.

TAUBENHAUS, J. J. *Diseases of Truck Crops and Their Control*, E. P. Dutton and Co., New York, 1918, 396 pp.

TUBEUF, K. F. VON. *Diseases of Plants Induced by Cryptogamic Parasites*, (English Edition by W. G. Smith), Longmans, Green and Co., London, 1897, 598 pp.

UNDERWOOD, LUCIEN MARCUS. *Moulds, Mildews and Mushrooms*, Henry Holt and Co., New York, 1899, 236 pp.

VEITCH, ROBERT AND SIMMONDS, J. H. *Pests and Diseases of Queensland*

Fruits and Vegetables, Dept. of Agriculture and Stock, Anthony James Cumming, Brisbane, Queensland, 1929.

VIOLA, PIERRE. *Maladies de la Vigne*, A. Delahaye et E. Lecrosnier, Paris, 1887.

WARD, H. MARSHALL. *Diseases in Plants*, Macmillan and Co., London, 1901, 309 pp.

WEBER, GUSTAVUS A. *The Plant Quarantine and Control Administration*, The Brookings Institution, Service Monograms of the United States Government, No. 59, Washington, D. C., 1930.

WEBSTER, A. D. *Tree Wounds and Diseases*, J. B. Lippincott and Co., Philadelphia, 1916, 215 pp.

WEISS, H. F. *The Preservation of Structural Timber*, Second Edition, McGraw-Hill Book Co., New York, 1916.

WHETZEL, H. H. *Laboratory Outlines in Plant Pathology*, W. B. Saunders Co., Philadelphia, 1925, 231 pp.

WHETZEL, H. H. *An Outline of the History of Phytopathology*, W. B. Saunders Co., Philadelphia, 1918, 130 pp.

INDEX

Bold face type indicates figures.

Abutilon chlorosis, 213, 225
Acetic acid, 274
Acid phosphate, 158
Acquired immunity, 38, 39
Actinomyces scabies, 201–205, **202, 203, 204**
Actinomyces spp., 160
Actinomycetales, 160
Aecidiospore, 35, 370
Aecidium, 369, 370
Agrostis spp., 378
Aira spp., 378
Airplane dusting, **86**
Albugo candida, 46, 133–136, **134, 135, 136, 137**
Alder, **47**
Alfalfa
  Anguillulina dipsaci, 445, 446, **447**
  Cuscuta spp., 429–433, **431**
  damping-off, 121–126, **122, 123, 124, 125, 126**
  dodder, 429–433, **431**
  leaf spot, 40, 279–283, **280, 281**
  Phymatotrichum omnivorum, 318
  phymatotrichum root rot, 318
  Pseudopeziza medicaginis, 279–283, **280, 281**
  Pythium debaryanum, 121–126, **122, 123, 124, 125**
  stem nematode, 445, 446, **447**
Algae, 31
Aluminum sulphate, 77, 78, 126
Ammonium sulphate, 78, 158, 205, 325, 444
Ancient records of
  fungous organs, 3
  plant diseases, 3, 4
Anguillulina dipsaci, 444–448, **445, 446, 447**
Angular leaf spot of cotton, 183–195, **183, 184**
Animal pathology, **39**
Antheridia, 3
Anthracnose
  of apple, 58
  of bean, 58
  fruit spot on cantaloup, **26**
  on raspberry, **27**
Anthrax, 19
Antibodies, 39, 40
Antitoxins, 39, 40
Aplanobacter stewartii, 161
Apothecium, 250
Apple
  anthracnose, 58
  Bacillus amylovorus, 19, 65, 162–170, **163, 164, 165, 166, 167**
  bitter pit, 450–453, **451**
  brown rot, 256–263
  callus knot, **460, 461**
  cedar-apple rust, 43, 44, 58, 110, 393–400, **395, 397, 398, 399**
  crown gall, 197–201, **198, 199**
  fire blight, 19, 65, 74, 162–170, **163, 164, 165, 166, 167**
  Fomes igniarius, 420, 422, **424**
  graft knot, 459–462, **460**
  Gymnosporangium juniperi-virginianae, 44, 393–400, **395, 397, 398, 399**
  Illinois blister canker, 28
  Nummularia discreta, **26**
  Pseudomonas tumefaciens, 197–201, **198, 199**
  scab, 49, 50, 56, 82, 96, 111, 283–289, **283, 284, 285, 286, 287, 288**
  scald, 27, 450, 453, 455, **454**
  Sclerotinia fructicola, 256–263
  specific diseases, 1
  Venturia inaequalis, 283–289, **282, 283, 284, 285, 286, 287, 288**
  white heart rot, 420, 422, **424**

Apricot
Bacillus amylovorus, 162
brown rot, 256–263, **261**
fire blight, 162
Sclerotinia fructicola, 256–263, **261**
Arceuthobium spp., 433–438, **435, 437**
Aristotle, 3
Armillaria mellea, 416–420, **417, 418, 419, 420**
Arsenate of lead, 263
Arsenic salts, 103
Ascomycetes, 27, 41, 250, 251
Ascospore, 49, 50, 56, 62, 250
Asparagus
Puccinia asparagi, 44, 389–393, **390, 391**
rust, 40, 389–393, **390, 391**
Aspen
Fomes igniarius, 420, **421, 424**
white heart rot, 420, **421, 424**
Association with host, smuts, 342–343
Aster
Fusarium conglutinans var. *callistephi*, **25**
wilt, **25**
yellows, 209, 214, 228, 229, 245–249, **246, 247**
Autoecius, 44, 369
Autotoxins, 39, 40
Avena spp., 386

Bacillus amylovorus, 65, 162–170, **163, 164, 165, 166, 167**
Bacillus carotovorus, 55, 170–178, **171, 172, 174, 175, 176, 178**
Bacillus tracheiphilus, **29**, 185–189, **186, 187, 188**
Bacteria, 19, 27, 31, 35, 159–205
Bacterium campestre, 161
Bacterium malvacearum, 161
Bacterium phaseoli, 161
Bacterium stewartii, 55, 189–194, **190, 191, 192, 193**
Banana
bunchy top, 209, 213
Barbak III, 98
Barberry
eradication, 68, **108**, 109, 368, 378, 379
Puccinia graminis, 43, 374–379, **375, 377**
stem rust, 43, 374–379, **375, 377**
Barley
Cladosporium herbarum, 294, 295
covered smut, 99
Erysiphe graminis var. *hordei*, 293–297, **294, 295, 296, 297**
leaf rust, 372
loose smut, 37
Barley—*Continued*
powdery mildew, 293–297, **294, 295, 296, 297**
Puccinia anomala, 372
Puccinia graminis var. *tritici*, 371, 378
seed treatment, 99, 100
stem rust, 37, **109**, 371, 378
Ustilago nuda, 100
Barrel
for treating seed, **97**
Basidiomycetes, 27, 341–425
Basidiospore, 370
Basidium, 341
Baumé, 87
Bayer dust, 98
Bean
black scurf, 414
blight, 70, 179–182, **179, 180, 182**
Corticium vagum, 414
mosaic, 39, 53, 54, 62, 63, 110, 209, 223–224, 230
Pseudomonas phaseoli, 179–182, **179, 180, 182**
rust, 44
Thielavia basicola, 269
Thielavia root rot, 269
Uromyces appendiculatus, 44
Beech
Fomes igniarius, 420, 422, **424**
white heart rot, 420, 422, **424**
Beet
Actinomyces scabies, 201
beet leaf hopper, 243–245, **244**
black scurf, 414
boron deficiency, 455–459, **456, 457**
Cercospora beticola, **34**, 57, 62, 63, 70, 71, 325–328, **326, 327, 328, 329**
Corticium vagum, 414
leaf spot, **34**, 57, 62, 63, 70, 71, 325–328, **326, 327, 328, 329**
Phoma betae, 455, 456
potato scab, 201
Beetles
elm, 306, **307**
Begonia
Thielavia basicola, 269
Thielavia root rot, 269
Beijerinck, 208
Binucleate mycelium, 341, 342, 369
Biologic forms, 33, 41
Biologic races, 33
Biotypes, 33, 42, 112
Birch
Fomes igniarius, 420, **422, 424**
white heart rot, 420, **422, 424**

Bitter pit, 450–453, **451**
Black-arm, 184
Blackberries
Gymnoconia interstitialis, 45, **46**
orange rust, 45, **46**
Black heart
potatoes, 27, 450
Black knot
plum, 45
Black leaf, 40, 189
Black leg of potato, 15, 37, 38, 54, 55, 173–178, **174, 175, 176, 178**
Black mustard
black rot, 194, **196**
Pseudomonas campestris, 194, **196**
Black rot of Crucifers, 194–197, **195, 196**
Black scurf of potato, 15, 52, 54, 69, 412–416, **413, 415**
Black stalk, 173
Black stem rust
on wheat, 14
Blasts, 3
Blight, 3
bean, 70, 179–182, **179, 180, 182**
chestnut, 2, 12, 13, 40
corn seedling, 60, **61**
wheat seedling, 53, 54, **59**, 60
Blossom-end rot of tomatoes, 27, 63, 450
Bolley, 96
Bordeaux, 87–93, 131, 143, 169, 289, 301
acid, 90, 91
alkaline, 90, 91
formula, 89, 91
instant, 91
introduction of, 19, 68, 88, 89
neutral, 90, 91
toxicity of, 92
Boric acid, 459
Boron
deficiency disease, 449, 455–459, **456, 457**
in control, 71, 459
Botrytis, 35
Bromus spp., 378
Broomrape, 427, **428**
Brown rot
plum, 37, 256–263, **257, 258, 259, 260, 261**
stone fruits, 58, 256–263, **257, 258, 259, 260, 261**
Brown spot of corn, 143–147, **144, 145, 146**
Brussels sprouts
black rot, 194, 196
Pseudomonas campestris, 194, **196**
Buckthorn
crown rust, 379–389, **382, 384, 385, 387**
Puccinia coronata, 379–389, **382, 384, 385, 387**
Bunchy top of banana, 209, 213
Bunt of wheat, 37, 52, 53, 54, 60, 96, 99, 360–364, **361, 362**
Bureau of Plant Industry
establishment of section of vegetable pathology in, 19
organizers of, **20**
Burrill, 19, 159, 166
Butternut
Fomes igniarius, 420, **424**
white heart rot, 420, **424**

Cabbage, 2
Bacillus carotovorus, 170–173, **172**
black bread mold, 32
black rot, 194–197, **195, 196**
club root, 54, 55, 56, 64, 72, 147–152, **148, 149, 150, 151**
damping-off, 121, **124**
Fusarium conglutinans, 52, **53**
Plasmodiophora brassicae, 54, 56, 147–152, **148, 149, 150, 151**
Pseudomonas campestris, 194–197, **195, 196**
Pythium debaryanum, 121, **124**
Sclerotinia spp., 263, **264**
soft rot, 170–173, **172**
yellows, 2, 52, **53**, 111, 112
Calamagrostis canadensis, 386
Calcium arsenate, 189
Callus knot, **460**, 461
Cancer root, 426
Cantaloup
antracnose, 26
Bacillus tracheiphilus, 186, **188**
powdery mildew, 112
wilt, 186, **188**
Capsella bursa-pastoris
white rust, 46
Carbohydrates, 1
Carbon bisulphide, 77, 78, 325
Carrot
Actinomyces scabies, 201
Bacillus carotovorus, 55, 170, **171, 172**
Heterodera marioni, **442, 443**
potato scab, 201
root knot, **442, 443**
Sclerotinia spp., 263
Cassytha, 429
Catabolic activities, 1

Catalpa, 269
Cauliflower
black rot, 194, **196**
club root, 147
Plasmodiophora brassicae, 147
Pseudomonas campestris, 194, **196**
Sclerotinia spp., 263
Cedar
Gymnosporangium juniperi-virginianae, 43, 44, 58, 108, 110, 393–400, **397, 398**
rust, 43, 44, 58, 108, 110, 393–400, **397, 398**
Cedar-apple rust, 43, 44, 58, 108, 110, 393–400, **395, 397, 398, 399**
Celery
Bacillus carotovorus, 170, **172**
black scurf, 414
Corticium vagum, 414
Jones' soft rot, 170, **172**
mosaic, 229
Sclerotinia spp., 263
yellows, 245, 248
Cephalosporium spp., 304
Ceratostomella ulmi, 108, 301–308, **302, 303, 304, 305, 307**
Cercospora beticola, 34, 57, 62, 63, 70, 71, 325–329, **326, 327, 328, 329**
Cereals, 3–6
Anguillulina dipsaci, 445, **447**
foot rots, 40
Gibberella saubinetii, 56
Helminthosporium gramineum, 56
Ophiobolus graminis, 55, 56
Puccinia graminis, 43
rusts, 33, 41, 42, 367–389
seed treatment, 96, **97, 98**, 100
smuts, 341–367
stem nematode, 445, **447**
Ceresan, 98
New Improved Ceresan, 98
Chaetocnema denticulata, 193
Chaetocnema pulicaria, 192
Checkrowing, 70, **71**, 328, 329
Chemicals, 77–78, 224–225, 450
Chenopodium murale, 221
curly top virus, 243
Cherry
Armillaria mellea, **419, 420**
brown rot, 256–263, **261**
mushroom root rot, **419, 420**
scab, 283
Sclerotinia fructicola, 256–263, **261**
Chestnut
blight, 2, 12, 13, 40, 50, 251–256, **252, 253, 254, 255**
Chestnut—*Continued*
Endothia parasitica, 251–256, **252, 253, 254, 255**
hybridization, 256
Tree Blight Commission, 256
Chickweed, 221
Chives, 356
Chlamydospore, 342
Chloropicrin, 77, 78, 325
Chloroses, 206
Chlorosis, 212
abutilon, 213, 225
white pickle of cucumber, 212, **213**
Cholera, 1
Chrysanthemum, 248
Citrus
Armillaria mellea, 416, **420**
canker, **26**, 106, 107
mushroom root rot, 416, **420**
Pseudomonas citri, **26**, 106, 107
Cladosporium herbarum, 294, 295
Claviceps purpurea, 5, 6, 274–279, **276, 278**
ergot poisoning, 5
rye, 5, 274–279
wheat, 6
Cleistothecium, 250
Clover
Anguillulina dipsaci, 445, **447**
broomrape, 427
clover rot, 72
clover rust, 44
Cuscuta spp., 429–433, **431, 432**
dodder, 429–433, **431, 432**
Erysiphi polygoni, 8
Orobanche, 427
powdery mildew, 8
stem nematode, 445, **447**
Thielavia basicola, 269
Thielavia root rot, 269
Uromyces trifolii, 44
Club root of cabbage, 54, 55, 56, 64, 72, 147–152, **148, 149, 150, 151**
Coffee rust, 10
Colletotrichum circinans Vog., 38
Colletotrichum lagenarium, **26**
Commandra, 426, 427
Commelina nudiflora, 229
Conidia, 3, **57**, 62, 251
Conifers, **43**
Conopholis, 426
Control
permanent measures of, 104–113
principles of, 68–113
temporary measures of, 70–104
virus diseases, 228–230

Copper, 71, 77, 78, 92, 99, 100
Copper carbonate, 94, 96, 99, 363
Copper salts, 94, 99
Copper sulphate, 92, 93, 99, 102, 103, 121, 268, 308, 324
Cork, 37, 38, 63
Corn, 14, 50, 51, 97, 98
Bacterium stewartii, 189
black bread mold, 32
brown spot, 143–147, **144, 145, 146**
Diplodia zeae, 32, 40, 60, 65, 66, 110, 310–317, **311, 312, 313, 314, 315, 316**
downy mildew, **24**
dry rot, 32, 40, 60, 65, 66, 110, 310–317, **313, 314, 315, 316**
frost injury, 450
Gibberella saubinetii, 60, **61**
mosaic, 212
Physoderma zeae-maydis, 57, 143–147, **144, 145, 146**
Puccinia sorghi, 44
rust, 44
scab disease, 60
seedling blight, 60, **61, 311**
smut, 343–350, **345, 346, 348, 349**
Stewart's disease, 55, 110, 189
streak disease, 225, 226, 228
sugar cane mosaic virus, 238
Ustilago zeae, 343–350, **345, 346, 348, 349**
Corn rootworm, 192
Corrosive sublimate, 200, 205
Cortical necrosis, **30**, 162
Corticium vagum, 54, 412–416, **413, 415**
Cotton, 2, 41
angular leaf spot, 183–185, **183, 184**
black root, 336
frenching, 336
Fusarium vasinfectum, 41, 336–340, **337, 338, 339**
Heterodera marioni, 440, **443**, 444
Phymatotrichum omnivorum, 56, 317–325, **317, 318, 320, 321, 323**
Pseudomonas malvacearum, 183–185, **183, 184**
root knot, 440, **443**, 444
root rot, 40, 56, 317–325, **317, 318, 320, 321, 323**
sore-shin, 66, 67
wilt, 2, 41, 336–340, **337, 338, 339**
Cottonwood
Fomes igniarius, 420, **424**
mistletoe, **433, 436, 437**
Cottonwood—*Continued*
Phoradendron spp., **433, 436, 437**
white heart rot, 420, **424**
Country Gentleman, 40, 110
Covered smut
of barley, 99
of oats, 113, 350–355, **352, 353**
Cowpeas
Heterodera marioni, 440, **442, 443**, 444
root knot, 440, **442, 443**, 444
Cowwheat, 426, 427
Coxe, William, 162
Creosote oil, 102, 103, 104
Cronartium ribicola, 44, 400–409, **403, 406, 407, 408**
Crop rotation, 72, 73, 75
Crosses of races, 43
Crown gall, 197–201, **198, 199**
Crown rust, 44, 58, 59, 113, 378–389, **380, 381, 382, 383, 384, 385, 387, 388**
alternate host Rhamnus, **42**, 378–389
on grasses, 386
on oats, 42, 43, 378–389
Cruciferae
Albugo candida, 133–136, **134, 135, 136, 137**
black rot, 194–197, **195, 196**
club root, 148
mosaic, 62
Plasmodiophora brassicae, 148
Pseudomonas campestris, 194–197, **195, 196**
white rust, 46, 58, 133–136, **134, 135, 136, 137**
Cucumber
Bacillus tracheiphilus, 185–189, **186, 187, 188**
mosaic, 40, 209, **212, 213, 220**, 223, 230
white pickle, 212–213, **213**
Sclerotinia spp., 263
stem rot, 63
Thielavia basicola, 269
Thielavia root rot, 269
wilt, **29**, 185–189, **186, 187, 188**
Cucumber beetle, 188
Cucurbit
Bacillus tracheiphilus, 185–189, **186, 187, 188**
curly top virus, 210
wilt, 185–189, **186, 187, 188**
Cultural characteristics, 41
Cuprous oxide, 99, 100

Curly top of sugar beets, 2, 11, 62, 230, **234**, 240–245, **239**, **240**, **241**, **242**, **243**, **244**
beet leaf hopper, **243–245**
Curly top virus
beans, 210
cucurbit species, 210
sugar beets, 209, 221, 225, **239**, 240–245, **240**, **241**, **242**, **243**, **244**
tomatoes, 210
Currants
Cronartium ribicola, 400–409, **406**, **407**, **408**
rust, 400–409, **406**, **407**, **408**
Current crop losses, 14–16
Cuscuta spp., 429–433

Dahlia
Armillaria mellea, **419**, **420**
Damping-off, 32, 40, 55, 63, 65, 70, 77, 121–126, **122**, **123**, **124**, **125**
Darnell-Smith, 99
Darwin, 18
Dasyscypha calicina, 108
de Bary, 18, 31, 43, 368
Deficiency diseases, 71
Degenerative diseases, 206
Delinting, 185
Denning, 162
Dent corn, 40
Devil's hair, 429
Diabrotica duodecimpunctata, 188, 193
Diabrotica longicornis, 192
Diabrotica vittata, 188
Dibotryon morbosum, 45
Dicotyledons, rust on, 43
Diplodia dry rot, 32, 40, 60, 65, 66, 110, 310–317, **311**, **312**, **313**, **314**, **315**, **316**
Diplodia zeae, 32, 40, 60, 65, 66, 110, 310–317, **311**, **312**, **313**, **314**, **315**, **316**
Diploid mycelium, 342
Direct general necrosis, **28**, 29, 162
Direct local necrosis, **27**, 29, 162
Disease, 3, 4, 5–17, 22, 23
classification, 24–30
definition, 22
influence of environment, 51–67
resistance, 36, 110–113
signs, 23–24
symptoms, 23–24
Disease-escaping, 78–80
Disease-free seed, 80, 81
Disease-free stock, 80, 81
Distortion, 213
Dodder, 26, 426, 429–433, **430**, **431**, **432**
Downy mildew, **24**, 41, 44
of grape, 9, 10, 19, 46, 50, 68, 137–143, **138**, **139**, **140**, **141**, **142**
of maize, **24**
Dry rot of corn, 32, 40, 60, 65, 66, 110, 310–317, **311**, **312**, **313**, **314**, **315**, **316**
Duchartre, 86
Dusting, 81–93
airplane, **84**
hand duster, **85**
Dutch elm disease, 301–308, **302**, **303**, **304**, **305**
Dwarfing, 213

Eelworms, 437–448, **442**, **443**, **447**
Egg plant, 100
Bacillus carotovorus, 170, **172**
soft rot, 170, **172**
Electrical pasteurization, 77
Elementary species, 33
Elm
beetle, 306, **307**
Cephalosporium spp., 304
Ceratostomella ulmi, 108, 301–308, **302**, **303**, **304**, **305**, **306**
eradication, 108
graphium wilt, 108, 301–308, **302**, **303**, **304**, **305**, **306**
Verticillium spp., 304
Endothia parasitica, 12, 13, 40, 50, 251–256, **252**, **253**, **254**, **255**
Environment
cause of plant diseases, 25, 27
influence on disease, 51–67, **53**
influence on host, 51, 59–63
influence on pathogen, 51, 52–59, 221
influence on resistance, 36, 51, 59–63
Enzyme
action on cell walls, 34
cytolytic, 168
in infection, 34
pectinase, 172, 176
Epidemic, 47
Epidermis penetration, 34
Epiphytic, 34
Epiphytotic, 44, 47–50, **48**, 57, 58, 62
definition, 47
extensive, **50**, 57, 58, 62
local, **49**, 50
Epizooic, 47
Eradication, 68, 106–110, 308, 368, 378, 379, 388, 389, 407, **408**, 409, 438

Ergot, 5, 62, 274–279, **276, 278**
ergot poisoning, 5
rye, 5, 274–279
wheat, 6
Erwinia amylovora, 161
Erwinia carotovora, 161
Erwinia tracheiphila, 161
Erysiphe graminis, varieties of, 297
Erysiphe graminis var. *hordei*, 294–297, **294, 295, 296, 297**
Erysiphe polygoni, 8
Ethyl alcohol, 169
Ethyl mercury chloride, 98, 104
Ethyl mercury phosphate, 98, 364
Etiology, 25–27
Eulalia, 238
Euphrasia, 426, 427
Evergreen, 32
Evolution, 18
Eyebright, 426, 427
Experiment stations, 19, 20, 21

False tinder fungus, 420–425
Farlow, 19
Fats, 1
Federal Quarantine Act, 68, 105
Federal plant quarantines, 100, 407
Fertilizers, 71
Festuca elatior, 386
Festuca spp., 378
Figwort, 426
Finger-and-toe disease, 147
Fir
Arceuthobium spp., **437**, 438
damping-off of seedlings, 121, **124**
mistletoe, **437**, 438
Pythium debaryanum, 121, **124**
Fire blight, 19, 65, **74**, 162–170, **163, 164, 165, 166, 167**
Fission fungi, 27
Flag smut, 39
Flax, 2, **16, 111**, 112
Cuscuta epilinum, 429
dodder, 429
Fusarium lini, 49, **50**, 53
heat canker, 450
Melampsora lini, 39, 44
rust, 39, 44
wilt, 2, 11, 16, 53, **111**, 112
Flea beetle, 192
Flint corn, 40
Flowering plants, 26, 27, 31, 426–438
Fog drive gun, **82**
Fomes igniarius, 420–425, **421, 422, 424**
Food, 27
Foot rots of cereals, 40
Formaldehyde, 19, 77, 78, 94–96, **95**, 126, 169, 205, 325, 354, 359, 363
Forms, 41
biologic, 33, 41
biotype, 33
new forms by crossing, 43
physiologic, 41
Frenching, 336
Frost injury, 27, 450
Fruit rot fungi, 35
Fungi, 31, 35, 114
algal, 114–158
cause of plant diseases, 25, 27
imperfecti, 27, 31, 41, 309–340
new races of by mutation, 43
palisade, 409–425
parasites of fungi, 55, 56
sac, 27, 250–308
stalk, 27, 341–425
Fungicides, 83–84, 450
Fusarium bulbigenum var. *lycopersici*, **54**
Fusarium bulbigenum var. *niveum*, 40, 53, 55, 65, 330–335, **331, 332, 333, 334, 335**
Fusarium conglutinans, 52, **53**
Fusarium conglutinans var. *callistephi*, **25**
Fusarium lini, 49, **50**, 53
Fusarium vasinfectum, 41, 336–340, **337, 338, 339**

Galls, 214, 369
General necrosis, **28**, 29, 162
Germ tube, 34, 35
Gibberella saubinetii, 53, 54, 56, **59**, 60, **61**
cereals, 56
corn, 60, **61**
wheat, 53, 54, **59**, 60
Gill fungi, 409
Ginseng
Thielavia basicola, 269
Thielavia root rot, 269
Golden Bantam, 40
Gooseberry
Cronartium ribicola, 400–409, **406, 407, 408**
powdery mildew, 8
rust, 400–409, **406, 407, 408**
Goosefoot, 221
Graft knot, 459–462, **460, 461**
Grape
crown gall, 197–201, **198**
downy mildew, 9, 10, 19, 46, 50, 68, 137–143, **138, 139, 140, 141, 142**

Grape—*Continued*
Plasmopara viticola, 9, 10, 46, 58, 62, 137–143, **138, 139, 140, 141, 142**
powdery mildew, 7, 8, 68, 86, 289–292, **290, 291**
Pseudomonas tumefaciens, 161, 197–201, **198, 199**
Uncinula necator, 7, 8, 289–292, **290, 291**
Graphium ulmi, 304
Graphium wilt of elm, 301–308, **302, 303, 304, 305, 307**
Grasses, 41, 43, 378
"Grey top" of tobacco, 231
Guatemala grass, 238
Gummosis, 183
Gymnoconia interstitialis, 45, 46
Gymnosporangium germinale, 394
Gymnosporangium globosum, 394
Gymnosporangium juniperi-virginianae, 44, 393–400, **395, 397, 398, 399**

Hairweed, 429
Hairy root, 197, 200
sugar beet, 241
Haploid mycelium, 341, 342, 370
Hartig, Robert, 18
Hatch Act, 19
Hawthorn
Bacillus amylovorus, 163
fire blight, 163
rust, 394
Heart rots, 411, 412, 420–425, **421, 422, 424**
Heat
canker, 450
in disinfecting soil, 75, 76, 77
in seed treatment, 94, 100, 126
Hell-bind, 429
Helminthosporium gramineum, 56
Hemp, 427
Heterodera marioni, 440–444, **441, 442**
Heteroecism, 43, 44, 369
Heterothallism, 341
Heterozygosity, 36
Hog cholera, 1
Holcus lanatus, 386
Hop, 39
mildew, 39
Sphaerotheca humuli, 39
Hopper burn, 15
Hosts, 25, 31, 36, 40, 42, 43, 59–63
alternate, **42**, 43
differential, 42
Hot water treatment, 94, 223, 367, 448
Human affairs, 3–18
Human pathology, 39
Humidity
influence on disease, 62–64
influence on host, 63
influence on parasites, 58, 62, 63
Hyacinth
Anguillulina dipsaci, 445, **447**
stem nematode, 445, **447**
Hybridization, 112, 113, 256
Hydathodes, 35
Hydnoraceae, 427
Hydrochloric acid, 169
Hydroxymercurichlorophenol, 98
Hydroxymercurinitrophenol, 97, 98
Hydroxymercuricresol sulphate, 98
Hylemyia cilicrura, 177, **178**
Hylurgopinus rufipes, 306
Hyperplasia, **47**, 214, 439
Hypertrophy, **30**, 369, 439
Hypha, **34**, 35
Hypoplasia, 211–212

Idaho Refugee beans, 39
Illinois blister canker, **28**
Immunity, 38–40, 113
definition, 38
in animal pathology, 39
in plants, 38
Immunization, 39, 40
Imperfecti (Fungi), 27, 41, 309–340
Inclusion bodies, 216
Indirect cortical necrosis, **30**, 162
Indirect vascular necrosis, **29**, 30, 162
Infected refuse, 74, 75
Infection, 34, 35, 36, **37**, 38, 52–56, **53**, 58
secondary, 44, 50
Inhibitors, 39
Inorganic salts, 1
Insecticides, injury by, 100, 101, 450
Insects, 75
cause of disease, 26
vectors, 62, 74, 225, 226
Intercellular, **33**, 34
Internal decline, 450
Internal symptoms, 24
Intracellular, **33**, 34
Inverted pan, **76**, 77
Iodent corn, 40
Iron, 71, 78
Ivy, 427
Iwanowski, 208, 222

Jones' soft rot of vegetables, 170–173, **171, 172**

Juneberry
Bacillus amylovorus, 163
fire blight, 163

Kale, 194
Koch, 19
Koch's postulates, 159
Kohlrabi, 194, **196**
Krameria, 426
Kühn, Julius, 18

Larch
canker, 108
Dasyscypha calicina, 108
Late blight of potato, 6, 7, 18, 31, 35, 44, **45, 49,** 50, 56, 70, 126–133, **127, 129, 130, 131, 132, 133**
Latitude of health, 22, **23**
Latitude of life, 22, **23**
Lead arsenate, 263
Leaf roll of potatoes, **215,** 220, 223
Leaf rust, 371–372
Leaf spot
alfalfa, 40, 279–283, **280, 281**
sugar beets, 72, 325–329, **326, 327, 328, 329**
Leek, 356
Legumes, 456
Lemons, 450
Lenticels, 35
Lettuce
Bacillus carotovorus, 170, **172**
black scurf, 414
Corticium vagum, 414
drop, 63, 263–269, **265, 267**
soft rot, 170, **172**
Sclerotinia spp., 263–269, **265, 267**
tip burn, 27
watery soft rot, 263
yellows virus in, 245
Light, 27, 59, 63, 449
Lime, 55, 77, 78, 88, 89, 152, 205
Lime sulphur, 86, 87, 263, 289, 301
Liming, 55
Lines, 33
Line selection, 111, 112
Local necrosis, 27, 162
Lodging, 450
Loeffler, 216
Loose smut, 100
of barley, 367
of oats, 37, 52–54, 113, 350 355, **352, 353**
of wheat, 364–367, **365, 366**
Loranthaceae, 426
Losses, 6–17, **109,** 251, **257,** 263–264, 269, 275, **282,** 298
Love vine, 429
Lump jaw, 1

Maggot, seed corn, 176, **178**
Maize, **24,** 40, 238
Mangels, 201
Mangelwurzel, 325
Maple
Fomes igniarius, 420, **424**
white heart rot, 420, **424**
Masked symptoms of virus, 61, 62, 221
Mayer, Adolf, 207, 216, 230
Meadow fescue, 386
Mechanical resistance, 36, 37, 38
Mechanical barriers, 37, 38
Melampyrum, 426, 427
Melampsora lini, 39, 44
Mercury, 94, 316, 325, 329, 335, 354, 363
mercuric chloride, 96, 97, 102, 103, 193, 194–197, 274
mercuric cyanide, 98
mercury ammonium chloride, 98
mercury poisoning, 97
Merko, 98
Microspecies, 33
Mildews, 3, 7, 8, 9, 10, 39, 41
Milkweed, 160
cucumber mosaic virus, **220**
Millardet, 19, 88, 89, 92
Mistletoe, 26, 426, 428, 429, 433–438, **433, 435, 436, 437**
Modern records of disease, 5–17
Moisture
air moisture, 57–59
soil moisture, 53–55
Monocots, 43
Mosaic, 39, 40, 61, 62, 63, 110, **209, 212, 213, 214, 216, 217,** 218, 223, 230–235
Mottle top of tobacco, 231
Mountain ash, 162
Mummy, plum, **259, 260,** 261, 262
Mushroom root rot, 416–420, **417, 418, 419, 420**
Mutation, 43
Mycelia, 3, 23, 44–46, 93, 341–343, 369–370
Mycorrhiza, 46, **47**

Narcissus
Anguillulina dipsaci, 445, **447**
stem nematode, 445, **447**
Natural immunity, 38, 39

Necrosis, 27–30, 251
direct general, **28**, 29
direct local, **27**, 29
indirect cortical, **30**
indirect vascular, **29**, 30
Nemas, 439
Nematodes, 26, 77, 78, 439–448, **441, 442, 443, 445, 446, 447**
Nettle-leaved goosefoot, 243
Nicotine sulphate, 189
Nightshade, 269
Non-parasitic diseases, 26, 27, 449–462
Norton, 126
Nummularia discreta, **26**

Oak
Fomes igniarius, 420, 422, **424**
white heart rot, 420, 422, **424**
Oats, 14, **16**, 17, **35**, 96, **109**
Anguillulina dipsaci, 445, 446, **447**
crown rust, **42**, 43, 44, 58, 59, 371, 379–389, **380, 381, 382, 383, 384, 385, 387, 388**
powdery mildew, 293
Puccinia coronata, **42**, 44, 371, 379–389, **381, 382, 383, 384, 385, 387, 388**
Puccinia graminis, 41, 371, 378
smut, 37, 52–54, 64, 65, 100, 113, 350–355, **352, 353**
stem nematode, 445, 446, **447**
stem rust, 371, 378
Ustilago avenae, 64, 65, 96, 100, 350–355, **352, 353**
Ustilago levis, 350–355, **352, 353**
Obligate parasite, 32, 369
Okra, 318
Olive, 4
Onions
Anguillulina dipsaci, 445, 446, **447**
black scurf, 414
Colletotrichum circinans, 38
Corticium vagum, 414
Puccinia asparagi, 44, 389
rust, 44, 389
Sclerotinia spp., 263
Sclerotium cepivorum, 52
smudge, 38, 110
smut, 52, 355–359, **356, 358, 359**
stem nematode, 445, 446, **447**
Urocystis cepulae, 355–359, **356, 358, 359**
white rot, 52
yellow dwarf, 209, **210, 211**, 223
Oogonia, 3
Oospora scabies, 161
Ophiobolus graminis, 55, 56
Orange rust, 45, 46
Orobanche, 427, **428**
Ozonium root rot, 317

Palisade fungi, 409–425
Parasite, 31, 32, 36, 40, 41, 55, 56
definition, 31
obligate, 32, 369
Parasitism, 18, 31–50, 341, 369, 409, 412, 426
definition, 31
intercellular, 34
intracellular, 34
Parsley, 263
Parsnip
Bacillus carotovorus, 170, **172**
soft rot, 170, **172**
Partial soil sterilization, 75–78, 325, 359, 444
Pasteur, 18
Pasteurization of soil, electrical, 77
Pathogenicity, 32, 33, 41
Peach
Armillaria mellea, **417, 420**
brown rot, 256–263, **257, 261**
crown gall, 197, **198**
leaf curl, 58, 298–301, **298, 299**
little peach virus, 40
mosaic, 230
phoney disease, 230
Pseudomonas tumefaciens, 197, **198**
root rot, **417, 420**
scab, 37
Sclerotinia fructicola, 256–263, **257, 261**
Taphrina deformans, 298–301, **298, 299**
yellows, 10, 40, 207, 209, 223, 226, 228, 230
Pear
Bacillus amylovorus, 162–170, **167**
fire blight, 162–170, **167**
Fomes igniarius, 422, **424**
scab, 283
white heart rot, 422, **424**
Pearl millet, 238
Pectase, 262
Pectinase, 262
Pedicularis, 426, 427
Penetration, **33**, 34, 35
Peppers
Bacillus carotovorus, 170, **172**
Sclerotinia spp., 263
soft rot, 170, **172**
Perennial mycelium, 44–46
Perithecium, 250
Permanent control measures, 104–113

Peronosporales, 3
Persoon, 368
Pfeiffer, 216
Phoma betae, 455, 456
Phoradendron spp., 428, 433–438, **437**
Phycomycetes, 27, 114–158
Phylloxera, 137
Phymatotrichum omnivorum, 56, 317–325, **317, 318, 320, 321, 323**
Phymatotrichum root rot, 317–325, **317, 318, 320, 321, 323**
Physiologic
  forms, 33, 41
  races, 33, 41
  species, 41
  varieties, 41
Physiological resistance, 36, 37
Physoderma zeae-maydis, 57, 115, 143–147, **144, 145, 146**
Phytomonas campestre, 161
Phytomonas malvacearum, 161
Phytomonas phaseoli, 161
Phytomonas rhizogenes, 161
Phytomonas stewartii, 161
Phytomonas tumefaciens, 161
Phytopathology, definition, 22, 23
Phytophthora infestans, 6, 7, **37, 49,** 56, **57,** 126–133, **127, 129, 130, 131, 132, 133**
Pine
  Arceuthobium spp., **435, 437**
  Cronartium ribicola, 400–409, **403, 406, 407, 408**
  damping-off, 121, **122, 124**
  mistletoe, **435, 437**
  Pythium debaryanum, 121, **122, 124**
  white pine blister rust, 400–409, **403, 406, 407, 408**
Pineapple
  Anguillulina dipsaci, 444, **447**
  root knot, 444, **447**
Pinus spp., 401
Plant diseases
  ancient records, 3, 4
  influence on human affairs, 3, 5
  modern records, 5–17
Plant importation, 105, 106
Plant pathology
  beginning of modern concept, 31
  development, 3, 5, 7, 18, 19
  introduction into educational institutions, 18
  section of vegetable pathology established, 19
Plant quarantine, 68, 104–106
Planting stock, 80, 81
Plasmodiophora brassicae, 54, 56, 147–152, **148, 149, 150, 151**
Plasmopara viticola, 9, 10, 46, 58, 62, 137–143, **138, 139, 140, 141, 142**
Plectodiscella veneta, **27**
Pliny, 4
Plum
  Bacillus amylovorus, 162
  black knot, 45
  brown rot, 37, 256–263, **258, 259, 260, 261**
  Dibotryon morbosum, 45
  fire blight, 162
  Fomes igniarius, 422, **424**
  Sclerotinia fructicola, 256–263, **258, 259, 260, 261**
  white heart rot, 422, **424**
Poa spp., 378
Poisonous gases, injury by, 450, 453, **454**
Polymorphism, 43, 369
Pop corn, 40
Pore fungi, 409
Potash, 340
Potassium chloride, 278
Potassium sulphate, 158
Potato, 6, 7, **15, 30,** 70
  Actinomyces scabies, 201–205, **202, 203, 204**
  Anguillulina dipsaci, 445, 446, **447**
  Bacillus carotovorus, 170, **172,** 173–178, **174, 175, 176, 177**
  beetle injury, 15
  black heart, 27, 450
  black leg, 15, 37, 38, 54, 55, 173–178, **174, 175, 176, 177**
  black scurf, 15, 52, 54, 96, 412–416, **413, 415**
  bread mold, 32
  common scab, 53, 54, 72
  Corticium vagum, 412–416, **413, 415**
  crinkle mosaic, 225
  curl, 207
  curly dwarf, 213
  dry rot, 129, **131**
  Heterodera marioni, 442, **443**
  hopper burn, 15
  late blight, 6, 7, 18, 31, 35, 44, **45, 49,** 50, 56, **57,** 70, 126–133, **127, 129, 130, 131, 132, 133**
  leaf roll, **215,** 220
  mosaic, 61, 62, 63, **209**
  non-parasitic diseases, 27

Potato—*Continued*
Phytophthora infestans, 6, 7, 35, **37, 49,** 56, **57,** 126–133, **127, 129, 130, 131, 132, 133**
powdery scab, 152–158, **153, 154, 155, 156, 157**
Rhizopus nigricans, 32, 117
root knot, 442, **443**
scab, 201–205, **202, 203, 204**
Sclerotinia spp., 263
Sebago, 133
seed treatment, **95,** 96, 98, 99
soft rot, 117, 170, **172**
spindle tuber, 62, 210, 213, 214
Spongospora subterranea, 152–158, **153, 154, 155, 156, 157**
stem nematode, 445, 446, **447**
sunscald, 450
Synchytrium endobioticum, **30,** 39
virus diseases, 15
wart disease, **30,** 39
wet rot, 129
Powdery mildew, 32, 34, 58, 63, 112
on barley, 293–297, **294, 295, 296, 297**
on clover, 8
on gooseberry, 8
on grape, 7, 8, 68, 86, 289–292, **290, 291**
Powdery scab of potatoes, 152–158, **153, 154, 155, 156, 157**
Prevost, 92, 99
Promycelium, 342, 369, 370
Propagating stock, 80, 81
Proteins, 1
Protocatechuic acid, 38
Protopectinase, 262
Protoplasm, 1, 38
Pruning, 74
Pseudomonas campestris, 194–197, **195, 196**
Pseudomonas citri, **26,** 106, 107
Pseudomonas malvacearum, 183–185, **183, 184**
Pseudomonas phaseoli, 179–182, **179, 180, 182**
Pseudomonas rhizogenes, 200
Pseudomonas tumefaciens, 161, 197–201, **198, 199**
Pseudopeziza medicaginis, 279–283, **280, 281**
Puccinia anomala, 372
Puccinia asparagi, 44, 389–393, **390, 391**
Puccinia coronata, **42,** 44, 371, 379–389, **380, 381, 382, 383, 384, 385, 387, 388**
Puccinia glumarum, 38, 371
Puccinia graminis, 37, 41, 42, 43, 44, 57, 371, 372–379, **373, 374, 375, 377**
Puccinia rubigo-vera, 42, 371–2
Puccinia sorghi, 44
Pumpkin
Bacillus tracheiphilus, 186, **188**
curly top virus, 240
wilt of cucurbits, 186, **188**
Pure line selection, 111, 112
Pycnium, 369
Pycniospore, 369, 370
Pyrus usuriensis, 418, **420**
Pythiaceous fungi, 55
Pythium, 55
Pythium debaryanum, 32, 115, 121–126, **122, 123, 124, 125**

Quarantine, 68, 104–106, 252, 308, 325, 407
Quince
Bacillus amylovorus, 162
fire blight, 162
Gymnosporangium germinale, 394
rust, 394

Races, 40–43
Racial strains, **33**
Radish
Actinomyces scabies, 201
Albugo candida, 133–136, **134, 135, 136, 137**
Bacillus carotovorus, 170, **172**
black rot, 194, **196**
club root, 147
Plasmodiophora brassicae, 147
potato scab, 201
Pseudomonas campestris, 194, **196**
soft rot, 170, **172**
white rust, 133–136, **134, 135, 136, 137**
Rafflesiaceae, 427
Rape
black rot, 194, **196**
club root, 147
Plasmodiophora brassicae, 147
Pseudomonas campestris, 194, **196**
Raspberry
anthracnose, **27**
crown gall, 197, **198**
Gymnoconia interstitialis, 45, 46
mosaic, 61
orange rust, 45, 46
Plectodiscella veneta, **27**
Pseudomonas tumefaciens, 197, **198**
Virus, 227, 230
Ratany, 426
Red cedar, 43, 44, 110
Red rattle, 426, 427

Reed grass, 386
Reid's yellow dent corn, 40
Resistance, 36–38, 110–113
  acquired, 30, 39
  definition, 36
  mechanical, 36, 37, 38
  physiological, 36, 38
Resistant varieties, 37, 43, 335, 340, 350, 354, 364, 367, 378, 379, 388, 392, 393, 400, 409, 444, 448, 455
Respiration, 1
Rhamnus
  crown rust, **42**, 44, 379–389, **382, 383, 384, 385, 387, 388**
  Puccinia coronata, **42**, 44, 379–389, **382, 383, 384, 385, 387, 388**
Rhinanthus, 426, 427
Rhizoctonia solani, 412
Rhizopus nigricans, 32, 58, 63, 117–121, **118, 120**
  on sweet potatoes, 63, 117–121, **118, 120**
Ribes, 110
  Cronartium ribicola, 44, 400–409, **406, 407, 408**
  white pine blister rust, 400–409, **406, 407, 408**
Rice stunt, 227
Ring spot of tobacco, 39
Roguing, 74, 80, 178, 230, 269
Root, **25, 33**
  mycorrhizal formations, 46, **47**
Root knot, 440–444, **441, 442, 443**
Root rot, 409–425
  mushroom, 416–420, **417, 418, 419, 420**
  of cotton, 40, 317–325, **317, 318, 320, 323**
  of tobacco, 37, 38, 269–274, **270, 271, 272**
  ozonium, 317
  Phymatotrichum, 317–325, **317, 318, 320, 323**
  Texas, 317
Rose
  Anguillulina dipsaci, **445, 446, 447**
  crown gall, 197, **198**
  Pseudomonas tumefaciens, 197, **198**
  stem nematode, **445, 446, 447**
Rotation, 72, 73
Roundworms, 439
Rugose mosaic, 214, 225
Rushes, 43
Rusts, 3, **35**, 41, **42**, 44–46, 48, 50, 113, 367–409
Rusts—*Continued*
  aeciospore, 35, 43, 369, 370
  asparagus rust, 40, 389–393
  black stem rust, 14, 37, 43, 371, 372–379
  cedar-apple rust, 43, 44, 393–400
  cereal rusts, 33
  coffee rust, 10
  crown rust, **42**, 44, 371, 379–389
  epiphytotics, 48, 50
  field characteristics, 370–372
  flax rust, 39
  obligate parasites, 32, 369
  promycelium, 369, 370
  spermatium, 369, 370
  sporidia, 369
  teleutospore, 43, 369
  uredospore, 35, 43, 369
  white pine blister rust, 400–409
Rutabaga
  Actinomyces scabies, 201
  Bacillus carotovorus, 170, **172**
  black rot, 194, **196**
  soft rot, 170, **172**
  potato scab, 201
  Pseudomonas campestris, 194, **196**
Rye, **109**
  Claviceps purpurea, 274–279, **276, 278**
  ergot, 5, 6, 274–279, **276, 278**
  leaf rust, 371–372
  powdery mildew, 293
  Puccinia graminis, 41, 371, 378
  Puccinia rubigo-vera var. *secalis*, 371–372
  stem rust, 371, 378

Salsify
  Bacillus carotovorus, 170, **172**
  soft rot, 170, **172**
Salt, 379
Sandalwood tree, 426, 427
  spike disease, 209, 214
Sanitation, 73–75
Saprophytism, 31, 32
Scab
  apple, 49, 50, 56, 82, 96, 111, 283–289, **283, 284, 285, 287, 288**
  cherry, 283
  common scab of potato, 53, 54, 72, 201–205, **202, 203, 204**
  peach, 37
  pear, 283
Scald of apples, 27, 450, 453–455, **454**
Schizomycetes, 27, 159
Schoeller, 368
Sclerospora graminicola, **24**
Sclerotia, 93

Sclerotinia fructicola, 256–263, **257, 258, 259, 260, 261**
Sclerotinia intermedia, 263
Sclerotinia minor, 263
Sclerotinia sclerotiorum, 263
Sclerotinia spp., 63, 263–269, **264, 265, 267**
Sclerotium cepivorum, 52
Scolytus multistriatus, 306
Scolytus scolytus, 306
Scrophulariaceae, 426
Sedges, 43
Seed, 49
  certified, 81
  disease-free, 80, 81
  plants as disease agents, 426–438
  seed-borne pathogens, 80, 81, 93, 225, 229, 343
  selection, 80, 81, 316
  treatment, 93–101, **95, 97, 98,** 274, 316, 329, 335, 343, 354, 359, 363, 367, 416
Seedlings
  damping-off, 32, 40, 55, 63, 70, 77, 121–126, **122, 123, 125**
Selection, 110–112
Semesan, 97
  New Improved Semesan Jr., 98
  Semesan Jr., 98
Sereh disease, 223, 228
Serum, 39, 40
Sex organs, 3
Shallots, 356
Shepherd's purse, 46
Signs of disease, 23, **24**
Smith, Erwin F., 160, 197, 207
Smut, 3, 18, 41, 44, 113, 341–367
  bunt of wheat, 360–364
  corn, 343–350
  flag smut, **39**
  loose smut
    of barley, 37, 367
    of wheat, 364, 367
  oat, 64, 65, 350–355
  onion, 355–359
  sorghum, 99
  stinking smut of wheat, 360–364
Smudge, onion, 38
Sodium bicarbonate, 103
Sodium borate, 459
Sodium cyanide, 78, 444
Sodium fluoride, 102, 103
Soft rot
  carrots, 70, 170–178, **171, 172, 173, 174, 175, 176, 178**
Soft rot—*Continued*
  sweet potatoes, 62, 117–121, **118, 120**
Soil, 49, 51–56, 60, 70, 75–78
  biological condition, 51, 55
  chemical condition, 51, **54**
  pasteurization, electrical, 77
  physical condition, 51, 55
  sterilization, 75–78, **76,** 268, 274, 325, 359, 444
Soil-inhabiting pathogens, 56, 97, 99
Sorauer, 18
Sore-shin of cotton, 66, 67
Sorghum
  smut, 99
  sugar cane mosaic virus, 238
Sow thistle, 248
Specialization, 33, 40, 41, **42**
Species, 33, 40, 41
  selection for disease resistance, 110, 111
Spermatium, 369, 370
Spermogonium, 369
Sphaerotheca humuli, 39
Sphere of health, 22, **23**
Sphere of life, 22, **23**
Spike disease of sandalwood, 209, 226
Spinach
  blight, 209, 215
  curly top virus, 240
  Sclerotinia spp., 263
Spindle tuber, 62, 210, 213, **214**
Sprochaeta, 160
Spirochaetales, 160
Spongospora subterranea, 152–158, **153, 154, 155, 156, 157**
Spores, 23, 43, 49, 52, 54, 56–58, 81, 93, 342, 369, 370
Sporidia, 342, 369, 370
Spray burn, 27, 101
Sprayers, **82**
Spraying, 81–93
  effectiveness, 81
  thoroughness, 83
  timeliness, 81, 82, 83
Spruce
  damping-off of seedlings, 121, **124**
  Pythium debaryanum, 121, **124**
Squash
  Bacillus tracheiphilus, 186
  curly top virus, 240
  wilt of cucurbits, 186
Stalk fungi, 341–425
Steam, 76, 325, 444
Stellaria media, 221
Stem nematode, 444–448, **445, 446, 447**

Stem rot
cucumbers, 63
potatoes, 173
Stem rust, 43, 44, 48, 57, 58, 68, 108, 109, 371, 372–379, **373, 374, 375, 377**
barley, 37, 371, 378
oats, 371, 378
rye, 371, 378
wheat, 14, **109,** 371, 372–379, **373, 374, 375, 377**
Sterilization of soil, partial, 75–78, **76,** 325, 359, 444
Sterocide, 98
Stewart's disease of corn, 55, 110, 189–194, **190, 191, 192, 193**
Stinking smut of wheat, 360–364, **361, 362**
Stomata, 35, 37, 62, 63
penetration, **34,** 35, 62, 63
Stone fruits, 58
Stowell's Evergreen Corn, 110
Strains, 33, 40, 41, 43, 112, 113
Strangleweed, 429
Strawberries
Bacillus amylovorus, 163
fire blight, 163
Rhizopus nigricans, 117, 118
soft rot, 117–118
Streak disease in corn, 225, 226
Stripe rust, 371
Stroma, 250
Sub-species, 40
Sudan grass, 238
Sugar beet, 2, 11, **34,** 328, 329
black scurf, 414
boron deficiency, 455–459, **456, 457**
Cercospora beticola, **34,** 57, 62, 63, 70, 71, 72, 325–329, **326, 327, 328, 329**
Corticium vagum, 414
curly top, 2, 11, 62, 209, 221, 240–245, **239, 240, 241, 242, 243**
damping-off, 121, **124**
leaf spot, 62, 63, 72, 325–329, **326, 327, 328, 329**
Phoma betae, 455, 456
Pythium debaryanum, 121, **124**
Sugar cane
galls, 214
mosaic, 212, 230, 235–240, **236, 237**
Sugar corn, 40
Sulphur, 68, 77, 78, 84–87, 205, 292, 297, 301, 324, 379, 393, 400
Sulphuric acid, 77, 78, 185, 324
Sumac, **430, 431**
Sunlight, 51
Sunscald, 450
Susceptibility, **35,** 36, 38, 41, 51, 59, 63
Sustained losses, 14–16
Sweet corn, 40, 110
Bacterium stewartii, 55, 189–194, **190, 191, 192, 193**
Stewart's wilt, 55, 110, 189–194, **190, 191, 192, 193**
Sweet potato
Rhizopus nigricans, 58, 63, 117–121, **118, 120**
seed treatment, 98, 99
soft rot, 62, 63, 117–121, **118, 120**
Swiss chard, 325
Symbiosis, 47
Symptoms, 23, **24, 25,** 27–40, 61, 62
Synchytrium endobioticum, **30,** 39
Synthetic activities, 1

Take-all of wheat, 70
Taphrina deformans, 298–301, **298, 299**
Teasel
Anguillulina dipsaci, 444, 446, **447**
stem nematode, 444, 446, **447**
Teleutosorus, 369
Teleutospore, 43, 58, 370
Temperature
air, 51, 55–58, **57, 59,** 60–62
cause of disease, 27, 449
in control, 70
soil, 52, **53,** 59, **60, 61**
Temporary control measures, 70–104
Teosinte, 143, 189
Texas root rot, 317
Thallophytes, 27
Theophrastus, 4, 68
Thielavia basicola, 55, 269–274, **270, 271, 272**
Thielavia root rot, 37, 38, 52, 269–274, **270, 271, 272**
Tilletia levis, 54, 360–364, **361, 362**
Tilletia tritici, 54, 360–364, **361, 362**
Timber rot, 409
Timothy, 41, 378
Tip burn of lettuce, 27
Tobacco
boron deficiency, 456
broomrape, 427
damping-off, 121, **124**
Heterodera marioni, 440, **442, 443**
mosaic, 39, **216, 217, 218,** 223–224
common, 230–235, **231, 232, 233**
Orobanche, 427
Pythium debaryanum, 121, **124**
ring spot, 39

Tobacco—*Continued*
root knot, 440, **442, 443**
root rot, 37, 38, 52
Thielavia basicola, 55, 61, 269–274, **270, 271, 272**
Thielavia root rot, 37, 38, 269–274, **270, 271, 272**
Tomato
Bacillus carotovorus, 170, **172**
black bread mold, 32
black scurf, 414
blossom-end rot, 27, 63, 450
broomrape, **428**
Corticium vagum, 414
crown gall, **198**
curly top virus, 210
Fusarium bulbigenum var. *lycopersici*, **54**
Heterodera marioni, **441, 442, 443**
late blight, 126
leaf roll virus, 220
mosaic, 223, 225
Orobanche, **428**
Phytophthora infestans, **37**, 126
powdery scab, 152
Pseudomonas tumefaciens, **198**
root knot, **441, 442, 443**
Sclerotinia spp., 263
seed treatment, 100
soft rot, 170, **172**
Spongospora subterranea, 152
tomato wilt, **54**, 56
western yellow blight, 240
Trichoderma lignorum, 55
Trillat, 19
Tuberculosis, 1
Tulip break, 207
Turnip
Actinomyces scabies, 201
Bacillus carotovorus, 170, **172**
black rot, 194, **196**
club root, 147
Heterodera marioni, **442, 443**
Plasmodiophora brassicae, 147
Pseudomonas campestris, 194, **196**
potato scab, 201
root knot, **442, 443**
soft rot, 170, **172**

Uncinula necator, 7, 8, 289–292, **290, 291**
United States Department of Agriculture section of vegetable pathology established, 19
Uredosorus, 369
Uredospore, 35, 43, 50, 370
Uredium, 369
Urocystis cepulae, 355–359, **356, 358, 359**
Urocystis tritici, 39
Uromyces appendiculatus, 44
Uromyces trifolii, 44
Uspulun, 97
Ustilago avenae, 64, 65, 350–355, **352, 353**
Ustilago levis, 350–355, **352, 353**
Ustilago nuda, 100
Ustilago tritici, 100, 364–367, **365, 366**
Ustilago zeae, 343–350, **345, 346, 348, 349**

Vaccination, 39, 40
Variation, 33, 41
Varieties, **35**, 36–43, 78–80, 110–113
Vascular necrosis, **29**, 30, 162
Vector, 226
Vegetables
Bacillus carotovorus, 170–173, **171, 172**
soft rot, 170–173, **171, 172**
Velvet grass, 386
Venturia inaequalis, 111, 283–289, **283, 284, 285, 287**
Verticillium spp. on elm, 304
Vine, 3, 4
Violets
Thielavia basicola, 269
Thielavia root rot, 269
Virus, 27, 35, 39–40, 60–62, 206, 208, 217, 220, 221, 222, **223**, 225
aster yellows, 209, 214, 228, 229, 245–249, **246, 247**
bean mosaic, 39
control, 228–230
cucumber mosaic, 40
curly top of sugar beets, 11, 62, 209, 221, 240–245, **239, 240, 241, 242, 243**
diseases of plants, 206–250
dissemination, 225
peach yellows, 10, 40, 207, 209
potato, 15
properties, 218–219
sugar cane mosaic, 212, 230, 235–240, **236, 237**
tobacco, 39, 223–224, **216, 217, 218**, 230–235, **231, 232, 233**
Viscum, 428
Von Martius, 18

Walnut, 420, **424**
Wandering jew, 229
Wart of potatoes, 39
Water, 27, 35, 36, 51, 449
Watermelon, 11, 12
Bacillus tracheiphilus, 186

Watermelon—*Continued*
Fusarium bulbigenum var. *Nsveum*, **33**, 40, 53, 55, 65, 330–335, **331**, **332**, **333**, **334**, **335**
Heterodera marioni, 440, **443**
root knot, 440, **443**
wilt, 11, 12, **33**, 40, 41, 53, 56, 110–112, 186, 330–335, **331**, **332**, **333**, **334**, **335**
Water molds, 28, 114
Watery soft rot of lettuce, 263
Weed eradication for virus control, 229
Weed seeds, 75
Wheat, 6, 14, 96, 100, **109**
black stem rust, 14, **109**, 371, 372–379, **373**, **374**, **375**, **377**
bunt, 37, 52–54, 60, 96, 99, 360–364, **361**, **362**
Clavsceps purpurea, 275
ergot, 6, 275
flag smut, 39
Gibberella saubinetii, 53, 54, **59**, 60
loose smut, 364–367, **365**, **366**
powdery mildew, 293
Puccinia glumarum, 38, 371
Puccinia graminis, 37, 41, 42, 43, 44, 371, 372–379, **373**, **374**, **375**, **377**
var. *tritici*, 42, 57
Puccinia rubigo-vera var. *tritici*, 42, 371
rosette, 213, 226, 230
scab, 2, 60
seedling blight, 53, 54, **59**, 60
stinking smut, 360–364, **361**, **362**
take-all, 70
Tilletia levis, 54, 360–364, **361**, **362**
Tilletia tritici, 54, 360–364, **361**, **362**
Urocystis tritici, 39
Ustilago tritici, 100, 364–367, **365**, **366**
variety Einkorn, 38
Wheat rosette virus, 213, 226, 230
White heart rot, 420–425, **421**, **422**, **424**
White pine
blister rust, 44, 110, 400–409, **403**, **406**, **407**, **408**
White pine—*Continued*
Cronartium ribicola, 400–409, **403**, **406**, **407**, **408**
White rust of crucifers, 46, 58, 133–136, **134**, **135**, **136**, **137**
on Shepherd's purse, 46
Wild carrot, 248
Wild mustard
black rot, 194, **197**
Pseudomonas campestris, 194, **197**
Willow, 420, **424**
Wilt
aster, **25**
cotton, 41, 336–340
cucumber, **29**, 185–189, **186**, **187**, **188**
elm, 301–308, **302**, **303**, **304**, **305**, **306**
flax, 11, 16
tomato, **54**, 56
watermelon, 11, 12, 40, 330–335, **331**, **332**, **333**, **334**, **335**
Wind, 51
Wisconsin Refugee bean, 39
Witches' broom, 214, 369
Wood preservation, 101–104
Wood rots, 32, 35, 409–425
Woronin, 149
Wound infection, 34, 35

X-bodies, 212, 216

Yellow dwarf
onion, 209, **210**, 211, 223, 229
Yellows
aster, 209, 245–249
cabbage, 52, **53**
peach, 10, 40, 207, 209
Yellow rattle, 426, 427
Yield tendencies and acreage, **15**, **16**

Zea mays, 40
Zinc, 78
Zinc chloride, 102–104, 169
Zinnia, 40
Zoospores, 54