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DISEASES OF FIELD CROPS

BY

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U. S. DEPARTMENT OF AGRICULTURE

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TO MY SON

PREFACE

The purpose of this volume is to present in as brief a space as feasible the important information pertaining to diseases of the field crops. Investigations on the diseases of this group of economic plants and upon the crop plants themselves are continually contributing new facts and modifying former points of view; therefore, a volume of this type can never be complete for the relatively large number of diseases included. Furthermore, investigations on many of the diseases are inadequate and much of the literature deals with limited phases of the diseases. Consequently, this volume is offered not as a complete treatise of subject matter or literature but rather as a convenient reference outline on current information concerning the diseases of this group of crop plants.

The diseases are listed on the basis of the primary cause of the disease under the crop plants included. While many of the diseases occur on more than one crop plant, especially in the cereals and grasses, the economic importance and varietal reactions within the various crop species differ considerably. The rather general tendency toward the specialization of agricultural investigations of crop plants argues further for the presentation of the diseases on this basis as a convenient reference volume for the investigator. The student using the volume as a text in diseases of field crops will acquire, perhaps, a better comprehension of the disease and more stimulation toward individual thinking by reference to several chapters for the information on a given disease. For the convenience of the teacher and as a guide to the student, the diseases are regrouped in the appendix on the basis of (1) the primary causal factor, a suggested list for class presentation on this basis, and (2) a list of the bacteria and fungi arranged by order and family. The body of the text is divided into three sections: (1) diseases of cereals and grasses, (2) diseases of legumes, and (3) diseases of fiber and other field crops. Several crop plants frequently included in field crops, such as field beans, field peas, potatoes, sugar beets, etc., are omitted, as they will be included in "Diseases of Vegetable Crops," by J. C. Walker, to appear in this series of texts. The crop plants are arranged alphabetically within the three sections. The diseases are arranged under each crop plant on the basis of the primary causal factor as follows: (1) nonparasitic, (2) virus, (3) bacterial, (4) phycomycetous, (5) ascomycetous, (6) hypomycetous

(fungi-imperfecti), and (7) basidiomycetous. The detailed discussion of a disease occurring on several crop plants is given under one crop only, and cross reference is made to this discussion in the other chapters.

A uniform plan of discussion of each disease has not been followed because of inadequate information in the case of certain diseases. In general, an attempt has been made to give the geographic distribution of the disease, the symptoms, the primary cause of each one, and the conditions that favor its development and dissemination, and, finally, to discuss palliative and control measures. Considerable emphasis has been given the morphology of the fungi concerned with the diseases, as they, perhaps, are the most reliable key to recognizing the disease and coping with its control. Physiologic specialization and biotypes of the parasites are discussed in some detail as an aid to both the plant pathologist and the plant breeder. Citation to pertinent literature is made with each chapter, and such publications frequently contain references to other papers, as important, possibly, as those cited. The references have been selected not only as the basis of the information used in the text but also as a convenient source for the student seeking more detailed information. Many references have been omitted in instances where good bibliographies have been given in general papers on the diseases. These bibliographical papers are noted after the citation of the article in the reference lists.

This volume has evolved from several mimeographed outlines over a period of more than 20 years: first, class outlines in the department of plant pathology at the University of Wisconsin and, later, several runs of "Outline of Diseases of Cereal and Forage Crop Plants of the Northern Part of the United States," published by the Burgess Publishing Company of Minneapolis, Minn. The facilities of the University of Wisconsin, of the various divisions of the Bureau of Plant Industry, Soils, and Agricultural Engineering of the Agricultural Research Administration in the U. S. Department of Agriculture, and of many state agricultural experiment stations, colleges, and universities have been used in the compilation and preparation of the volume. The book is therefore being offered without author's royalties in order to make the material available to students and investigators in this and allied fields of the plant sciences at the minimum cost.

The volume represents the contributions of many colleagues in mycology, plant pathology, agronomy, and plant breeding who have cooperated generously through conference, correspondence, and supplying data or illustrations. Each chapter has been reviewed by several investigators specializing on certain diseases or upon the maladies of certain crop plants. Although the list of these reviewers is too long to be

included, their cooperation is appreciated and their contributions gratefully recognized. Some of the experimental results included herein are from the unpublished work of specific investigators, quite frequently the reviewers. Where practical, this credit is designated by footnotes, although in many instances the information is involved in statements and discussions and cannot therefore be recognized except by a general appreciation of the cooperation. Appreciation is expressed to the author's wife, Leah A. Dickson, for her suggestions and for reading the manuscript and proof.

In nomenclature, the author has followed the U. S. Department of Agriculture wherever possible. Unfortunately, there is no general agreement nationally or internationally on binomials for the man-made species of plants and animals; to be consistent, therefore, some system must be adopted arbitrarily.

Illustrations contributed by the various cooperators are indicated in the legends. The remainder are from original photographs taken largely by Eugene Herrling of the department of plant pathology of the University of Wisconsin.

Whatever merit this volume may have is due in large part to the cooperation of the many who contributed to its preparation; whatever its deficiencies, the author assumes full responsibility.

JAMES G. DICKSON

MADISON, WIS.,
May, 1947.

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SECTION I

GENERAL INTRODUCTION

CHAPTER I

INTRODUCTION

The history and development of field crops have been associated with plant diseases. Throughout the years, one or another plant malady has threatened the economy of crop production, with the result that the malady was brought under control or the crop shifted to other evironments. Economic pressures frequently have been involved in these adjustments. The history of these battles has been an interesting one, and it has illustrated the ability of man and the adaptability of plants. Undoubtedly the same battle existed in the past when subsistence farming was the problem of the family, although generally the conflict was neither so spectacular nor so broad in its implications. With the advent of commercial farming and the subsequent production of large continuous acreages of the same variety of a given crop and the product's entry into commerce, these adjustments in crop production assumed regional and frequently national or international significance. The history of plant pathology also has been shaped by such conflicts.

Diseases of the cereal crops were among the first studied. The early studies of plant diseases were dominated by dogma and tradition. Knowledge was vague regarding the nature of disease, and the role of microorganisms was unknown. Many of the diseases of crop plants were attributed to weather conditions, frequently a conspicuous factor associated with the unhealthy condition of the crop. Later, when fungi were observed to be associated with diseased plants, they were interpreted, at first, as the result rather than the cause of the unhealthy conditions. For a period, emphasis was placed upon the collection and classification of the fungi. In the latter part of the eighteenth century and continuing into the early part of the nineteenth century, increasing emphasis was placed on systematic mycology. Bulliard, Persoon, Nees von Esenbeck, Schweinitz, Léveille, Fries, and Berkeley contributed much to the early classification of the fungi. During the latter half of the nineteenth century Fuckel, Karsten, the Tulasnes, Corda, Saccardo, Thaxter, and many others advanced the mycological study.

Economic pressures for more food in the heavily populated European countries was indirectly the force that stimulated the investigation of diseases and their control. Prévost and later De Bary proved by experimentation the nature of parasitism, the epidemic character of fungus diseases, the importance of methods of inoculation and infection, and the practical application of disease control. These two investigators founded the science of phytopathology. Kühn was among the first to organize a general, practical attack on the diseases of cultivated crops with special emphasis on disease control. During the latter half of the nineteenth century, publications were numerous. Among many others who contributed special service to plant pathology or indirectly through mycological publications were Brefeld, Frank, Hartig, Schroeter, Sorauer, and Winter in Germany; Oudemans in Holland; Cornu, Millardet, and Prillieux in France; Comes in Italy; Woronin in Russia; Eriksson, Henning, and Jensen in Sweden; Massie, Plowright, and Ward in England; Burrill, Clinton, Farlow, Halstead, Hitchcock, Kellerman, Selby, Swingle, and many others in the United States.

The investigations on diseases of field crops in the United States started early with the development of the U. S. Department of Agriculture and the state agricultural experiment stations. Much of this early work was directed more especially toward immediately practical problems of disease control. Seed treatments, sanitation, disease reaction of varieties of the different cereals, and study of the various fungi constituted the earlier investigations. The rediscovery of Mendel's laws of heredity and their application to genetics soon offered new techniques for disease control. The difference in disease reaction of specific varieties and specialization of the fungus parasites first recognized by Eriksson and Ward and later studied in detail by Salmon, Freeman, Stakman, Reed, and others directed attention to disease resistance as a control measure. Orton and Bolley working with the wilt diseases developed the early techniques and proved the practicability of disease resistance as a control measure. Nilsson-Ehle and Biffin soon after proved that the characters for rust resistance were inherited on a definite factorial basis. The advances in this field during the past three decades have placed disease resistance as of first importance in the control of diseases and the improvement of field crops.

The study of the life cycle of the fungi, especially in relation to the fusion of gametes and the genetical implications of these fusions to variation in pathogenicity, has become increasingly important in the development of basic plant pathology. Knowledge of the mechanism of variation, the rapidity with which new physiological races develop, and means of limiting or controlling them all assume increasing importance with the use of disease resistance as a control measure.

The investigations on the influence of environmental conditions upon the development of the diseases of field crops have expanded largely since Sorauer's early work. The earlier advances in general plant physiology and the study of the normal physiology of cultivated plants led naturally to the study of the abnormal physiology of diseased plants. The environmental complex that predisposes the plant to attack in the case of parasitic diseases or induces abnormal functioning or development in nonparasitic diseases was studied especially by Ward and Blackman in England, by L. R. Jones and associates in the United States, and more recently by many others. The application of special environments to the production of epiphytotics has materially increased the accuracy of the tests for disease resistance and the factorial analysis of resistance. With the more intensive cultivation of field crops the so-called "deficiency diseases" have become of increasing importance.

The virus diseases first studied by Mayer, Ivanowski, and Beijerinck and demonstrated as transmissible by insects by Takami and E. D. Ball are becoming of increasing importance in the pathology of field crops. Improved techniques in transmission and identification of viruses on various plants have demonstrated their presence in most of the cultivated crop plants.

The science of plant pathology is young. New diseases of cultivated plants are being reported. New physiological races of many of the important parasites are being found. Fundamental knowledge concerning the nature of parasitism, the physiology of suscept, parasite, and the disease, the life cycle of the parasite, variability in the pathogen, sources of disease-resistant parental material, the nature of disease resistance, epidemiology, and the action of seed-treatment compounds is increasing rapidly. Therefore, a text on diseases of field crops, including a wide group of cultivated plants, cannot be completely up-to-date on all phases of the subject. The organization of the factual information, a systematic discussion of the research reported in the numerous publications, and listing of the more important reference material appears essential if plant pathologists, plant breeders, and agronomists are to continue rendering the maximum service in the development of field crops.

CHAPTER II

PHYSIOLOGICAL ANATOMY OF PLANT GROUPS IN RELATION TO DISEASE

Disease development and, conversely, disease resistance are determined in part by morphological structures and physiological processes in the plants involved. The morphology and physiology of the plant are governed by the genetic complement (genotype) operating within the plant structures (phenotype) in the external environment. In other words, the expression of the complex of genetic factors through structure (morphology) and function (physiology) are conditioned within limits by the external environment. The importance of a basic understanding and experienced familiarity with the physiological anatomy of the plants upon which the disease occurs is essential in the comprehension of disease development and in the application of disease control. In the nonparasitic diseases, the investigator is concerned with the plant and its reaction to excesses or deficiencies in the environment. In the diseases caused by bacteria and fungi, the interplay of two plants, the suscept and parasite, and the conditioning environment are involved in disease development. Or stated in more detail, the potential capacity of the bacterium or fungus to incite disease is dependent upon genetic constitution expressed through morphological structures and physiological processes and the environmental complex, including the suscept, under which the potential parasite is developing. Likewise the susceptibility, tolerance, or resistance, of the suscept is conditioned by a similar complex. The presence of natural avenues of entrance for the parasite, the existence of tissue barriers, the type of embryonic development, the rate of tissue maturation, and the presence or absence of suitable nutrient complexes or single compounds are illustrative of a few of the anatomical and physiological factors that determine the course of disease development, economic importance, and control. Therefore, it is essential that the plant pathologist be familiar with the physiological anatomy of the suscepts as well as the morphology and physiology of the parasite.

1. Physiological Anatomy of the Gramineae.—The developmental anatomy of the cereals and grasses is associated with disease development and type of disease in several phases of growth and maturation. Space does not permit a comprehensive discussion of the subject. However, reference should be made to Avery (1930), Haberland (1914), Hayward

(1938), McCall (1934), Percival (1921), and others for the developmental anatomy of the Gramineae.

The mature grain is a caryopsis. The kernel either threshes free from the floral bracts as in most wheats, or it is enclosed in the lemma and palea, as for example, in barley and oats. The caryopsis consists of the adherent pericarp and remains of the integuments and nucellus, the protective tissues enclosing the endosperm, and the embryo embedded against the endosperm. A semipermeable membrane of lipoid composition is deposited on the outer surface of the nucellar tissue and the inner epidermis of the inner integument. At maturity the membrane is continuous on these compressed residual tissues and extends through the conductive tissue of the chalaza. The membrane (cuticle) over the back of the scutellum is joined with this semipermeable membrane, sealing in the epithelial surface in contact with the endosperm (Fig. 1). The role of this differentially permeable membrane system is discussed by Brown (1907), Dickson and Shands (1941), Tharp (1935), and others. The pericarp tissues when moist constitute a suitable medium for many of the fungi parasitic on the cereals and grasses. The semipermeable membrane functions not only in holding soluble reserves within the kernel, but also as a barrier to prevent fungi and bacteria in the pericarp entering the endosperm and embryo, as described by Johann (1935), Pugh *et al.* (1932), and others. Mechanical injury of the membrane results in rapid invasion of the endosperm reserves and embryonic tissues by soil microorganisms during the early stages of germination. Parasites invading through the ovary wall, as the fungi causing ergot and the loose smuts, must enter early after pollination or this avenue is barred. The erect position and compact nature of the spike in many of the Gramineae tend to increase fungus and bacterial invasion of the pericarp as free moisture is held in the floral bracts enclosing the caryopsis, resulting in conditions favorable for invasion of the basal portion of the kernel by blight and rot organisms. In grain enclosed permanently in the floral bracts, the lemma and palea provide a protective covering for spores and mycelium held between the pericarp and bracts. The inoculum in this position is in close proximity to the embryonic tissues of the young seedling, and infection occurs before the tissues become resistant. The attached floral bracts protect the seed-borne inoculum from the direct action of fungicides, as trapped air prevents complete wetting of the inner surfaces. Consequently, the volatile compounds are more effective as seed fungicides in such grains than toxic substances acting by direct contact of the solutions with the fungi and bacteria.

The physiological anatomy and development of the seedling of the Gramineae frequently is favorable for disease development. The anatomy of the seedling and differences in development in the various

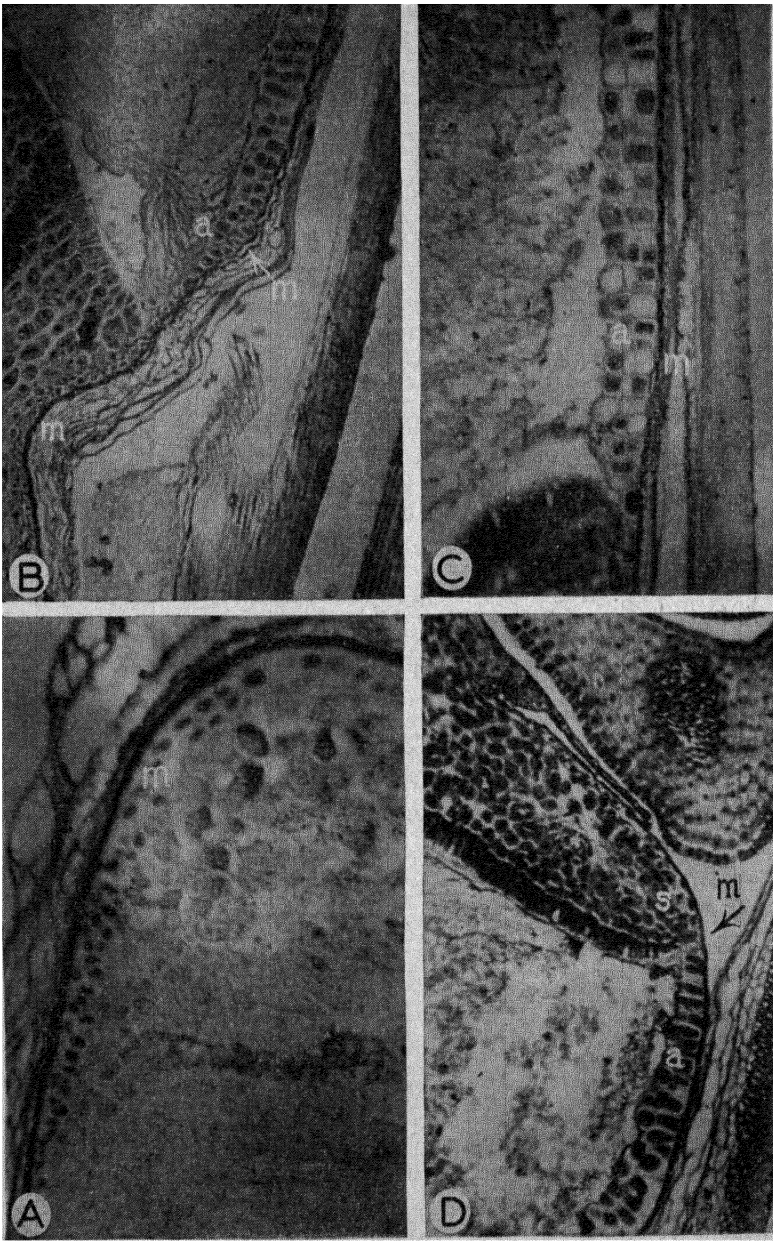

FIG. 1.—Longisections and transection of a barley kernel showing the semipermeable membrane enclosing the endosperm and joining with the surface membrane on the back of the scutellum. (*A*) The heavy membrane (*m*) enclosing the distal end of the kernel. (*B*) The membrane (*m*) on the crease side of the kernel along the base of the scutellum. (*C*) The membrane (*m*) on embryo side of the kernel along the apex of the scutellum. (*D*) Transection showing membrane (*m*) along aleurone cells (*a*) and the back of scutellum (*s*).

genera of the family are discussed fully by Avery (1930), McCall (1934), Sargent and Arber (1915), and others. Brown (1936), Jones *et al.* (1926), Kolk (1930), Pearson (1931), and others describe the avenues of entrance and tissues invaded as well as the physiology of disease development. The composition and maturation of seedling tissues, especially as influenced by environment, are important in relation to disease development and the expression of resistance to seedling blights, seedling smut infection, and other diseases. The emergence of the seminal roots by digestion and mechanical pressure through the coleorhiza and especially the cortex of the lower internode and the cotyledonary node offer avenues of entrance to cortical-invading fungi. This is especially significant in the seedling type illustrated by corn, in which internodal elongation occurs below the coleoptilar node or in tissues unprotected by the coleoptile (Fig. 2).

The role of the coleoptile as a protective sheath is important. Seedling infection by the smut fungi is largely through the coleoptile while it is very young. The tissues soon mature and prevent entrance into the enclosed growing point. The formation of crown and tiller primordia early in the seedling development is important in the establishment of the "systemic" type of infection common in the cereal and grass smuts.

The structure and development of the basal tissues of the culm or crown are important in the development of crown rot, foot rot, and stalk rot diseases. In winter cereals and perennial grasses the crown is the important structure associated with winter killing. The crown consists of secondary culms arising from axillary buds at the basal nodes of the main axis and lateral culms. Under ordinary environments this complex of branches occurs in the upper inch of soil. In certain of the perennial grasses, new culm primordia that develop from the axillary buds require one or two seasons before culm elongation occurs (Bond, 1940). This delayed culm development apparently is associated with the time interval between infection and spore development in certain of the grass smuts. The crown roots arise from the pericyclic region of the first internode and in the intercalary meristems at the base of the lower internodes. The crown roots rupture the cortex by pressure and digestion. If wound response and deposition of suberin, lignin, or lipoid substances in the adjacent cortical cell walls is delayed, the root ruptures offer avenues of entrance for soil-infesting organisms. Insect injuries in these tissues also offer channels of entrance of crown rot parasites. Stuckey (1941) discusses seasonal growth of the grass roots, and Weaver (1926) published good descriptions of the root systems of the cereal crops.

The anatomy and development of the culm is such as to function in protecting the primordia and embryonic tissues. Newton and Brown (1934), Griffiths (1928), Zehner and Humphrey (1929), and others have

shown that introduction of spores of rust fungi and of the corn smut fungus into the differentiating embryonic tissues results in a general

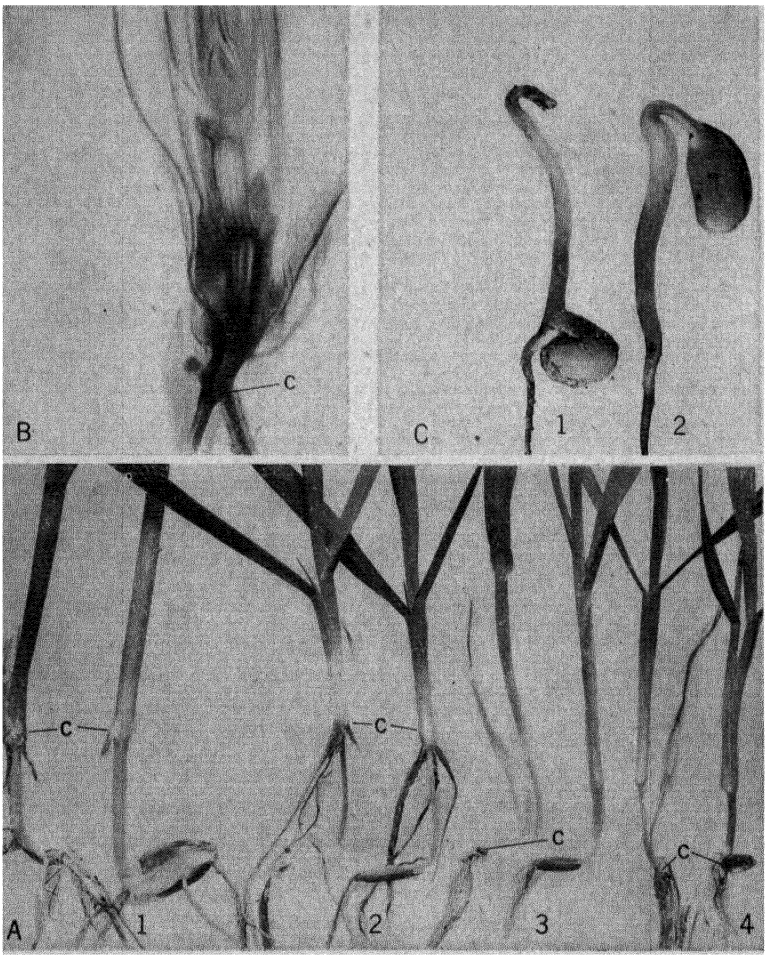

FIG. 2.—Seedling development and internodal elongation in cereals and legumes. (*A*) Seedlings of corn, oats, barley, and wheat illustrating the two common types of internodal elongation in relation to the coleoptilar node (*c*): left, corn (1) and oats (2) in which the internode between the cotyledonary and coleoptilar nodes elongates leaving the internode unprotected by the coleoptile; right, barley (3) and wheat (4) in which the internodal elongation is above the coleoptilar node or enclosed within the protective sheath. The coleoptile is split and pulled aside in the left-hand seedling of each pair to show the base and the internode enclosed. The development and position of seminal and crown roots is shown also. (*B*) Longisection of the crown of the oat seedling. (*C*) Pea (1) and soybean (2) seedlings illustrating the two types of seedling development in the Leguminosae.

heavy infection. Inoculum entering the funnel at the top of the leaf whorl frequently comes in contact with embryonic or susceptible tissues and results in infection. The leaf sheath with the ligule tightly encircling

the culm at the juncture of the sheath and the base of the leaf blade prevents inoculum from reaching the younger tissues of the basal part of the culm internode and the axillary bud. The position of the leaf blade and the basal region of elongation frequently result in leaf blotch diseases, killing the entire blade.

The type of inflorescence and development of the reduced flowers varies in the family. Generally the compact spike is damaged more by blights and rots than the open panicle. The floral bracts protect the young ovary from flower-infecting organisms. The cross-pollinated species in which the floral bracts are open longer are damaged more by ergot than the self-pollinated species. Humid, wet weather prolongs the period of open floral bracts in both self- and cross-pollinated species, as well as providing conditions favorable for germination and infection by the parasite and, therefore, usually results in higher infections with ergot, loose smut, and kernel blights. Dead anther, pollen, and stylar tissues all furnish excellent nutritive media for the initial development of many of the blight and rot fungi, and these organisms, once established as saprophytes, invade the adjacent living tissues by contact infection to produce disease.

The development of exterior and interior membranes in association with tissue maturity or wound response are important in water economy, gaseous exchange, and the invasion of parasitic organisms. The development of such membranes in the Gramineae is not greatly different from that in the other families of crop plants. Therefore, the discussion of this important phase of physiological anatomy and that of natural openings in relation to disease development will be presented later in the chapter.

2. Physiological Anatomy of the Leguminosae.—The leguminous crops included in the discussion of field-crop diseases are largely perennial or biennial in growth habit. The annual crop plants of this family differ somewhat in root and stem development from the perennial or biennial types. However, anatomically, they are sufficiently similar to be included in the general discussion for this family. Compton (1912), Hayward (1938), Jones (1928), Lute (1928), Martin (1914), Pammel (1899), Wilson (1913), and Winton (1914) discuss various phases of the structure and morphology of this group.

The seeds are borne in pods, the latter varying in shape and structure. The pod tissues frequently are invaded by fungi. The infections in many instances extend through the wall, rotting the pod and enclosed seeds or resulting in seed infection. The epidermis of the seed in most members of this family consists of a row of palisade cells varying in cell-wall thickness, permeability, and durability in different species and under different environments. Apparently the walls of the palisade cells

function in a manner somewhat similar to the semipermeable membrane in the caryopsis of the Gramineae. Mechanical injury such as scarification overcomes the impermeability but frequently increases seedling loss by cracking the seed coat. Breaking the seed coat in harvesting and handling the seed increases the losses from seedling blights during the early stages of germination. The seed at maturity consists of the two cotyledons in which the reserves are stored and the embryonic epicotyl and hypocotyl all enclosed within the integuments. Parasites established in the seed coat or in the cotyledons or gaining entrance to the young seedling through cracks in the seed coat during the very early stages of germination cause severe damage.

In germination, the primary root emerges near the hilum or in the microphylar area. In the subsequent development, two types of germination are represented in the family: (1) epigeal, in which the cotyledons, usually enclosed within the seed coat, are pushed above the soil surface by the rapid elongation and straightening of the hypocotyl; (2) hypogeal, in which the hypocotyl elongation is limited, the cotyledons remain in position within the seed coat in the soil, and the epicotyl, more fully differentiated prior to germination, pushes up through the soil. In this latter type of development, seedling blight and lesions below the soil level are the common type of disease. In the former, seedling blight during the very early stages of germination occurs followed by a later lesioning or "damping-off" from invasion of the hypocotyl near the soil surface or by a blighting of the embryonic epicotyl. Disease lesions on the cotyledons develop, and fungus sporulation frequently is abundant.

Root development consists of a primary tap root with numerous laterals. Weaver (1926) illustrated the root system of several members of this family. Elongation, branching, and secondary thickening of the root system is rapid and rather continuous during the first growing season. As described by Fred *et al.* (1932), nodule formation is abundant during this period of active root growth. Secondary cambial roots develop during the following season. In both biennials and perennials, noncambial smaller root branches develop in the spring of the second and in the succeeding years in the case of perennials, as described by Jones (1943). These apparently function in increasing absorbing area during the period of rapid spring growth and deteriorate during the summer to reappear in limited numbers in the fall. Decay is severe in these transient roots during the summer period, at least in the vicinity of Madison, Wis.

Jones (1928), Jones and McCulloch (1926), Jones and Weimer (1928), Weimer (1927), and others have described winter injury in the older roots and crown and the relation of these injuries to disease development, especially in alfalfa. The capacity of the plant to recover from such

injuries through the formation of phellogen and periderm or by wall thickening and the deposition of suberin-like substances into the walls is related to both crown rots and bacterial-wilt development.

The crown in the older plants consists of the basal stem branches and the axillary and secondary buds. The development of the crown is influenced by environmental conditions and age of the plant. Diseases also modify the number of buds developing as in crown wart and witches'-broom of alfalfa. The annual stems developing from the crown buds are generally angular in shape with a fairly large outer layer of collenchyma tissue. Secondary thickening and changes in turgor in the collenchyma frequently result in longitudinal splitting of the epidermis. These openings unless rapidly closed by periderm serve as avenues of entrance for fungi and bacteria.

The stem elongation and development of the inflorescence in many legumes is affected by insects. Species of aphids and leaf hoppers cause severe damage through retarded apical bud development and yellowing of the foliage. The blighting of the flower buds and dropping of flowers is associated with these and other insects.

3. Physiological Anatomy of Other Dictoyledonous Plants.—The physiological anatomy of cotton, flax, and hemp is discussed in detail by Hayward (1938). As in the legumes, seedling structure and development is associated closely with disease development. Both seed-borne and soil-borne organisms cause blighting, lesioning, and damping-off during early seedling development. In cotton and flax, the structure, composition, and development of the seed is associated with seed infection and damage by microorganisms. The industrial quality of the fibers is frequently damaged by disease in the more humid climates. Conant (1927), Jewett (1938), and Johnson (1924) in the discussion of tobacco diseases gave consideration to structure and development of the plant in relation to disease.

4. General Discussion of Plant-defense Mechanisms.—The crop plants in economical production are, generally speaking, the result of long years of selection and adaption to environment. In the relatively short-lived herbaceous plants, the reaction to major diseases plays an increasingly important role in the selection and survival of a variety. The cost of protective fungicidal sprays and dusts is too great in field-crop economy to permit their general use; therefore, crop-plant development is based upon selecting disease-escaping, disease-tolerant, or disease-resistant varieties. These adaptations in plants generally are associated with gradual or abrupt changes in morphology and physiology. The nature of these changes, their dependence upon the general or more specific genetic composition of the plant for their stability, and the relation of composition and physiology of the plant as a whole or of

specific tissues for their expression are important factors in the economy of the plant species or variety. The difference between susceptible and resistant plants may not be primarily dependent upon a particular structure or substance, but rather, upon the more basic metabolic or maturation processes or upon some inherent quality or characteristic that functions even more directly. Certain of these morphological and physiological characteristics apply more or less generally to all plant species, and, therefore, they are presented briefly at the end of this chapter.

Natural openings are important avenues of entrance for certain organisms producing disease in plants. In field crops, the stomata are probably the most important, as hydathodes and lenticels are less extensively developed than in other plant groups. The rust fungi constitute the large group of parasites entering the tissues by way of the stomata. Although extent of infection is associated perhaps with stomatal size and the period the stomata are open, as shown by Hart (1929, 1931) and Peterson (1931), the more basic type of resistance to the rusts is associated with cellular physiology and composition.

The rate and character of cuticle formation on the epidermal surfaces of the plant are important in disease development. A relatively large number of fungi are capable of penetrating the young cuticle. In general, the resistance to mechanical penetration increases with the maturation and thickening of this surface membrane. Priestley (1921, 1943) summarized the literature and suggested the aggregate nature of cutin and suberin. Blackman and Wellsford (1916), Brown and Harvey (1927), Dey (1919), Leach (1923), Young (1926), and others have studied the penetration of the cuticular membrane by fungi. This surface membrane is important in the water econony of the plant, as discussed by Maximov (1929) and Priestley and coworkers (1922, 1923, 1930). The cuticle and surface structures, hairs, etc., prevent wetting and uniform distribution of water over the exposed tissue surfaces. This phenomenon is important in distribution of inoculum and entrance through natural openings as well as direct surface penetration by plant parasites. Apparently the quantity and quality of cuticle formation is associated with the physiological maturity of not only the epidermal cells, but also those of the underlying tissues. The deposition of the similar aggregate of substances, suberin, on or in the walls of cells within certain tissues is associated with the physiology of the cells.

Suberized cell walls function not only in water economy, but also in retarding or checking the advance of parasitic organisms. Periderm, which serves as a protective tissue after the loss of epidermis and cortex, contains this suberin aggregate. The periderm, whether derived from phellogen of pericyclic origin, secondary phloem parenchyma, or from the subepidermal cortical parenchyma, is similar in its function and the

general presence of suberin. The endodermis is impregnated with suberin as the roots and underground stems mature. The radial and end walls of the endodermal cells are suberized first, followed frequently by suberization of the tangential walls. Collenchyma and sclerenchyma cell walls frequently contain suberin as the tissues approach maturity. Lignification also is commonly associated with many of these thickened walls. The development of these protective and strengthening tissues occurs in the sequence of tissue differentiation, development, and maturation. Environment, however, influences the rate and frequently the extent of wall thickening.

The rate and type of thickening of cellular structures is activated frequently by wound response. The regular sequence in development of these anatomical structures apparently is hastened and expanded by mechanical injuries, invasion of parasites, etc. Such injuries usually stimulate cell-wall thickening and suberization, periderm formation, or ultimately both, in cells of cambium, parenchyma, pericycle, and less frequently collenchyma. An active type of defense mechanism is stimulated in such responses. The role of such a defense mechanism is described by Conant (1927), Dickson *et al.* (1923), Fellows (1928), Jones (1928), Pearson (1931), Weimer and Harter (1921), and others. The response in the corn root by the deposition of suberin-like substances in the cortical cells around secondary-root ruptures and the thickening of the endodermis and suberization of cells of the pericycle and cortex in advance of fungus invasion illustrate this type of reaction (Fig. 3).

The composition of the cell walls and the middle lamellae affect the rate of advance of the parasite and tissue necrosis. The changes in composition associated with maturation of many tissues is correlated apparently with susceptibility or resistance to disease. Harlow (1932), Ritter (1925), and others suggested the change in composition of the middle lamella, in woody tissues at least, from pectin-like compounds in the embryonic state to lignin upon maturity. Brown (1915, 1916, 1917), De-Bary (1886), Hawkins and Harvey (1919), Jones (1909), Pearson (1931), and Ward (1888) discussed the general physiology of intercellular advance of fungi and bacteria in both vegetative and storage tissues. Gäuman (1927), Ritter and Fleck (1926), Sponsler and Dore (1926), and others suggested that the degree of hydration of the cellulose molecular aggregates and the associated substances, lignin and suberin, is correlated with tissue maturation. The physical and chemical changes in the cell membranes are a direct or indirect response of the cell protoplasm. The rate of these changes is influenced by the genetic constitution and the influence of the environment upon the plant metabolism.

The physical and chemical properties of the cell protoplasm are associated directly with plant-defense mechanisms. Cold resistance and

drought resistance are due partly to the composition and physical state of the cell protoplasts, as discussed by Akermann (1927), Harvey (1935), Levitt (1941), Maximov (1929), and Newton and Martin (1930). Specific compounds existing in the plant cells or more commonly present in less active molecular combinations are important in the defense mechan-

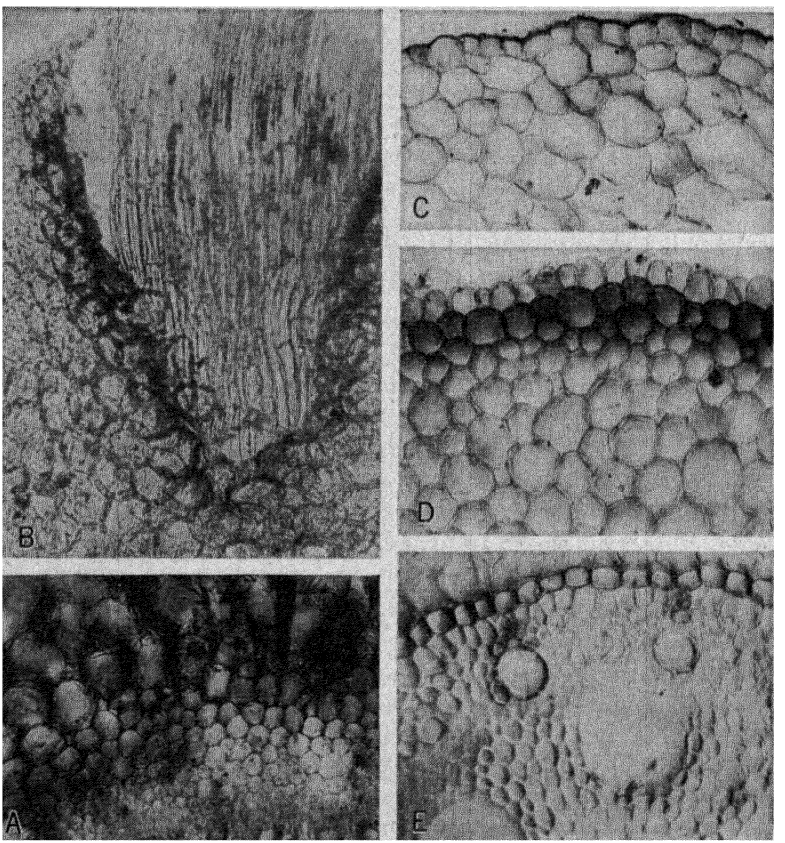

FIG. 3.—The deposition of suberin-like materials in the cells of cortex, pericycle, and endodermis of the corn seedling. (*A*) The reaction of cells of cortex and pericycle in "corking off" a fungus. (*B*) The reaction of cortical cells in "corking off" a root rupture in the internode above the scutellar node. (*C—E*) The reaction to Sudan IV of cells of epidermis of subcoleoptilar internode (*C*), cortex (*D*), and endodermis (*E*) of mature seedling root. The cells with the suberin-like deposition stain red with Sudan IV in dilute sulfuric acid.

ism of the cell, as discussed by Walker (1924, 1929, 1941). The cell protoplasts are concerned directly in the nutrition of the obligate parasite. In this group of parasites the hyphae or specialized branches, haustoria, enter the cells of the suscept and function in a balanced type of metabolism during the cycle of fungus development. The living cells of the suscept, apparently influenced by the stimulus of the parasite, supply the

essential nutrients for the balanced metabolism of both. In the obligate parasite, as illustrated by the rusts and powdery-mildew fungi, this is accomplished at the expense of the suscept. In the symbionts, as in the nodule bacteria, there is a mutual beneficial relationship. Parasitism of this type is usually very specialized. Physiologic races of the fungi are restricted to species or even varieties in the plants capable of functioning in this compatible physiological or nutritional relationship. Conversely, disease resistance is concerned directly with the incompatible relation of the two protoplasts. The fungus hyphae coming in contact with the suscept cells or protoplasm either cease to develop further, or, more commonly, the protoplasts of the contacted cells of the suscept are disorganized, leaving the parasite isolated from living suscept tissue. This response is described by Allen (1923, 1926, 1927), Gibson (1904), Humphrey and Dufrenoy (1944), Stakman (1914), Ward (1902, 1905), and others. The development of disease-escaping, disease-tolerant, or disease-resistant plants is concerned with the morphology and physiology of the suscept plants and, in the case of parasitic diseases, the parasite.

The variation of the parasite in its pathogenic characteristics is equally important. The basis of variation in fungi, bacteria, and viruses is still in the exploratory stage of development. Some variation is due to segregation following nuclear fusion, some to mutation, and some to nuclear reassortment. Variation is expressed in physiological changes and less commonly in alterations in morphology. Stakman (1940) summarizes the information on variability in this group of lower plants.

Insect damage and the relation of anatomy, physiology, and composition of field-crop plants to insect attack parallels somewhat that of plant diseases. Leach (1940) discusses the relation of insects to the transmission of diseases and gives a good summary of many types of insect damage. Metcalf and Flint (1939) present some of the more important insect problems of field crops. Resistance to insect attack and damage is important in field-crop economy as well as in disease control.

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SECTION II

DISEASES OF CEREALS AND GRASSES

CHAPTER III

BARLEY DISEASES

The cultivated barleys comprise chiefly two species: *Hordeum vulgare* L., six-rowed, and *H. distichon* L., two-rowed barleys. Varieties of the former species are grown more extensively than the latter. In the genus Hordeum, as in Triticum and Avena, the basic chromosome number is seven pairs. Multiples of this basic number occur in the wild species. The cultivated barleys all have seven pairs of chromosomes. Barley has been used extensively in the study of linkage relations, including resistance to certain diseases, as summarized by Robertson, Wiebe, and Immer (1941).

The cultivated barleys are grouped into three classes based on the character of growth: winter, intermediate, and spring. Winter and spring barleys comprise the major economic groups (Åberg and Wiebe, 1946). The winter barleys are not winter hardy; therefore, they are grown in a belt extending east and west across the United States approximately south of 42° latitude. They occupy similar areas with mild winters throughout the world. In the extreme southern United States and especially the Southwest, fall-sown spring barleys predominate. Spring varieties occupy the important barley acreage in the northern United States and Canada and similar areas throughout the world.

Barley is grown under a wide range of environmental conditions, chiefly for grain, although it is used as a forage crop in limited sections. The crop is most productive in regions of cool climate during the growing season and in well-drained finer silt or clay soils. The wide range in growth characters, period required for development, and barley types largely account for its very extensive distribution throughout world agriculture (Carlton, 1920, Harlan, 1936, and Weaver, 1943).

The developmental anatomy of the barley plant is relatively similar to wheat (Chap. II). The development of brown pigmentation in association with tissue injury and necrosis results in disease symptoms somewhat different from those in wheat and oats.

Barley diseases cause large losses in both yield and quality of grain. Estimated average annual losses in the United States for the 10-year period 1930 through 1939 amounted to 7.1 per cent of the crop, or over

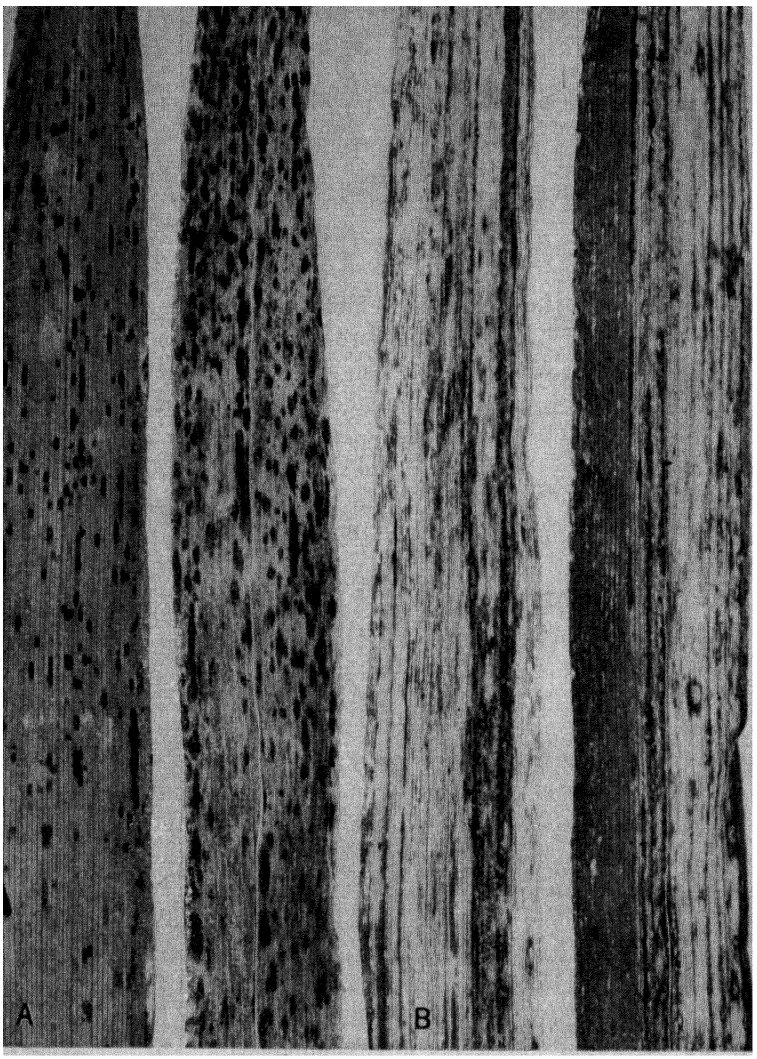

FIG. 4.—Two nonparasitic maladies of barley: false stripe (*A*) and brown spot (*B*).

14 million bushels (Plant Disease Survey). Two years in which scab occurred in epiphytotic form and one of stem rust are included in the 10-year period. Losses from barley stripe are much lower in the United States and Canada than prior to the general use of resistant varieties and organic mercury-seed treatments.

1. Nonparasitic Diseases.—The nonparasitic disturbances in barley are manifest by leaf spotting, striping, or yellowing and frequently the incomplete emergence of the spike from the boot. False stripe and brown spot are the two most common leaf manifestations of the nonparasitic maladies.

False Stripe.—The symptoms appear first as a light-brown linear mottling that frequently develops into stripes with irregular margins (Fig. 4). The necrosis and browning of the cells of the mesophyll of the leaf blade apparently are the only symptoms. The condition usually results in little damage to yields. The leaf stripes are different in character from those of the parasitic stripe disease of barley. The spikes develop free from discoloration and blighting, a condition that further differentiates it from the stripe disease. The conditions causing the malady are not known.

Brown Spot.—Small, brown to black, circular to oblong, dry spots with a regular margin develop on the leaf blades (Fig. 4). Many cultivated varieties and several wild species of *Hordeum* show the spotting. The necrosis occurs, sporadically, at any period from tillering to just prior to maturity. The leaf symptoms frequently occur on otherwise normally developing plants, although they commonly are associated with incomplete emergence of the spikes and twisting of the leaves. This type of spotting is differentiated easily from the parasitic spot-blotch disease of barley. Frequently the symptoms are associated with deficiencies of certain mineral elements, notably boron or copper (Christensen, 1934, Eaton, 1944, Schropp, 1940).

2. Bacterial Blight, *Xanthomonas translucens* (L. R. Jones, A. G. Johnson, and Reddy) Dowson.—The bacterial blights of the cereals and grasses are differentiated into two groups. (1) The bacterial colony is in a gelatinous matrix and advances between the cells of the tissues of the suscept. This group is relatively numerous. (2) The bacterial colony develops without the gelatinous matrix and is localized somewhat in tissue cavities with water soaking and chlorosis of cells surrounding the colony. The bacterial exudate is conspicuous on the lesions in the former and absent in the latter. The bacterial blight of barley is typical of the first.

The bacterial blight occurs widely on many of the economic varieties and some wild *Hordeum* species. A similar disease occurs on the other cereals and many grasses (Wallin, 1946). The disease is distributed widely throughout North America (Jones *et al.*, 1917) as well as northern Europe and Asia (Jaczewski, 1935). Bacterial blight is more prevalent through the spring barley areas where usually it is of minor importance, although local or general epiphytotics on very susceptible varieties are reported as reducing yields of foliage and grain.

Symptoms and Effects.—Small linear water-soaked areas, frequently quite numerous on localized areas of the leaf blade and sheath, develop after several days of rainy, damp weather. These lesions elongate and coalesce into irregular narrow glossy-surfaced stripes. The stripe frequently shows water-soaked, light-yellow, light-brown, and dark-brown regions, depending upon the age of the coalesced lesions (Fig. 5). The center of the lesion is translucent in the later stages of development. Minute drops of white resinous exudate or a thin film of the exudate are characteristic on the lesion surface. Numerous lesions usually result in a slow yellowing and death of the leaf blade progressing from the apex downward. Similar lesions develop on the leaf sheath and floral bracts. Severe late infections usually result in retarded spike elongation and in abundant exudate and blighting of the spike and adjacent tissues. Lesions on the kernels are small and inconspicuous. The characteristic symptom is the narrow, linear, translucent, and glossy lesion.

The Organism.—*Xanthomonas translucens* (L. R. Jones, A. G. Johnson, and Reddy) Dowson[1].
[*Phytomonas translucens* (L. R. Jones, A. G. Johnson, and Reddy) Bergey *et al.*]
(*Bacterium translucens* L. R. Jones, A. G. Johnson, and Reddy)
[*Pseudomonas translucens* (L. R. Jones, A. G. Johnson, and Reddy) Stapp.]

The cylindrical rod-shaped bacteria with rounded ends are motile by means of polar flagella, and no spores are formed. The bacterial colonies develop in a gelatinous matrix. Specialized varieties and races occur on the cereals and grasses (Hagborg, 1942, and Wallin, 1946).

Etiology.—The bacteria enter the young tissues through natural openings and wounds. Advancement in the mesophyl and parenchymatous tissues is between the cells especially when the tissues are water-soaked. First infections occur during the seedling stage early in the spring. The secondary infections occur on the younger tissues throughout the growing season whenever high moisture prevails. The exudate is splashed by meteoric water, transmitted by contact, and carried extensively by insects. Sucking and biting insects are important in dissemination and infection (Leach, 1940). The bacteria imbedded in the exudate remain dormant under unfavorable conditions and resume active development when conditions become favorable. The bacteria remain viable over long periods when dehydrated and sealed in the gelatinous matrix in

[1] Several systems of nomenclature are in use for the bacterial plant pathogenes. The writer has followed the classification presented by Bergey *et al.* (1939, 1946). The reader is referred also to Elliott (1930, 1943) and Riker and Baldwin (1942).

the lesions. The organism is carried from season to season in crop residue and on the seed.

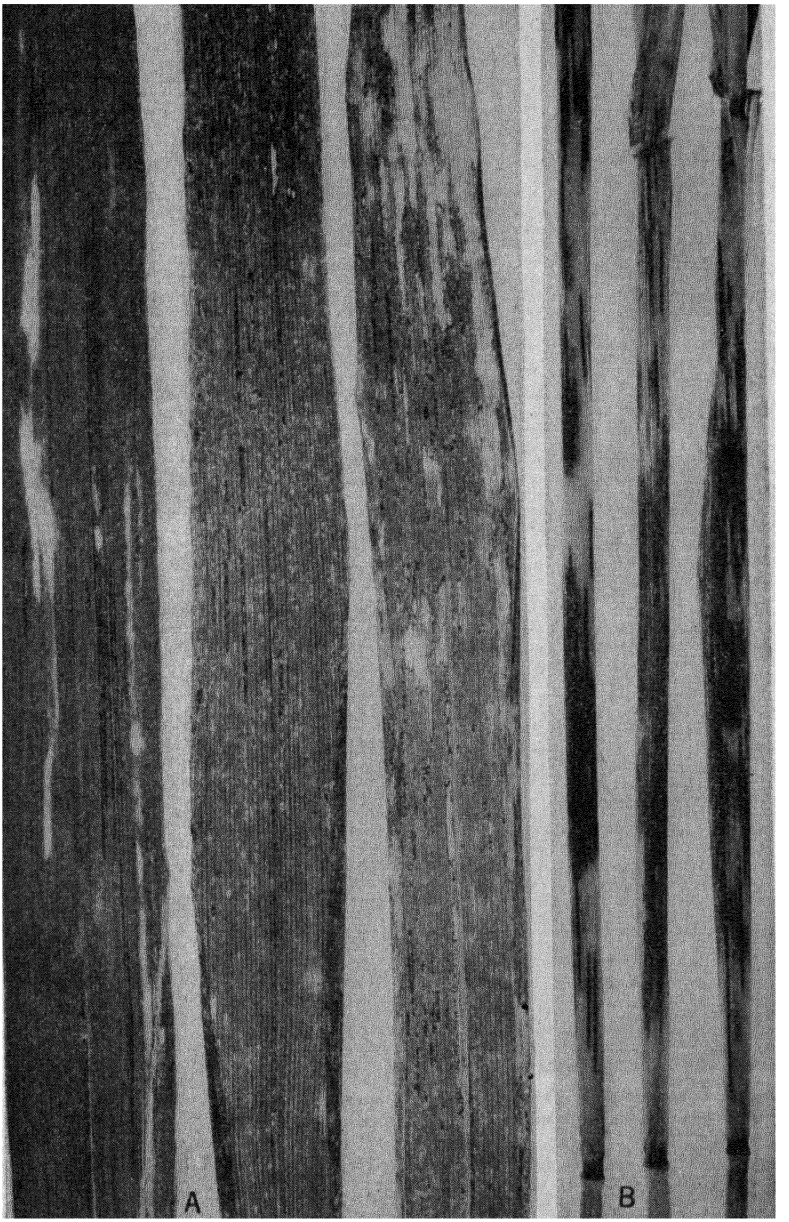

FIG. 5.—Leaf (*A*) and sheath (*B*) lesions of bacterial blight of barley caused by *Xanthomonas translucens*.

Control.—Crop rotation and seed treatment with mercury compounds assist in the control of the disease. Sporadic outbreaks occur, however, regardless of the use of control measures. Varietal reaction to the disease

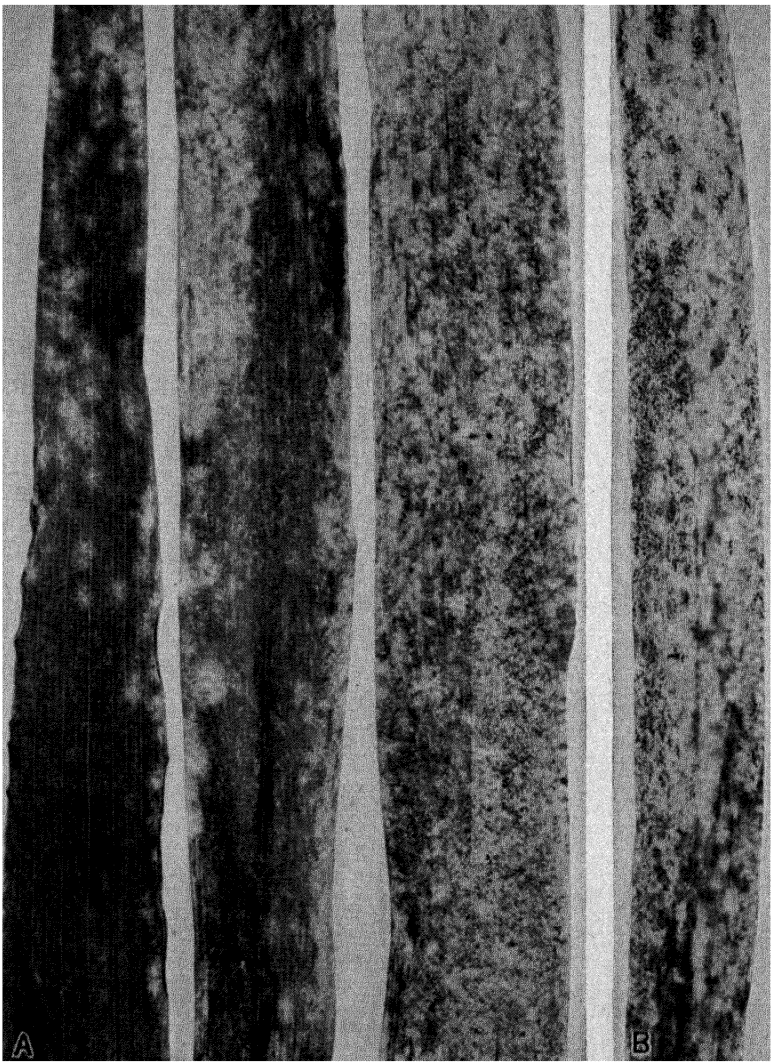

FIG. 6.—Powdery mildew of barley (*A*) and wheat (*B*) showing the powdery, superficial mycelium and conidia and perithecial initials of *Erysiphe graminis*.

varies greatly. Jones and Johnson (1917) reported Oderbrucker (C. I. 1272)[1] and Chevalier as the most resistant of about 40 varieties. In the

[1] Accession numbers of the Division of Cereal Crops and Diseases, U. S. Department of Agriculture.

epiphytotic of 1944, Oderbrucker (C. I. 4666), a Chevron (C. I. 1111) × Bolivia (C. I. 1257) selection, and many selections of the Manchuria group showed resistance. Some of the smooth-awned varieties were low in infection.

3. Powdery Mildew, *Erysiphe graminis* DC.—The disease occurs generally on the cereals and grasses with the exception of those in the tribes Maydeae, Andropogoneae, Zoysieae, Paniceae, and Oryzeae (Mains and Dietz, 1930, Marchal, 1902, 1903, Reed, 1909, and Salmon, 1900, 1904). Economically the disease is generally of more importance on barley than on the other cereal crops.

Many varieties of the cultivated barleys as well as wild species of *Hordeum* are susceptible to powdery mildew. The disease is general in distribution throughout the humid and semihumid areas of the world (Honecker, 1934, 1935, 1937, Marchal, 1903, and Salmon, 1904), and it is generally more severe on both winter and spring barleys in the areas where cool, humid, and cloudy weather persists during the growing period.

Heavy infection with powdery mildew increases respiration and reduces yield. Yarwood (1934) has shown an increase in rate of respiration with the clover powdery mildew. Honecker (1937) reported an increased protein content in barley from mildew-infected plants. Other investigators have studied the disease on wheat (See Chap. XI).

Yields are reduced when mildew infection is severe during the period of active plant growth and grain development. The effect upon yield has been demonstrated by using dusts and sprays as a control on a mildew-susceptible variety. Similar effects have been demonstrated by the use of resistant backcross material. Under a severe mildew epiphytotic in 1942,[1] Oderbrucker homozygous for waxy and mildew resistance yielded about 30 per cent more than Oderbrucker homozygous for a starchy endosperm and mildew susceptibility or than the susceptible Oderbrucker parent. The lines heterozygous for mildew resistance and waxy were intermediate in yield.

Symptoms and Effects.—The powdery mildew develops on the epidermis of blades, leaf sheaths, and floral bracts. The superficial mycelium and conidia are first light gray in color, the mycelium darkens with age, and later numerous round dark perithecia develop on these areas (Fig. 6). The tissues of the suscepts beneath the mycelium vary in response to the fungus. In the more susceptible varieties, chlorosis and browning accompany the aging of the mycelium. In many varieties, light- to dark-brown pigmentation and frequently necrosis occur beneath or adjacent to the superficial mycelium (Fig. 7). The response is similar to the so-called "flecking" in the reaction to rust. The characteristic symptom is

[1] Unpublished data from H. L. Shands, department of agronomy, University of Wisconsin, Madison, Wis.

the gray powdery-surfaced lesions scattered or completely covering the leaf blade with yellowing, browning, and gradual drying out of the leaf tissue. The symptom is also an indication of the damage to the suscept.

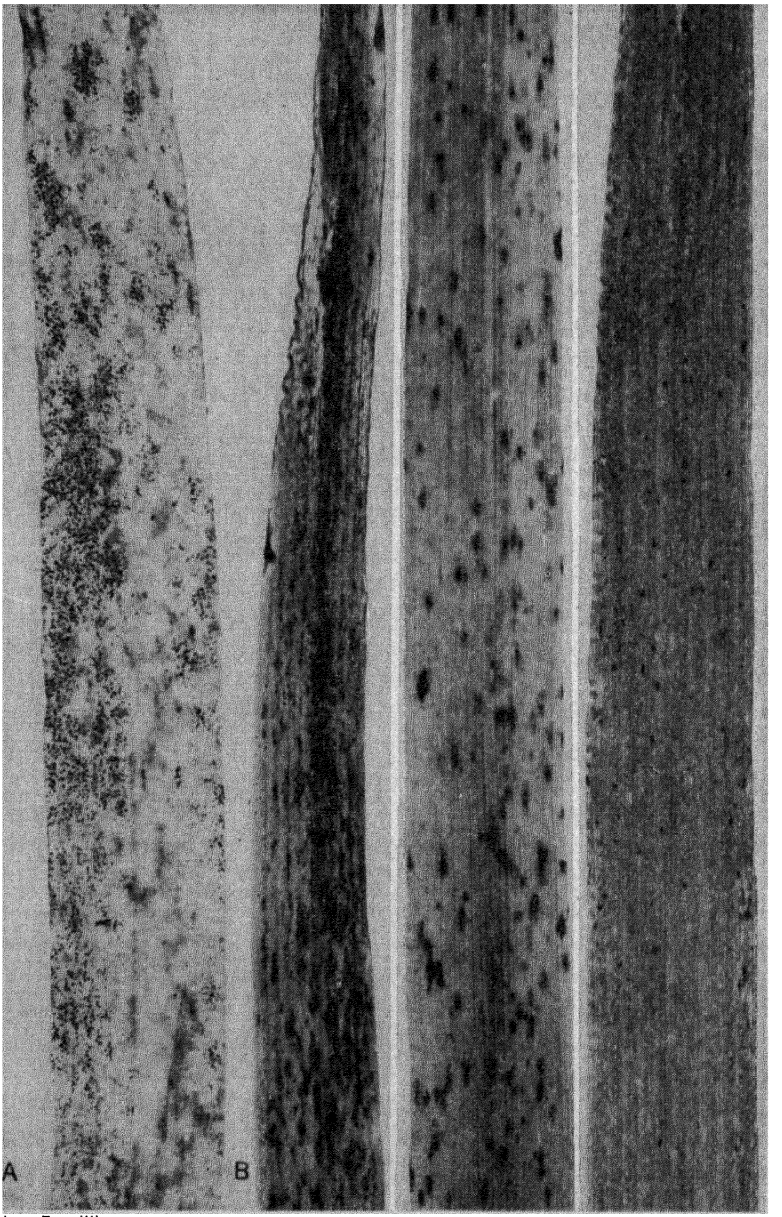

FIG. 7.—The response of barley varieties to *Erysiphe graminis hordei*. (*A*) Susceptible. (*B*) Resistant, sparce mycelial development with, left, abundant cell necrosis and, right, restricted necrosis without parasitic establishment of the fungus.

The Fungus.—*Erysiphe graminis* DC.

Specialized varieties of *Erysiphe graminis* occur on the cereals and related grasses as well as on certain groups of the grasses. These varieties of the fungus are similar morphologically, but they are restricted parasitically to certain genera of the Gramineae; trinomials are used for their designation. The variety occurring on barley is *E. graminis hordei* El. Marchal.

The mycelium is superficial, branched, white, and later turns gray to brown. Conidiophores form soon after the mycelium is established. They are medium in length with a terminal generative cell. The conidia are light gray, ovoid, measure 25–30 by 8–10 microns, and are borne in chains. Perithecia are dark brown, round to subspherical, about 220 microns in diameter, and scattered. The appendages are rudimentary, short, and pale brown. The asci are numerous, cylindric to ovate-oblong, with usually eight spores. In the North Central and Central area of the United States, many perithecia are empty or contain incompletely developed asci.

Etiology.—The primary infection occurs from ascospores or conidia. Perithecia and ascorpores are produced on mature tissues of the suscept under favorable weather conditions. Ascospore development occurs in late summer on the cereal crops and grasses in the North Central United States although, apparently, relatively few are available for spring inoculum. The mycelium persists from season to season in the areas where the winters are mild enough for infected leaves to survive; however, ascospores are the important source of primary inoculum in the spring-grain area. The spores are largely wind-borne. Conidial formation, dissemination, and germination are best in a humid, cool atmosphere, but in the absence of free water, according to Cherewick (1944) and Yarwood (1936).

Infection in *Erysiphe graminis* is by direct cuticular penetration of haustoria forming branches into the epidermal cells. The mycelium spreads on the epidermis of the leaves and floral bracts. Conidiophores are formed, and abundant diurnal development of conidia follows. The spread of the initial mycelium is rapid, and secondary infections occur in great abundance, especially during periods of cool, cloudy weather. As the suscepts approach maturity perithecia develop in the mycelial matts (Harper, 1905, Reed, 1909, Smith, 1900). Powdery-mildew development is aggressive during the period of rapid growth and spike development of the cereals.

Control Measures.—The disease can be controlled by the use of sulphur dusts, and the inoculum is reduced by the use of potassium or sodium sulphide (1.0 per cent solution in water) or copper sulphate (1.05 per cent in water) sprays to which is added a suitable spreader, such as 0.03 per cent glyceryl alkyl resin (Yarwood, 1945). These methods of control are not economical except on an experimental basis; therefore, the use of mildew-resistant varieties represents the most practical method of control in both the cereals and grasses.

Disease Resistance and Physiologic Races.—A relatively large number of barleys within the different species and groups are resistant to one or more of the physiologic races of *Erysiphe graminis hordei.* According to Mains and Martini (1932), Shands (1939), and Tidd (1937), Arlington (C. I. 702), Duplex (C. I. 2433), Chevron (C. I. 1111), unnamed selection (C. I. 2444), and others are resistant to at least five races of the parasite. Briggs (1935, 1937, 1938), Dietz (1930), Tidd (1937), and others have studied the inheritance of resistance. A number of factor pairs are involved in the expression of resistance in different barley varieties and to the various physiologic races, as summarized by Briggs and Stanford (1943).

The reaction in the seedling stage of six differential varieties of barley to 10 physiologic races of *Erysiphe graminis hordei* as reported by Cherewick (1944), Mains and Dietz (1930), and Tidd (1937) is shown in the following table.

Physiologic race	Reaction on barley varieties					
	Nepal (C.I. 595)	Heils Hanna (C.I. 682)	Goldfoil (C.I. 928)	Peruvian (C.I. 935)	Black Hull-less (C.I. 666)	Chevron (C.I. 1111)
1	I	I	R	R	I	R
2	S	S	R	I	I	R
3	S	S	R	S	S	R
4	S	S	R	I	S	R
5	S	S	S	S	S	R
6	R	S	R	R	I	R
7	R	S	S	R	R	R
8	S	S	R	R	S	S
9	I	S	R	R	I	S
10	I	S	R	S	I	R

R—resistant; I—intermediate; and S—susceptible.

4. Fusarium Blight or Scab, *Gibberella zeae* (Schw.) Petch [*G. saubinetii* (Mont.) Sacc.] and *Fusarium* Spp.—The Fusarium blight occurs on all the cereals and many of the grasses. The head blight is especially severe on rye, barley, and wheat. The complete discussion of the disease is included in Chap. XI.

Distribution and Importance on Barley.—The head blight occurs in the Eastern and Central United States and adjacent Canada. The area includes the humid spring-barley and the northern portion of the humid winter-barley sections. Damage to both yields and quality frequently is high in this area, and losses become more sporadic and less frequent in the drier prairie and plains areas. The seedling blight and root rot phase

of the disease extends into the drier areas. The disease is common on barley in the humid regions of Europe and Asia.

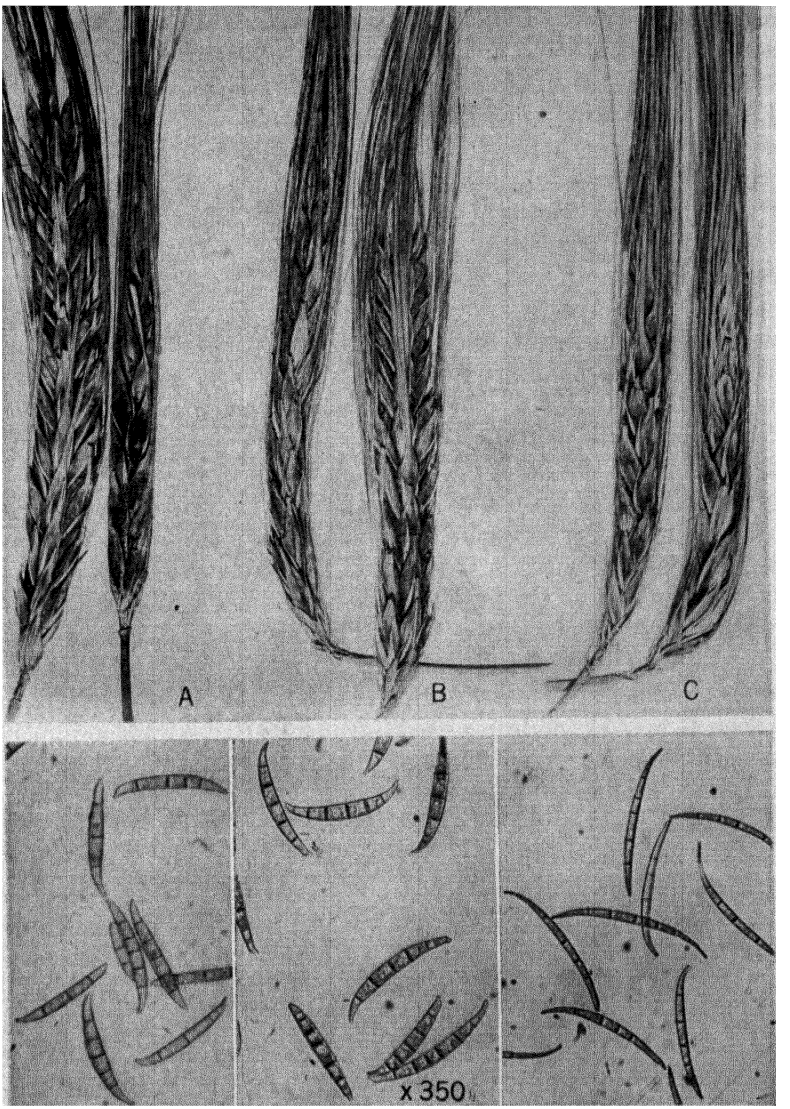

FIG. 8.—Barley spikes infected with *Gibberella* and *Fusarium* species and the conidia of the three species. (*A*) *G. zeae*, (*B*) *F. culmorum*, and (*C*) *F. avenaceum*. Conidia highly magnified.

The fungi causing the disease show a geographical distribution within the different regions. In the corn-belt section of the Eastern and Central United States, *Gibberella zeae* (Schw.) Petch is the common species. In

the more northern area, *Fusarium culmorum* (W. G. Sm.) Sacc. and *F. avenaceum* (Fr.) Sacc. and some other species predominate. In northern Europe, *F. culmorum* and in southeastern Europe, *G. zeae* are the chief organisms on barley. In northeastern Asia, *G. zeae* causes heavy annual losses in barley, as reported by Atanasoff (1923), Bennett (1932, 1933, 1935), Dickson (1930), Naumov (1914), and Palchevsky (1891).

The disease causes reduction in yield of grain, and it damages quality. The blighted barley kernels are shriveled or lighter in weight, yet cannot be separated completely from the sound grain. The infected kernels contain substances causing acute emesis in pigs, dogs, or humans. Cattle, sheep, and mature chickens seemingly are not affected by the diseased grain, according to Christensen and Kernkamp (1936), Dickson (1942), Hoyman (1941), Mains *et al.* (1930), Mundkur (1934), Roche and Bohstedt (1931), and others. Barley containing 4 per cent of blight-damaged kernels, including blight caused by various fungi, is given the special designation "blighted" in the Federal grain-grading procedure. Blighted barley is discounted heavily on the commercial markets of the United States and cannot be exported to foreign markets.

Symptoms.—Spikes are dwarfed and compressed with infected spikelets closed rather than spreading. All or part of the spike is infected (Fig. 8). Hulls (lemma and palea) are light to dark brown with a dead, lusterless surface. Conidial or perithecial masses commonly develop on the surface, especially during moist weather (Fig. 9). The kernels are shrunken and light brown in color. The pericarp surface is rough or scabby in appearance. The starch mass is grayish in color and flour-like in texture. The scab is not distinguished easily from the Helminthosporium blight unless symptoms are very characteristic or spore masses are present on the kernel. Plating the kernels on acidified agar is a reliable method of differentiating the two.

Seedling infections in barley are primarily from seed-borne inoculum. Restricted reddish-brown cortical lesions occur when the infected seed is sown in cool, moist soil. Seedling blight before or after emergence occurs in a warm soil. Seedling infection of clean seed from mycelium in the soil is common when the soil temperatures are high. Crown and basal culm rot occur commonly in the later stages of development of the barley plant.

Control.—Clean seed and seed treatment help control seedling infection. The mercury dusts are effective in controlling seedling damage from infected seed. Sanitation, rotation, and early planting help in reducing crown infection and head blight. Soil preparation to obtain complete coverage of barley, wheat, and corn-crop residues helps reduce inoculum for head blight infection.

Varieties resistant to head blight offer the best means of control. The commercial varieties commonly grown in the areas where scab is pre-

valent are susceptible to the disease. No variety highly resistant to scab has been found. The more resistant barleys are: Svansota (C. I. 1907) (a two-rowed barley), Chevron (C. I. 1111), Korsbyg (C. I. 918), Cross

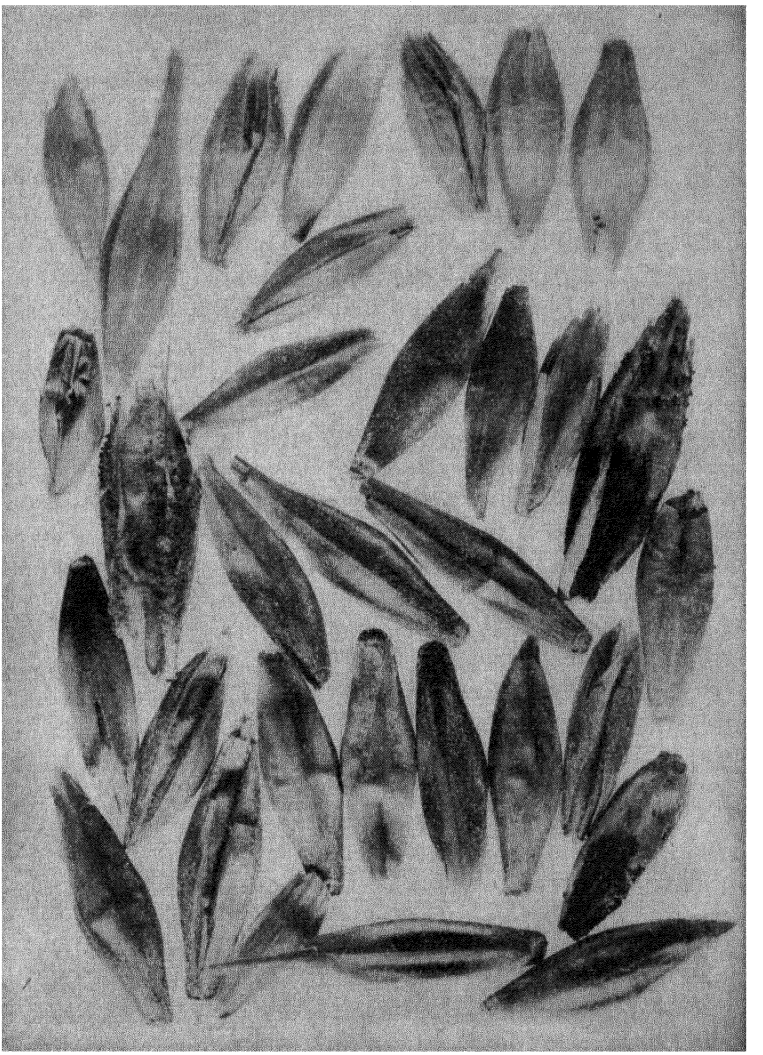

FIG. 9.—Barley kernels blighted by *Gibberella zeae* showing discoloration and conidial and perithecial development

(C. I. 1613 and 2492), an unnamed selection (C. I. 1918), and Peatland (C. I. 5267) (six-rowed barleys). These barleys are being used in breeding for scab resistance (Shands, 1939). See Fusarium Blight, Chap. XI, for the complete discussion.

5. Ergot, *Claviceps purpurea* (Fr.) Tul.—Ergot is found frequently on barley through the North Central United States and Canada. The disease occurs in other similar areas where rye or common grass suscepts are infected. Damage is rarely as high as in rye, durum wheats, and many grasses. See Ergot on Rye, Chap. VIII, for the complete discussion.

6. Net Blotch, *Pyrenophora teres* (Died.) Drechs.—The disease is common on all the cultivated barleys and apparently is restricted to these species. Net blotch is distributed with barley culture throughout the temperate humid regions of the world. The disease occurs more abundantly in the cooler climates or where barley is grown in the cooler period of the year. It is very common throughout Northern Europe. Like the stripe disease, it was introduced with barley into North America. Under most conditions the disease is of minor importance, although under favorable conditions it causes considerable reduction of foliage, especially as the crop approaches maturity.

Symptoms.—The first symptom on the seedling leaf is the development of brown reticulate blotches at or near the tip of the blade. The lesions rarely develop from the base of the seedling leaf as in spot blotch. Local lesions on the young leaves develop from the seedling stage until maturity. The young infections show the characteristic netted blotch. The darker brown necrotic areas of the blotch are distributed irregularly in narrow, indefinitely margined lines both parallel and perpendicular to the leaf axis. This makes a dark-brown reticulate pattern within the areas of lighter brown (Fig. 10). Later as the necrosis of the mesophyl tissue expands and the blotches coalesce longitudinally, the characteristic appearance changes to dark brown limited stripes with irregular margins. The net-like pattern is evident in these older lesions only along the margins. In the advanced stages of infection, a series of several to as many as 10 irregularly margined stripes extend parallel, frequently the full length of the blade. The stripes do not continue into the leaf sheath as in the stripe disease. Conidiophores and conidia develop sparsely on the lesions. Small linear brown lesions occur on the floral bracts. The light-brown discoloration of the lemma without the conspicuous netted appearance is characteristic of kernel infection. Seed-borne infection is determined accurately only by plating the kernels on acidified potato dextrose agar. Seed-borne infection is higher in the more northern areas; Machacek and Wallace (1942) reported 0 to 64 per cent in a Canadian survey.

The Fungus.—*Pyrenophora teres* (Died.) Drechs.
(*Helminthosporium teres* Sacc.) (Conidial stage)
(*Pleospora teres* Died.)
(*Helminthosporium hordei* Eidam.)

The mycelium is white to olivaceous in the tissues and makes a very sparse tufted growth on media. Bodies resembling vegetative resting spores appear in culture.

Conidial development on suscept and in culture usually is limited. The conidiophores are light brown to olivaceous, occurring singly or in groups of two to three, and the swollen basal cell is usually larger than in the other *Helminthosporium* spp. on barley. The average dimensions are 120–200 by 7–9 microns. Conidia are yellowish olivaceous, never dark olivaceous, thin-walled, constricted at the septa with much rounded apical

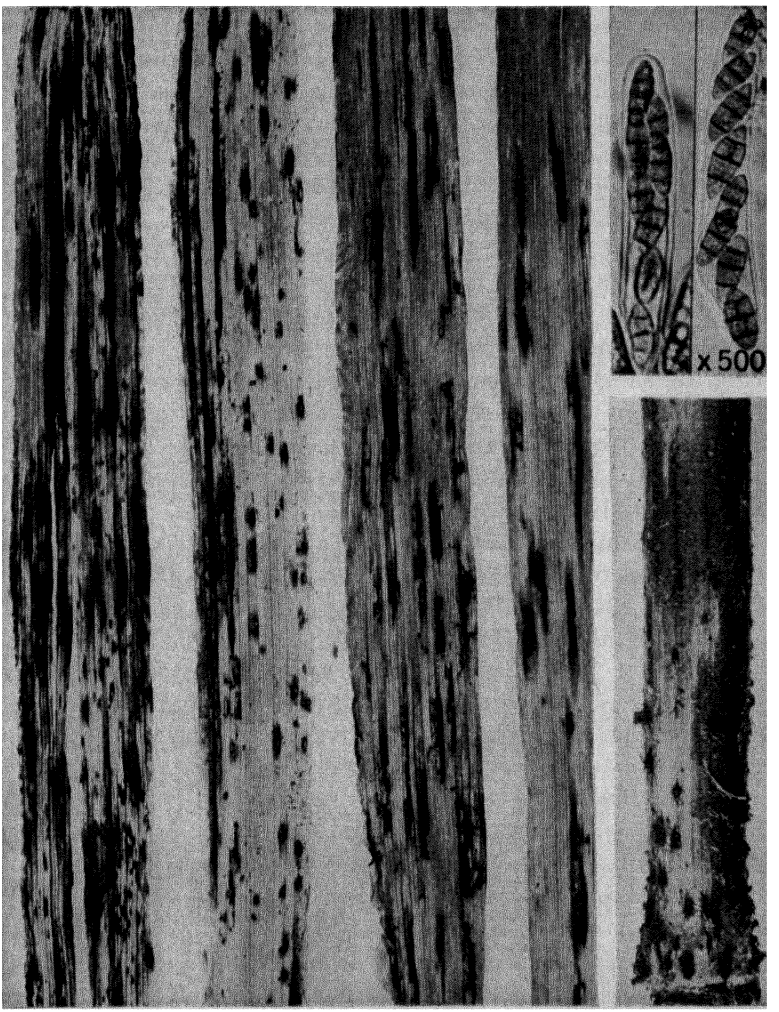

FIG. 10.—Net blotch of barley caused by *Pyrenophora teres* showing the characteristic leaf lesions at different stages of development and the perithecia and ascospores of the fungus.

cells. The basal cell is larger, resulting in a subcylindrical shape. Germ tubes develop from all cells of the conidium. The perithecia develop abundantly on barley stubble and straw, especially the following spring. They are superficial or partly submerged, elongated, irregular in shape, and about 0.5 mm. in diameter. Setae and conidiophores are abundant on the surface. The ostiolar beak is not common. The numerous asci are subcylindrical with a ring-like thickening at the apical end, and each contains

eight spores. The ascospores are light brown, 3-septate, with the center cell usually divided longitudinally in the mature spores. The spores are much constricted at the septa (Fig. 10). Germ tubes are formed from all cells (Drechsler, 1923).

Etiology.—The early seedling infection of *Pyrenophora teres* is from seed-borne mycelium or ascospores produced on old straw and stubble. Ascospores or conidia are responsible for continued infection whenever conditions are favorable. The infection on fall or spring barley is abundant during cool, humid weather. The lesions enlarge and coalesce throughout the growing season. The mycelium grows into the sheath and culm tissues as the disease progresses and the plants mature. Local infections on the floral bracts occur from spike emergence to shortly after flowering. Perithecia develop on the barley stubble and straw in the late fall and again in the early spring. The disease is usually very abundant on volunteer barley late in the fall.

Control.—Seed treatment with the standard mercury dusts controls the seed-borne inoculum. Sanitation and crop rotation are important in reducing the ascosporic inoculum. Barley sown on or near fields with barley stubble on the surface is infected heavily the following spring and summer. Differences in varietal reaction to the disease are reported by Geschele (1928) and Ravn (1900). Most commercial varieties in North America are relatively susceptible.

7. Stripe Disease, *Helminthosporium gramineum* Rabh.—The barley-stripe is today a relatively minor disease in spring-barley culture in the United States and Canada. It is prevalent and causes considerable damage in the California barley area and in the South Central winter-barley section of the United States. Damage from stripe is severe in limited sections of northern Europe and in northern and eastern Asia as well as in portions of Turkey, Iran, and Transcaucasia. The distribution of the disease is widespread, as reported by Mitra (1931), Nishikado (1929), Ravn (1900), and Smith (1929, 1930).

Symptoms and Effects.—The symptoms of the disease are conspicuous from the late tillering stage until the crop is mature (Drechsler, 1923, Ravn, 1900). The first symptoms at the tillering stage are yellow striping of the older leaf blades and sheaths. Some seedling blight occurs in severely infected seed of susceptible varieties. The yellow stripes soon turn brown as tissue necrosis progresses, and finally the tissues dry out and fray as the leaves mature. During the period of culm elongation, the symptons are distinctive: the young leaves unfolding show the yellow striping with the successive necrosis and browning conspicuous on the leaves below (Fig. 11). The elongation of the culms of the striped plants varies from rosette-like development to fully elongated plants. Varieties of barley, races of the parasite, and environmental conditions influence culm elongation in diseased plants, as discussed by Arny and Shands

(1945), Christensen and Graham (1934), Isenbeck (1937), Leukel *et al.* (1933), Mitra (1931), Shands (1934), and Shands and Dickson (1934). The spikes fail to emerge in many diseased plants. Those that emerge

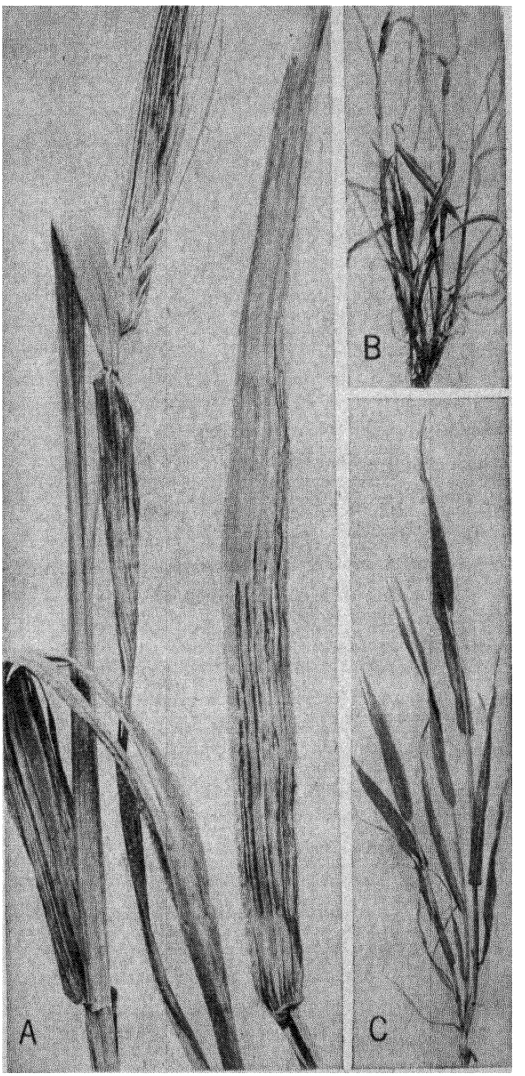

FIG. 11.—Barley plants showing the yellow to brown stripes on leaf blades and sheaths of plants infected with *Helminthosporium gramineum*. (*A*—*B*) The brown stripes, shredding of the leaves, and brown blighted condition of the spikes of mature plants. (*C*) The brown stripes on the lower leaves and yellow stripes on the upper leaves of younger plants.

are blighted, twisted, compressed, and brown in color. On the Pacific Coast, the kernels are damaged less and the brown infected kernels fre-

quently appear in the threshed grain. When the healthy plants are heading, the striped plants show a gray to olive-gray color due to the development of conidiophores and conidia over the mass of lesioned tissue. After conidial development of the fungus, the tissues of the infected plants split, fray out, and collapse. The florally infected kernels rarely show indications of infection, although the fungus can be plated from infected kernels on suitable media.

The Fungus.— *Helminthosporium gramineum* Rabh.
[*Brachysporium gracile* Wallr. var.
gramineum (Rabh.) Sacc.]
(*Napicladium hordei* Rost.)
(*Heterosporium gramineum* Oud.)

Diedicke (1902, 1904) described the ascigerous stage under the binomial *Pleospora graminea* Died., Noack (1905) described it and used *P. trichostroma* Noack, and Paxton (1922) described what he considered to be the perfect stage from material collected in California. Ito and Kuribayashi (1931) discussed the taxonomy and described *Pyrenophora graminea* (Rabh.) Ito and Kuribay.; however, the connection with the conidial stage was not demonstrated and the morphology was similar to *P. teres*. Later investigations have not confirmed these reports.

The morphology of this and other species is given in detail by Dreschsler (1923). The mycelium in the tissues is abundant, subhyaline to light yellow. In culture the mycelium varies greatly in color from gray through olive to black. The mycelium in culture does not produce conidia unless exposed to cyclic changes of light and darkness. The conidiophores and conidia develop on the plants only as the suscepts are heading. Conidiophores are borne in clusters, usually three to five. The basal segment is enlarged, the distal portion is slender, the color is gray to olivaceous. The conidia are subhyaline to yellowish brown, straight, subcylindrical to slightly tapering, have rounded ends, thin-walled, 1- to 7-septate without constrictions at the septa, and average 105 by 20 microns in size. Both end cells and less commonly the central cells form germ tubes. Secondary conidia form from the germ tubes, as described by Dreschler (1923). Christensen and Graham (1934) have shown morphological differences between some races.

Etiology.—Natural infection from conidia of *Helminthosporium gramineum* occurs at or soon after flowering of the barley spikelet. The mycelium is established on or in the pericarp or in embryo tissues before maturity of the kernel. The final establishment of the mycelium in parasitic relationship with the young seedling tissues generally occurs during seed germination. Seedling infection can be induced in high percentages by placing the actively developing mycelium of the fungus in contact with the germinating barley kernels. This method of inoculation is used extensively in stripe-resistance studies, as reported by Arny (1945), Isenbeck (1930), and Shands (1934). After seedling infection, the parasite develops in the culm primordia and grows with the differentiating tissues during seedling development. The systemic distribution of the mycelium continues with the differentiation and development of the plant structures. This apparently is the only species in the genus *Hel-*

minthosporium in which a systemic type of infection occurs. Conidial production is synchronized with the heading, blossoming, and early stages of kernel development of the suscept. The conidial formation is probably a direct response to the physiology of the suscept tissues, as the same response occurs under greenhouse culture during the winter months. The fungus is carried over from season to season on or in infected seed. The mycelium remains viable in dry seed for an indefinite length of time.

Environmental conditions influence floral infection and disease development in the seedling. Isenbeck (1937) and Ravn (1900) reported that sufficient moisture to wet the spores is necessary for floral infection. This limits the regions where the stripe disease occurs to areas where dew or precipitation occurs during the period of barley flowering. Christensen and Graham (1934), Isenbeck (1937), Kiessling (1916), Leukel *et al.* (1933), Ravn (1900), Shands (1934), and others reported on the influence of moisture, temperature, and fertility on seedling development of stripe. A cool, moist, fertile soil favors stripe development in the seedling and developing plant.

Control Measures.—Seed treatment with the organic mercury dusts, such as Ceresan, control the seed-borne infection. Resistant varieties offer the best means of control of the disease.

Disease Resistance and Fungus Specialization.—Physiologic specialization occurs, although investigations are not comprehensive enough to determine the number or stability of the races or arrange a key for their identification, as discussed by Christensen and Graham (1934), Shands and Dickson (1934), and others. Resistance in varying degrees is common in varieties of most of the barley types. Arny (1945), Isenbeck (1930), Shands and Arny (1944), and others reported varieties intermediate to highly resistant, and they have discussed the inheritance of resistance. The evidence indicates more than one factor pair determining resistance to stripe in many varieties and a single factor pair in certain varieties and cultures of the fungus. Lion (C.I. 923), Peatland (C.I. 5267), and Chevron (C.I. 1111) have been used extensively in breeding stripe-resistant varieties. Resistance has been incorporated into many commercial varieties, with the result that stripe is now a minor disease in the spring-barley area of the United States. The following commercial varieties are resistant enough for practical stripe control: Wisconsin Barbless (C.I. 5105), Glabron (C.I. 4577), Trebi (C.I. 936), Regal (C.I. 5030), Newal (C.I. 6088), Velvon (C.I. 6109), and Mars (C.I. 7015), six-rowed; and Spartan (C.I. 5027), Vance (C.I. 4586), and Hannchen (C.I. 531) two-rowed.

8. Spot Blotch.—*Helminthosporium sativum* Pam., King, and Bakke.—The spot blotch disease is different in several respects from many of the other diseases caused by species of *Helminthosporium*. The fungus

attacks a wide range of grasses and is common on wheat and barley. The mycelium is very resistant to unfavorable conditions and is abundant in gramineous crop residues both in and on the soil surface. Local lesions occur on seedlings, plant crowns, culms, leaves, floral structures, and kernels. The seedling and crown damage occurs in relatively dry, hot areas as well as abundant infections of all tissues in humid, warm regions.

Geographic Distribution and Importance.—The disease is widespread

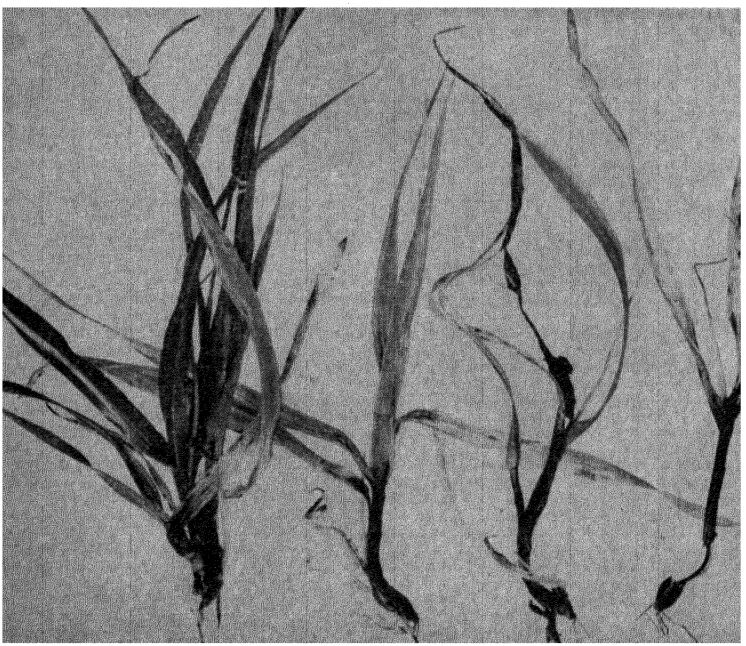

FIG. 12.—Seedling blight of barley caused by *Helminthosporium sativum*. Symptoms range from stunted seedlings with dark green leaves and brown lesions on the basal leaf sheaths to blighted seedlings.

in North America, and it is common although probably not so extensively distributed in South America, Europe, Asia, Australia, and Africa as reported by Dickson (1930), Hynes (1935, 1937), Ito and Kuribayashi (1931), Kuribayashi (1917), Lindfors (1918), Smith (1930), Sorokin (1890), and others. Damage on both barley and wheat are severe where susceptible varieties are grown extensively.

Symptoms and Effects.—The seedling blight is characteristically a dry rot type of tissue necrosis. The dark-brown to black lesions usually occur first on the coleoptile and progress inward. The seedling is killed before emergence or more frequently after emergence. The seedling leaves of infected plants are dark green, erect, with dark-brown lesions near the soil line that soon extend into the leaf blade. Development of

infected seedlings is retarded, and tillering is excessive. The seedling symptoms are similar in barley, wheat, and some grasses. The crown

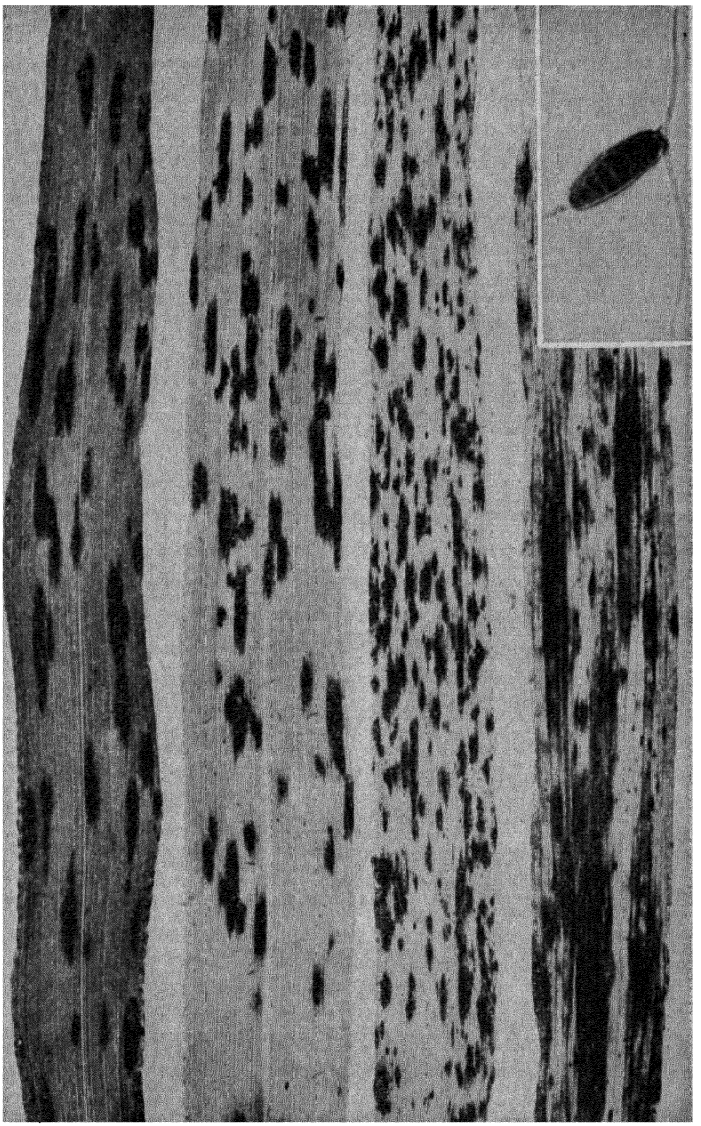

FIG. 13.—Spot blotch of barley caused by *Helminthosporium sativum* and the germinating conidium of the fungus. The elongate brown spots with definite margin coalesce later to form brown irregular stripes. The conidia germinate by germ tubes from the apical cells only.

rot develops at or below the soil surface. Cortical and leaf-sheath tissues are lesioned, tiller buds are blighted, and the crown-root system

is invaded. The necrosis is accompanied by a dark-brown discoloration of the tissues (Fig. 12).

The leaf spot varies in size and shape. The individual lesions are round to oblong with definite margins. The color is a uniform dark brown without conspicuous water-soaking accompanying necrosis. The spots coalesce to form the blotches that frequently cover large areas of leaf blade. They are more restricted on the leaf sheath. The older lesions develop an olivaceous cast due to the abundant development of conidiophores and conidia. The heavily infected leaves dry out and mature early. The fungus continues to sporulate on the dead tissues (Fig. 13).

Lesions on the floral bracts and kernels range from small black spots to dark-brown discoloration of the surface. The characteristic "black point" or blackened embryo end of the kernel is one of the common symptoms on wheat and barley (Fig. 14). The extensive invasion of crown and culm tissues usually results in shorter culms, partial emergence of the spike, and sterility or poorly filled kernels. Head blight occurring early also causes sterility or killing of individual kernels soon after pollination (Figs. 13 and 14). The dark-gray to black mycelium with abundant conidial development in most cultures is characteristic when the diseased tissues are plated on acidified potato-dextrose agar.

The Fungus.— *Helminthosporium sativum* Pam., King, and Bakke
(*Helminthosporium acrothecioides* Lindf.)
(*Helminthosporium inconspicuum* Peck)
(*Helminthosporium sorokinanum* Sacc.)

Ito and Kuribayashi (1931) reported the acigerous stage as *Ophiobolus sativus* Ito and Kuribay., although the relationship was not established definitely.

The mycelium is olivaceous to black when mature and develops abundantly, including conidial production on media. Conidiophores emerge from stomata or between epidermal cell walls, singly or two to three, rarely more. The basal cell is swollen, has a heavy wall, is dark olivaceous, and the conidial scars are conspicuous. Conidiophores on agar cultures are short modified branches of hyphae. The conidia are slightly to distinctly curved, thick-walled, reddish to dark olivaceous brown, 1- to 10-septate, widest near the middle, and the ends round off abruptly. The size, shape, and color vary greatly, depending upon the culture and the environment. The conidia germinate from the apical cells only, Drechsler (1923, 1934) (Fig. 13). The ascigerous stage was described by Ito and Kuribayashi (1931) as follows:

Perithecia are black to brown, pseudoparenchymatous, flask-shaped with ostiolar beak, and measure 340–470 by 370–530 microns. Many hyphae and conidiophores are associated with the young perithecia, which disappear as the perithecia mature. Asci are numerous fusiform or cylindrical, straight or slightly curved, rounded at the apex, shortly stipitate at the base with hyaline wall, and contain usually four to eight ascospores coiled in a close helix. The spores are flagelliform or filiform, obtusely pointed at both ends, somewhat broader at the base, hyaline or light olive, and measure 160–360

by 6–9 microns. Drechsler (1934) transferred the group of species with coiled ascospores to the new genus *Cochliobolus*.

Etiology.—Seedling and crown infections occur from seed-borne mycelium or from crop residues in the soil. The organism develops aggressively as a saprophyte on crop residue or mature tissues of the cereals and grasses. Disease development is usually more severe in

FIG. 14.—Spike blight (*A*) and kernel blight (*B*) of barley caused by *Helminthosporium sativum*.

late-sown grain or in a warm soil. Infection of embryonic tissues is by direct penetration, natural openings, or injuries. Frequently insect injury of crown tissues is followed by the invasion of *Helminthosporium sativum*. Leaf infections develop under warm, moist conditions, and they spread rapidly from secondary conidial infections. The abundant conidial inoculum results in severe infection of young tissues whenever environmental conditions are favorable. Plants that are retarded in development by injuries or unfavorable growing conditions are usually

more susceptible to attack. Seed infection is frequently quite high. Extensive platings of both barley and wheat kernels from the North Central area during the past 5 years show an average of over 5 per cent infection. In some seasons samples show as high as 75 per cent infection. Similar surveys in Canada indicate high infections in years favorable for the disease, as reported by Greaney and Machacek (1942).

Control.—The control of the disease is difficult and should be given more attention than in the past. Sanitation and crop rotation are important, but in the spring-grain area, where grains and grasses comprise such a large percentage of acreage, suitable rotations offer difficulties. Seed treatments with the mercury compounds have been effective in increasing stands of vigorous seedlings. Resistant varieties offer the best means of control. Hayes *et al.* (1923), Christensen (1945), and others have shown differences in susceptibility, although most commercial barleys and wheats are diseased under favorable conditions. The most resistant six-rowed barleys found to date are in the Manchuria group and Peatland (C.I. 5267). Hannchen (C.I. 531) and Svansota (C.I. 1907) are moderately resistant two-rowed commercial varieties. Resistance apparently is conditioned by several single factor pairs inherited independently. Christensen (1922, 1925, 1937) reported specialization and variation in the fungus.

Mackie and Paxton (1923) have described a species of *Helminthosporium* on barley in California differing slightly from *H. sativum* which they have described as *H. californicum* Mackie and Paxton. Mackie (1928) reported Chevalier as resistant to this rusty blotch.

9. Rhynchosporium Scald, *Rhynchosporium secalis* (Oud.) J. J. Davis.—The scald disease occurs commonly on barley, rye, and *Bromus inermis* Leyss. Many other grasses are infected by this species and *Rhynchosporium orthosporum* Cald. Specialized races of the parasites occur which limit the distribution of inoculum between suscepts. The disease is distributed in the cooler, humid and semihumid sections of North and South America, Europe, and Asia (Caldwell, 1937, Dickson, 1930). The disease is of minor importance in the North Central North American spring-barley area, as the commercial barleys are moderately resistant. Scald causes considerable defoliation in the barley section of the Pacific Coast and in areas in Europe and Asia where susceptible varieties are grown.

Description.—The blotches are conspicuous on the leaf blades and sheaths. The lesions are first water-soaked ovate to irregular scald-like blotches. The color of the lesions changes rapidly from bluish green to zonated scald and brown pigmented rings and finally to a bleached straw color with a brown margin on barley and straw color on rye (Fig. 15).

The Fungus.—*Rhynchosporium secalis* (Oud.) J. J. Davis
(*Marsonia secalis* Oud.)
(*Rhynchosporium graminicola* Hein.)

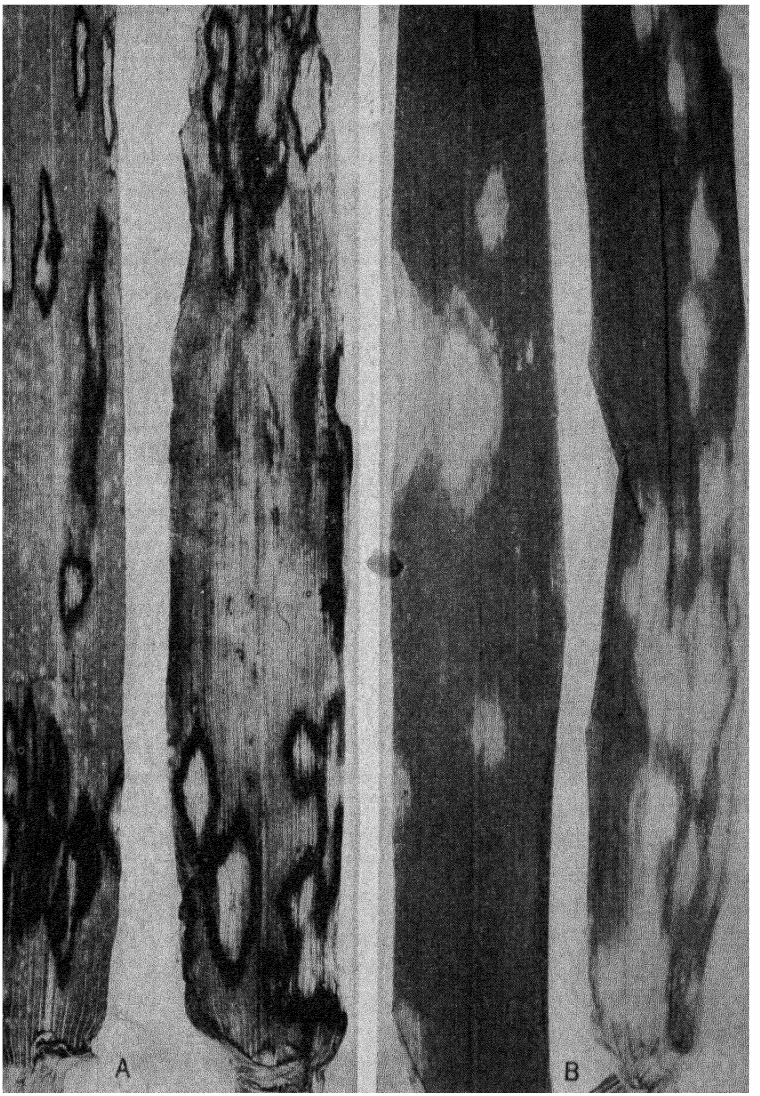

FIG. 15.—Leaf scald caused by *Rhynchosporium secalis* on barley (*A*) and rye (*B*).

The mycelium is hyaline to light gray, developing sparsely as a compact stroma under the cuticle of the suscept. The conidia, borne sessilly on cells of the fertile stroma, are hyaline, aseptate, cylindrical to ovate, with a short oblique apical beak on most spores, and measure 12–20 by 2–4 microns. *Rhynchosporium orthosporum* Cald. occurring on

a number of grasses is differentiated by the longer, more cylindrical conidia without the apical beak.

Etiology.—The leaf spots develop abundantly during cool weather. The mycelial stroma on the leaves apparently persists on living or dead leaf tissues to furnish the primary conidial inoculum. Leaf scald on winter rye from stroma formed the previous autumn frequently occurs during the first frost-free days of spring. Lesions develop on barley throughout the winter-growing season in the Pacific Coast barley area. Infection is usually abundant in late autumn on volunteer spring barley. The fungus overwinters on the infected dead leaves and probably on the crop residue to produce new infections on barley seedlings the following spring. Secondary conidial inoculum is abundant during the cool, humid spring-growing period and again in late autumn. The conidia are air-borne, frequently from considerable distances, and are distributed by rain as well. Infection is by direct penetration of the cuticle on the young leaf. The subcuticular mycelial stroma develops which soon ruptures the cuticle, and conidial production ensues. In the winter-barley sections, lesions occur infrequently on the floral bracts, although seed-borne inoculum has not been demonstrated.

Control.—Crop rotation, elimination of crop residue, and resistant varieties offer the best means of control. Sporadic local occurrence of the disease is common on susceptible varieties even though local crop rotation and sanitation measures are used. Spring burning of residue on perennial grasses helps reduce the overwintered inoculum on the grasses.

Resistant Varieties and Specialization.—Most of the commercial barley varieties grown in the North Central United States are moderately resistant to the disease. Otrada Beardless (C.I. 5631) and a Hanna selection are resistant at Davis, Calif. Resistance apparently is conditioned by a single factor pair[1] (Mackie,1929). Reports in the literature are conflicting regarding specialization of the fungus. The evidence in the United States and southern Russia indicates specialized races in both species of the genus. Six races of *Rhynchosporium secalis* are reported, each restricted to a single or closely related species, according to Caldwell (1937).

10. Anthracnose, *Colletotrichum graminicolum* (Ces.) Wils.—The disease is common on the cereals and grasses. It is of little importance on barley unless the crop is grown on sandy dry soils low in fertility. See Anthracnose, Chap. VIII, for the complete discussion.

11. Septoria Leaf Blotch, *Septoria passerinii* Sacc.—The Septoria leaf blotches are common on the cereals and grasses. A large number of

[1] Unpublished information from Dr. F. M. Briggs, department of agronomy, California Experiment Station, Davis, Calif.

species have been described with the differentiation based largely on spore morphology (Sprague, 1944). The etiology of this group of parasites is similar; therefore, the complete discussion of the diseases is given in Chap. XI.

The blotch caused by *Septoria passerinii* occurs sparingly on cultivated barleys and several wild *Hordeum* spp. The disease is distributed widely in North and South America, and it is common in both Europe and Asia. It causes little damage on barley, as defoliation is rare.

Description.—The lesions are linear with indefinite margins as the yellowish-brown area blends into the green of the leaf blade and sheath. Numerous small dark-brown pycnidia, imbedded in the tissue, develop on the straw-colored older portion of the blotch.

The Fungus.—*Septoria passerinii* Sacc.
(*Septoria murina* Pass.)

The mycelium in culture is scant and hyaline to buff colored. Conidia formed in culture are cylindrical or slightly curved, 3-septate, and hyaline. Pycnidia are subepidermal, dark brown, smooth, globose or mostly subglobose, 80 to 150 microns in diameter, with a smooth ostiole formed under the stomata of the suscept. Pycnospores are 3-septate, usually measure 1.7–3 by 23–46 microns, are hyaline, straight or slightly curved, and rounded at the ends. Microconidia develop in the pycnidia under some conditions.

Septoria nodorum Berk. is reported on barley kernels, Machacek (1945), Chap. XI.

12. Loose Smut, *Ustilago nuda* (Jens.) Rostr. Introductory Discussion.—The smuts are relatively numerous on the cereals and grasses; for example, three species occur on the cultivated barleys. Mycologists and plant pathologists have named the *Ustilago* spp., in many instances, on the basis of the parasitic potentialities of the fungus rather than on differentiating morphological characters. This tendency makes it impossible to identify the species in certain of the cereal smuts without knowing the suscept upon which the fungus occurs. The consolidation of these species on sound morphological criteria might be desirable, especially with the information now available on life cycle, cytology, and genetics of this group of plant pathogens. This is in accord with the concept of the early mycologists before information on specialization was more complete and knowledge on the type of germination was available or when the classification was based largely upon gross morphology. Persoon's (1801) grouping of subspecies under the binomial *Uredo segetum* is an example. More recently, Cunningham (1924), Fisher (1943), Rodenhiser (1926), Tapke (1943), and others have discussed such recombinations based on morphology. Specialization could be designed then within the species by the use of trinomials indicating the suscept specialization. This method is in common use in the case of other plant

pathogens of the cereals and grasses, notably in *Puccinia graminis* Pers., *P. rubigo-vera* (DC.) Wint., *Erysiphe graminis* DC., and others. Long usage of many of the accepted binomials for these smut fungi, however, argues against this combination of species and the resultant confusion. Stevenson and Johnson (1944) revised certain of the binomials to comply with the International Rules of Botanical Nomenclature. The following list of the cereal smut fungi includes their revisions. These are accepted by many authors, although the binomials used previously occur in some current publications and in the older literature.

LIST OF COMMON NAMES OF THE DISEASES AND BINOMIALS OF THE CEREAL SMUT FUNGI

Common name	Scientific name according to—	
	The International Rules of Botanical Nomenclature	Previous American practice
Leaf smut of rice	*Entyloma lineatum* (Cke.) J. J. Davis	*E. oryzae* H. & P. Syd.
Loose kernel smut of sorghum	*Sphacelotheca cruenta* (Kuehn) A. A. Potter	*S. cruenta* (Kuehn) A. A. Potter
Smut of broom corn, millet	*Sphacelotheca destruens* (Schlecht.) Stevenson & A. G. Johnson	*S. panici-miliacei* (Pers.) Bub.
Head smut of corn and sorghum	*Sphacelotheca reiliana* (Kuehn) Clint.	*S. reiliana* (Kuehn) Clint.
Covered kernel smut of sorghum	*Sphacelotheca sorghi* (Lk.) Clint.	*S. sorghi* (Lk.) Clint.
Rough-spored bunt of rye and wheat	*Tilletia caries* (DC.) Tul.	*T. tritici* (Bjerk.) Wint.
Smooth-spored bunt of rye and wheat	*Tilletia foetida* (Wallr.) Liro	*T. foetens* (Berk. & Curtis) Trel. or *T. levis* Kuehn
Kernel smut of rice	*Neovossia horrida* (Tak.) Pad. & A. Kahn	*T. horrida* Tak.
Stalk smut of rye	*Urocystis occulta* (Wallr.) Rab.	*U. occulta* (Wallr.) Rab.
Flag smut of wheat	*Urocystis tritici* Koern.	*U. tritici* Koern.
Black loose smut of oats	*Ustilago avenae* (Pers.) Rostr.	*U. avenae* (Pers.) Jens.
Millet smut	*Ustilago crameri* Koern.	*U. crameri* Koern.
Covered smut of barley	*Ustilago hordei* (Pers.) Lagerh.	*U. hordei* (Pers.) Lagerh.
Covered smut of oats	*Ustilago kolleri* Wille	*U. levis* (Kell. & Swing.) Magn.
Corn smut	*Ustilago maydis* (DC.) Cda.	*U. zeae* (Beckm.) Ung.
Black semiloose smut of barley	*Ustilago nigra* Tapke	*U. nigra* Tapke
Loose smut of barley	*Ustilago nuda* (Jens.) Rostr.	*U. nuda* (Jens.) Kell. & Swing.
Loose smut of wheat	*Ustilago tritici* (Pers.) Rostr.	*U. tritici* (Pers.) Rostr.

Suscepts and Distribution of the Loose Smut.—The true loose smut or "deep-borne" loose smut occurs on barley and a limited number of grasses. Many of the cultivated barleys are relatively susceptible as well as the selections of *Hordeum spontaneum* Koch tested. The loose smut is distributed widely in regions where humid, cool weather occurs during the period barley is heading. The disease causes reductions in yield approximately equivalent to the percentage of loose smut (Semeniuk and Ross, 1942). The smutted plants develop sufficiently well to compete for moisture and soil nutrients and yet produce no grain. Losses are higher, therefore, than occur in diseases in which the plants are killed before the competition for moisture becomes acute.

Description.—The smutted spikes emerge from the boot slightly earlier than the spikes on healthy plants. The sori are enclosed in a fragile membrane which soon ruptures, releasing the brown to dark-brown dusty spore mass (Fig. 16). The sori frequently develop in the leaves when the plants make a rank vegetative growth. The smutted spikes are conspicuous when the barley is heading, as the erect smutted spikes stand above the healthy plants during this period of spore spread. The brown spore mass is wind-borne over the field, while the healthy plants are pollinating and extruding the dehisced anthers. At maturity of the crop, the bare rachises of the smutted plants frequently stand erect above the level of the filled heads.

The Fungus.—*Ustilago nuda* (Jens.) Rostr.
(*Ustilago segetum* var. *hordei* f. *nuda* Jens.)
(*Ustilago segetum* var. *nuda* Jens.)
(*Ustilago hordei* var. *nuda* Jens.)
[*Ustilago nuda* (Jens.) K. & S.]

The mycelium in culture is hyaline changing to buff, sparse, and predominantly binucleate. Chlamydospore-like cells develop in the old cultures on malt or barley-seedling agar. The mycelium in suscept is hyaline changing to brown, irregularly lobed, and predominantly binucleate. The fertile mycelial cells become subspherical to spherical and develop finely echinulate brown epispore walls in the formation of the chlamydospores. The mature spores are lighter colored on one side, finely echinulate, and 5 to 9 microns in diameter (Fig. 16, insert *A*). The fresh spores germinate readily to form a one- to four-celled promycelium (basidium). No sporidia are produced (Fig. 16, insert *B*). Fusions occur between compatible cells of the promycelium by means of short or long conjugation tubes. Branches or hyphae develop from the fused cells or the conjugation tubes. Limited branching also occurs from unfused (haploid) cells, according to Christensen (1935), Dickson and Johann (1946), and Lange (1909, 1917). The earlier literature is summarized by Roemer *et al.* (1938). The sporidial development at low temperatures originally described by Huttig is questionable.

Etiology.—The wind- or air-borne chlamydospores of *U. nuda* which lodge in the susceptible barley flowers germinate, conjugation occurs between the compatible haploid cells of the promycelium, and the

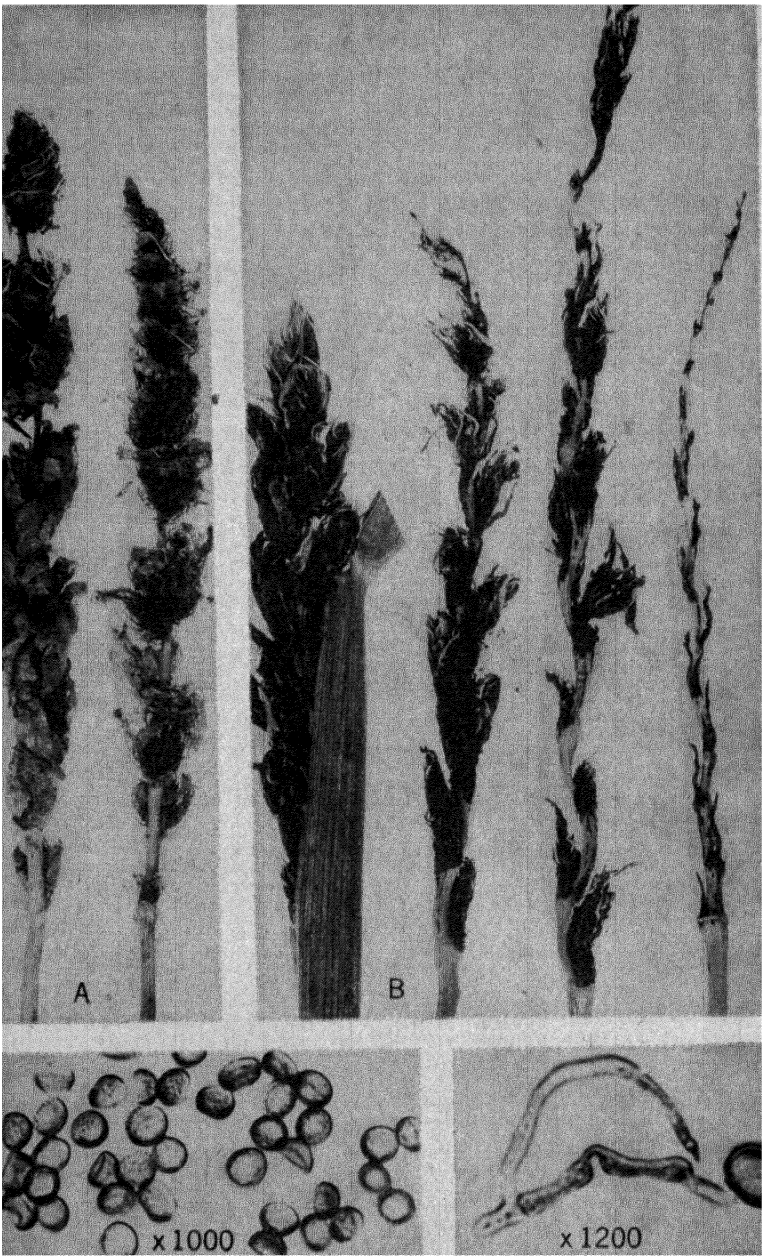

FIG. 16.—Spikes of barley (*A*) and wheat (*B*) with the spore masses of *Ustilago nuda* and *U. tritici* respectively replacing the floral tissues. Spore morphology and type of germination of the spores, greatly magnified, are shown in the inserts.

binucleate parasitic hyphae penetrate through the stigma or the young ovary wall. In most instances, the binucleate mycelium enters the suscept tissues in initiating the parasitic phase of the life cycle. The mycelium becomes established in the pericarp, remains of the integuments, and embryo tissues before the infected kernels are mature, and it remains dormant chiefly in the scutellum of the infected kernel until germination of the grain, as described by Brefeld (1903), Brioli (1913), Freeman and Johnson (1909), Hecke (1904), Hori (1907), Lang (1917), Simmonds (1946), and others. The chlamydospores usually lose their germinative power after a period of a few months, although spores stored at low temperatures frequently remain viable for longer periods, as reported by Dickson and Johann (1946), Stakman (1913), and others. Maddox (1896) was probably the first to demonstrate floral infection in studies with the loose smut of wheat. Environmental conditions, during the period the flower tissues of the barley plant are susceptible, are important in relation to infection. Humid, cool weather with light showers and dews is favorable for dissemination of spores and infection. Inoculation by introducing the spores, either dry or in suspension, into the flowers before pollination to 3 to 4 days after pollination results in high percentages of infected kernels. The following table representing the averages of a large number of inoculations on barley indicates the relation of stage of kernel development to infection.[1]

Days before or after pollination	1	Pol.	1	2	3	4	5	6
Number of spikes inoculated	10	1237	419	482	472	92	33	4
Average loose smut, per cent	43.0	34 2	25 1	20.8	20.1	9.0	9 6	3.3

The development of *Ustilago nuda* in the infected seed is resumed with the germination and growth of the seedlings. Environmental conditions show only a limited influence on the development of the fungus during the seedling stage. The mycelium enters the culm primordia and develops with the tissues during the formation and differentiation of the crown buds and finally the floral tissues where spores develop prior to spike emergence from the boot. Spores are formed in the leaves when vegetative development is rank. The spores are distributed for some days before and after pollination of the healthy flowers. Floral infection again establishes the fungus in the kernel. Seedling infection from dormant spores carried over on the seed or in the soil, as in the other smuts of the cereals, does not occur naturally.

[1] From unpublished data of H. L. Shands, University of Wisconsin, Madison, Wis.

Tisdale and Tapke (1924) described what they considered a seedling infection with loose smut. Tapke (1932, 1943) later showed this to be a loose type of smut with dark echinulate spores that germinate to form basidia and sporidia, which he described as *Ustilago nigra* Tapke. This fungus is capable of producing seedling infection. Biedenkopf (1894) first described, inadequately, this type of spore germination and considered the fungus an intermediate type of specific rank, *U. medians* Bied.

Control.—The hot-water seed treatment is the only satisfactory method of controlling the seed infection. Jensen (1888) first used the hot-water treatment for loose smut control. Kellerman and Swingle (1890), Leukel *et al.* (1938), Tapke (1924, 1926), and others have modified the treatment in an effort to obtain better smut control with the minimum reduction in seed germination. According to Dickson (1946), presoaking in cold water 6 hours and treating the wet grain 13 minutes at 53°C. (127.4°F.) does not give complete loose smut control. The loose smut infection in a large number of treated barley samples ranged from a trace to several per cent, depending upon the seed lot. Presoaking 12 to 18 hours in cold water and treating 13 minutes at 53 to 54°C. (127.4 to 129.2°F.), cooling immediately, and drying gave complete smut control without greatly reducing germination of the seed. Variety, season, and location where grown influence seed damage by the treatment.

Resistance and Specialization.—Many of the commercial smooth-awned barleys are susceptible to loose smut, but several recently developed smooth-awned selections are resistant. The Manchurian varieties, O.A.C. 21 (C.I. 1470), Minn. 184 (C.I. 2330), N. D. 2121 (C.I. 2947), and Dorsett (C.I. 4821) are moderately resistant. Trebi (C.I. 936), Tregal (C.I. 6359), and Warrior (C.I. 6991) constitute some of the more resistant commercial six-rowed varieties. Jet (C.I. 967), a two-rowed black barley, is resistant to the known races of this fungus. Resistance apparently is conditioned by several single factor pairs occurring in the different varieties with modifying factors associated with resistance in some instances, according to Livingston (1942), Nahmmacher (1932), Schaller (1946), and Zeiner (1932). Preliminary linkage data indicate that these factor pairs occur on several chromosomes. Shands (1946) has shown a close linkage between stem rust and loose smut resistance in the cross Chevron (C.I. 1111) X Trebi (C.I. 936) with resistance to each disease conditioned by a single factor pair. Specialized races of *Ustilago nuda* occur, but insufficient data are available to determine the number or their differentiation.

Specialization in the smut fungi was first demonstrated by Kniep in 1919. The difference in the use of the term in relation to the smut fungi as compared with those causing the rust and powdery mildew diseases was discussed by Christensen and Rodenhiser (1940). The

rust and powdery mildew fungi are propagated by urediospores or conidia, respectively, and thereby true biotypes are maintained. In the smut fungi the mature chlamydospores represent the diploid phase in the life cycle of the fungus which cannot be propagated independently. The chlamydospores germinate to form basidia or promycelia, which represent the beginning of the haploid phase as the reduction divisions occur during this so-called "germination" process. Therefore, the resultant cells of the promycelium and sporidia are comparable to the sporidia and ascospores of the rust and powdery mildew fungi, respectively. Before infection occurs in the smut fungi, there must be fusion between compatible haploid lines. Consequently, each new generation of chlamydospores may consist of a new group of related biotypes. For practical purposes, however, the terms "physiologic races" or "specialized races" are used in the smut fungi for collections of chlamydospores having the same general virulence on certain differential suscepts.

13. Black or Semiloose Smut, *Ustilago nigra* Tapke.—Two species, *viz.*, *U. avenae* (Pers.) Rostr. on oats and certain grasses and *U. nigra* Tapke on barley are similar in morphology, life cycle, and the symptoms they produce on the respective suscepts. The two differ only in the plants they are capable of parasitizing.

The distribution of the black semiloose smut on barley probably is extensive in Europe, Asia, and North and South America. As this smut can be differentiated from the true loose smut only by means of spore germination, the data on distribution are not extensive.

Description.—The characteristic symptoms of the black semiloose smut on barley are the relatively dark-brown to black spore mass and the variation in the range of looseness of the spore mass. This smut is not distinguishable by symptoms alone; microscopic examination of the chlamydospores and germination of the spores of the fungus are necessary. The membranes enclosing the sori vary from relatively fragile to persisting or semicovered types. There is apparently considerable variation in the membrane texture due to the influence of the suscept (Fig. 17). The smutted spikes generally appear later than in the case of the loose smut or more nearly comparable to the time of appearance of the covered smut. The spores are shed later and over a longer period than in the case of the loose smut.

The Fungus.— *Ustilago nigra* Tapke

Ustilago medians Bied. is used also as the binomial for this fungus, as discussed by Tapke (1943).

Mycelium is not formed abundantly in culture, although in certain isolates, mycelium is produced sparsely. The fungus can be carried for relatively long periods in culture by the continuous budding of the sporidia. This is especially characteristic on culture media high in nutrients. Mycelium in the tissues is hyaline at first and gradually

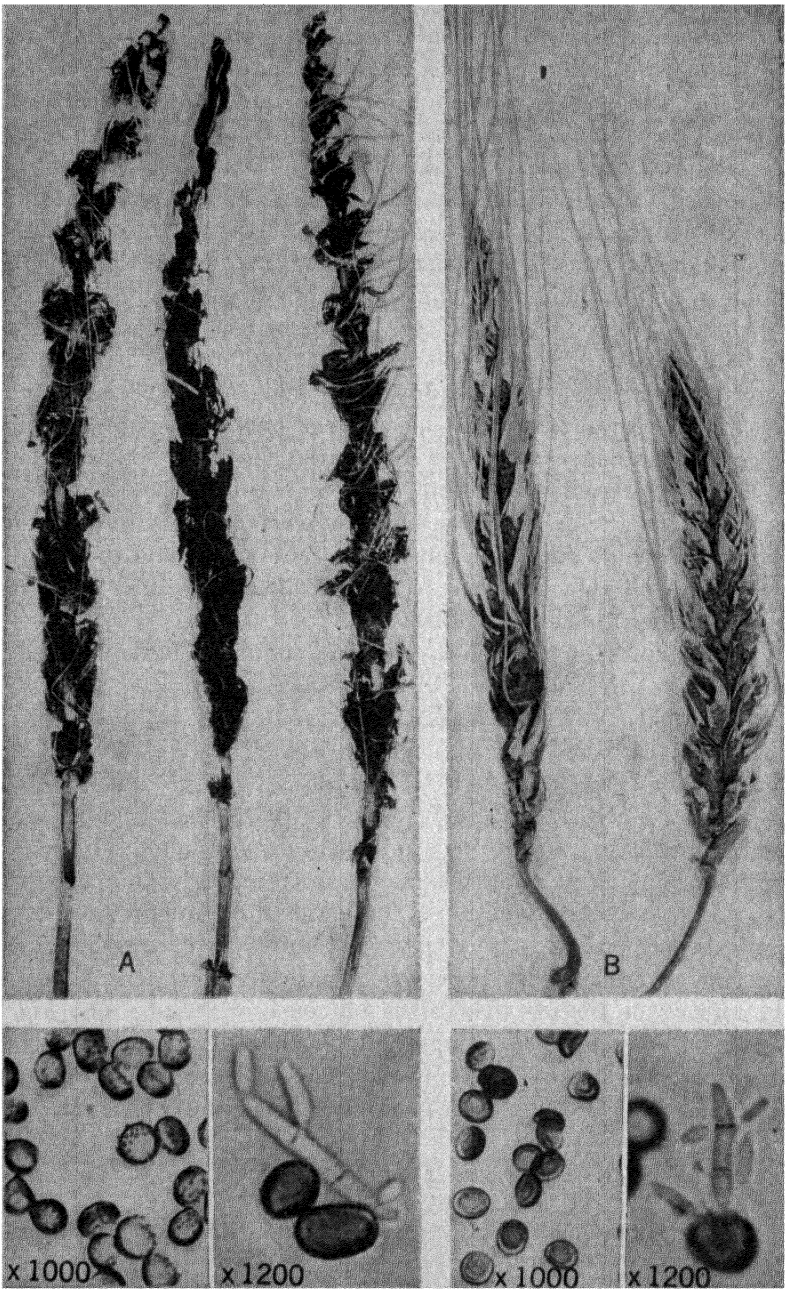

FIG. 17.—The black or semiloose smut (*A*) and the covered smut (*B*) of barley caused by *Ustilago nigra* and *U. hordei* respectively. The chlamydospores and type of germination of each are shown in the inserts.

darkens to black at maturity. Chlamydospores are formed in the floral structures, and the spore mass is covered by a membrane varying in its persistence. The chlamydospores are spherical to subspherical, 6.5 by 7 microns, dark brown to black, with echinulations varying from slight to pronounced (Fig. 17, insert *A*). The spores germinate to form a promycelium (basidium) and oblong to elongate sporidia (Fig. 17, insert *A*). The sporidia increase by yeast-like budding or function as gametes to initiate the binucleate stage of the fungus. Allison (1937) and Bever (1945) reported hybrids between this species and *Ustilago hordei* (Pers.) Lagerh.

Etiology.—The chlamydospores are carried over on the seed. The inoculum consists of spores carried on the floral bracts or frequently enclosed within the lemma and palea. The spores may be distributed during the flowering period or later during harvesting procedures. The dry chlamydospores remain viable for long periods. Spores introduced into the flowers by inoculation do not show the influence of young flowers on infection as in the case of the loose smut, as indicated in the following table.[1]

Days before or after pollination	1	Pol.	1	2	3	4
Number kernels inoculated	629	812	1012	1039	963	902
Average black loose smut, per cent	5.8	17.9	18.4	15.3	16 3	14.7

Treatment of a similar number of inoculated kernels with formaldehyde (1 part commercial formalin to 250 parts water) gave complete control of this smut. Environmental conditions during and following the heading of the barley plant have little influence on smut infection by *Ustilago nigra*. Dormant spores may be carried over in the soil under dry conditions, notably in winter barley areas.

The chlamydospores germinate under conditions favorable for the germination of the barley kernels. Sporidial formation and gametic fusions occur under the same conditions. Penetration is chiefly through the coleoptile into the growing point of the very young seedling. The seedling infection and systemic establishment of the parasite in the culm primordia are influenced by environment, as reported by Josephson (1946). The fungus mycelium develops with the growth and differentiation of the plant tissues, and finally spores are produced replacing the ovaries and the floral bracts.

Control.—This smut, as in the case of the covered smut, is controlled by the ordinary seed treatments. The organic mercury dusts, such as Ceresan, have been used extensively for the control of the seed-borne spores of this fungus. Formaldehyde, in liquid or dust form, is also fairly effective as a treatment. Inasmuch as the spores are frequently

[1] From unpublished data of L. M. Josephson, formerly University of Wisconsin, Madison, Wis.

within the floral bracts (hulls), the seed-treatment compound must be relatively volatile to be effective in the control of the seed-borne spores.

Resistance and Specialization.—The reaction of barley varieties to this fungus is different from that in the loose smut. The varietal reaction is in general more similar to that of the covered smut. The commercial varieties of barley showing considerable resistance to this smut are Manchurian varieties as a group and certain of the smooth-awned varieties, notably Newal (C.I. 6088), Wisconsin Barbless (C I. 5105), and Glabron (C.I. 4577). Certain varieties have been resistant to most races of the fungus, *viz.*, Persicum (C.I. 2448), Pannier (C.I. 1330), and Lyallpur (C.I. 3403). Tapke (1943) reported seven distinct physiological races of *Ustilago nigra* on five varieties of barley, as shown in the following table. Race 4 occurred more frequently in its distribution in the United States than all others combined.

Physiologic race	Reaction on barley varieties					
	Excelsior (C.I. 1248)	Hannchen (C.I. 531)	Himalaya (C.I. 1312)	Lion (C.I. 923)	Nepal (C.I. 595)	Odessa (C.I. 934)
1	R	R	R	R	R	S
2	R	S	R	R	R	S
3	R	R	R	I	R	S
4	R	S	R	S	R	S
5	R	S	S	I	S	S
6	S	I	I	I	S	S
7	R	S	I	R	S	S

Josephson (1946), using three additional varieties as differentials—Lompoc (C.I. 1213), Manchuria O.A.C. 21 (C.I. 1470), and Wisconsin Barbless (C.I. 5105)—differentiated nine races.

14. Covered Smut, *Ustilago hordei* (Pers.) Lagerh.—The covered smuts occurring on barley and oats are listed under *Ustilago hordei* (Pers.) Lagerh. and *U. kolleri* Wille or formerly *U. levis* (K. & S.) Magn., respectively. The covered smuts on these two cereals are similar in symptoms as well as in life cycle and morphology of the fungi.

The covered smut of barley is common on many of the cultivated varieties of barley and has been reported by Fischer (1938, 1945) on *Agropyron cristatum* (L.) Gaertn., *A. caninum* (L.) Beauv., *Elymus glaucus* Buckl., and *E. canadensis* L. in the Northwestern United States. The covered smut is world wide in its distribution and is perhaps more extensively distributed than either of the other two species on barley. The disease causes losses in the barley crop in the United States averaging approximately as much as the other two smuts combined.

Description.—The characteristic symptom of the covered smut on barley is a rather persistent membrane enclosing the sorus until the plants are fully mature. Quite frequently the grayish-white sori are enclosed within the partly modified floral bracts of the spikelet. The awn on the awned varieties also develops in most instances (Fig. 17). The smutted heads emerge at about the same time as those of the healthy plants and are conspicuous especially as the crop reaches maturity. The smut masses and dark-brown to black smooth-walled spores on the kernels are conspicuous in the threshed grain from fields where smut infection is more than a few per cent. The Federal grain grades designate such barley "smutty," and the barley grade carries the designation "smutty."

The Fungus.— *Ustilago hordei* (Pers.) Lagerh.
(*Uredo segetum* subsp. *hordei* Pers.)
(*Ustilago carbo* var. *hordei* DC.)
(*Ustilago segetum* var. *hordei* Rab.)
(*Ustilago segetum* var. *hordei* f. *tecta* Jens.)
(*Ustilago segetum* var. *tecta* Jens.)
(*Ustilago hordei* var. *tecta* Jens.)
(*Ustilago jensenii* Rostr.)
(*Ustilago hordei* Kell. and Swing.)

Mycelium in culture is sparse, most of the development being by yeast-l of the numerous sporidia. In the tissues the mycelium is at first hyaline, later turning to dark brown or black. The chlamydospores are lighter colored on one side, subspherical to angular with a smooth outer membrane, and range from 5 to 9 microns in diameter (Fig. 17, insert *B*). The spores germinate to form characteristically a four-celled promycelium (basidium) and four ovate to oblong sporidia with the abundant development of secondary sporidia (Fig. 17, insert *B*).

Etiology.—Infection of the very young seedlings occurs from the seed-borne chlamydospores. In some drier areas the infection may occur from spores in the surface soil. Penetration is through the young coleoptile into the embryonic growing point, and the further development of the parasite occurs in association with the differentiating tissues of the crown buds and floral structures especially. Spores are produced, replacing the kernel and, less commonly, the floral bracts. The seedling infection is influenced by soil environmental conditions, especially soil temperature, moisture, and to a lesser extent, soil fertility, as reported by Faris (1924), Schaffnit (1926), and others.

Control.—The seed-borne spores are controlled by the use of the organic mercury dusts as well as by formaldehyde dusts and solutions.

Resistant Varieties and Specialization.—The varietal reaction to this smut is similar to that of the black semiloose smut. There are, however, other varieties that show some differential response. The following

varieties are relatively resistant to this fungus: Persicum (C.I. 2448), Pannier (C.I. 1330), Hillsa (C.I. 1604), Lyallpur (C.I. 3403), and a hull-less barley (C.I. 2448). Specialization in pathogenicity of the barley races of this parasite has been shown by Aamodt (1935), Faris (1924), Rodenhiser (1928), Tapke (1937, 1943), and others. The reaction of 8 barley varieties to the 13 physiologic races as reported by Tapke (1937, 1945) follows:

Physiologic race	Reaction of barley varieties							
	Excelsior (C.I. 1248)	Himalaya (C.I. 1312)	Hannchen (C.I. 531)	Lion (C.I. 923)	Nepal (C.I. 595)	Odessa (C.I. 934)	Pannier (C.I. 1330)	Trebi (C.I. 936)
1	R	R	I	R	R	S	R	I
2	R	R	R	I	S	S	R	R
3	I	R	R	I	S	S	R	R
4	R	I	I	R	S	S	I	I
5	R	R	R	I	R	S	R	I
6	R	R	I–S	I	R	S	R	S
7	R	R	R	R	S	S	R	I
8	R	R	R	R	R	S	R	R
9	I	R	I	I	S	S	R	R
10	R	I	I	I	S	S	I	I
11	R	I	I–S	I	I	S	R	S
12	R	I	I	R	S	S	R	R
13	I	R	I	I	S	S	R	I

The physiologic race 6 has been reported by Tapke (1937, 1945) as the most prevalent through the spring-barley area of the United States.

15. Stem Rust, *Puccinia graminis* Pers.—Stem rust on *Hordeum* spp. is caused by one or more of three of the specialized varieties, *viz.*, *Puccinia graminis tritici* Eriks. and Henn., *P. graminis secalis* Eriks., and certain specialized races that are confined more particularly to barley. Stem rust is distributed extensively on both cultivated and wild species of *Hordeum* throughout the humid and semihumid temperate regions of the world. Losses generally are not so heavy on barley over a period of years as those occurring on wheat. Barley, however, rates second in stem rust losses in the United States and, in certain years, as in 1937, losses are very severe over relatively wide areas. The disease is more prevalent in the North Central spring-barley area of Canada and the United States than in any of the other barley sections of North America. Stem rust is also severe on barley across northern Europe and Asia. The important stem rust resistant varieties used for breeding purposes are Chevron (C.I. 1111), Peatland (C.I. 5267), and several plant selections

similar to these two varieties. Kindred (C.I. 6969) and Mars (C.I. 7015) are stem rust resistant commercial varieties. Stem rust is discussed more fully in Chap. XI.

16. Stripe Rust, *Puccinia glumarum* (Schm.) Eriks. & Henn.—The stripe rust occurs on cultivated and wild *Hordeum* spp. as well as being distributed extensively on *Triticum* spp. and many grasses. In the United States and Canada this disease is of relatively minor importance on barley, as it is restricted to the Pacific Coast and Intermountain area where most of the commercial varieties grown are moderately resistant. Considerable damage is reported on barley in northern Europe, parts of northern Asia, and in Argentina in South America.

Description.—The stripe rust on barley is different in appearance from either the leaf rust or stem rust. The linear citron-yellow uredia are conspicuous on the leaf blades and sheaths as well as on the floral bracts when conditions are favorable for infection. Frequently the uredia unite end to end to form narrow stripes extending considerable distances on the leaf blades and sheaths of susceptible varieties. The telia form narrow, fine lines, dark brown in color, and covered by the epidermis. The uredia are conspicuous from very early spring until mid-summer; the telia develop sparsely in late summer to maturity of the crop. A relatively large number of barleys are resistant to this rust, as reported by Bever (1938), Newton and Johnson (1936), Straib (1935), and others. For a complete discussion of the stripe rust see Chap. XI.

17. Leaf Rust, *Puccinia anomala* Rostr., or preferably *P. hordei* Otth.—The leaf rust of barley occurs extensively in both the winter and spring barley areas of the Eastern and Central United States. In this area the rust is found almost every season, and in some seasons it develops in epiphytotic form, especially in the southern spring-barley area. This rust is distributed generally in most of the countries where barley is grown. *Puccinia hordei* is probably confined to cultivated barley species and a few closely related wild grasses. The aecial host of this rust fungus is found on *Ornithogalum umbellatum* L. (the common star-of-Bethlehem) and some other species, as reported by Mains and Jackson (1924) and Tranzschel (1914). Although *O. umbellatum* is distributed generally, the aecial infection is uncommon in North America. The main distribution of the rust apparently is a northward spread of uredial inoculum from the southern winter-barley area. Certain races of the wheat leaf rust fungus, *P. rubigo-vera tritici* (Eriks.) Carleton, also infect barley in the Mississippi Valley area and westward (Johnston, 1936).

Description.—The uredia are small, round, and light yellowish-brown in color. This rust is relatively inconspicuous until uredial development is quite abundant. The telia are round to oblong, brown, and covered by the epidermis. The telial stage is less abundant, especially in the

more northern sections of the barley area. Both uredia and telia develop on the leaf blades and leaf sheaths and rarely occur on the floral structures.

The Fungus.—*Puccinia hordei* Otth. or *P. anomala* Rostr.
(*Puccinia rubigo-vera simplex* Koern.)
(*Puccinia simplex* Eriks. and Henn.)
(*Aecidium ornithagaleum* Bubak.)

Buchwald (1943) has shown that *Puccinia hordei* Otth. was used in 1871, and *P. anomala* Rostr. in 1878.

The pycnia and aecia occur as elevated light orange-yellow areas on the leaves of *Ornithogalum*. The aeciospores are globoid, hyaline, and minutely verrucose. The uredia are round, small, yellowish brown in color, and they show little or no rupturing of the epidermis. The urediospores are broadly ellipsoid, light yellow in color, finely echinulate, with germ pores distributed on all faces of the spore. The telia are round to oblong, are covered by the epidermis, and produce mainly one-celled spores. The teliospores germinate to form characteristically the four-celled basidium and sporidia.

Etiology.—In the more southern winter-barley regions, the leaf rust develops in the uredial stage throughout the winter growing period. The urediospores do not overwinter in the spring-barley area. The northward spread of the uredial infection is prevalent as the crop develops. Infections in the more northern sections frequently are not evident until relatively late in the spring or early summer. Secondary spread from urediospores is abundant during warm, humid, summer weather. Telial development is limited in the spring-barley area. The aecial stage does not develop abundantly, naturally in the United States, although Mains and Jackson (1924) reported it in Indiana, and it is produced at Madison, Wis., when barley straw containing telia is placed near *Ornithogalum umbellatum*.

Control and Resistance.—The economical control of this rust is through the use of resistant varieties. Many of the cultivated barleys grown in the United States are moderately resistant. Barleys resistant to the leaf rust are Callas (C.I. 2440), Mecknos Moroc (C.I. 1379), Peruvian (C.I. 935), Quinn (C.I. 1024), Bolivia (C.I. 1257), Juliaca (C.I. 1114, C.I. 2329), and many others in the barley collection of the U. S. Department of Agriculture. Two selections of Orge, —B100 and —B101, were resistant to the two physiologic races in the United States and to the Australian race. Two physiologic races of *P. anomala* have been reported by Mains and Martini (1932) in the United States, and Waterhouse (1928) reported one additional race in Australia.

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CHAPTER IV

CORN DISEASES

Corn, or maize, is represented by the single species, *Zea mays* L. All the cultivated varieties of corn are included in this species, and no wild or uncultivated forms are known. Botanical varieties or subspecies of *Zea mays* are pod corn, *Z. mays* var. *tunicata*; flour corn, *Z. mays* var. *amylacea*; flint corn, *Z. mays* var. *indurata*; pop corn, *Z. mays* var. *everta*; sweet corn, *Z. mays* var. *saccharata*; and dent corn, *Z. mays* var. *indentata*. Dent corn comprises the major acreage in the United States and flint corn in South America, Europe, and Asia. The United States grows approximately three-fourths of the world's supply of corn.

The basic chromosome number in corn is 10 pairs. The inheritance of some 350 genes is known, as reported by Emerson, Beadle, and Fraser (1935), Jenkins (1936), Mangelsdorf and Reeves (1939), and others. The extensive development of inbred lines of corn stimulated by the practical program of hybrid corn production has resulted in the isolation and stabilization of many characters, including some conditioning disease resistance. Practical disease control for seedling blights, root and stalk rot diseases, ear rots, smut, and a few other diseases is being accomplished through resistance in the development of inbred lines. In some instances, diseases of minor importance have become ephiphytotic in areas where susceptible hybrids have been distributed, as in the case of bacterial blight and Helminthosporium blight.

Corn is a warm-climate annual. However, it is grown under a wide range of environmental conditions due to its great adaptability. Changes in the length of growing period enable the crop to be grown economically from the frost-free southern tropics to an 80-day, frost-free period in the Northern United States and southern Canada. The use of hybrid corn is increasing the productivity for both grain and silage over this wide geographical range.

Corn is damaged by a relatively large number of diseases. The developmental anatomy and physiology of the plant play an important role in many of these diseases. Estimated annual losses in the United States for the 10-year period 1930 to 1939 was 12.5 per cent of the annual grain crop, or over 250 million bushels (Plant Disease Survey). The losses in sweet corn, an important canning and garden crop in the United States, averaged 9.5 per cent, or over 38 thousand tons of canning corn. These

losses are being reduced appreciably in recent years with the development of better adapted disease-resistant hybrids.

1. Nonparasitic Diseases.—The nonparasitic diseases in corn are manifest by various combinations of symptoms. Usually stalk and leaf symptoms are closely associated. The symptoms under field conditions are frequently similar to those caused by parasitic organisms. The group of maladies is divided into two general classes based upon cause: (1) temperature and moisture and (2) mineral deficiencies.

Low temperatures, especially during the period of seedling growth, produce chlorophyll disturbances and retarded growth. Many of the virescent types are expressed more commonly at low light intensity and at low temperatures (Demeric, 1924, Smith, 1935). Continued low temperatures result in browning of leaf tissue, depletion of endosperm reserves, and blighting of seedlings by soil-borne organisms. Warm weather usually results in the formation of chlorophyll in the younger leaves and recovery, although the plants frequently are retarded in growth. Low temperatures and frost before the mature corn is dried fully cause a bleached wrinkled pericarp and low or weakened germination (Kiesselbach and Ratcliff, 1918).

High temperatures and drought cause firing of upper leaves and tassels. The affected tissues are conspicuous as they bleach and dry out.

Corn is relatively sensitive to certain mineral deficiencies or balance of inorganic elements. Leaf spotting, blotching, or chlorosis is frequently accompanied by necrosis of stalk tissue below the apical growing point. In other instances, deposits of inorganic-organic complexes occur in the basal stalk tissues. External symptoms are usually reduced internodal elongation, yellow to brown leaf lesions, and barren stalks or nubbin ears. This condition occurs commonly on acid marsh or peat soils or on soils high in soluble aluminum and iron compounds. Low potash, magnesium, and other elements are associated with the condition. Abbott *et al.* (1913), Hartwell and Pember (1918), Hoffer and Carr (1923), Jones (1929), and Magistad (1925) have investigated this type of injury in corn.

2. Mosaic, Streak, and Stripe, Infectious Viruses Transmitted by *Aphis maidis* (Fitch) and Several Other Aphids, *Cicadulina* Spp. and *Peregrinus maidis* (Ashm.), Respectively.—Corn, sugar cane, sorghums, and related grasses are damaged by a complex of virus diseases. Some of these are transmitted to a number of crop plants in these groups, while others are restricted in range of suscepts. Damage from these diseases apparently is limited to the warmer climates or outside the main corn-producing areas. The maladies are common on corn in the sugar-cane sections and their distribution on corn is essentially the same as on sugar cane (Brandes 1920, Brandes and Klaphaak, 1920, Cook, 1936, Stahl, 1927, Storey, 1928 and 1937, Storey and McClean, 1930).

The symptoms of the various maladies are somewhat similar. The numerous mosaic strains are manifest on corn by dark-green or light-green mottling and striping of the leaves and epidermis of the stalks. On the older leaves, the symptoms are less conspicuous and characteristic. The corn plants are dwarfed by several of the mosaics, and ear production is reduced. Phloem necrosis is common in the corn mosaics. Intracellular bodies were reported by Kunkel (1921). The streak and stripe diseases are differentiated by the green or yellow fine stripes with definite margins and the absence of mottling on the leaves.

Several viruses are associated with these diseases. The corn mosaic viruses are transmitted by *Aphis maidis* (Fitch) (Brandes 1921), at least three other aphids, and perhaps by *Peregrinus maidis* (Ashm.). Apparently, the aphids persist on sugar cane to infect the annual corn and sorghum. Continuous growing of corn in the mild climates enables the aphids to develop continuously on corn. Certain of these viruses are transmitted by mechanical means. Storey (1928, 1933) reported a streak disease on corn and sugar cane transmitted by the leaf hoppers *Cicadulina mbila* (Naude), *C. zeae* China, and *C. storeyi* China. Stahl (1927) differentiated stripe from sugar cane mosaic and demonstrated its transmission by *P. maidis*. Carter (1941) reported the transmission of corn mosaic by this leaf hopper, although the stripe disease may have been involved.

These diseases are controlled best by resistant varieties as in the sugar cane. However, relatively little breeding for resistance in corn is reported to date. Certain Peruvian flint varieties are resistant to the streak disease (see also Chap. X).

3. Bacterial Wilt (Stewart's Disease), *Bacterium stewartii* E. F. Sm.—The bacterial wilt of corn is a destructive disease of susceptible varieties of sweet, flint, and dent corn. Before the use of resistant varieties, large acreages of sweet corn in the United States were devastated by epiphytotics of this disease. The wilt earlier was largely responsible for the northern shift in the canning-corn acreage in this country. The use of the resistant single-cross hybrid, Golden Cross Bantam, and other resistant hybrids, has reduced this hazard and made possible the economical production of the Bantam-type sweet corn in the corn-belt area (Smith, 1933).

The disease is common in the warmer areas. It occurs infrequently in the northern tier of states in the United States. It has been reported from Mexico, Puerto Rico, South Africa, and Italy (Elliott, 1941). The major damage is confined to the United States, where sweet corn is an important commercial crop.

Symptoms and Effects.—The disease is a typical bacterial vascular wilt. The bacteria develop in a gelatinous matrix inside the vascular

bundles of the stalks, inflorescences, and leaves. The bacterial mass breaks out of the bundles in the later stages of disease development. In susceptible varieties of sweet corn, the disease is a typical wilt, especially

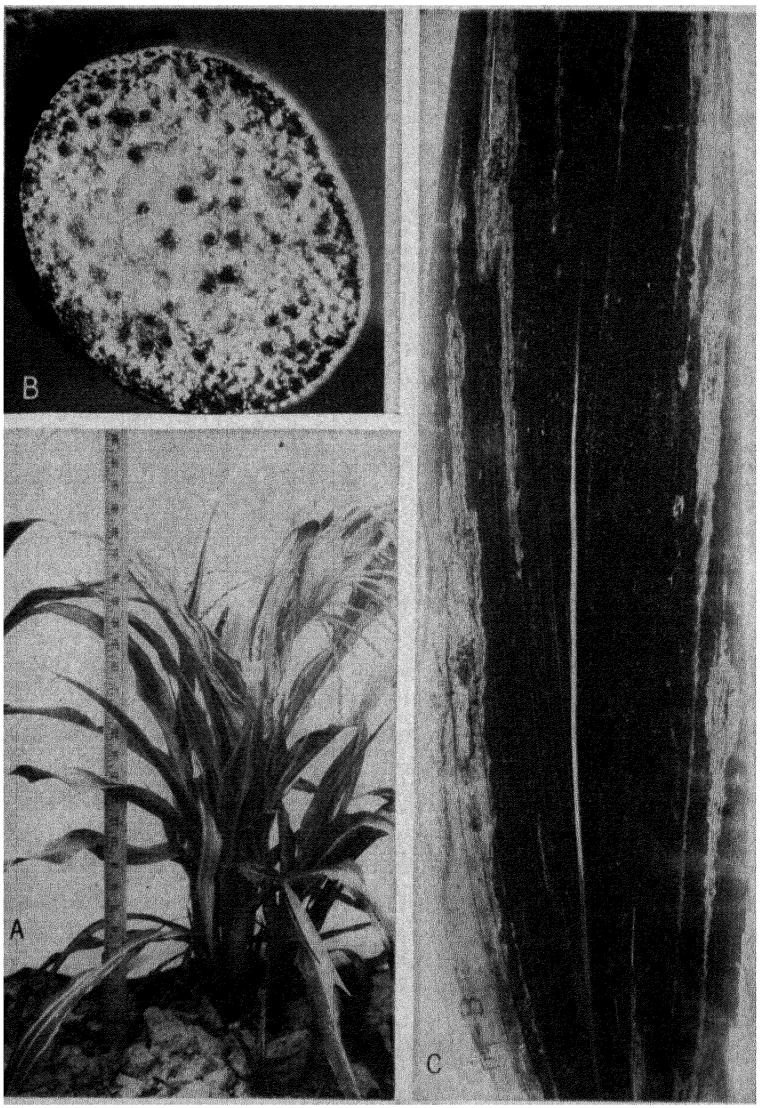

FIG. 18.—Bacterial wilt of Golden Bantam sweet corn (*A*) and bacterial lesions on a blade of dent corn (*C*). Bacterial exudate in bundles is shown in (*B*). (*Courtesy of Bureau of Plant Industry, Soils, and Agricultural Engineering, U. S. Department of Agriculture.*)

in the early stages of plant growth. Plants not killed are stunted, form tassels and nubbin ears early, and show considerable leaf necrosis. Later

leaf infections from insect injuries result in pale-green wilted streaks, which frequently extend into the stalk (Fig. 18). On dent corn the leaf lesions are the characteristic symptom. Abundant lesions result in the killing of the leaf blade. The yellow bacterial exudate is conspicuous in the vascular bundles of stalks and leaves. In dent corn, stalk infection and the formation of the exudate is not so extensive as in the more susceptible sweet corn varieties.

The Bacterium.—*Bacterium stewartii* E. F. Sm.
[*Phytomonas stewartii* (E. F. Sm.) Bergey *et al.*]
(*Pseudomonas stewartii* E. F. Sm.)
[*Aplanobacter stewartii* (E. F. Sm.) McCull.]

The nonmotile rods with rounded ends develop in a gelatinous matrix.

Etiology.—The corn plants are infected either from the seed or through the feeding injuries of insects carrying the bacteria. Apparently, infection direct from the soil is not common. The corn flea beetle is the common insect carrier of the inoculum both in primary and secondary infections (Elliott, 1941, and Rand and Cash, 1924). Once inside the tissues, the bacteria develop in the conductive tissues, frequently filling the vessels with the bacterial mass. The bacteria spread through the vascular bundles to tassels and ears. Kernel infection occurs under favorable conditions. The bacteria imbedded in the matrix survive the winter in diseased stalk tissues. This source of inoculum apparently is not important in initiating the infection the following year, as infested beetles carry the bacteria for long periods inside their bodies. From 10 to 20 per cent of the beetle populations carry the bacteria when they come out of hibernation in the spring. The infestation and infection increase simultaneously during the summer. Bacterial lesions, following beetle feeding on dent corn, develop abundantly after the plants are in tassel. This is the period when beetle infestation usually is highest.

Resistance to bacterial wilt is the best means of control. Usually the early-maturing varieties of corn are more susceptible. The majority of the resistant inbred lines and hybrids possess a type of resistance associated with height and lateness of the plants. A few highly resistant lines have a type of resistance not correlated with either height or lateness (Ivanoff and Riker, 1936). Golden Cross Bantam is the most resistant yellow sweet corn hybrid in extensive commercial production. A few dent corn hybrids are very susceptible to bacterial wilt.

4. Cobb's Disease of Sugar Cane, *Xanthomonas vasculorum* (Cobb) Dows. [*Phytomonas vasculorum* (Cobb) Bergey *et al.*].—Ivanoff (1935) inoculated corn, sugar cane, and sorghum with *Xanthomonas vasculorum.* Symptoms and development of the disease were similar to wilt, although the organism was sufficiently different to retain the species. The discussion of this disease is given in Chap. X.

5. Bacterial Blights.—Several bacterial blights have been described on corn. Certain of these are more common on sorghums and related crops than on corn. Local water-soaked lesions occur on leaf sheath and culm tissues. The discussion of this group of diseases is included in Chap. IX.

6. Physoderma Brown Spot, *Physoderma zea-maydis* Shaw.—Corn and teosinte are the only known hosts of this parasite. The disease is of minor importance in the extreme southern portion of the corn belt of the United States and other warm humid regions of corn culture (Eddins, 1933, Tisdale, 1919, 1920). The lesions occur on leaf and stalk tissues. The oblong to round lesions are slightly water-soaked, light-green, later turning reddish brown. The small spots coalesce to form brown blotches, especially at the base of the leaf blade and adjacent sheath tissue. The lesions also occur on the stalk beneath the leaf sheath. Later the epidermis and parenchyma collapse to form small pockets of dusty-brown sporangia in the leaf sheath and stalk tissues. The stalks frequently break at these infection centers.

The Fungus.—*Physoderma zea-maydis* Shaw.

The sporangia are smooth, brown, flattened on one side, and measure 18–24 by 20–30 microns. The lid of the sporangia opens on germination, liberating the uniciliate zoospores, 3 to 4 microns wide by 5 to 7 microns long. The fusion of gametes and the formation of a true sporangium is described by Sparron (1934).

Etiology.—The fungus develops as an obligate parasite on corn, and the spores persist in the old stalks. The sporangia are distributed by wind and other agencies. Those falling within the leaf whorl and leaf axils germinate to form zoospores. The zoospores become attached to the young corn tissues, usually within the leaf whorl, and form an infection hypha that enters the mesophyl or parenchymatous cells. Larger vegetative cells develop from the fine mycelium, and sporangia are formed which ultimately fill the cell. Sporangia are discharged when the tissues disintegrate, or they are carried over unfavorable conditions in the corn tissues. Abundant moisture and high temperature are essential to the development of the disease (Tisdale, 1919, Voorhees, 1933).

Control is largely through sanitation and the use of resistant hybrids, according to Eddins and Voorhees (1935).

7. Pythium Root Rot, *Pythium arrhenomanes* Drechs. and *P. graminicolum* Subr.—Species of *Pythium* are parasitic on the rootlets of many of the grasses as well as other crops throughout the world. The behavior on this former group of suscepts is somewhat different from that on the dicotyledonous crop plants, mainly because of plant structure and development. Root systems are depleted from the seedling stage until maturity. Under favorable conditions, seedling blight of corn is more common than damping-off; the latter being more general in seedlings with small

delicate hypocotyls. This group of fungi is associated with crop sequences and soils high in moisture.

The disease is common on corn, although not so destructive as it is on sugar cane, sorghums, and related plants. The root rot of corn is widely distributed, from the tropical to northern limits of corn culture and extending into the heavier soils of even the semihumid areas (Branstetter, 1927, and Rands and Dapp, 1934).

Symptoms and Effects.—The disease is primarily a rootlet rot. Light-brown water-soaked lesions develop on the finer rootlets and at the root ruptures. In the earlier stages of germination, the rot is in close proximity to the seedling tissues; at later stages it may be some distance from the crown. The rot advances into the main roots and seedling or crown tissues in wet, cold soils. Lobulate sporangia occur in the cortex of the rotted tissues, usually near the surface. Later, oöspores are formed throughout the invaded tissues. In wet, cold soils seedling blight occurs, as reported by Johann *et al.* (1928) and others (Fig. 19). Plating on tissue media is useful in identification of the species involved (Johann, 1928).

The Fungus.—*Pythium arrhenomanes* Drechs.
(*Pythium butleri* Subr.)
[*Pythium aphanidermatum* (Eds.) Fitzp.]
(*Rheosporangium aphanidermatum* Eds.)

Drechsler (1936) retains the species *P. graminicolum* Subr. as morphologically distinct from *P. arrhenomanes*, although they are similar morphologically. Both attack corn, sugar cane, sorghum, etc.; the former is more common on the small grains.

The differentiating morphology of these two species is given by Drechsler (1936) and Middleton (1943). *Pythium arrhenomanes* forms numerous large lobulate sporangia in tissues or culture. Oögonia are formed in tissue with crook-necked antheridia and remote connections between antheridia and oögonial stalks. Numerous antheridia are attached to the oögonium. The antheridial walls do not persist and in the mature oöspore are nearly indiscernible (Fig. 19). This is in contrast to the close mycelial connection between antheridium and oögonium, fewer antheridia attached to an oögonium, and the persistence of the antheridial membranous parts on the wall of the mature oöspore in *P. graminicolum*. Elliott (1943) described a stalk rot of corn caused by the similar species, *P. aphanidermatum* (Eds.) Fitzp. (*P. butleri* Subr.).

Etiology.—The mycelium develops in the soils in association with crop residue and invades the plant roots whenever environmental conditions are favorable (Carpenter, 1934). The fungus develops in root tissues throughout the growing season.

Control.—Crop sequence and soil preparation, including balanced fertility, are remedial measures. The use of resistant varieties and hybrids is becoming an important control measure, as reported by Bowman *et al.* (1937), Johann *et al.* (1928), and Rands and Sherwood (1927). Early seedling and root infections in infested soils are prevented by seed treatment with Arasan.

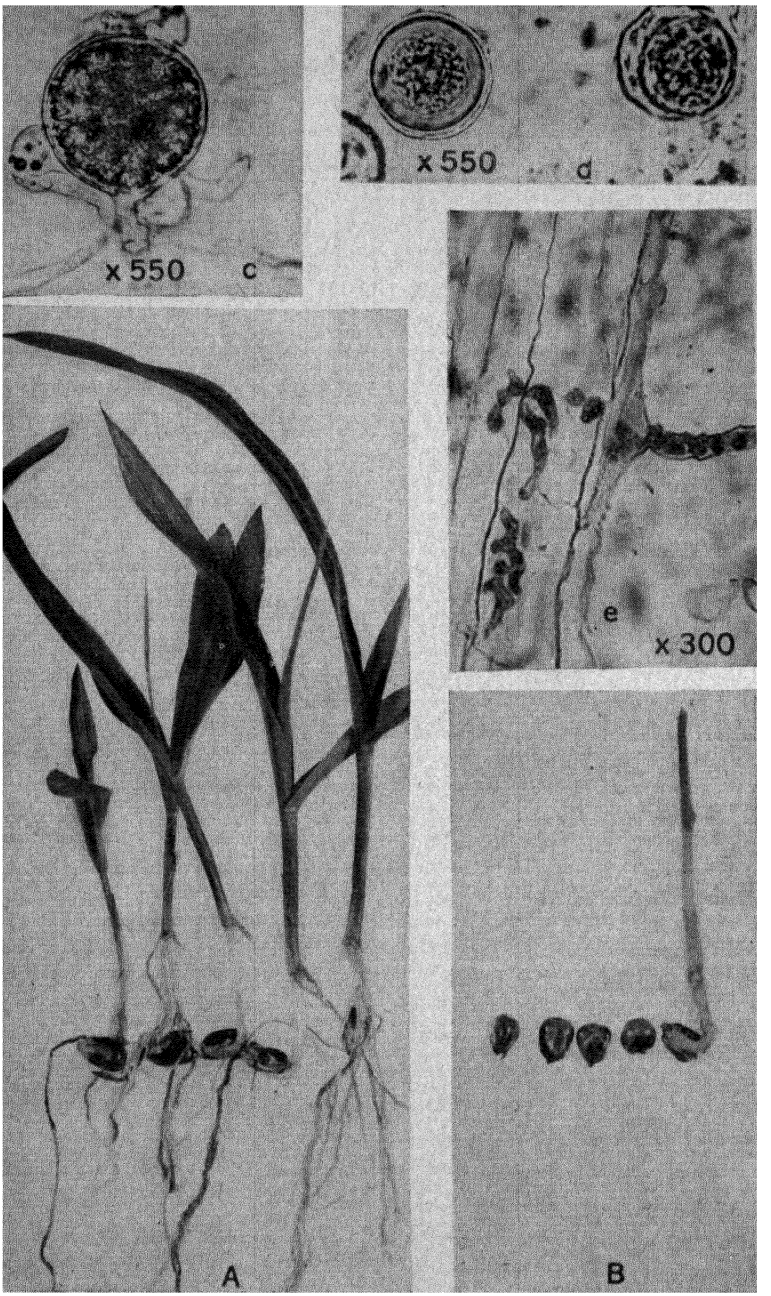

FIG. 19.—(*A*) Pythium root rot of corn in a cold dry soil and (*B*) seedling blight in a cold wet soil. The lobulate sporangia in the roots (*e*), oögonium (*c*), and oöspores (*d*) of *Pythium arrhenomanes* are shown, highly magnified, in the inserts. (*Courtesy of Helen Johann.*)

8. Downy Mildews, *Sclerospora philippinensis* Weston and Other Species.—Corn is attacked by several species of *Sclerospora*. Only three are of major economic importance on this crop. The disease on corn is of economic importance only in tropical areas, especially the South Pacific Islands, southern Asia, and South Africa. Infection by *Sclerospora graminicola* (Sacc.) Schroet. is reported occasionally on corn in the temperate zones. The age of the plant, tissues invaded, and environment all modify the symptoms. White to yellow streaking of the leaves, development of the downy mildew on the streaks, followed by necrosis and browning of the streaks, are the characteristic leaf symptoms. Dwarfing, reduced elongation of upper internodes, suckering, and retarded development of the plants are typical where the infection occurs early, as reported by Butler (1913), Weston (1920). According to Melhus *et al.* (1928), the symptoms on corn caused by *S. graminicola* are similar to those caused by the other species.

The Fungi.—Several species occur on corn in various parts of the world.

Sclerospora philippinensis Weston. Philippine species on corn
S. maydis (Rac.) Butl. Java species on corn
(*S. javanica* Palm)
S. indica Butl. and Bisby. Indian species on corn
S. spontanea Weston. Philippine on *Saccharum spontaneum* and corn
S. sacchari Miyake. South Pacific on sugar cane and corn
S. sorghi (Kulk.) Weston and Uppal. Asia, Africa on sorghum and corn
S. graminicola (Sacc.) Schroet. General on *Setaria* spp. and corn
S. macrospora Sacc. General on Gramineae

According to Weston (1920, 1921), the first three species are characterized by the conidial stage only having been found. An oöspore stage of a Sclerospora occurs on *Saccharum spontaneum* L., but it has not been connected definitely with *S. spontanea*. The morphology of the conidial stage of *S. philippinensis*, *S. maydis*, and *S. sacchari* is similar. Conidiophores are erect, varying slightly in length and shape for the different species, and all three have a conspicuous basal cell. They are dichotomously branched, with the conidia borne on rather long conoid-subulate curved sterigmata. Conidia are ellipsoid to ovoid, slightly rounded at the apex, and vary in size in the different species (see following table). The conidia germinate by forming a germ tube (Uppal and Weston, 1936). Leece (1941), Lyon (1915), and Miyake (1911) reported oöspores of *S. sacchari* on sugar cane. According to Weston (1921), the conidia of *S. spontanea* are narrower and longer than in the other species. The species *S. sorghi*, occurring on sorghum and corn in southern Asia and South Africa, is intermediate in morphology between *S. graminicola*, described in Chap. V, and the tropical group of species, as suggested by Weston and Uppal (1932). The conidiophores are erect, with a bulbous basal cell and branch in regular close succession. The sterigmata are tapering and long (16 microns). The conidia are broadly rotund, the ends are bluntly rounded, and they germinate by the formation of a germ tube. The oögonial stage is similar to *S. graminicola*. *S. macrospora* is discussed in Chap. X.

Etiology.—The group of species, occurring in tropical climates, persists in the mycelial and conidial stage. Primary and secondary infections from conidia occur when weather conditions are favorable. Epiphytotics are common on corn and less common on sugar cane. The sorghum downy mildew extends into the drier areas, as the oöspores are long-lived and produce primary infections on the new crop. No very satisfactory control measures have been reported. Some differences in response of varieties are evident.

A summary table of the *Sclerospora* species on the cereals and grasses appears on the opposite page.

9. Kernel Mold, Scutellum Rot, and Seedling Injury, *Rhizopus*, *Aspergillus*, and *Penicillium* Spp.—This type of damage to corn is common in the more humid areas throughout the world. The disease is associated with the maturation and moisture loss in the field and with storage conditions after harvest. Losses from this type of damage effect yield somewhat, but more especially the quality of the grain. The organisms involved are generally secondary, as they are semiparasitic in nature. The primary cause is immature corn of high moisture content left in the field too long or stored with high moisture.

The symptoms of this type of damage are not easily differentiated from kernel damage by Gibberella, Fusarium, and Diplodia. The mold damage in the kernel is more superficial, with less rotting of the kernel and a characteristic moldy odor. Frequently, the two groups of diseases are combined. Damaged corn under the Federal grain standards includes both types. Koehler and Holbert (1930, 1938) and others have discussed the scutellum rot in which these organisms invade the scutellum and adjacent endosperm to damage the stored reserves and weaken the embryo.

Damage of this type in seed corn is reduced by early harvesting and artificial drying. Better drying and storage facilities for the commercial corn crop are necessary and economical in preventing this type of loss. Corn hybrids that mature and dry rapidly in the field are important in reducing the damage.

10. Gibberella Ear Rot, Kernel Rot, Stalk Rot, and Seedling Blight, *Gibberella* and *Fusarium* Spp.—Two species of *Gibberella* are common on corn, and they produce different symptoms as well as differing in their distribution. *Gibberella zeae* (Schw.) Petch [*G. saubinetii* (Mont.) Sacc.] causes a pink ear rot, stalk rot, and seedling blight on corn. This fungus also occurs commonly on the other cereal crops where it causes more damage perhaps than on corn. In the United States, *G. zeae* is distributed on corn and wheat, barley and rye through the eastern and central sections of the corn belt, and it is less common in the southern and western corn-producing sections. *G. fujikuroi* (Saw.) Wr. produces a kernel rot and stalk rot of corn throughout the entire corn belt and southward into

	Sclerospora spp.							
	S. macrospora	*S. graminicola*	*S. sorghi*	*S. noblei*	*S. spontanea*	*S. philippinensis*	*S. maydis*	*S. sacchari*
Plants reported suscepts	*Triticum* spp. *Zea mays* *Avena* spp. *Secalis* sp. *Oryza sativa* *Bromus commutatus* *Phalaris* spp. *Phragmites* spp. *Glyceria* spp. *Andropogon* spp. *Panicum* sp. *Lolium* spp. *Alopecurus* spp.	*Setaria viridis* *S. italica* *S. magna* *S. glauca* *S. verticillata* *Zea mays* *Euchlaena mexicana* *Sorghum vulgare* *Saccharum officinarum* *Pennisetum* spp.	*Sorghum vulgare* *S. arundinaceum* *Zea mays* *Euchlaena mexicana*	*Sorghum plumosum* *Andropogon australis*	*Saccharum spontaneum* *S. officinarum* *Zea mays* *Euchlaena luxurians* *Miscanthus japonicus* *Sorghum* sp.	*Zea mays* *Euchlaena luxurians* *Sorghum vulgare* *Saccharum spontaneum* *Miscanthus japonicus*	*Zea mays* *Teosinte-corn hybrids*	*Saccharum officinarum* *Zea mays* *Euchlaena* spp.
Common suscepts in North America	Wheat, *Bromus*, *Avena*	Millets	Unknown	Unknown	Unknown	Unknown	Unknown	Unknown
Distribution	General	General	South Asia, Africa	Australia	Philippines	Philippines	Java	South Pacific
Conidiophores: length, microns	Reported by Peglion but not common	268	180–300	300–450	350–550	150–400	150–300	190–280
Basal cell		Absent	Present	Present	Present	Present	Present	Present
Conidia, microns		14–23 × 11–17	15–29 × 15–27	21–37 × 13–29	39–45 × 15–17	17–57 × 11–27	28–45 × 16–22	25–41 × 15–23
Conidial germination	Zoospores	Zoospores	Germ tube	Germ tube	Germ tube	Germ tube	Germ tube	Germ tube
Oöspores, microns	60–65	30–60	31–69	19–35	Connection not certain	Unknown	Unknown	Oögonia 11–23
Germination	Germ tube	Germ tube	Germ tube	Germ tube				

* In addition to the species listed in the table, the following species have been described: *Sclerospora miscanthi* Miya. on species of *Miscanthus* and *Saccharum* from Japan and the Philippines with oöspores (47 to 49 microns) only described, *S. farlowii* Griff. on *Chloris elegans* from the Southwestern United States with oöspores (28 to 45 microns) the only stage known, *S. magnusiana* Sorok. on Equisetum from Russia, *S. butleri* Weston on *Eragrostis aspera* from Nyasaland, *S. northi* Weston on *Erianthus maximum* from Fiji, and *S. oryzae* Brizi on rice from Formosa.

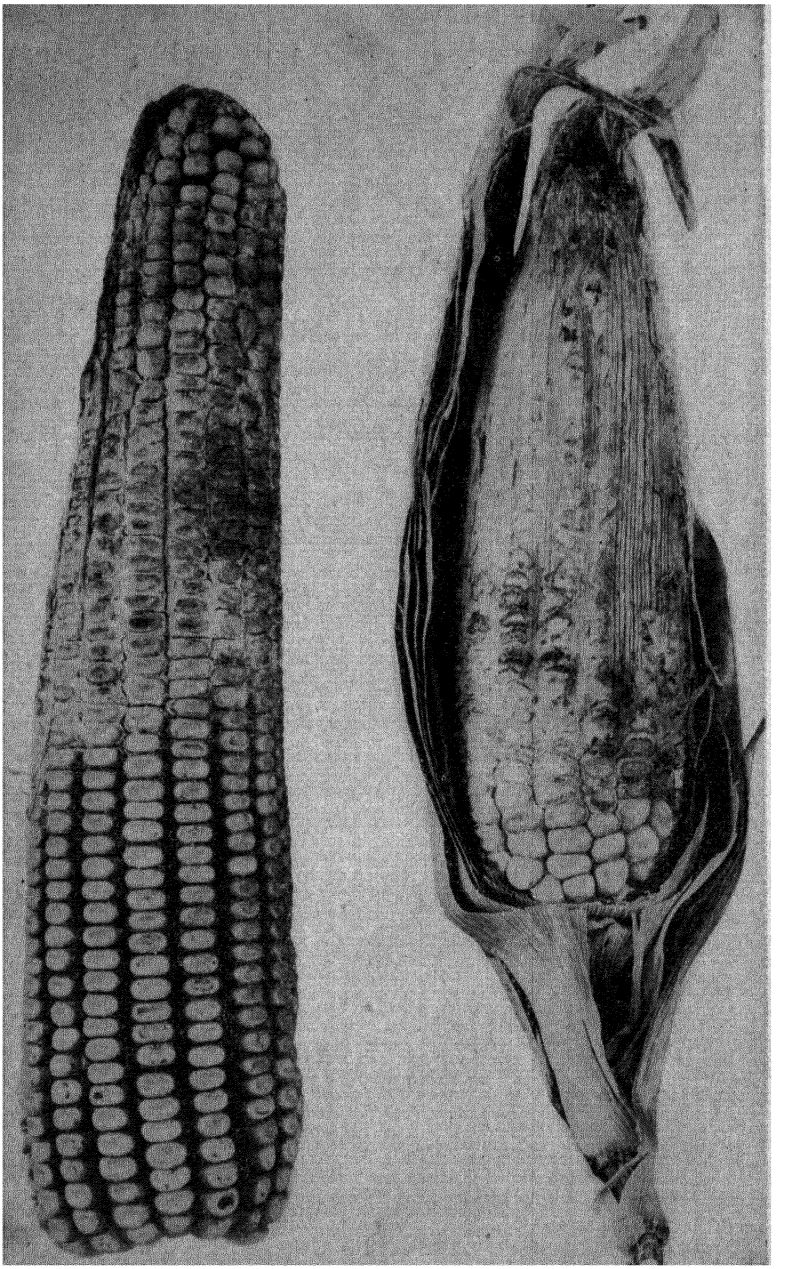

FIG. 20.—Gibberella or pink ear rot of corn caused by *Gibberella zeae:* left, tip portion of the ear rotted; right, rot involving the entire ear.

the subtemperate and tropical zones. The distribution of the two species is similar in the other corn-producing areas of the world; the former species being common in the humid temperate zones, the latter extending over the humid and semihumid temperate zones and into the subtemperate and tropical zones (Blattny, 1931, Edwards, 1937, Maher, 1931, Mendiola, 1930, Nishikado, 1933, Stevens and Wood, 1935, and others).

Symptoms and Effects.—The symptoms of the ear rots vary with the fungus and severity of attack. The Gibberella ear rot caused by *G. zeae* is typically a pink or reddish rot progressing from the tip of the ear downward. A small portion of the tip to all the ear is rotted, depending upon age of the plant when infection occurs and the environmental conditions (Fig. 20). The Fusarium kernel rot caused by *G. fujikuroi* is typically a rot of individual or groups of kernels. The symptoms produced by *G. fujikuroi* and *G. fujikuroi subglutinans* Ed. are similar. The color of the rotted kernels is pink to reddish brown or gray, depending upon the general prevalence of mycelium of the fungus and weather conditions (Fig. 21). In both types of rots, the symptoms are evident after the husks are removed.

The diseased kernels are not all distinguishable in the shelled grain. The badly rotted kernels are pink to reddish brown, with mycelium conspicuous on the surface. The less damaged kernels are evident by a lusterless pericarp surface and shrunken, sometimes brown, area over the embryo. Many kernels infected with these fungi show no external symptoms. Plating the kernels on agar or germinating the kernels in a moist atmosphere soon shows the pinkish-white mycelium on the kernel surface. Grain infected with *Gibberella zeae* causes emesis in pigs and humans.

The stalk rots, more commonly, are associated with the roots, crown, and lower nodes of the stalk. They cause reddish lesions, premature ripening, and stalk breaking. Local sheath and node lesions, pinkish in color, occur frequently with stalk rotting. *Gibberella zeae* frequently causes premature ripening of the stalks, but both fungi develop aggressively on the dead stalks late in the autumn or in the following spring. Bluish-black round perithecia develop abundantly on these stalks in the late autumn in mild climates or in the following spring in the main corn belt of the United States (Fig. 22). The perithecia of *G. fujikuroi* and the variety *G. fujikuroi subglutinans* Ed. are not so common in the United States as they are in more tropical areas (Edwards, 1935, 1936, Ullstrup, 1936).

The seedling blight of corn is characteristically a water-soaked rotting of the cortical tissues. The blight occurs before emergence or when the seedlings are in the first to third leaf stage, rarely later. Brown water-soaked lesions on the subcrown internode and roots are characteristic, as described by Dickson (1923), Pearson (1931), Voorhees (1933). Environ-

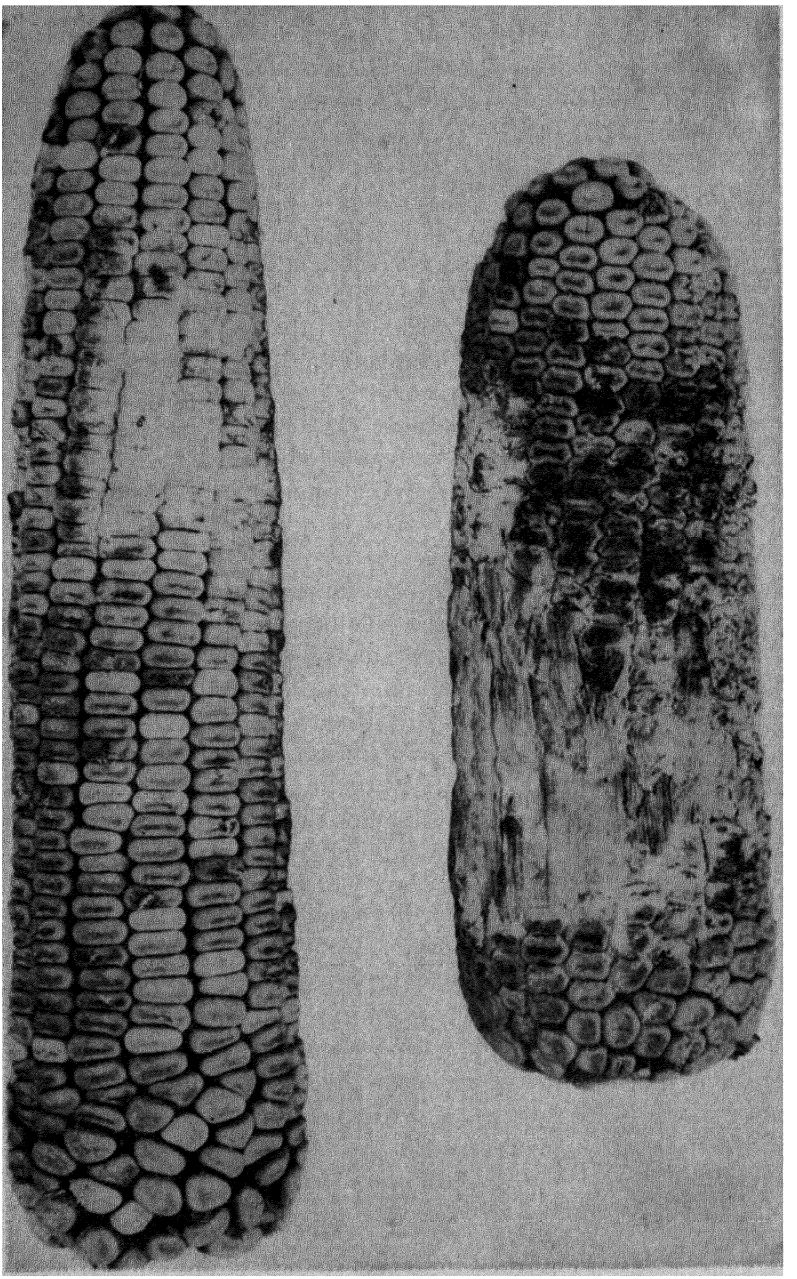

FIG. 21.—Fusarium or pink kernel rot of corn caused by ***Fusarium monoliforme***, the conid stage of *Gibberella fujikuroi*.

mental conditions especially low temperatures influence the amount of blighting and symptoms, according to Dickson (1923), Dufrenoy and Fremont (1931). Seedling blight is common with *Gibberella zeae*, whereas

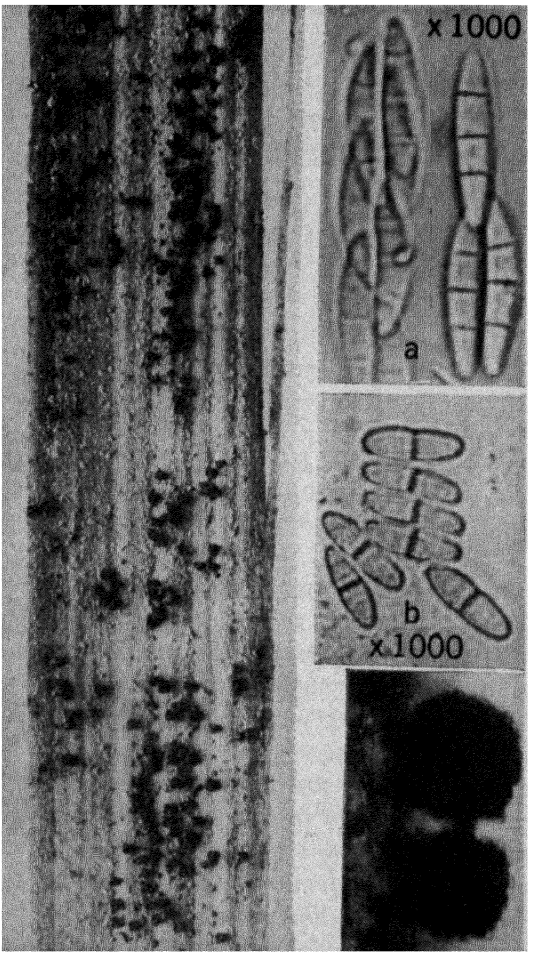

FIG. 22.—Perithecia of *Gibberella zeae* (*G. saubinetii*) on cornstalk tissue. Highly magnified ascospores of *G. zeae* (*a*) and *G. fujikuroi* (*b*) are shown in the inserts.

root lesions and slender weakened plants occur more frequently with *G. fujikuroi* infections.

The Fungi.—1. *Gibberella zeae* (Schw.) Petch or [*Gibberella saubinetii* (Mont.) Sacc.] Used until recently (*Fusarium graminearum* Schw.). Conidial stage

The detailed synonymy and morphology are given in Chap. XI.

Macroconidia are produced in sporodochia or pseudopionnetes, 3- to 5-septate, curved with gradual tapering toward tip. Microconidia and vegetative resting cells (chlamydospores) are not produced.

Perithecia are borne superficially, are globose, smooth, and blue black. Asci are oblong clavate, contain eight spores, arranged obliquely in one row. Ascospores are 3-septate, slender, tapering uniformly to the ends, and slightly curved (Fig. 22).

2. *Gibberella fujikuroi* (Saw.) Wr.
[*Gibberella moniliformis* (Sheld.) Wine.]
(*Fusarium moniliforme* Sheld.) Conidial stage

Macroconidia are produced sparingly, 3- to 5-septate, and curved toward the tip. Microconidia are borne in chains or false heads on branches of the hyphae, usually nonseptate except when germinating. Perithecia are similar to former species. Asci are less clavate, more oblong than in former species, contain eight spores, arranged in two irregular rows. Ascospores are straight, tapering to tips, 1- to 3-septate, usually 1-septate.

3. *Gibberella fujikuroi* (Saw.) Wr. var. *subglutinans* Ed.
(*Fusarium moniliforme* Sheld. var. *subglutinans* Wr. and Reinking.) Conidial stage

Macroconidia are similar to the species, but less curved toward the tip, usually 3-septate. Microconidia are borne singly or in false heads, never in chains. Asci are long, narrow, subclavate, contain usually eight spores, less commonly four or six arranged in one oblique row. Ascospores are straight, rounded at tips, short and thick, and 1-septate.

Etiology.—The fungi develop on crop residue of the cereal crops that remain in and on the surface of the soil. Spore development is confined largely to residue on the soil surface. Mycelium, conidia, and ascospores are produced during the growing season. Primary and secondary infections occur when environmental conditions are favorable. Seed infection, even in artificially dried corn, is frequently high, especially with *Gibberella fujikuroi*. Seedling blight occurs during germination and the early seedling stage when the soil is cold while the seed is germinating. Root and stalk rots become evident soon after pollination and increase in severity as the plants mature. Ear infections occur through and around the silks as these tissues decline in physiological activity after pollination. Windborne spores apparently are the main source of inoculum for ear infection.

Control.—Crop rotation and plowing under crop residue aid in reducing the volume of inoculum. Resistant hybrids are important in reducing seedling blight and stalk rots. Commercial hybrids also vary in susceptibility to ear rot, according to Hayes *et al.* (1933), Holbert *et al.* (1924, 1926, 1929), McIndoe (1931).

11. Diplodia Ear Rot, Stalk Rot, and Seedling Blight, *Diplodia zeae* (Schw.) Lév. and *D. macrospora* Earle and *Physalospora* Spp.—The Diplodia rot is of major importance in economical corn production. The disease occurs rather generally wherever the crop is grown intensively.

In the United States, the disease is important in the corn belt and diminishes in amount and severity in the western drier sections and in the northern, cooler areas. The disease produced by the less common species *D. macrospora* occurs in the more humid, warmer climates. The distribution of the disease and damage from it vary considerably depending upon climatic conditions.

Symptoms and Effect.—Seedling blight is generally less prevalent than in the Fusarium diseases. The cortical lesions and blighted seedlings resulting from infected seed are characterized by a brown dry rot, especially below the soil surface.

The stalk rot and leaf-sheath lesions are generally not conspicuous until after pollination of the corn plant. Reddish-purple to dark-brown blotches occur on the leaf sheath and extend into the nodes and basal portion of the internodes. Mycelial development frequently is extensive between the leaf sheath and stalk; however, other organisms, including saprophytes, develop beneath the leaf sheath. Stalk rot is initiated from lesions within the leaf sheaths and from rotted roots. Stalk rot more frequently develops from the adventitious roots and crown upward into the stalk, causing premature ripening and chaffy or rotted ears. The brown discoloration of the internodes and nodes is concealed by the dead bleached leaf sheaths (Fig. 23). The rotted stalks break over as the plants mature.

The ear rot varies from an inconspicuous infection of the kernels to a complete rotting of the ear and husks. The age of the ear tissues when infection occurs and environmental conditions influence the ear rot symptoms. The ear rot usually progresses upward from the base of the ear. The white to grayish-brown mycelium occurs between the kernels on the rotted ears (Fig. 23). The dark-brown pycnidia occur late in the season in the invaded husks, the floral bracts, and the pericarp of rotted kernels. Pycnidial development on the rotted stalks occurs in late autumn, and they are abundant on the old stalk tissues the following spring and summer.

The Fungi.—1. *Diplodia zeae* (Schw.) Lév.
[*Diplodia maydis* (Berk.) Sacc.]
(*Sphaeria striaeformis* var. 4 Schw.)
(*Sphaeria zeae* Schw.)
(*Sphaeria maydis* Berk.)
[*Sphaeria Hendersonia zeae* (Schw.) Curr.]
[*Hendersonia zeae* (Curr.) Hazsl.]
(*Diplodia zeae* Lév.)
[*Dothiora zeae* (Schw.) Benn.]
[*Macrodiplodia zeae* (Schw.) P. and S.]
[*Phaeostagonosporopsis zeae* (Schw.) Wor.]

Shear and Stevens (1935) discuss the confused nomenclature for this fungus and suggest the continued use of *Diplodia zeae* on the basis of long usage, although *D. maydis* is the more correct binomial.

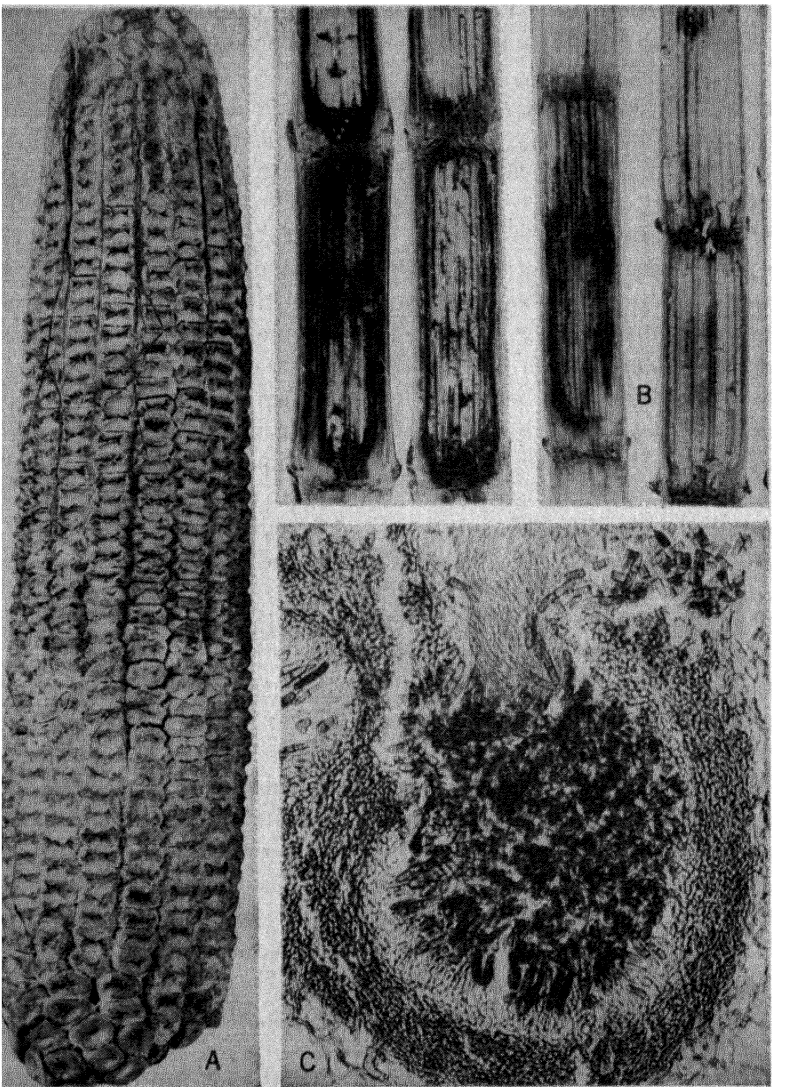

FIG. 23.—Ear rot (*A*) and stalk rot (*B*) of corn caused by *Diplodia zeae*. (*C*) Pycnidium and conidia highly magnified.

Globose flask-shaped or irregular pycnidia develop below the surface with a well-defined beak protruding through the epidermis. The conidiophores are simple, short, and pointed. The conidia are ovate, two-celled, straight to slightly curved with rounded to bluntlytape red ends, measure 25–30 by 6 microns, and are olivaceous in color. Slender, thread-like hyaline scolecospores are described by Butler (1913) and Johann (1939).

2. *Diplodia macrospora* Earle.
[*Dothiora zeae* var. *macrospora* (Schw.) P. and S.]

The morphology of this species is similar to the former except that the mycelium is coarser and the conidia are about twice the length of *Diplodia zeae*, or 70–80 by 6–8 microns. Scolecospores are reported associated with this species.

Etiology.—These fungi develop saprophytically on dead corn tissue on which inoculum is produced in abundance. The physiologically active corn tissues are infected more generally by contact infections from seed-borne mycelium, mycelium developing on pollen or organic materials collected within the leaf sheaths, and mycelium in crop residue in the soil. Mechanical injuries also offer avenues of entrance into susceptible tissues. As the corn plants approach maturity, the fungus established in the parenchymatous tissues is capable of advancing intercellularly through the stalk and into the ear, although most of the ear rot results from local infections. According to Clayton (1927), Durrell (1923), Holbert (1935), Koehler and Holbert (1930), McNew (1937), and Young (1926), the aggressive development of the fungus is associated with tissues approaching physiological maturity. Conidia and mycelia in the old stalk tissues are the important source of inoculum. Infected seed is the principal cause of seedling blight.

The disease is reduced by the use of stalk rot resistant hybrids, sanitation and rotation, and seed treatment (Hoppe and Holbert, 1936, Smith and Trost, 1934). The organic mercury dusts are very effective in controlling the seed infection (Hoppe, 1945, Raleigh, 1930), but they do not control the stalk and ear rots. An inhibitor or antibiotic substance produced by the fungus is suggested by Kent (1940).

Physalospora zeicola Ell. and Ev. (*Diplodia frumenti* Ell. and Ev.) and *P. zeae* Stout (*Macrophoma zeae* Tehon and Daniels) occur on corn in the Southeastern and Central United States (Eddins and Voorhees, 1933, and Ullstrup, 1946). These fungi also occur on other crops and crop residues. Stalk lesions and a minor ear rot are produced by the former, and leaf lesions, ear rot, and stalk and tassel lesions on corn are caused by the latter fungus. Early symptoms on the stalks and ears are similar to the more widely distributed Diplodia rot of corn. The darker colored mycelium, the slate-gray color of the rotted ears, and the frequent presence of sclerotia differentiate the Physalospora rot from the Diplodia rot. Sterile mycelium is more commonly plated from the tissues rotted by *P. zeae* than by *P. zeicola* and *Diplodia* spp., which usually produce pycnidia in culture.

3. *Physalospora zeicola* Ell. and Ev.
(*Diplodia frumenti* Ell. and Ev.)

The black perithecia are gregarious, covered by the epidermis, with conical necks terminating in glossy black ostioles protruding through the cuticle. The asci are

clavate-cylindrical, nearly sessile, measure 95–140 by 10–13 microns, are double walled, and appear white in section. Ascospores, usually eight and arranged biseriately, are ellipsoid in shape, unicellular, hyaline, and measure 20–23 by 8–9 microns. Pycnidia are submerged, black, and contain dark-brown striate aseptate spores. Pycnidia are produced in cultures on media.

4. *Physalospora zeae* Stout
(*Macrophoma zeae* Tehon and Daniels)

The black perithecia with minute papillate ostioles through the cuticle are formed in the mesophyll of the leaves and on tassel necks and branches. The asci are long, clavate to cylindrical, stalked, double walled, and measure 85–175 by 17–22 microns. Ascospores, usually eight and arranged subbiseriately, are hyaline to dilute amber, narrow ellipsoid, unicellular, and measure 19–25 by 6.5–8 (mean 26.8 by 10) microns (Ullstrup, 1946). Pycnidia of the imperfect stage occurring in association with perithecia (Ullstrup, 1946) are submerged, black, globose, protruding, usually with a short neck, and produce unicellular ellipsoid tapering conidia. Pycnidium-like structures that produce numerous nongerminating, hyaline, unicelled microconidia extruded in droplets in a mucous-like matrix are produced in nature and occasionally in culture.

12. Nigrospora Cob and Stalk Rot, *Nigrospora oryzae* (Berk. and Br.) Petch and *N. sphaerica* (Sacc.) Mason.—Corn is the common suscept, although the above species and *Nigrospora sacchari* (Speg.) Mason occur on a group of Monocotyledonous plants. The disease causes losses of corn in some years; however, it is of minor importance on corn and other cereals in North America, Europe, and Asia, as reported by Durrell (1925), Mason (1927), Petch (1924), Savulescu and Rayss (1930, 1931), and Standen (1945).

Symptoms and Effects.—The stalk and cob rots are not conspicuous until about harvest. The thin-walled cells of the stalk and cob of the ear are rotted away, leaving the vascular and sclerenchyma tissue. The kernels are chaffy, and the cob is shredded and easily broken (Fig. 24). The stalks break over at any point below the ear. The cob rot is the most important manifestation of the disease, as local losses frequently occur in the northern section of the corn belt.

The Fungi.—*Nigrospora oryzae* (Berk. and Br.) Petch and
Nigrospora sphaerica (Sacc.) Mason
(*Basisporium gallarum* Moll.)
(*Coniosporium gacevi* Bubak.)

The two species were differentiated tentatively on spore size: 13.5 to 14.9 microns for *Nigrospora oryzae* and 16.5 to 17.8 microns for *N. sphaerica* (Mason, 1927). Standen (1943) studied the variability of cultures from corn and other crops and demonstrated the difficulty of differentiating by spore size. If combined, the binomial *N. oryzae* should be used. The conidia are spherical, 10 to 20 microns in diameter, black, and borne on short lateral branches.

Durrell (1925), Savulescu and Rayss (1932), and Standen (1944, 1945) suggest that the disease develops under conditions of slow or checked

development of the corn plant, such as light frosts and sudden and acute drought.

13. Helminthosporium Leaf Blight and Leaf Spots, *Helminthosporium*

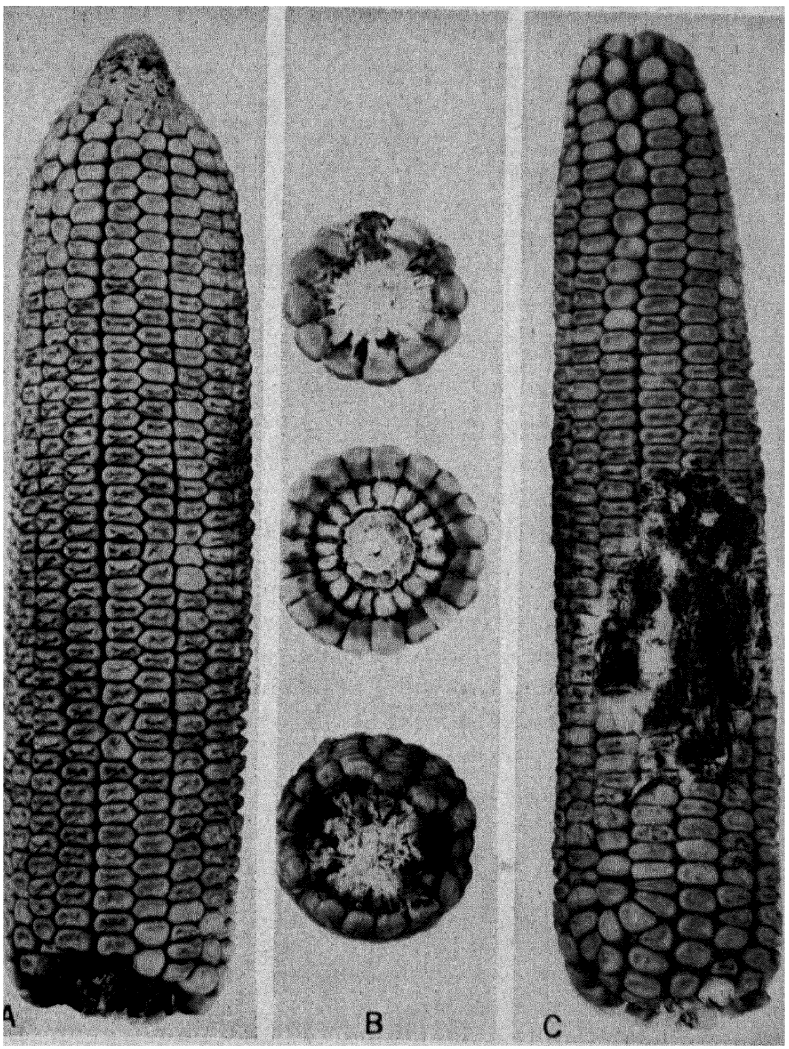

FIG. 24.—Cob rot of corn caused by *Nigrospora sphaerica*. The chaffy light-colored ear (*A*) is the characteristic appearance. (*B*) The rotted basal portion of the cob and dark masses of spores on the glumes. (*C*) Surface mycelium, less common.

turcicum Pass., *Cochliobolus heterostrophus* Drechs. (*H. maydis* Nishikado and Miyake), and *H. carbonum* Ullstrup.—Three Helminthosporium diseases occur rather generally on corn. These are associated predomi-

nantly with the corn leaves, and they differ in geographic distribution, suscept range, and damage.

The northern leaf blight caused by *H. turcicum* is distributed widely over the world on corn, Sudan grass, Johnson grass, and other sorghums. Epidemics on corn occur more generally in the Eastern United States, and the fungus extends north and west on Sudan grass. The characteristic symptoms are large linear to irregular, somewhat elliptical, lesions on the leaf blades and extending into the leaf sheaths. The lesions are first water-soaked, then light olivaceous to brown, and finally black to straw color as the tissues dry out (Drechsler, 1923, Koehler and Holbert, 1938, Ullstrup, 1943). Narrow bands of pigmentation occur along the margins of the lesions on Sudan grass (Fig. 25). Under favorable environment, the entire leaf blade is killed. Conidia are produced abundantly on the older portions of the lesions. Tassel infection on corn is less conspicuous, and ear infection is rare, although infection of the floral bracts in Sudan grass is more common.

The southern leaf spot caused by *Cochliobolus heterostrophus* (*Helminthosporium maydis*) is distributed widely over the world on corn and teosinte in the warmer climatic zones. The disease is common in the Southern United States, and it occurs associated with the former disease in the southern edge of the corn belt. The lesions on the leaves are distinct from those caused by *H. turcicum*, as they are smaller, more definite, and different in pattern and color (Fig. 25). The numerous spots are elongated between the veins with limited and parallel margins, usually less than 0.5 mm. long, buff to reddish brown in color, with a zonated or target-like color pattern (Drechsler, 1925). Less conspicuous spots occur on the floral bracts of the tassels. Ear infection is not common.

The leaf spot caused by *Helminthosporium carbonum* occurs on corn in the United States. The leaf spots are similar to the southern leaf spot in race 1 of the parasite, and narrow irregular chocolate-brown spots are produced by race 2 (Fig. 25). Both races of the parasite produce a black moldy growth over the kernels of susceptible varieties and result in a charred appearance of the infected ear (Ullstrup, 1941, 1943, 1944).

The Fungi.—1. *Helminthosporium turcicum* Pass.
(*Helminthosporium inconspicuum* Cke. and Ell.)

Conidial production is abundant on the leaf lesions. Conidiophores emerge two to six from the stomata, are olivaceous in color, 2- to 4-septate, and measure 7–9 by 150–250 microns. The cells of the conidiophore are longer than in most species. Conidia vary greatly in size and shape, ranging from 45–132 by 15–25 microns. The spores are straight or slightly curved, pale olivaceous, widest near the middle and tapering toward both ends, usually 3- to 8-septate with a protruding hilum (Fig. 25). Germination is characteristically by polar germ tubes. Mycelium and conidia develop on culture media. Specialized races occur on corn, some of which infect Sudan grass, and on Sudan grass; the latter do not generally infect corn.

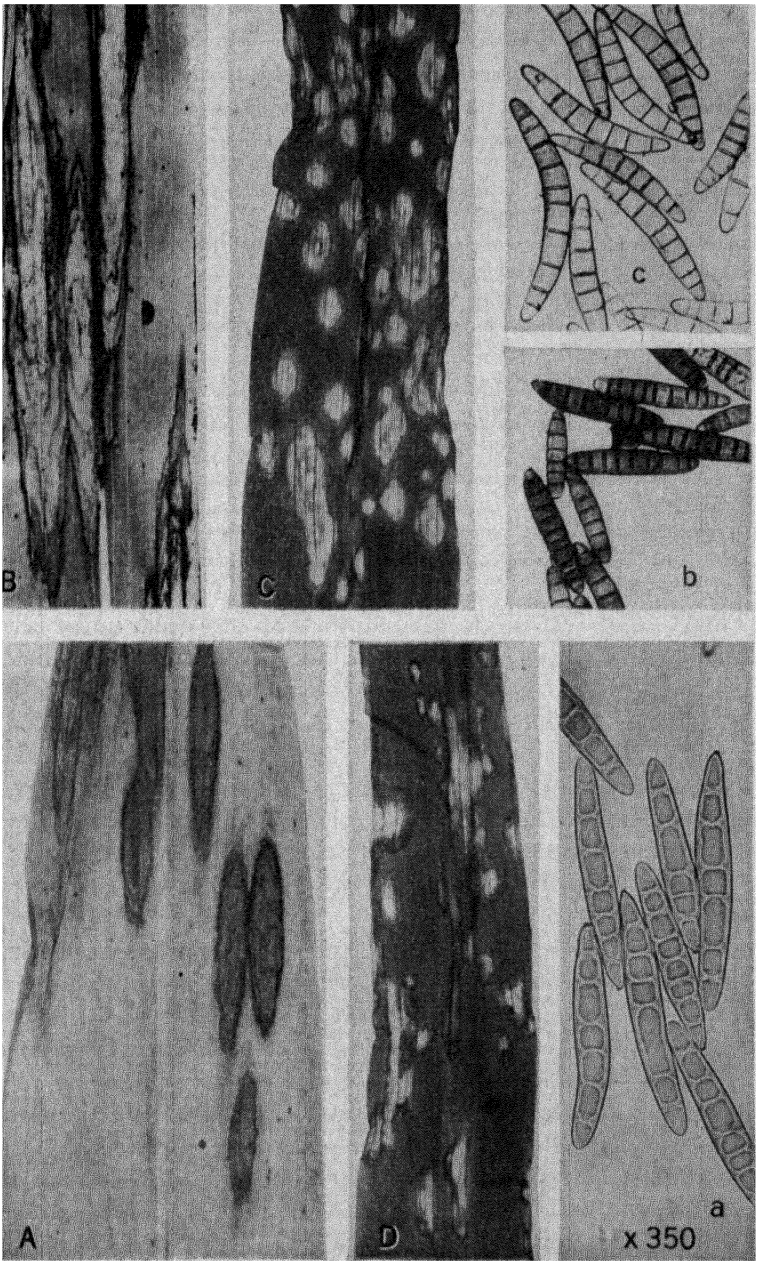

FIG. 25.—Leaf blights of corn (*A*) and Sudan grass (*B*) caused by *Helminthosporium* ***turcicum***, and of corn caused by ***H. carbonum*** (*C*) and ***Cochliobolus heterostrophus*** (*D*). Inserts, at same magnification, show the conidia of (*a*) *H. turcicum*, (*b*) ***H. carbonum***, and (*c*) *C. heterostrophus*. (*Courtesy of A. J. Ullstrup.*)

2. *Cochliobolus heterostrophus* Drechs.
(*Ophiobolus heterostrophus* Drechs.)
(*Helminthosporium maydis* Nishik. and Miy.)

The conidiophores arise 2 to 3 from the stomata on the older portion of the spot, 120 to 170 microns long, olivaceous. Conidia are light olivaceous, 30 to 115 microns long by 10 to 17 microns wide, often curved, tapering toward the rounded ends (Fig. 25). They germinate by polar germ tubes. Perithecia are numerous in old tissue, ellipsodial, about 0.4 mm. in diameter, black, with conidiophores and mycelium on the surface, ostiolate beak well defined. Asci are numerous, short stipitate, with a rounded end, are 160 to 180 microns long, and contain typically four spores. Ascospores are filamentous, arranged in parallel multiple coils, usually four coils. Drechsler (1925, 1934) gives the details of the morphology and the taxonomic significance of the new genus Cochliobolus.

3. *Helminthosporium carbonum* Ullstrup.

This species occurs on a few susceptible corn inbreds and varieties. Originally described by Ullstrup (1943, 1944) as *Helminthosporium maydis* Nishik. and Miy., it is relatively similar in conidial morphology to the above species. Conidia are straight to slightly curved, widest in the center tapering toward the rounded ends, measure 25–100 by 7–18 microns, are 2- to 12-septate, have an inconspicuous hilum, and germinate from polar cells (Fig. 25). *H. zeicola* Stout was described on corn stalks, but if a separate species, it is not common.

Etiology.—The leaf infections of all three species occur under favorable environmental conditions throughout the growing season. The source of primary inoculum is largely that produced on the crop residues. Secondary conidial infection is general after the leaf infections are established. Mitra (1923), Nishikado and Miyake (1926), and Nishikado (1927) discuss the general etiology and distribution of the former two diseases. The southern leaf spot disease develops at somewhat higher temperatures than the leaf blight. Apparently only *Helminthosporium carbonum* is seed-borne on corn (Ullstrup, 1943, 1944). *H. turcicum* is seed-borne on Sudan grass (Chilton, 1940).

Control measures consist largely of sanitation, rotation, proper covering of crop residue, and use of resistant corn hybrids. The inbred lines N.C. 34, K. 155, K. 175, Ky. 114, T. 49B, T. 105B, T. 206, Mo. 21A, C.I. 15, C.I. 16, and C.I. 23 are moderately resistant to *Helminthosporium turcicum*. Many of the commercial hybrids in use are relatively resistant or tolerant to all three diseases. Resistance to the destructive race 1 of *H. carbonum* is inherited as a monogenic recessive; therefore, hybrids are resistant unless susceptible inbreds are combined. Seed treatment of Sudan grass seed with the mercury dusts reduces the infection.

14. Rhizoctonia Rot, *Rhizoctonia* Species.—Rhizoctonia especially in the warmer climates causes a root, stalk, and ear rot of corn. Many of the species of *Rhizoctonia* are capable of attacking corn. The disease is of minor importance on this crop. Voorhees (1934) described an ear rot of corn in which the rotted ears are covered by a salmon pink and later a

gray mycelium and brown to black sclerotia are formed on the husks. He described the fungus on corn as a new species, *Rhizoctonia zeae* Voorhees.

15. Smut, *Ustilago maydis* (DC.) Cda.—The common corn smut is one of the most widely distributed diseases on this crop. Teosinte is the only other plant infected. Corn smut has been distributed with the spread of the crop; yet the disease commonly is not carried over on the grain as in many other cereal smuts. Evidently sufficient inoculum is carried on the seed to introduce the parasite. Once the parasite is introduced and established, the extremely hardy spores, produced in countless numbers, perpetuate the fungus. The corn smut eradication program in Australia is still uncertain, as new infections occur even after 1 or 2 years of apparent absence of the disease. The use of the mechanical corn harvester increases materially the amount of inoculum carried on the grain.

The losses from corn smut average high each year. The losses are generally greater in the warmer and somewhat drier areas. According to Garber and Hoover (1928), Immer and Christensen (1928, 1931), Johnson and Christensen (1935), and Jorgensen (1929), the location of the galls on the plant and the time of development and size influence the effect on yield. Hurd-Karrer's (1926, 1927) reports on the effect of smut infection on sap concentration and sugar content partly explain the location effect of the gall on yield.

Symptoms.—The smut galls occur on any part of the corn plant where embryonic tissues are exposed. Gall formation is induced by the fungus. As shown by Knowles (1889), the mycelium developing between the cells of the embryonic thin-walled tissues induces hyperplasia and hypertrophy and the excessive development of the phloem elements of the bundles. The galls are common in axillary buds, individual flowers of ear and tassel, and leaves and are less common on stalk tissue unless mechanical injuries occur (Fig. 26). The white membrane of modified corn epidermal tissue ruptures during gall enlargement, releasing the black mass of spores. Galls on corn seedlings usually result in the dwarfing and blighting of the plants. The later gall development rarely kills the plant or plant parts.

The Fungus.—[*Ustilago maydis* (DC.) Cda., or *Ustilago zeae* (Beckm.) Ung.]

(*Lycoperdon zeae* Beckm.)
(*Uredo zeae* Schw.)
(*Ustilago maydis* Cda.)
(*Ustilago schivenitzii* Tul.)
(*Ustilago zeae-maydis* Wint.)
(*Ustilago euchlaenae* Arcong.)
(*Ustilago mays-zeae* Magn.)

The spores are spherical to ellipsoidal, 8 to 11 microns in diameter, black, with heavy spine-like echinulations. The germination is typically by the formation of a basidium

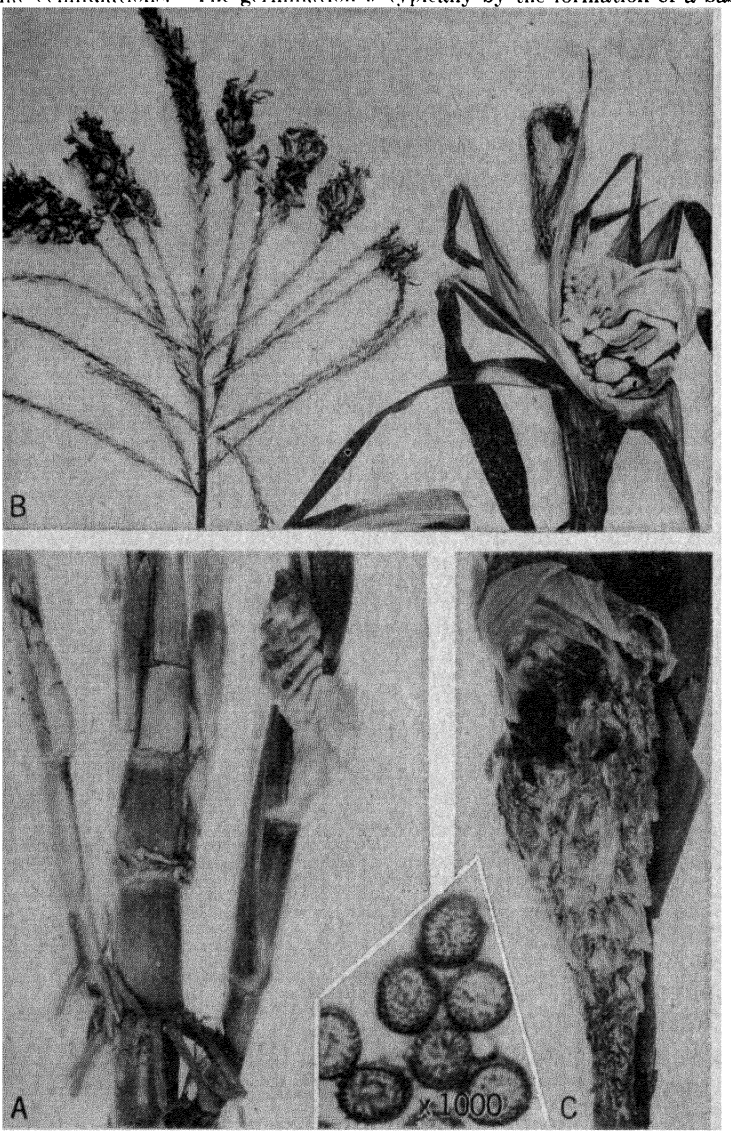

FIG. 26.—Corn smut galls on tassel and ear (*B*) and axillary buds (*A*). Infection before the tissues are fully differentiated results in a sorus involving the entire ear (*C*). Chlamydospores of *Ustilago maydis*, highly magnified, are shown in insert

(promycelium) and sporidia. According to Chilton (1940), the type of germination and sporidial development is variable between strains of the fungus. Sporidia increase by budding. According to Christensen and Rodenhiser (1940), Sampson (1939), and Stakman (1940), fusions occur between compatible sporidia.

Etiology.—Infection of the corn plant occurs during the period of vegetative development. Davis (1936) and Melhus and Davis (1931) describe a systemic infection of the plant occuring in the late seedling stage and sori development as the tissues differentiate throughout the season. Local infections of embryonic tissue occur at any time during the vegetative development of the corn plant. Injuries, such as hail injury and detasseling, frequently result in late localized infections. The source of inoculum is largely air-borne sporidia from chlamydospores on the soil and crop residue. The spores are very resistant to extremes of environment. Delayed germination is common in a fair proportion of the spore population, this greatly lengthens the period of abundant inoculum. Sporidial fusion and the development of the dikaryotic infection hyphae are characteristic, although Chilton (1940) and Christensen (1931) report exceptions. Gall formation and spore dissemination are continuous during the summer growing period. The use of the mechanical corn harvester increases the spread of spores. The smut galls are not deleterious to animals other than increasing the dust content of dry fodder.

Control.—In areas where corn is grown continuously or in short rotations on contiguous acreages, crop rotation and sanitation do not control the disease. This is especially true where the ripe corn is harvested mechanically and the stalks are left on the fields. Under such conditions, resistant hybrids offer the only satisfactory means of control. Cutting the green corn and ensiling it reduce the spore inoculum. Under such conditions with crop rotation, it is possible greatly to reduce the damage from corn smut. Seed treatment with the mercury dusts is important where corn is introduced into smut-free areas.

Smut-resistant hybrids offer the best means of control. Hayes *et al.* (1924), Hoover (1932), Immer (1927), Jones (1918), and others summarize the problems and the reaction of hybrids and inbreds. Many of the more recent hybrids developed for the corn belt are moderately resistant to many of the variants of this parasite. Some few inbreds in both sweet and dent corn are resistant over a wide range of conditions. The variability of the parasite, as shown by Christensen and Stakman (1926), Stakman *et al.* (1929, 1933, 1940), makes the control problem more difficult.

16. Head Smut, *Sphacelotheca reiliana* (Kuehn) Clint.—The head smut occurs on corn and sorghums. In the Central United States, the disease is more common on the latter crop group. In the United States, the disease on corn is localized in limited sections in the Western Intermountain and Southwestern areas. It is the more prevalent smut on corn in southern Russia, India, and South Africa, according to Bressman

(1933), McAlpine (1910), Pole-Evans (1911), and Potter (1914). Losses from this smut are high in areas favorable for its development.

Symptoms.—The large smut sori replace the tassel and ear in corn and the panicle in the sorghums. The sorus consists of the conductive tissues of the suscept surrounded by the spore mass and the fragile exterior fungal membrane. Frequently, only part of the tassel or panicle is replaced by the sorus. In such cases the floral bracts frequently develop into leaf-like proliferations. The black dusty spore mass is conspicuous soon after the tassels or panicles emerge.

The Fungus.—*Sphacelotheca reiliana* (Kuehn) Clint.
[*Sorosporium reilianum* (Kuehn) McAlp.]
(*Ustilago reiliana* Kuehn)
(*Ustilago pulveracea* Cook)
[*Ustilago reiliana* forma *zeae* (Kuehn) Pass.]
[*Cintractia sorghi* (Link) Hirsch.]

The sorus is composed of loosely united spores and the conductive tissues of the suscept at first enclosed by a fragile fungal membrane. The reddish-brown to black chlamydospores are finely echinulate, irregular to spherical, and 9 to 12 microns in diameter. The spores germinate by forming basidia and lateral sporidia. Germination is frequently irregular with branching promycelia.

Etiology.—Infection occurs in the seedling or young plant, resulting in a systemic distribution of the mycelium in the apical primordial tissues. Sori develop in the floral structures. Potter (1914) demonstrated the prevalence of seedling rather than floral infection. Christensen (1926) and Reed *et al.* (1927) have demonstrated the importance of environmental conditions to the seedling infection. Zehner and Humphrey (1929) produced infection by introducing the spore suspension through the leaf whorl into the growing point in seedlings. Spores are distributed widely during the latter part of the growing season. Soil-borne inoculum is the important source of natural infection. Reed (1927) has shown that separate physiologic races occur on corn and the sorghums.

Control.—Sanitation and rotation are important in preventing seedling infection. In areas where the disease is distributed extensively, these measures are not sufficient to prevent its occurrence. Seed treatment with the mercury dusts helps prevent seedling infection. Differences in susceptibility in corn occurs in south Russia and in Kansas. Reed (1927) has shown that the sweet sorghum and sorgo varieties are relatively susceptible and that the feterita, milo, broom corn, kaffir, and kaoliang varieties are resistant.

17. Leaf Rust, *Puccinia sorghi* Schw.—The rust of corn is of very minor importance on both field and sweet corn varieties. Corn and teosinte

are the uredial and telial hosts. Several species of *Oxalis*, chiefly *O. stricta* L., *O. cymosa* S., *O. corniculata* L., distributed in the United States and *O. europe* in Europe, are the aecial hosts. The aecial infection is limited in the United States. Allen (1933, 1934), Arthur (1904), Cummins (1931), Pole-Evans (1923), and Tranzschel (1907) have reported on the life cycle and heterothallism in this parasite.

The Fungus.—*Puccinia sorghi* Schw.

The pycnia (spermatia) and aecia are citron yellow to orange and elevated on the suscept tissues. Aeciospores are angular, hyaline. The uredia form light-brown oblong lesions with the epidermis turned back around the margins. Urediospores are globose to ovate, slightly verrucose, with several pores unevenly distributed. Telia usually form in the uredia and are not covered by the epidermis. The teliospores are ovate to oblong, constricted at the septum, dark brown, and have a long persistent pedicel.

Specialization has been reported by Mains (1934) and Stakman *et al.* (1928). Mains (1924, 1926, 1931) reports resistant varieties and the inheritance of resistance on a single-factor pair basis. Rhoades (1935) reports the location of a gene for rust resistance on the tenth or shortest chromosome. Smith (1926) and Wellensiek (1927) have studied infection and the nature of resistance.

Two other rusts are reported on corn from Central and South America. Cummins (1941) reported *Puccinia polysora* Underw. from the above areas and in the Southern United States on corn and teosinte and *Angiopsora zeae* Mains on corn from four locations in Central and South America.

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CHAPTER V

MILLET DISEASES

The millets are not grown extensively in the United States. Two species, *Setaria italica* (L.) Beauv. [*Chaetochloa italica* (L.) Scrib.] and *Panicum miliaceum* L., comprise the major acreage. Several additional species are grown less commonly in the United States, but extensively elsewhere: *Echinochloa crusgalli* var. *frumentacea* (Roxb.) Wight, *E. colonum* (L.) Link, *Pennisetum glaucum* (L.) R. Br., and *Eleusine coracana* (L.) Gaertn. The cultivated millets are important food crops in parts of southern Europe, Asia, and Africa.

In both Setaria and Panicum, polyploid series exist. The basic chromosome number in both genera is apparently nine pairs. The cultivated Setaria millet is in the nine-chromosome pair and the Panicum millet is in the 18-pair group, based on the reports of Arenkova (1940), Avdulov (1931), Kishimoto (1938), and Rau (1929).

The cultivated millets are summer annuals adapted to warm climates. Most of the species are drought resistant. Downy mildew and smuts are the more important diseases causing losses in this crop.

1. Bacterial Blights.—Two bacterial blights are reported on the millets.

Bacterial spot caused by *Pseudomonas alboprecipitans* Rosen [*Phytomonas alboprecipitans* (Rosen) Bergey *et al.*] occurs on the Setaria millet and some wild species. Small grayish-green spots with brown pigmentation are the common symptoms, as described by Rosen (1924).

A *bacterial stripe* of the Panicum millet caused by *Bacterium panici* Elliott [*Phytomonas panici* (Elliott) Bergey *et al.*] was reported by Elliott (1923). The brown stripes on the leaves are water-soaked and later show scales of exudate on the surface of the lesions.

2. Pythium Root Rot, *Pythium* Spp.—Rootlet rot and blighting of seedlings occurs under certain soil conditions. The Graminicolous species of Pythium parasitize these crop plants.

3. Downy Mildew, *Sclerospora graminicola* (Sacc.) Schroet.—The cultivated millets as well as many wild millet-like grasses are damaged by the disease, as described by Melhus and Van Haltern (1924). The disease is world wide in its distribution and causes heavy losses, notably in Asiatic countries where the millets constitute an important cereal crop. The disease is generally distributed in the United States on the weed grass, *Setaria viridis* (L.) Beauv.

Symptoms and Effects.—The symptoms are very characteristic on these millets. The plants are dwarfed chiefly through reduced internodal elongation of the culms. Excessive tillering from the crown and development of branches from the axillary buds along the culm are generally

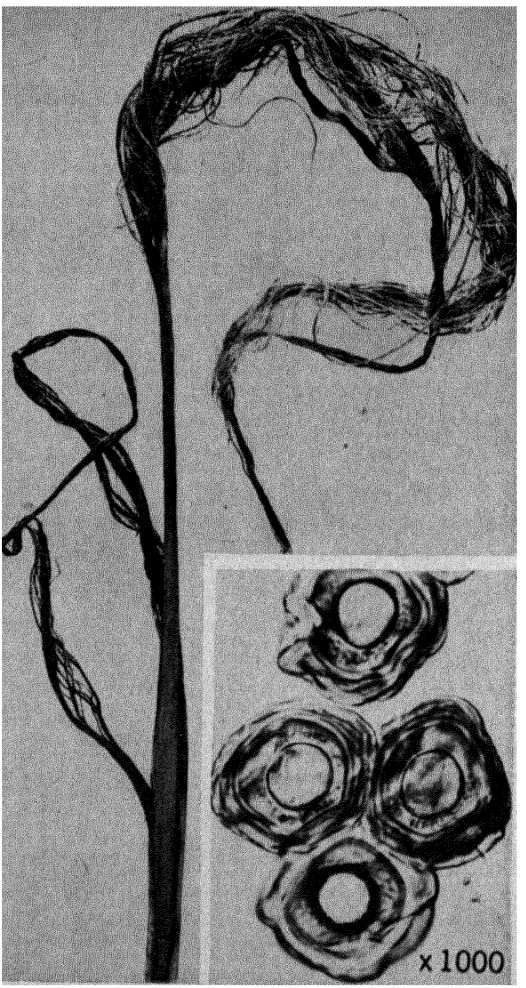

FIG. 27.—Downy mildew on Setaria caused by *Sclerospora graminicola*, and the oöspores of the fungus highly magnified.

characteristic. The development of leaf-like malformations of the floral bracts and failure of kernel development are further common symptoms. The downy mass of conidiophores and conidia are usually common on suscepts grown in a humid climate and are less common under dry conditions. Oöspores develop abundantly in the mesophyl and paren-

chyma. Leaf necrosis and browning are followed by splitting and shredding of the invaded tissues, especially as the plants approach maturity. Axillary bud development and shredding of the leaves is less pronounced in the *Pennisetum* spp. The excessive proliferation of buds and inflorescences during vegetative development combined with little or no kernel development causes a serious reduction in yield where infection is high (Fig. 27).

The Fungus.—*Sclerospora graminicola* (Sacc.) Schroet.
(*Protomyces graminicola* Sacc.)

The conidiophores emerge singly or in groups through the stomatal openings. They are short (average 268 microns), thickened, without a basal cell, and produce numerous short branches near the apex. The conidia are borne apically on short (8 microns) sterigmata. The conidia are elliptical, slightly pointed with a conspicuous dehiscent papilla, smooth-walled, hyaline, and range from 14 to 23 microns long by 11 to 17 microns wide. Occasional larger conidia occur. The conidia germinate to form three or more kidney-shaped two-ciliate zoospores. Oöspores are produced in large number in the cells of the mesophyl and parenchyma. The oöspores are irregular to round (30 to 60 microns in diameter), thick-walled, have a smooth outer wall, and are reddish brown in color. They germinate by the formation of a germ tube, as described by McDonough (1937).

Etiology.—The oöspores remain viable in the soil and crop refuse for long periods. The oöspores frequently are carried with the seed. Only a small percentage of the spores germinate at one time; therefore, the oösporic inoculum is present over long periods. Infection of young plants occurs from the spores in the soil. Wind-borne spores also may serve as inoculum when they come in contact with embryonic tissues. Conidia cause secondary spread when the plant tissues are still susceptible and weather favorable. The mature plant tissues are relatively resistant.

Control.—Control of the disease is difficult in areas where these crops are grown continuously over large areas, due to the general soil infestation. In the United States where millets are not grown extensively, the control is easier unless the crop is sown in areas where the wild *Setaria viridis* is infected. Seed treatment with formaldehyde, sulphuric acid, and organic mercury compounds are reported as the best treatments. Tasugi and Akaishi (1933, 1935) report resistant varieties of the millets.

4. Helminthosporium Leaf Spots.—Several species have been reported on the millets and closely related species. Drechsler (1923) described *Helminthosporium monoceras* Drechs. on *Echinochloa crusgalli* (L.) Beauv. in the United States. Nishikado (1929) reported *H. panici-miliacei* Nishikado and *H. yamadai* Nishikado on the Panicum millet and Ito (1930) *H. setaria* Saw. with the perithecial stage, *Cochliobolus setariae* (Ito and Kuribay.) Drechs. on Setaria millets in Japan. Mitra and Mohta (1934) reported *H. nodulosum* (Berk. and Curt.) Sacc. and *H. leucostylum* Drechs. on millet-like grasses in India.

5. Long Smut, *Tolyposporium penicillariae* Bref.—This smut is not reported in the United States but occurs in Asia and Africa, according to Britton-Jones (1922) and Butler (1918). The pear-shaped brown to

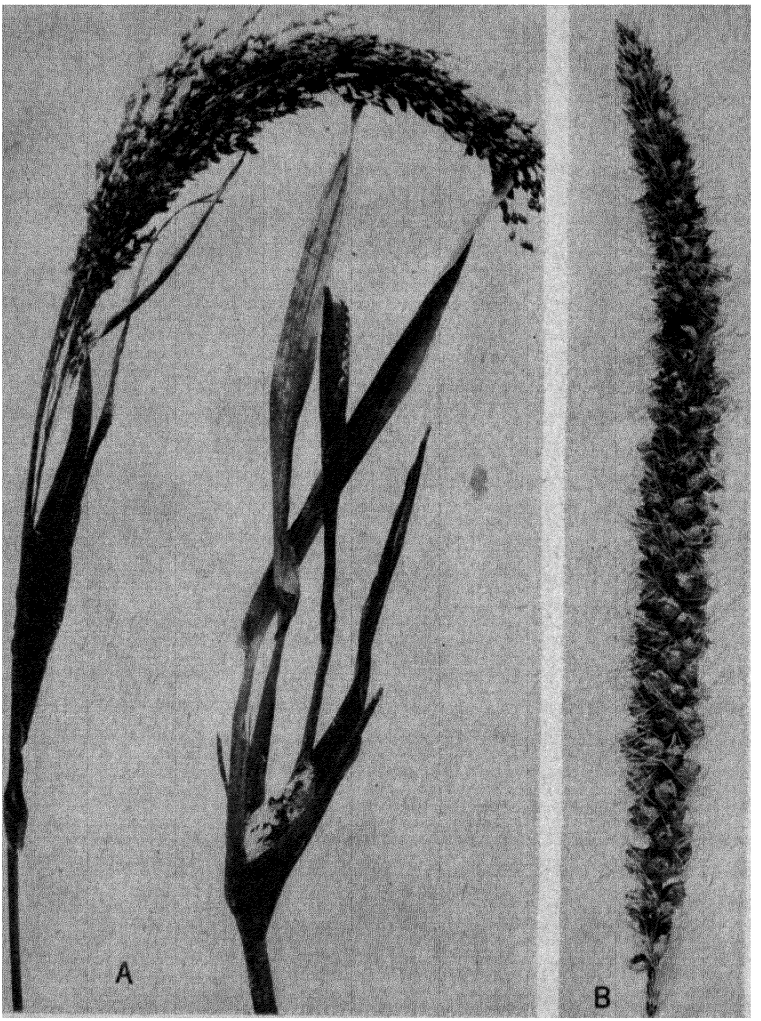

FIG. 28.—(*A*) Head smut of millet caused by *Sphacelotheca destruens;* (*B*) kernel smut of Setaria caused by *Ustilago neglecta.*

black sori protrude from the floral bracts. The membrane ruptures exposing the greenish-brown spores.

6. Kernel Smut, *Ustilago crameri* Koern.—This smut is common on the cultivated Setaria millets in Asia and Africa and uncommon in the

United States. It is similar in appearance to the kernel smut caused by *Ustilago neglecta* Niessl. on *Setaria lutescens* (Weigel) Hubb [*S. glauca* (L.) Beauv.], a common weed grass (Fig. 28). The disease is severe in some of the Asiatic areas, especially where the spores persist in the soil. The ovaries are replaced by the spore mass. The sori are enclosed in the floral envelopes and are similar in shape to an enlarged kernel. The brittle floral bracts break, releasing the loose spore mass as the plants reach maturity, or they persist, enclosing the spores in the threshed grain.

The Fungus.— *Ustilago crameri* Koern.

The spores are enclosed by the ovary wall and floral bracts. Spores are subspherical to irregular, 8 to 11 microns in diameter, and reddish brown in color. The spores germinate to form a basidium and irregular branching or sporidia.

Porter (1928), Vasey (1918), Yu *et al.* (1934) have shown that formaldehyde solutions, copper carbonate, and the mercury dusts control the seed infection and reduce infection from soil infestation. Tu and Li (1935) report resistant varieties.

7. Head Smut, *Sphacelotheca destruens* (Schlect.) Stevenson and A. G. Johnson.—The head smut of the Panicum millets is widespread on this crop. Similar-appearing smuts occur on several of the wild species. The smut sori are first evident as the panicles emerge. The entire inflorescence is modified into a sorus enclosed by a grayish-white false membrane. The membrane ruptures as the plants mature, exposing the dark-brown spore mass and the vascular tissues of the smutted panicle (Fig. 28).

The Fungus.—*Sphacelotheca destruens* (Schlect.) Stevenson and A. G. Johnson
(*Sphacelotheca panici-miliacei* Bubak.)
(*Uredo segetum* var. *panici-miliacei* Pers.)
(*Uredo segetum* var. *panici* Alb. and Schw.)
(*Uredo carbo* var. *panici-miliacei* DC.)
(*Caeoma destruens* Schlect.)
(*Uredo destruens* Duby.)
(*Tilletia destruens* Lév.)
(*Ustilago panici-miliacei* Wint.)
(*Sorosporium panici-miliacei* Tak.)

The sori are formed in the inflorescence, completely destroying all but the vascular elements. The sorus is covered by a grayish-white false membrane of fungus origin. Hyaline angular sterile cells of false membrane adhere in masses to the surface of the sorus. Spores are reddish brown, spherical to subspherical, 7 to 10 microns in diameter, and mostly smooth. The chlamydospores germinate to form basidia and sporidia.

The etiology and control are similar to the kernel smut.

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CHAPTER VI

OAT DISEASES

The cultivated oats are derived mainly from two wild species. The common oat, *Avena sativa* L., apparently is the principal commercial species originating from the wild oat, *A. fatua* L., and *A. byzantina* Koch. originates from the wild red oat, *A. sterilis* L. The varieties of *A. sativa*, the common oat, grown in the cooler climates comprise the largest acreage devoted to this crop. The red-oat varieties are grown chiefly in the warmer climates. The basic chromosome number in Avena is seven pairs. The wild and cultivated species mentioned above and *A. nuda* L. and *A. orientalis* Schreb. all have 21 pairs of chromosomes. Kihara and Nishigama (1932), Malzew (1930), Stanton (1936), and others discuss the genetics and cytology of this genus.

The more recent oat hybrids, combining disease resistance and high yielding capacity, represent a significant advance in disease control. Murphy (1942) and Stanton (1936) list the source of these selections and their reaction to the smuts and rusts. This parental material represents an available collection of superior germ plasm for rapid advance in oat breeding.

The cultivated oats are grouped into two classes—winter and spring—based on character of growth. Varieties of the common oat and hybrids including it and closely related species comprise the main group of spring types. Varieties of the red oat or combinations with it comprise the more important winter types grown. In oats, like barley, the winter types are distributed in the milder climates and the spring varieties occupy the larger acreage in the temperate zones. Fall-sown spring oats are grown on limited acreages in the extreme Southern United States.

Oats occupy an extensive acreage, and they are grown chiefly for feed grain. Oats rank third in acreage devoted to the cereal grains in the United States. Limited acreages are produced for pasture and hay. The oat plant is predominantly a low-temperature crop, especially during the seedling and early vegetative period. The red oat type will develop at somewhat higher temperature than the common oat, particularly in the later vegetative stages.

Oat diseases cause large losses in the United States. These losses are being reduced rapidly by the use of disease-resistant varieties. There is still, however, a large acreage of older varieties susceptible to one or more

of the major oat diseases. Estimated average annual losses in the United States for the 10-year period 1930 to 1939 amounted to 10 per cent of the crop, or over 103 million bushels annually (Plant Disease Survey). Losses from the oat smuts and crown and stem rusts have been appreciably lower during the past two years, *i.e.*, since the general use of the newer disease-resistant varieties.

1. Nonparasitic.—Two nonparasitic maladies are common on oats in limited areas. The blasting of spikelets of the panicle is conspicuous and causes a reduction in yield. The gray speck or dry leaf spot disease is important on oats and other cereals and grasses.

Blast.—Blast of oats is caused by a number of environmental conditions. Species and varieties vary greatly in their tendency to produce white empty spikelets, especially near the base of the panicle. The blasting is apparently associated with a disturbed plant metabolism either when the panicle tissues are differentiating or near the period of pollination. Derick and Forsyth (1935), Elliott (1925), and Huskins (1931) discuss the influence of moisture, unbalanced fertility, and other factors upon the development of blast. These factors influence blasting when they occur at the tillering stage or later near the period of flower pollination. Derick and Hamilton (1939) show the influence of blast on yield. There is an indication that the conditions associated with blasting of the spikelets tend to reduce the potential yield of the remaining spikelets. The better adapted varieties are relatively free from blast.

Gray Speck or Dry Leaf Spot.—The gray speck is not common in the major oat-growing sections of North America, although it occurs in alkaline organic soils (Hageman *et al.*, 1942, MacLachlan, 1941, 1943, and Sherman and Harmer, 1941). The malady is severe in parts of Europe and Australia, as reported by Davies and Jones (1931), Lundegårdh (1931, 1932), Rademacker (1935), and Samuel and Piper (1928). The disease occurs with less severity on wheat, barley, and some grasses.

Symptoms and Effects.—Light-green to gray irregular to oblong flecks occur on the leaves, especially the leaf blades. The areas enlarge, dry out, and change to a buff or light-brown color (Fig. 29). The size and extent of the spots is modified by variety, severity of the manganese deficiency, and soil moisture. The plants are reduced in height; the leaf blades are narrow and more erect; and the plants are chlorotic as well as showing leaf spots in severe manifestations of the disease. Yields are reduced greatly when plant development is retarded. The dry leaf spot stage symptoms are easily differentiated from the heritable blotch described by Ferdinandsen and Winge (1929).

The disease is associated with certain soil types. Alkaline soils, low in soluble manganese, and frequently high in organic matter, are conducive to leaf spot development. Unbalanced soil nutrients and their

direct or indirect effect upon soluble or available manganese appear to be among the basic contributing factors. In organic soils, certain bacteria are associated with the conversion of soluble manganese compounds to insoluble oxides (MacLachlan, 1941, 1943). According to

FIG. 29.—Gray speck and dry leaf spot caused by a deficiency of manganese. (*A*) gray speck and dry spot on oat leaves and (*B*) a similar disturbance on winter wheat.

Lundegårdh (1931) all factors that cause a decrease in manganese absorption by the plant influence the development of the disease. Indirectly then, the addition of lime, humus or colloids, nitrates, or alkaline phosphates increases the development of gray speck. In addition to the manganese deficiency, a disturbed ion balance in the plant, especially

extremely low or high potassium-calcium ratios, increases the manifestation of gray speck. There is apparently no distinct relation between the absolute manganese content of the leaves and the appearance of the necrosis. However, the direct cause of the disease is a deficiency of manganese. Fertilization with ammonium salts facilitates the solution of the manganese in the soil. Blatty (1932), Gerretsen (1937), Hiltner (1924), Samuel and Piper (1928, 1929), and others report control by the use of manganese sulphate, although in certain soils the results are temporary as the added manganese is changed quickly to insoluble oxides. MacLachlan (1941, 1943) obtained quicker and more permanent results by the use of 1 per cent manganese sulphate spray with bentonite and soap. MacLachlan (1941, 1943), Sherman and Harmer (1941), and Rademacker (1935) report differences in the tolerance of various crop plants.

2. Mosaic, Viruses.—The mosaic on oats occurs in the southern winter oat area of North America, and it is reported in similar sections of Europe and Asia. The oat mosaic occurring in Southern Russia and reported to be transmitted by *Delphacodes straitellus* (Fall.) has not been compared fully with that occurring in North America. Apparently certain of the newer winter oat varieties originating from Victoria × Richland and Boone hybrids are susceptible, whereas the Red Rustproof and Fulghum varieties are resistant to the mosaic in the Southern United States. The disease is discussed in more detail in Chap. XI.

3. Halo Blight, *Pseudomonas coronafaciens* (Elliott) Stapp.—The cultivated oats and several grasses (*Agropyron, Avena, Bromus* spp.) are susceptible in varying degrees. The disease is common in North America, and it is reported in northern Europe. The severe attack early in the season apparently does not cause an appreciable reduction in yield of grain. Severe infection just previous to emergence of the panicles reduces yield.

Description.—The lesions are more common on the leaf blades, but they occur on the leaf sheaths and floral bracts in severe late infections. The initial lesion is an oval to oblong water-soaked small spot, changing gradually from green to buff or light brown. The initial infection is associated with the stomata or more frequently aphid or other insect punctures. The tissues surrounding the small spot gradually lose the green color and become slightly water-soaked and light yellow in color. The light-yellow zone forms a halo area around the restricted brown lesion. As the number of infections increase, the lesions coalesce, forming an irregular halo area (Fig. 30). The tissues dry out and fade to light-brown and straw-colored mottling. No exudate is present on the lesion. The spots on the floral bracts are less conspicuous, owing chiefly to the reduction in size of the halo area.

The bacterial colony is restricted to the stomatal cavity or mechanical puncture and between the adjacent mesophyll cells. Spread between the cells in the tissues is limited. The cells of the adjacent tissue change in composition and function. Chlorophyll regeneration is stopped; the permeability of the membranes is changed; and the intercellular spaces

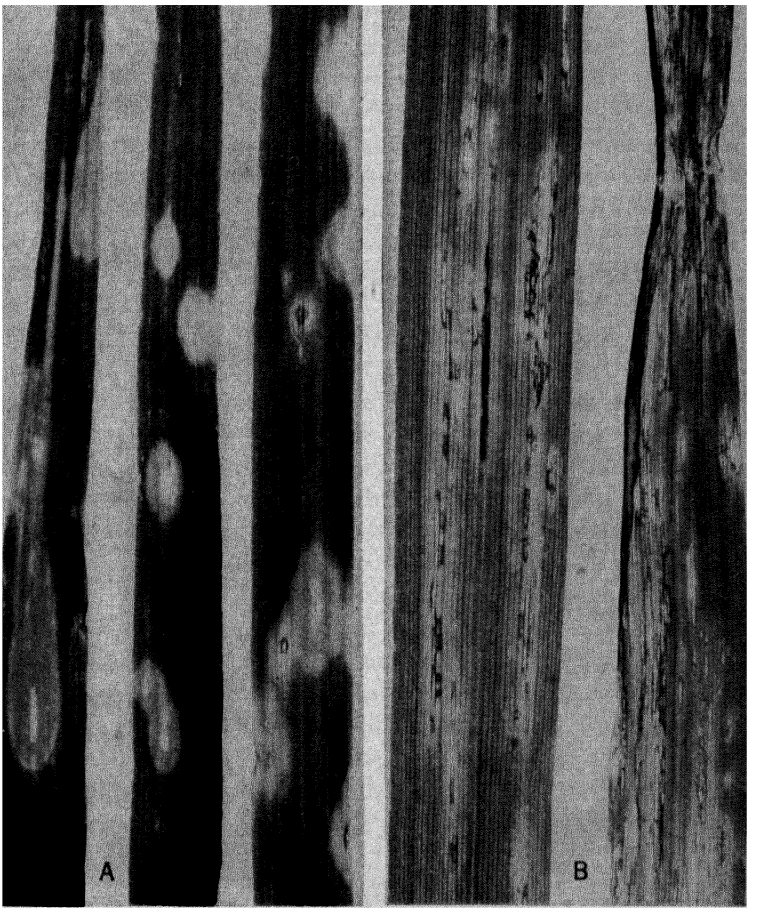

FIG. 30.—Halo blight (*A*) and stripe blight (*B*) of oats caused by *Pseudomonas coronafaciens* and *P. striafaciens* respectively, showing the progressive stages in lesion development.

become water-soaked. The bacterial colony apparently modifies the physiology of the adjacent cells without rapid necrosis of the tissue and in this manner establishes a nutritive balance with the suscept tissues without rapid advance between the cells. The pathological histology in this type of infection is in marked contrast to that of the bacteria in the gelatinous matrix advancing between the cells of the suscept, as in

the bacterial blight of barley caused by *Xanthomonas translucens* and the stripe blight of oats caused by *Pseudomonas striafaciens* (Elliott) Starr and Burk.

The Bacterium.—*Pseudomonas coronafaciens* (Elliott) Stapp
[*Phytomonas coronafaciens* (Elliott) Bergey *et al.*]
(*Bacterium coronafaciens* Elliott)
(*Pseudomonas avenae* Manns)

The motile rods with rounded ends and one or more polar flagella develop without a gelatinous matrix. The colony is white on nutrient media. See Elliott (1920, 1930) for the detailed description of the organism.

Etiology.—The bacteria enter the tissues through natural openings or injuries. Insects are important factors in the distribution and infection. Abundant moisture is necessary for the rapid development and spread of the disease. Johnson (1937) discusses the importance of high moisture content of the tissues (water-soaking) and the development of the disease. Secondary infections occur when conditions are favorable. Spikelet infection frequently occurs as the panicle is emerging from the leaf whorl. The organism is capable of existing considerable periods in crop residue. Infections of the hull and pericarp carry the bacteria over on the seed.

Control.—Seed treatment, sanitation, and rotation reduce the general abundance of the disease. The disease occurs rather generally in the humid areas despite the practice of these control measures. Apparently, insects play an important role in the general spread and establishment of the disease. Varieties show differences in susceptibility. Victoria × Richland selections are intermediate to susceptible in reaction, and Bond hybrid selections, especially D69 × Bond, are resistant.

4. Bacterial Stripe Blight, *Pseudomonas striafaciens* (Elliott) Starr and Burk.—The stripe blight occurs sparingly on oats in various sections of North America. The lesions first appear as sunken water-soaked minute spots that coalesce to form long water-soaked stripes. Bacterial exudate is apparent on the surface of the lesion. The disease is similar in appearance and the etiology is essentially the same as the bacterial blight of barley.

The Bacterium.—*Pseudomonas striafaciens* (Elliott) Starr and Burk.
[*Phytomonas striafaciens* (Elliott) Bergey *et al.*]
(*Bacterium striafaciens* Elliott)

The small rods with rounded ends and polar flagella are smaller than *Pseudomonas coronafaciens*. The colony is white on media. See Elliott (1927, 1930) for the detailed description.

5. Downy Mildew, *Sclerospora macrospora* Sacc.—The downy mildew occurs on oats in scattered locations in Europe, Australia, and the United States. See Chap. XI, Downy Mildew on wheat.

6. Powdery Mildew, *Erysiphe graminis avenae* El. Marchal.—The powdery mildew is not common on most of the cultivated varieties of oats. Reed (1920) demonstrated that many of the older varieties of oats are susceptible to certain specialized races of the fungus. This variety of the fungus is specialized on species of *Avena* and *Arrhenatherum*. See Powdery Mildew of barley (Chap. III) for the detailed discussion of the disease.

7. Fusarium Blight, *Gibberella* and *Fusarium* Spp.—The seedling blight occurs on all varieties of oats, especially in the Northern United States, Canada, and northern Europe. According to Greany *et al.* (1938) and Simmonds (1928) foot rot is also common. Losses from the kernel blight are limited, due largely to the open panicle and closed, generally pendent, spikelets and flowers. Occasionally, individual spikelets or kernels are infected. The blighted kernels are straw colored and generally have a pinkish cast from the mycelial and conidial masses. See Fusarium Blight of barley (Chap. III) and wheat (Chap. XI) for details of the disease.

8. Helminthosporium Leaf Blotch, *Pyrenophora avenae* Ito and Kuribay. Conidial stage *Helminthosporium avenae* Eidam.—The disease is distributed generally on the cultivated oats, although it is of minor importance, as reported by Dennis (1935), Drechsler (1923), O'Brien and Dennis (1933), Ravn (1901), and Turner and Milliard (1931).

Description.—The blotches are oblong to linear with an irregular margin. They are light reddish brown, frequently with a sunken center, and conidia are fairly abundant on the older portion of the lesion (Fig. 31). The blotches are generally on the leaf blade. The infected leaf blades turn yellow and dry out as the infection advances.

The Fungus.—*Pyrenophora avenae* Ito and Kuribay.

Helminthosporium avenae Eidam. Conidial stage

(*Helminthosporium teres forma avenae-sativae* Bri. and Cav.)

(*Helminthosporium avenae-sativae* Bri. and Cav.)

The morphology of the conidial stage is similar to *P. teres* (Drechsler, 1923). According to Dennis (1935), Dickson (1946), and Ito and Kuribayashi (1931), the perithecial stage is not extensively distributed. The perithecia are partly submerged, irregular in shape, and less than 0.5 mm. in diameter. Setae and conidiophores are common on the surface. Asci, when fully developed, are clavate to cylindrical, slightly curved, rounded at the apex, characteristically eight-spored, many with two to four spores. Many asci are without organized spores. Ascospores are light brown, 5-septate, constricted at the septa with the center two or three cells divided longitudinally in the mature spores (Fig. 31).

Etiology.—The primary infection occurs on the coleoptile or seedling leaves from seed-borne inoculum or mycelium, conidia, or ascospores from crop refuse. Secondary infections occur throughout the growing

season. Ascosporic inoculum is a possible source of secondary infection as well as conidia from lesions on the leaves and crop refuse. Seed

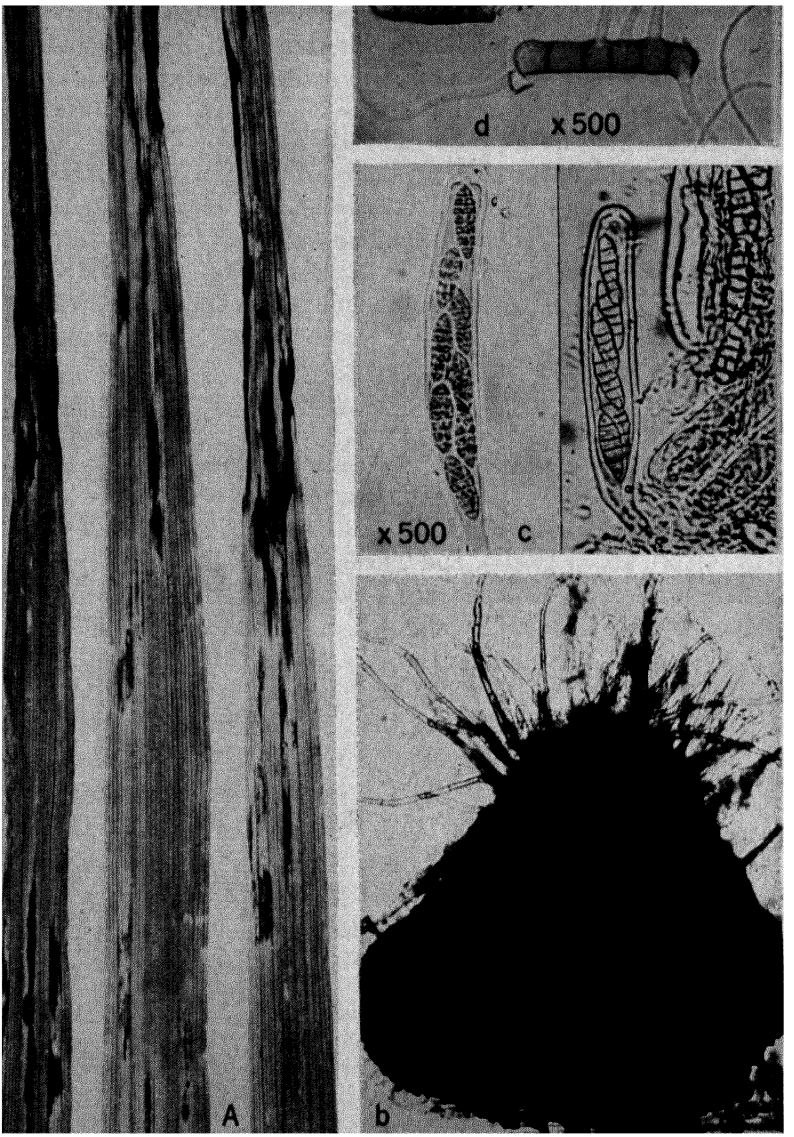

FIG. 31.—Helminthosporium leaf spot of oats caused by *Pyrenophora avenae* and the perithecium, ascospores, and conidia of the fungus. (*A*) typical reddish-brown leaf lesions, (*b*) perithecium, (*c*) asci and ascospores, and (*d*) germinating conidium.

infection is common where the blotch develops abundantly, according to O'Brien and Dennis (1933) and Ravn (1900).

A leaf blight and culm and crown rot caused by *Helminthosporium victoriae* M. and M. similar to *H. sativum* in morphology occurs on oat varieties derived from crosses with Victoria (Meehan and Murphy, 1946, and Murphy and Meehan, 1946). The disease is prevalent in the winter and spring oat areas in the Central United States, and it causes leaf blight, premature ripening, and lodging in these newer susceptible varieties.

9. Scolecotrichum Leaf Blotch, *Scolecotrichum graminis* var. *avenae* Eriks.—This leaf blotch is common throughout the world on a large group of the grasses and less general on oats. The disease is distributed widely on the grasses. The oblong to linear reddish-brown to brownish-purple blotches with regular margins develop on the leaves. The necrotic area is dry and sunken with conspicuous rows of the tufts of conidiophores that emerge through the stomata. Conidial production on most of the oat varities is sparce. For the full discussion of the disease refer to Chap. XII.

10. Anthracnose, *Colletotrichum graminicolum* (Ces.) Wils.—The disease generally is not so common on oats as on the other cereals. As reported by Sanford (1935), the disease develops on the root, crown, and basal culm tissues. In severe attacks the plants are killed prematurely. The disease is associated with dry soils low in fertility. See Chap. VIII for the detailed discussion.

11. Septoria Leaf Blotch or Speckled Blotch, *Leptosphaeria avenaria* G. F. Weber.—The disease occurs sparingly on oats and some closely related wild grasses. The blotches on the leaves are inconspicuous, with an indefinite margin and light-yellow to straw-colored areas intermingled with the green. Later the area turns light brown, and the small black pycnidia develop on the necrotic portion. Perithecia form later in the dead tissue, according to Weber (1922).

The Fungus.—*Leptosphaeria avenaria* G. F. Weber
Septoria avenae Frank. Conidial stage

The pycnidia are scattered over the necrotic area, subepidermal, globose with well-developed ostiole. Spores are rod-shaped, usually straight, 3-septate, hyaline, and guttulate. Perithecia are submerged, smooth, and black; the ostiole does not protrude. Asci are narrowly clavate with rounded apex. Ascospores are fusoid, straight, with rounded ends, 3-septate, and yellow to light olive colored.

According to Sprague (1934), a race of *Septoria tritici* Rob. occurs on oats in the Pacific Northwest.

Etiology.—The fungus persists from one season to the next in the crop residue. The mycelium develops saprophytically on the dead oat tissue, and the pycnospores remain viable during unfavorable periods. Ascosporic inoculum probably is not important in the etiology of the fungus. Leaf infections occur during cool wet weather. The fungus is capable of producing a culm rot when conditions are favorable.

12. Black Loose Smut, *Ustilago avenae* (Pers.) Rostr.—Two species of *Ustilago* cause black loose smuts on the cereals and grasses. The black loose smut on oats and some grasses is caused by *Ustilago avenae* (Pers.) Rostr. The morphology and life cycle of this fungus is similar to *U. nigra* Tapke (*U. medians* Biedenkopf) on barley. A physiologic race of *U. avenae* formerly classified as *U. perennans* Rostr. occurs on certain grasses. See Chap. III, Black Semiloose Smut of barley, for detailed discussion.

The black loose smut is world wide in its distribution on the cultivated oats and wild *Avena* spp. and related grasses. This smut is less prevalent perhaps than the covered smut in the major oat-producing areas of the United States. Separate estimates of losses from the two oat smuts are difficult to make because of variations in symptoms of the two smuts on different oat varieties (Fig. 32). The combined losses, however, are high but are being reduced rapidly in the past few years by the use of resistant varieties.

Description and Effect.—The individual flowers of the oat panicle are replaced, in large part, by the spore mass. The smut sori vary from the loose powdery black spore mass replacing the floral structures, except the rachilla, to a semiloose spore mass enclosed within the lemma and palea. Oat variety and physiologic race of the parasite largely determine the difference in symptoms. The sori at first are covered by delicate gray membranes as the panicles emerge, but these membranes soon rupture, releasing the black spore masses. The smutted panicles are the first conspicuous evidence of the disease as they appear simultaneously with the emergence of the healthy inflorescences.

The Fungus.— *Ustilago avenae* (Pers.) Rostr.
(*Uredo segetum* subsp. *avenae* Pers.)
(*Uredo carbo* var. *avenae* DC.)
(*Ustilago segetum* var. *avenae* Jens.)
(*Ustilago avenae* Jens.)
[*Ustilago avenae* (Pers.) Jens.]

The fungus is distinguished by the finely echinulate chlamydospores that germinate to form a promycelium (basidium) and sporidia. The echinulations vary from distinct to very fine thickenings on the exospore wall. See Chapter III, Black Semiloose Smut of barley, for the morphology.

Etiology.—The cycle of development is similar in both of the black loose smut fungi. Seedling infection from seed-borne chlamydospores occurs during the early stages of germination and seedling development. Systemic invasion of the growing point of the seedling results, and spores are formed in the individual flowers. Environmental factors play an important role in smut infection and development, as reported by

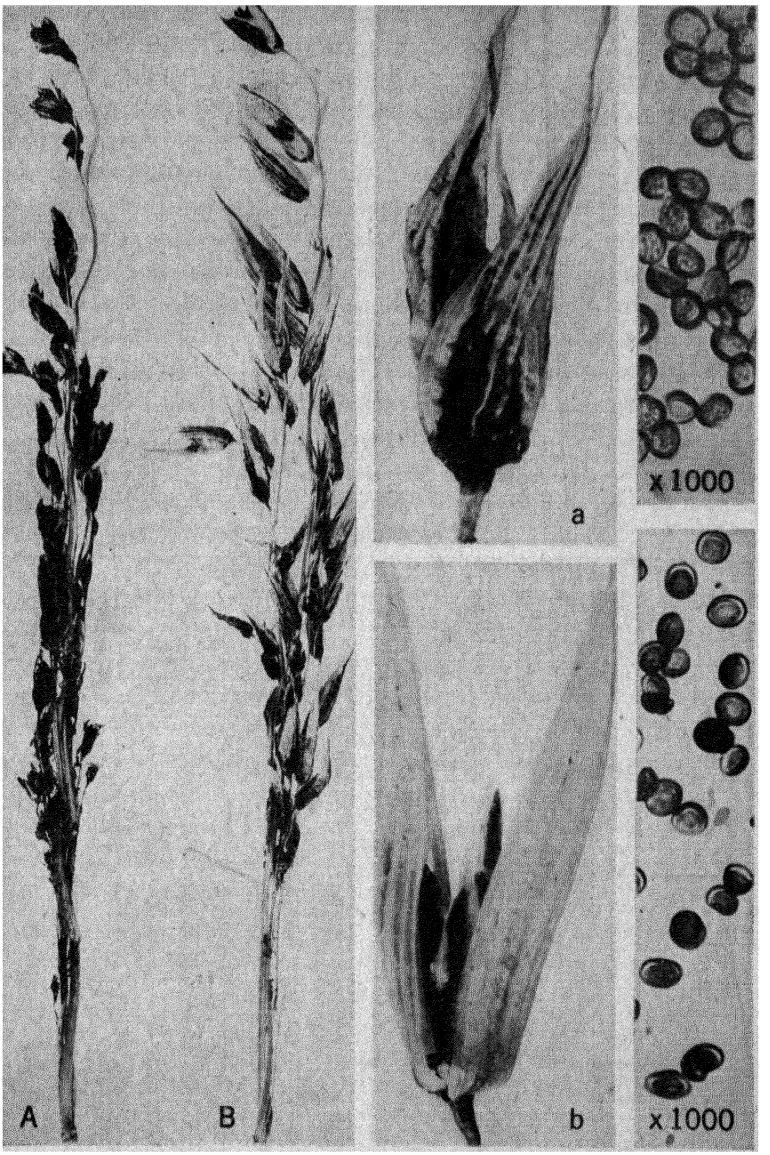

FIG. 32.—Oat panicles and spikelets infected with the black loose smut caused by *Ustilago avenae* (*A*) (*a*) and covered smut caused by *U. kolleri* (*B*) (*b*), showing the characteristic differences in symptoms and spores. The symptoms produced by the two species vary with oat varieties.

Bartholomew and Jones (1923), Johnson (1927), Jones (1923), Reed and Faris (1924), and Reed (1938). The spores are distributed from anthesis through harvesting of the crop. The spores within the floral bracts are

the best situated for seedling infection and the most difficult to reach by seed treatment. The spores germinating beneath the hulls frequently are in close contact with the young coleoptile of the seedling that the smut fungus penetrates most readily before the coleoptile is 1 cm. long. The chlamydospores located between the hulls and the pericarp are protected from the direct action of fungicides in solution as trapped air prevents complete wetting of the inner surfaces.

Control.—Seed treatment, especially with volatile fungicidal compounds, controls the loose and covered smuts of oats. Smut-resistant oat varieties are the most effective and economical methods of control. Seed treatment is advised by many pathologists and plant breeders even though smut-resistant varieties are used. This advice is based upon two main premises: (1) seed treatment to prevent the possible establish-

THE REACTION OF 10 DIFFERENTIAL VARIETIES OF OATS TO 15 PHYSIOLOGIC RACES OF *Ustilago avenae*

Physiologic race no.	Smut reaction on differential varieties									
	Anthony (C.I. 2143)	Black Diamond (C.I. 1878)	Victory (C.I. 560)	Gothland (C.I. 1898)	Monarch (C.I. 1876)	Fulghum (C.I. 708)	Black Mesdag (C.I. 1877)	Camas (C.I. 2965)	Nicol (C.I. 2925)	Lelina (C.I. 3404)
A										
1	S	S	S	R	R	R	R	R	R	R
2	S	S	S	R	R	R	R	S	R	R
3	S	S	S	R–	S	R	R	R	R	R
4	S	S	S	R	S	R	S	R	R–	R
5	S	S	S	S	R	R	R	R	R	R
6	S	S	S	S	S	R	R	R	R	R
7	S	S	S	S	S	R	R	S	R–	R
8	S	S	R	R	R	R	R	R	R	R
9	S	S	R	R–	R	S	R	R	R	R
10	S	R	S	S	R	R	R	R	R	R
11	S	R	S	S	R	R	R	R	S	R
12	R	S	R	R–	R	S	R	R	R	R
13	R–	R	R	R	S	R	R	R	R	R
14	S	S	S	R	R	S	R	R	R	S
15	R	S	R	S	S	S	R	R	R	S

R= resistant; mean, 0 to 5 per cent. R– = resistant to susceptible; mean 5 to 10 per cent. S= susceptible; 10 per cent or above.

ment and accumulation of races of the smut fungi capable of attacking the resistant variety, and (2) seed treatment to protect the seedlings from soil-borne and other seed-borne organisms parasitic on oat seedlings. Seed-treatment experiments with smut-resistant varieties in the North

Central humid area indicate increased stand in many instances, but relatively few show significant increases in yield at the standard rate of seeding (2 bushels per acre). Economizing on seed by reducing the rate of seeding, combined with seed treatment, is probably desirable in many areas.

Specialization and Resistance.--Physiologic specialization has been investigated extensively since Reed (1924) first described the two physiologic races for each species. Reed (1940) differentiated 29 races of *Ustilago avenae*; of these, 2 races (10 and 11) infected a few species and varieties, 6 races attack Fulghum (C.I. 3211), 6 races are unable to attack Fulghum but infect Gothland (C.I. 1898), and 15 races are unable to infect either Fulghum or Gothland. Roemer *et al.* (1937) and Vaughan (1938) reported a race capable of attacking Black Mesdag (C.I. 1877) and Sampson (1929, 1938) one infecting *Avena brevis*. Reed and Stanton (1942) reported a subrace 30A infecting Victoria (C.I. 4201) and Lee × Victoria selections. Races 30 and 31 infect Victoria but not the Victoria × Richland varieties; however, Hansing *et al.* (1946) reported a race infecting this latter group of varieties. Holton and Rodenhiser (1946) have reviewed the literature and reported on extensive experiments with the specialized races collected in the United States and Canada. Using 10 differential varieties of oats, they report 15 races of *U. avenae* and 7 races of *U. kolleri*. These include many of the races differentiated by others, although the equivalents are indicated in only a few. The table on the opposite page is condensed from the data presented by Holton and Rodenhiser (1946).

The practical application of information on specialization is in relation to determining smut resistance. Holton and Rodenhiser (1946) list the following oat varieties and hybrid selections, which were smut-free in field tests at Pullman, Wash., with the 22 individual races of *Ustilago avenae* and *U. kolleri*.

Variety or Selection with Pedigree	C. I. Number
Benton (D69 × Bond)	3910
Boone (Victoria × Richland)	3305
Clinton (D69 × Bond)	3971
Huron (Markton × Victory)	3756
Marion (Markton × Rainbow)	3247
Markton	2053
Marvic (Markton × Victory)	2597
Neosho (Fulghum × Markton × Victoria-Richland)	4141
Rangler (Nortex × Victoria)	3733
Bond × Anthony Sel.	4004
D69 × Bond Sel.	3662

Variety or Selection with Pedigree	C. I. Number
D69 × Bond Sel.	3663
D69 × Bond Sel.	3841
D69 × Bond Sel.	3846
D69 × Bond Sel.	4285
D69 × Bond Sel.	4272
Fulghum-Markton × Victoria-Richland Sel.	4001
Markton × Rainbow Sel.	3350
Red Rustproof × (Victoria-Richland) Sel.	3720
Richland × Fulghum Sel.	3966
Victoria-Richland × Markton-Rainbow Sel.	3609
Victoria-Richland × Morota-Bond Sel.	4301

They report many other of the newer varieties and selections as resistant or with only small percentages of smut produced by a few physiologic races. Certain varieties, however, were highly susceptible to specific races.

Resistance to the two oat smuts is conditioned by several factors and modifiers or inhibitors acting independently or in combinations. The oat varieties and physiologic races of the parasites determine the factors functioning in the expression of resistance. Single factor pairs, single factors and modifiers, and multiple factors are reported by Hayes *et al.* (1939), Reed and Associates (1934, 1935, 1937, 1938, 1941, 1942), Stanton *et al.* (1934, 1943), Torrie (1939), and many others. The nature of smut resistance and invasion of the resistant plants is discussed by Western (1936, 1937) and Zade and Arland (1933). The genetics of the oat smut fungi and inheritance of characters in crosses is summarized by Christensen and Rodenhiser (1940), Holton (1931, 1932, 1936), and Sampson (1939).

13. Covered Smut, *Ustilago kolleri* Wille.—The covered smuts of oats and barley are similar in symptoms, morphology of the fungi, and etiology. The persistence of the membrane enclosing the sorus varies with oat variety. Hybridization between *U. avenae* and *U. kolleri* results in normal segregation for both fungus characters and disease symptoms, according to Holton (1931, 1932, 1936).

The covered smut is world wide in its distribution on cultivated oats and wild species of *Avena*. Losses in general probably are greater than those caused by the black loose smut, owing to the greater prevalence of the covered smut. Varieties resistant to the covered and black loose smuts are effective in reducing losses from these diseases in the major oat-producing regions of the world.

Description and Effect.—The smut sori replacing the kernels are enclosed in a fairly permanent membrane composed of pericarp and floral bracts. The smutted panicles are the first conspicuous evidence of

the disease. In varieties in which the floral bracts are not modified greatly by the smut, the smut sori are not conspicuous until the crop is mature. The bleached lusterless lemma and palea then appear gray due to the spore mass within. The smut sori are broken during ripening of the grain and threshing, releasing the spores over the surface of the healthy kernels. In many varieties of oats, the sori develop in the lemma and palea as well as in the kernel, and in such varieties the smut is more apparent (Fig. 32).

The Fungus.— *Ustilago kolleri* Wille
(*Ustilago avenae* var. *levis* Kell. and Swing.)
[*Ustilago levis* (Kell. and Swing.) Magn.]

The fungus is distinguished by the small smooth chlamydospores which germinate to form a promycelium (basidia) and sporidia. See Chap. III, Covered Smut of barley, for morphology and detailed discussion.

Etiology.—Seed-borne chlamydospores and seedling infection occur as in the black loose smut.

Specialization and Resistance.—Reed (1940) differentiated 14 physiologic races of *Ustilago kolleri* on 10 species and varieties of *Avena*. Holton and Rodenhiser (1946) differentiate 7 races, using the same 10 differential varieties employed for determining the races of *U. avenae*, as condensed in the following table.

THE REACTION OF 10 DIFFERENTIAL VARIETIES OF OATS TO 7 PHYSIOLOGIC RACES OF *Ustilago kolleri*

Physiologic race no.	Smut reaction on differential varieties									
	Anthony	Black Diamond	Victory	Gothland	Monarch	Fulghum	Black Mesdag	Camas	Nicol	Lelina
K										
1	S	S	S	R	R	R	R	R	R	R
2	S	S	S	R	S	R	R	R	R	R
3	S	S	S	S	R–	R	R	R	R	R
4	S	S	R	R	S	S	S	R	R	R
5	S	S	S	R	S	R	S	R	R	R
6	S–	S	R	R	R	R	R	R	R	R
7	S	S	S	S	R	R	R	R	R	S

R = resistant; mean, 0 to 5 per cent.
R– = resistant to susuptible; mean, 5 to 10 per cent.
S = susceptible; 10 per cent or above.

THE REACTION IN THE SEEDLING STAGE OF 6 GROUPS OF OAT VARIETIES TO THE UREDIAL STAGE OF 12 PHYSIOLOGIC RACES OF *Puccinia graminis avenae*

Varietal groups	C.I. no.	Seedling infection type by races of *P. graminis avenae*											
		1	2	3	4	5	6	7	8	9	10*	12	13
Group 1:													
Anthony	2143	2	2	4	4	2	4	4	2	2	2	4	4
Green Mountain	1892	2	2	4	4	2	4	4	2	2	2	4	4
Minn. 742	2874	2	2	4	4	1	4	4	2	1	2		
Minrus	2144	2	2	4	4	2	4	4	2	2	2	4	4
White Tartar	551	2	2	4	4	2	4	4	2	2	2	4	4
Group 2:													
Hajira × Joanette	4023	1	1	2	1	1	1	1	1	1	1	1	1
Hajira × Joanette	4024	1	1	2	1	1	1	1	1	1	1	1	1
Hajira × Joanette	4025	1	1	2	1	1	1	1	1	1	1	1	1
Group 3:													
Victoria × (Hajira × Banner) Selections 1225, etc.		1	1	1	1	1	2	1	1	1	1	1	1
Group 4:													
Hajira	1001	1	1	1	4	1	4	2	4	x	4	1	4
Hawkeye	2464	2	1	2	4	2	4	2	4	x	4		
Iogold	2329	1	1	1	4	1	4	1	4	x	4	1	4
Iowa D67	2870	1	1	2	4	2	4	2	4	x			
Rainbow	2345	1	1	2	4	1	4	1	4	x	4		
Richland	787	1	1	1	4	1	4	1	4	x	4	1	4
Group 5:													
Joanette strain	2660	1	4	1	1	x	4	4	4	4	x	x	x
Sevnothree	3251	1	4	1	1	x	4	4	4	4	x	x	x
Group 6:													
Alber	2766	4	4	4	4	4	4	4	4	4	4		
Belar	2760	4	4	4	4	4	4	4	4	4	4	4	4
Bond	2733	4	4	4	4	4	4	4	4	4	4	4	4
Cassel	2911	4	4	4	4	4	4	4	4	4	4		
Cowra	2761	3	4	4	4	4	4	4	4	4	4		
Fulmer	2912	4	4	4	4	4	4	4	4	4	4		
Glabrota	2630	3	4	4	3	4	4	4	4	4	4	4	4
Gopher	2027	4	4	4	4	4	4	4	4	4	4	4	4
Kareela	2774	4	4	4	4	4	4	4	4	4			
Ruakura	2025	4	4	4	4	4	4	4	4	4	4	4	4
Sterisel	2891	4	4	4	4	4	4	4	4	4	4	4	4
Swedish Select	134	4	4	4	4	4	4	4	4	4	4		
Victoria	2401	4	4	4	4	4	4	4	4	4	4	4	4
Victory	1145	4	4	4	4	4	4	4	4	4	4	4	4

*Race 10 A shows a resistant (1) reaction on group 5 instead of the mesothetic reaction.

1—Resistant (minute uredia surrounded by solid necrotic areas). 2—Moderately resistant (uredia small with hypersensitive areas around them). 3—Moderately susceptible (medium-size uredia with slight chlorosis but no necrosis). 4—Extremely susceptible (large confluent uredia). x—Mesothetic (infection and uredial size heterogenous and undefined). 1 and 2—Resistant to practically resistant. 3 and 4—Susceptible.

The table of smut-free varieties and selections given under the black loose smut (page 117) indicates the wide range of selections resistant to both species. In addition, some of the newer oat varieties are reported by Holton and Rodenhiser (1946) as highly resistant to black loose smut and free from covered smut, notably Marida (C.I. 2571) (Markton × Idamine), Sac (C.I. 3907) (Bond × Iogold), Bannock × Victoria-Richland Sel. (C.I. 4181), and D69 × Bond Sel. (C.I. 4532); whereas a number of the newer varieties showed a low percentage of smut produced by race 7 of *Ustilago kolleri.*

14. Stem Rust, *Puccinia graminis avenae* Eriks. and Henn.—Stem rust occurs on some varieties of all *Avena* spp. and on many related grasses. The disease is world wide in its distribution on oats, and it reduces the forage value and yield of grain. Stem rust resistant varieties are reducing the importance of this disease on oats. According to Stakman and Loegering (1944), physiologic races 8 and 10 of the fungus are a potential danger to the many rust-resistant varieties originating from the Victoria × Richland cross.

The red rust stage of the disease is conspicuous on the leaves and culms. In the northern spring-oat area, the disease occurs relatively late on most oat varieties. The black rust stage is apparent late in the season on the leaf sheaths and culms. See Chap. XI, Stem Rust of wheat, for the detailed discussion of the disease.

Specialization and Resistance.—Specialization occurs within the physiologic variety *Puccinia graminis avenae.* As reported by Levine and Smith (1937), Newton *et al.* (1940), and Stakman *et al.* (1935), 12 physiologic races are differentiated on three groups of oat varieties. Fischer and Claassen (1944) reported race 14 obtained from *Poa ampla* Merr. A modification of the tabulations by Levine and Smith (1937) and Newton *et al.* (1940) is given on page 120.

Three varieties in group 1 (Anthony, Minn. 742, and Minrus), two varieties in group 4 (Rainbow and Richland), and two varieties in group 6 (Gopher and Victoria) give the same reaction in the seedling and adult plant stage. The group 5 varieties are influenced in their reaction by light and temperature, as has been shown by Gordon (1933). The varieties in groups 1, 4, and 5 are used for differentiating the physiologic races on oats. The reaction of the 0 to 4 types is discussed in Chap. XI.

No variety of oats known is resistant to all physiologic races of the stem rust parasite. Selections from Hajira (C.I. 1001) × Joanette (C.I. 2660) combine resistance or moderate resistance to all known races, as reported by Welsh (1937). According to Murphy *et al.* (1942) and Newton *et al.* (1940), Richland (C.I. 787) and Iogold (C.I. 2329) are resistant to the more common races in North America. Rainbow (C.I. 2345), Hajira (C.I. 1001), and Rusota (C.I. 2343) are similar in reaction to stem rust.

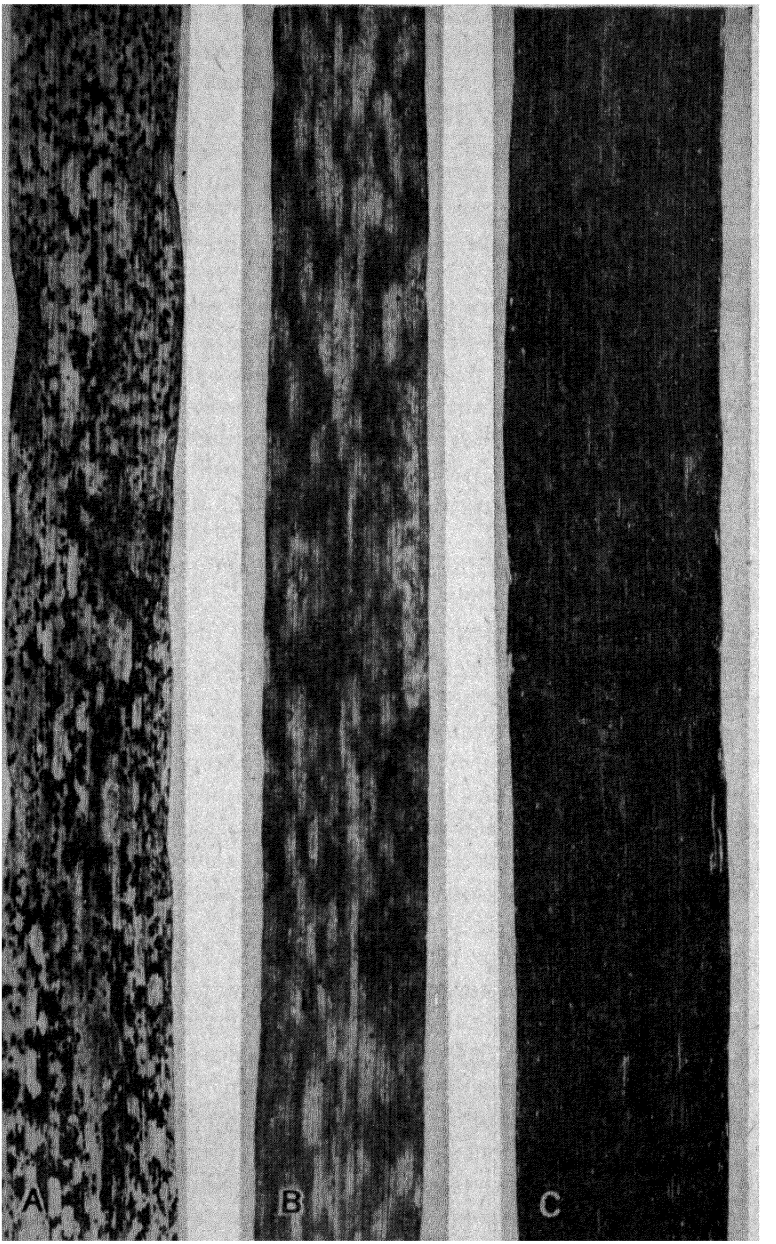

FIG. 33.—Crown rust reaction of oat varieties under field conditions. (*A*) Sixty day, numerous uredia and telia, (*B*) Vicland, necrosis and minute uredia, and (*C*) Bond absence of necrosis and uredia with common races of parasite.

The varieties in group 1 (see table above) are moderately resistant, and the selections in groups 2 and 3 are resistant to races 8 and 10 discussed by Stakman and Loegering (1944). Resistance to stem rust in oats is dominant and inherited on a single-factor basis in most crosses studied, as summarized by Smith (1934).

15. Crown Rust, *Puccinia coronata* (Pers.) Cda.—The crown rust or leaf rust occurs on most species of *Avena* and many related grasses. The aecial stage occurs more commonly on *Rhamnus cathartica* L. and *R. lanceolata* Pursh., according to Melhus *et al.* (1922) and Tranzschel (1934). The distribution of the rust is world-wide in temperate humid and semi-humid areas. The disease causes heavy losses, that are probably larger than those from stem rust in most oat-growing areas.

Description and Effects.—All stages of this heteroecious long-cycle rust are common symptoms in the temperate zones. The aecial infections on the *Rhamnus* spp. are common and conspicuous, the orange-yellow elevated lesions occurring on leaves, young stems, and fruits. The uredia develop on the leaves and floral structures of oats and grasses. The uredia are round to oblong but soon spread and coalesce to form irregular orange-yellow patterns. The epidermis of the suscept is not turned back as in the stem rust uredia. The telia frequently form a dark border around the uredia and develop independently, especially on the leaf sheath in linear dark-brown spots covered by the epidermis of the suscept (Fig. 33). Oats heavily infected with crown rust lodge badly and ripen prematurely.

The Fungus.—*Puccinia coronata* (Pers.) Cda.
(*Puccinia coronifera* Eriks.)
(*Puccinia lolii* Niel.)
(*Puccinia coronifera Kleb.*)

The aecial stage consists of the raised pycnia (spermatia) with exudate usually on the upper surface of the leaf and the aecia with large peridia on the lower surface (Fig. 34). The aeciospores are subglobose, finely verrucose, and light orange-yellow. The uredio-spores are globose or ovate, markedly echinulate, have three to four germ pores located irregularly, and are orange-yellow in color. The telia are covered by the epidermis except when forming directly in the uredia. The teliospores are constricted slightly at the septum, the apex is thickened with several blunt processes forming a crown-like apex, the pedicels are short and thickened, the color is dark brown (Fig. 34).

Etiology.—In the major spring-oat areas, the aecial host plays an important role in the cycle of development of the fungus. Heavy aecial infection occurs in the early spring usually when the spring-sown oats are in the late seedling or early tillering stage of development. Aeciospore spread to the oats and grasses occurs early. The development of the uredial stage from primary infections from these wind-borne spores is scattered considerable distances from the *Rhamnus*. In agricultural

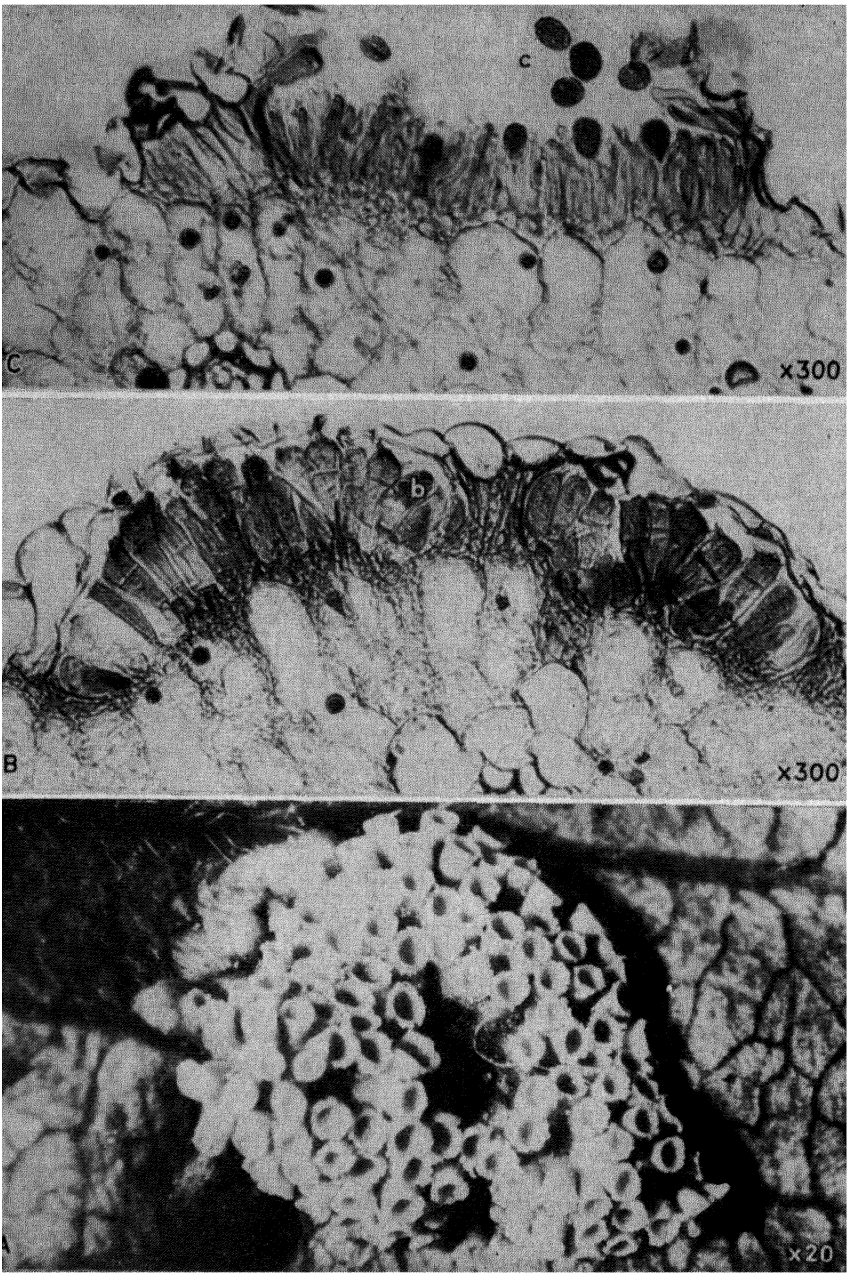

FIG. 34.—The spore forms of *Puccinia coronata*. The aecium on *Rhamnus cathartica* showing the peridia of the aecial cups (*A*). The urediospores (*c*) and teliospores (*b*) are produced in the uridium (*C*) and telium (*B*) shown in transections of the oat leaf.

areas where the *Rhamnus* is used as a hedge plant, as occurred some years ago in Rock County, Wisconsin, the early spread of crown rust to the oat plants is general. The uredial stage develops rapidly, and secondary infections from urediospores occur whenever weather is favorable. Telia develop later as the oats and grasses mature. The teliospores overwinter on the old leaves and germinate to form the sporidia that infect the *Rhamnus* spp. the following spring. Allen (1932) and Buller (1941) studied the cytology and sexual fusion of the aecial stage. Murphy (1935) investigated physiologic races from *Rhamnus* spp. inoculations. In the southern oat areas, the uredial stage develops continuously on the oats and grasses. Northern spread of the urediospores occurs from these southern sections. The spring infection from this source of inoculum is usually much later in the season than where the aecial infection occurs. Extensive investigations on the overwintering of the uredial stage in the northern spring-oat area of North America and Europe indicate the rarity of its occurrence.

Control.—Eradication of the *Rhamnus* is practiced in some sections. Some states combine *Rhamnus* and *Berberis* eradication. *Rhamnus* hedges around fields are generally removed by farmers when they realize the connection between the *Rhamnus* and the crown rust in their oat fields. Rust-resistant varieties are rapidly replacing the older susceptible varieties.

Specialization and Resistance.—Specialized races of *Puccinia coronata* are numerous. According to Murphy *et al.* (1942), 51 races are known to occur in North America. Straib (1937) using 15 varieties, differentiated 142 races in Europe, whereas, Vallega (1942) using the 13 standard varieties described 4 races from Argentina where the rust persists largely in the uredial stage. The races in North and South America and the oat varieties upon which they are differentiated are given in the following table. The types of reaction of the different varieties to the uredial stage of *P. coronata* are shown in (Fig. 33).

Crown rust resistant parental material is available for oat breeding. According to Murphy *et al.* (1942), Bond (C.I. 2733) is resistant to 48 of the known races in North America and Victoria (C.I. 2401) to 47. One or the other of these two introduced varieties is involved in most of the smut- and rust-resistant varieties distributed during the past few years, notably Boone (C.I. 3305), Tama (C.I. 3502), Vicland (C.I. 3611), and several others from the Victoria × Richland and more recent selections from Bond hybrids. These new varieties are making Murphy's (1942) regression of yield on coefficient of crown rust a reality in the spring-oat area of the United States. Other varieties and selections are being used also in various recombinations.

THE REACTION IN THE SEEDLING STAGE OF 13 VARIETIES OF OATS TO THE UREDIAL STAGE OF 56 PHYSIOLOGIC RACES OF *Puccinia coronata* FROM NORTH AND SOUTH AMERICA

Physiologic race no.	Reaction of differential varieties to races of *P. coronata*												
	Avena sativa						*A. sativa orientalis*		*A. byzantina*				*A. strigosa*
	Ruakura (C.I. 2025)	Green Russian (C.I. 2890)	Hawkeye (C.I. 2264)	Anthony (C.I. 2143)	Sunrise (C.I. 982)	Victoria (C.I. 2401)	Green Mountain (C.I. 1892)	White Tartar (C.I. 551)	Appler (C.I. 1815)	Sterisel (C.I. 2991)	Bélar (C.I. 2760)	Bond (C.I. 2733)	Glabrota (C.I. 2630)
1	4	4	4	4	4	1	4	4	4	4	3	0	0
2	1	0	4	0	0	0	0	0	0	1	0	0	0
3	0	4	4	4	0	1	4	4	0	0	0	0	0
4	3	1	4	0	4	0	x	1	4	4	4	0	0
5	0	1	4	0	4	0	0	0	4	3	4	..	0
6	0	4	4	4	4	2	4	4	4	4	4	0	0
7	4	4	0	0	4	1	0	0	4	4	4	0	0
8	1	3	4	4	0	0	4	4	1	3	2	..	0
9	1	2	3	3	2	0	3	3	1	1	2	..	0
10	4	4	4	4	1	1	1	1	4	4	4	..	0
11	4	4	0	0	2	0	0	4	4	4	4	0	4
12	4	4	0	3	4	1	4	0	4	3	3	0	0
13	4	4	4	4	2	0	4	4	4	0	0	..	3
14	1	4	4	4	1	0	4	4	4	0	0	..	0
15	0	4	4	4	4	0	4	4	4	4	1	..	0
16	1	4	0	0	0	1	1	4	0	0	0	0	4
17	0	4	4	0	4	1	1	4	4	..	4	..	0
18	0	4	0	0	0	0	0	4	0	..	0	..	0
19	0	4	4	1	1	0	1	4	1	0	1	0	0
20	4	4	0	4	0	0	0	0	4	4	4	0	0
21	0	0	4	4	0	0	4	4	4	4	4	..	0
22	0	4	4	0	4	0	0	0	4	4	4	..	0
23	4	4	4	4	0	1	4	4	0	4	2	..	0
24	0	4	4	4	0	0	4	4	0	0	0	0	4
25	4	4	4	0	4	0	4	0	4	4	4	0	0
26	4	4	4	0	4	0	0	4	4	4	4	0	0
27	4	4	4	0	4	0	4	0	4	0	4	..	0
28	4	0	4	1	0	0	4	4	0	3	3	0	0
29	0	2	0	0	0	0	0	0	2	2	0	0	0
30	4	4	4	3	4	1	4	4	4	3	4	0	3

THE REACTION IN THE SEEDLING STAGE OF 13 VARIETIES OF OATS TO THE UREDIAL STAGE OF 56 PHYSIOLOGIC RACES OF *Puccinia coronata* FROM NORTH AND SOUTH AMERICA (*Continued*)

Physiologic race no.	Reaction of differential varieties to races of *P. coronata*												
	Avena sativa						*A. sativa orientalis*		*A. byzantina*				*A. strigosa*
	Ruakura (C.I. 2025)	Green Russian (C.I. 2890)	Hawkeye (C.I. 2264)	Anthony (C.I. 2143)	Sunrise (C.I. 982)	Victoria (C.I. 2401)	Green Mountain (C.I. 1892)	White Tartar (C.I. 551)	Appler (C.I. 1815)	Sterisel (C.I. 2991)	Belar (C.I. 2760)	Bond (C.I. 2733)	Glabrota (C.I. 2630)
31	3	3	3	x	3	1	3	x	4	4	4	0	0
32	4	x	4	x	x	1	x	x	3	2	x	0	0
33	4	0	3	3	4	0	3	3	4	3	x	4	1
34	4	0	0	0	4	0	0	0	4	4	4	4	0
35	3	0	3	3	2	0	4	4	2	0	1	0	3
36	3	3	4	3	0	0	4	3	0	0	x	0	4
37	4	3	2	2	3	0	2	2	4	0	4	0	0
38	0	0	4	0	0	..	0	0	0	0	0	..	4
39	0	0	4	0	0	..	0	0	0	0	4	..	0
40	4	4	0	0	4	1	4	0	4	4	4	0	0
41	1	2	4	4	4	3	4	4	3	3	4	0	0
42	0	3	3	3	0	..	4	4	1	0	3	..	4
43	3	3	4	3	3	..	4	4	3	4	0	1	0
44	0	1	4	0	0	..	1	3–4	0	1	1	2	4
45	4	4	3	3	4	2	3	3	4	4	4	4	0
46	3	0	0	0	4	0	0	0	3	3	3	0	0
47	1	4	0	1	4	2	0	1	4	4	4	0	0
48	2	3	2	2	3	2	3	2	4	4	4	0	0
49	1	4	1	2	3	0	4	4	4	4	3	0	0
50	2	2	1	2	4	3	2	4	4	4	4	0	0
51	0	4	4	4	4	0	4	4	0	2	2	0	0
52	2	4	0	0	3	3	3	3	4	4	4	0	0
53	3	2	4	0	4	3	1	3	4	4	4	0	0
54	0	0	0	0	4	0	0	0	4	4	3	0	4
55*	1	3	4	4	4	4	4	4	3	3	4	4	0
56*	3	4	4	4	3	4	4	3	4	3	3	0	0

*Vallega, J. from Argentina, two additional races (1 and 45) are reported from Argentina.

The Victoria variety and the majority of the varieties selected from hybrids in which it was involved are susceptible to the Helminthosporium blight caused by *H. victoriae* (see p. 113). There is apparently a close association or linkage in the Victoria variety between the factors for resistance to crown rust and susceptibility to this Helminthosporium blight. Evidence indicates, however, that some few hybrid selections involving this variety in the cross are resistant to the blight. The recent damage in some locations caused by this disease on oat varieties derived from crosses with Victoria has directed attention to other sources of crown rust resistance, notably Bond. However, Bond is susceptible to physiologic races 33, 34, and 45 of *Puccinia coronata*. These races are distributed sparsely at present, chiefly in the Southern United States' winter-oat area. Race 45 of *Puccinia coronata* is also found in Argentina. More recently, race 45 has increased in prevalence and damage in the north-central portion of the winter-oat area[1]. In this area, Bond and its derivatives are damaged also by the anthracnose disease. Ukraine (C.I. 3259), a variety of *Avena sativa* introduced from Russia by the author is resistant to races 33, 34, 45 and equally resistant to form 41, the only form found at present to which Victoria is susceptible[2]. This variety is only moderately resistant to a biotype of physiologic race 1 of *Puccinia coronata*. Ukraine is being used as a further source of crown rust resistance.

The following table taken from Murphy *et al.* (1942) summarizes the more important varieties used in breeding oats for disease resistance.

THE MORE IMPORTANT OAT VARIETIES USED IN BREEDING FOR DISEASE RESISTANCE

Smuts		Crown rust		Stem rust	
Variety	Relative resistance	Variety	Relative resistance	Variety	Relative resistance
Victoria.....	+++	Bond.....	+++	Richland...	+++
Markton.....	++++	Victoria....	+++	Iogold.....	+++
Navarro.....	+++	Rainbow...	+	Rainbow...	+++
Bond.....	++	Alber.....	+	White Tartar	++
Black Mesdag..	++	Capa.	+	Jostrain....	+

++++ highly resistant to all known races.
+++ highly resistant to all races tested or except to certain rare races.
++ adequate resistance under most field conditions.
+ moderately resistant.

[1] ROSEN, H. R., *Phytopath.* 35:143-144, 1945, and more recent information given the author.

[2] MURPHY, H. C., *Iowa Agr. Exp. Sta. Rpt.* 1936, part 1, 98-99.

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CHAPTER VII

RICE DISEASES

The cultivated rice varieties belong to the one species *Oryza sativa* L. The rice plant is a warm-climate annual with both spring and intermediate to winter types of growth response, or more correctly expressed in the case of rice, long- and short-day photoperiodic response are represented. The crop is grown without irrigation (upland) and with irrigation (lowland). The major commercial crop is grown under irrigation or where the plants are grown in water the greater part of the growing season. The basic chromosome number in the cultivated rice varieties and most wild varieties of this species is 12 pairs. Multiples of this basic number occur in the wild *Oryza* spp. According to Jones (1936), haploid, triploid, and tetraploid rice plants occur.

The rice plant is adapted to relatively warm climates. Jones (1936) gives a mean temperature of 70°F. or above during the entire growing season as essential for successful rice culture. The physiological anatomy of the plant is similar to the other Gramineae. The high silicon content of the cell membranes especially of leaf and floral bracts is notable for rice and closely related species. Attempts have been made to correlate silicon content with disease resistance in some instances. The anatomy of the leaf is different than many of the grasses, as discussed by Tullis (1935). Long-, medium-, and short-grain types are represented in the commercial varieties.

Diseases are important in the economical production of rice. Seedling blight and culm rot maladies are of major importance. Estimated average annual losses from rice diseases in the United States are not tabulated. The total reduction in yield and quality is probably less than in the other cereal crops. Losses in the more intensive rice areas of the Pacific are frequently higher than those in the United States.

1. Nonparasitic.—*Straighthead* of rice is prevalent in certain sections of the United States. The disease apparently occurs on new rice land or on soils where large amounts of organic material are plowed under. Tisdale (1921) reported it as one of the most destructive diseases of irrigated rice in the Southern United States. According to Tullis (1940) more recently, the occurrence of the trouble is limited and practical control is practiced. The leaves are dark green and stiff. Frequently one or both glumes are absent and the grain does not fill. Large roots, without

branches and root hairs, develop rather than the fine well-branched fibrous root system. The plants remain green with erect panicles as the crop matures. The disease is prevented under most conditions by draining off the irrigation water after 6 weeks and allowing the soil to dry thoroughly, after which the water is applied and left on for the remainder of the season. Proper crop rotation and avoidance of excessive amounts of raw organic material turned into the soil, constitute the best means of preventing soil conditions conducive to straighthead.

White tip, due partly to soil mineral deficiencies (Martin and Allstatt, 1940, and Tullis, 1940), is prevalent in many rice areas of the United States. The white tip or upper half of the leaf blade and distortion of the panicle by the tightly clasping leaf sheath with some sterility are the important symptoms. Practical control of the disease is difficult and requires crop rotation as well as the use of balanced fertilizers.

2. Dwarf, or Stunt, Insect-transmitted Virus.—The stunt disease of rice occurs only in the Japanese Islands and eastern Asia insofar as present distribution is recorded. The disease is important historically both from the early records of damage and the first recorded insect transmission of a virus. As summarized by Fukushi (1934, 1940) and Katsura (1936), the insect-transmission studies on dwarf started by Takami in 1883 and reported from 1895 to 1908 antedated the report of Ball in 1905 on the insect transmission of the virus causing curly top of beets.

Description and Effect.—The dwarf disease of rice is a streak and rosette combination of symptoms. The plants are dwarfed, tillering excessively, leaves and stunted panicles are erect and late, and leaves are streaked. The leaves frequently show white specks along the veins. These develop into narrow interrupted to continuous light-yellow streaks on a dark-green leaf background. Intracellular bodies are present in the cells of the chlorotic tissues and in the cells of crown and roots. According to Fukushi (1934, 1940), *Panicum miliaceum* L., *Echinochloa crusgalli* var. *edulis* Hon., *Alopecurus fulvus* L., and *Poa pratensis* L. are subject to attack but not extensively diseased. Rye, wheat, and oats are slightly susceptible.

The insect vector is the rice leaf hopper, *Nephotettix apicalis* var. *cincticeps* (Uhl.). Fukushi (1933, 1935, 1937, 1939) has studied the insect and virus relationship in great detail. Control is accomplished in part by destruction of the leaf hopper. The insects overwinter on *Astragalus sinicus* L., which is common as a winter crop and weed in the rice areas.

Kuribayashi (1931) described a stripe virus on rice that is characterized by yellowish stripes on the leaf and some stunting of the plant. The virus is transmitted by another leaf hopper, *Delphacodes striatellus* (Fall.).

3. Bacterial Kernel Rot, *Pseudomonas itoana* Toch.—Tochinai (1932) described a black kernel rot of the rice grain similar to the black chaff of

wheat. The disease is distributed sparsely in the Japanese Islands. He described the short rod-shaped bacterium with polar flagella as *Pseudomonas itoana* Toch.

4. Fusarium and Gibberella Blight or "Bakanae Disease", *Gibberella fujikuroi* (Saw.) Wr. and *G. zeae* (Schw.) Petch [*G. saubinetii* (Mont.) Sacc.].—Rice in the more humid Asiatic regions is damaged by this group of fungi. Seedling blight and kernel blight are reported by Seto (1935), including a good summary of the literature. The disease on rice is similar in general symptoms and etiology to the disease on the other cereal crops. Certain cultures, notably of *Gibberella fujikuroi*, stimulate internodal and root elongation. Extracts of these fungus cultures produce the same effect not only on rice, but also on many other species of the Gramineae. The Japanese investigators have studied this phase of the problem extensively.

Tullis (1936, 1940) reported the isolation of *Fusarium* from pink and yellow discolored rice kernels and from those with a chalky endosperm from the humid rice-producing areas in the United States. *Fusarium* species ranked second in frequency of occurrence in the discolored grain. Germination and vigor of seedling development is reduced in the discolored kernels according to Tisdale (1922).

Although the Gibberella blight on corn, wheat, and barley has been studied extensively in the United States, the disease on rice has received little attention. Probably *Gibberella fujikuroi* occurs commonly on this crop in the humid rice sections of the United States as is the case in Asiatic rice areas.

5. False Smut, *Ustilaginoidea virens* (Cke.) Tak.—The sclerotia occur, replacing the flowers similar to ergot. Usually only a few flowers are infected in a panicle, and the sclerotia are large enough to be removed in cleaning the threshed rice. According to Butler (1918) and Fulton (1908), the disease is widely distributed but of little economic importance.

The Fungus.— *Ustilaginoidea virens* (Cke.) Tak.
[*Ustilaginoidea oryzae* (Pat.) Bref.]

According to Brefeld (1895), this parasite as well as *Ustilaginoidea setariae* Bref. on Setaria are related closely to Claviceps. Gäuman and Dodge (1928) state that the genus, based on *Ustilaginoidea setariae*, differs from Claviceps chiefly in the imperfect forms. The conidia are borne over the surface of the pseudomorphs replacing the ovary. The conidia are spherical, echinulate, and 4 to 6 microns in diameter. They germinate to form secondary spores. The sclerotia, protruding from the floral bracts, are spherical, 5 to 8 mm. in diameter, and olive green in color due to the layer of conidia. Germination of the sclerotia of *U. virens* is undescribed.

The etiology is indefinite. The scattered sclerotia in individual flowers indicates a cycle similar to that of *Claviceps purpurea.*

6. Culm Rot, *Leptosphaeria salvinii* Catt.—The culm rot is an impor-

tant disease in most rice-producing sections of the world. The disease causes lodging and lightweight grain. The lesions, on the leaf sheath near the water line, appear first, a month to 6 weeks before the plants head. The black discolored areas spread around the culm and inward

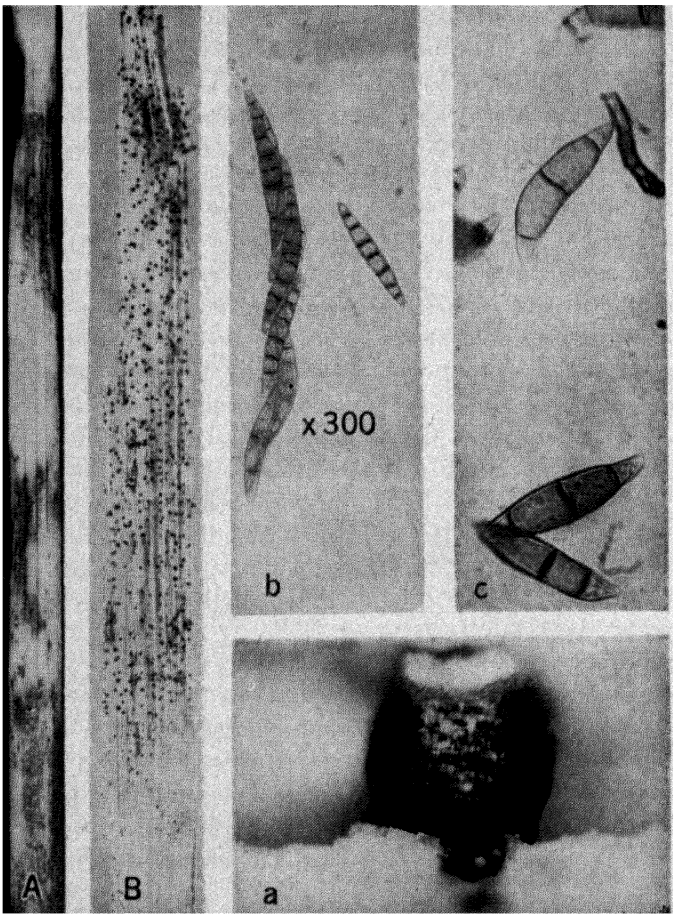

FIG. 35.—Culm rot of rice caused by *Leptosphaeria salvinii* showing the lesion on the culm (*A*) and the sclerotia on the old straw (*B*). The perithecium (*a*), the ascus and eight ascospores (*b*), and conidia (*c*) are shown in the inserts. (*Courtesy of E. C. Tullis.*)

into the culm tissue. Numerous black sclerotia develop inside the leaf sheath and later in the culm. The fungus mycelium forms dark threads on the epidermis of the culm. The lesion extends in dark-brown streaks above the older black necrotic area. As the panicles fill, the stalks break over in the necrotic area and early infection results in poorly filled grain.

Butler (1913) reported excessive late tillers as a symptom. Tullis (1933) indicated that excessive tillering is not associated with the disease in the United States. The characteristic symptoms are shown in (Fig. 35).

The Fungus.—*Leptosphaeria salvinii* Catt.
(*Sclerotium oryzae* Catt.). Sclerotial stage
(*Helminthosporium sigmoideum* Cav.). Conidial stage
(*Helminthosporium sigmoideum* var. *microsphaeroides* Nakata)

The mycelium is white inside the tissues and olivaceous on the surface of the tissues with numerous olivaceous irregular appresoria. The spherical sclerotia are black, nearly smooth, and mostly 230 to 270 microns in diameter. Conidiophores are dark, septate, simple or sparsely branched. The conidia are borne singly on a sharp-pointed sterigmata. Conidia are fusiform, slightly curved, typically 3-septate. The two intercalary cells are dark brown; apical cells are lime green. Germination of the apical cells is characteristic. Perithecia are dark, globose, imbedded in the outer tissues of the sheath, with a short beak flush with the epidermis of the sheath, and average 381 microns in diameter. Asci are narrowly clavate, short stalked, with delicate wall. Ascospores are arranged biseriatly, fusiform, 3-septate with the center septum constricted when mature, brown with two end cells lighter brown, mostly 44 to 48 microns long by 8 microns wide (Fig. 35).

Etiology.—The sclerotial stage occurs commonly on rice and certain wild grasses. This stage has been described from most rice-growing sections. The conidial stage is usually associated with the sclerotial stage, but it is known to occur in fewer areas. The ascigerous stage is less common and is reported from fewer locations than the sclerotial stage. The fungus persists in the sclerotial stage and develops mycelium and conidia from these structures. Primary infections are commonly from the sclerotia.

Control.—According to Tullis (1940) the most satisfactory control is to drain the water from infected fields before the infections reach the rice culm. Only enough water is added, after draining to maturity, to keep the soil saturated. This control measure results in a slight reduction in yield of healthy fields, but it prevents lodging in heavily infected fields. No highly resistant commercial varieties are available. Cralley (1936) and Tullis and Cralley (1933) noted the difference in susceptibility of varieties and that the Japanese short-grained varieties are more resistant than the long-grained types. According to Cralley (1939), a complete fertilizer containing some excess of potassium increased yields without increasing stem rot. According to Cralley and Tullis (1935), a similar culm rot is caused by *Helminthosporium sigmoideum* var. *irregulare* Cralley and Tullis. The disease is not so severe as in the former. The sclerotia are numerous and irregular in shape and size and smaller than those of *Leptosphaeria salvinii*. The conidia are similar in both, except that

those of the variety, *H. sigmoideum irregulare* commonly germinate before maturity. No perfect stage of this latter fungus is described.

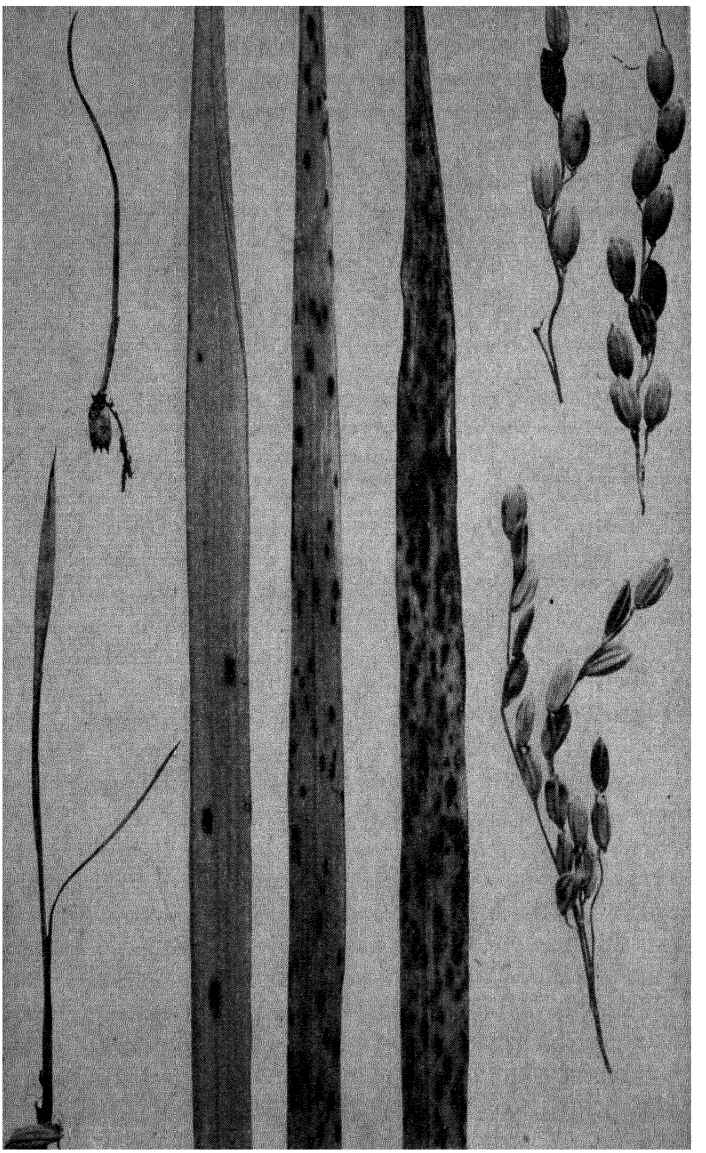

FIG. 36.—Leaf and kernel lesions and seedling blight on rice caused by *Cochliobolus miyabeanus* (*Helminthosporium oryzae*).

7. Black Sheath Rot, *Ophiobolus oryzinus* Sacc.—Tullis (1933) has reviewed the literature on the several species of *Ophiobolus* reported on rice.

Certain of these more common on wheat and other cereals are discussed in Chap. XI. Miyake (1910) described *Ophiobolus oryzae* Miyake, which apparently is different morphologically from *O. oryzinus* and *Cochliobolus* (*O.*) *miyabeanus* Ito and Kuribay. The latter was described by Ito and Kuribayashi (1927) as the ascigerous stage of *Helminthosporium oryzae* Breda de Haan.

The black sheath rot is of minor importance, as the culm tissues are rarely invaded. The disease appears as a black rotted lesion on the sheath tissues near the water line. The fungus perithecia are formed on rice straw and stubble and on the sheaths of *Typha latifolia* L., the common cattail.

8. Helminthosporium Blight, *Cochliobolus* (*Ophiobolus*) *miyabeanus* Ito and Kuribay. (*Helminthosporium oryzae* Breda de Haan).—The disease is apparently world wide in distribution on rice. Damage is frequently severe. Losses in stand due to seedling blight, in yield due to leaf and culm infection, and in quality and yield by kernel infection are frequently high.

Description and Effect.—The seedling blight is similar in appearance to that caused by *Helminthosporium sativum* on wheat and barley. The brown cortical lesions appear on the coleoptile, subcrown internode, and seminal roots. Seedling blight occurs before or after emergence. Brown leaf spots and leaf-sheath lesions develop on the less severely infected seedlings. Circular to elongate brown leaf spots are first small without marked water-soaking and later spread with reddish-brown margins and gray centers (Fig. 36). On severely infected plants the leaves dry out before the plants are mature. Conidiophores and conidia develop on the lesions. Lesions on the culm, below the panicle or at the base of the panicle, are common when weather conditions are favorable. The brown necrotic areas result in shriveled kernels and broken panicles. Blighted kernels result from early flower infections. Small brown lesions on the floral bracts and pericarp cause discoloration of the milled rice.

The Fungus.—*Cochliobolus* (*Ophiobolus*) *miyabeanus* Ito and Kuribay. (*Helminthosporium oryzae* Breda de Haan). Conidial stage
(*Helminthosporium macrocarpum Thuem*)
(*Helminthosporium oryzae* Miy. and Hori)

Ito and Kuribayashi (1927) described the ascigerous stage produced in culture from single conidia and named it *Ophiobolus miyabeanus* Ito and Kuribay. They state (1931) that perithecia were secured only in culture and have not been collected in the field. Tullis has collected the perfect stage under field conditions in the United States.

The mycelium forms grayish-brown to dark-brown mycelial mats in and on the plant parts and in culture. The conidiophores form in mats or singly and are light brown to olivaceous and vary greatly in both width and length. The conidia are brown, slightly curved, tapering toward the round apex and toward the base, and vary greatly in shape

and septation. The peripheral wall is moderately thin, and the hilum is inconspicuous. Spores germinate characteristically from the two apical cells. Perithecia in culture are

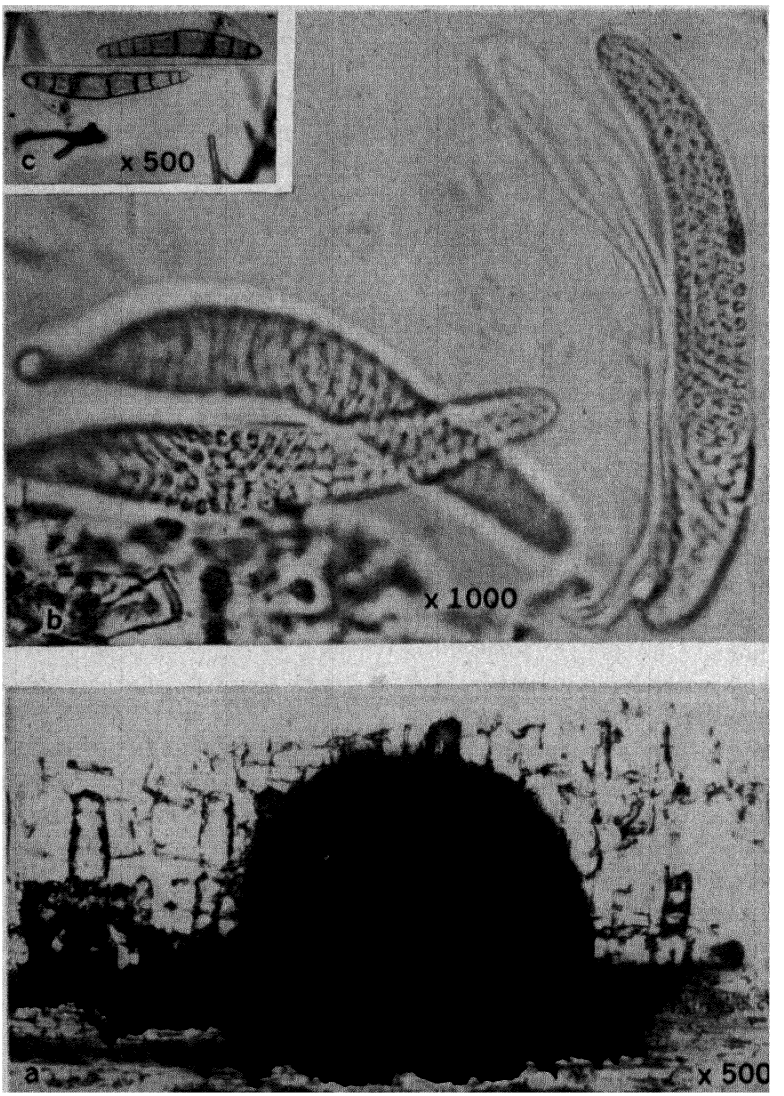

FIG. 37.—Perithecium (*a*), asci and coiled ascospores (*b*), and conidia (*c*) of *Cochliobolus miyabeanus* (*Helminthosporium oryzae*), highly magnified.

globose, pseudoparenchymatous, black, and form an ostiolar beak. Asci are cylindrical to long, fusiform, slightly curved, and contain mostly four to six ascospores. Ascopores are filiform and form in a close helix in the ascus (Fig. 37). *Helminthesporium sativum* Pamm., King, and Bakke also produces a seedling blight on rice.

Etiology.—The conidia and mycelium are common on crop residue. The fungus persists over unfavorable periods in both the conidial and mycelial phases. Primary infection is from one or the other of these sources. Ocfemia (1924), Nishikado (1923), and Wei and Lin (1936), have studied the influence of temperature and moisture on seedling blight and conidial morphology. Seedling blight is more prevalent in the cooler soils. Lesions on the aerial parts develop rapidly at the higher temperatures and moistures. Ocfemia (1924) and Nishikado (1927) have reported on the regional variation in the fungus. Tochinai and Sakamoto (1937) found specialization in pathogenicity and differences in physiology and morphology.

Control.—Control by sanitation and crop rotation is unsatisfactory, as the fungus develops abundantly on crop residue. Seed treatment with mercury or copper compounds will help reduce seedling blight, but it is not effective alone in the control of the disease. Resistant varieties offer the best means of control. Wei and Lin (1936) reported that all Chinese rice varieties tested were infected, although there was a difference in relative susceptibility. Several Japanese varieties are moderately resistant, according to Adair (1941) who has used the resistant variety, Mubo Aikoku, and others in breeding for resistance. He reported that resistance is governed by at least two factor pairs and that suscept reaction is similar in the seedling and mature plant. Tullis (1935) studied the leaf invasion by *Helminthosporium oryzae* in moderately resistant and susceptible strains of rice. The physiological response of the cells, the relation of large intercellular spaces in the mesophyll, and the sclerenchyma in the bundle sheath determine the type of fungus infection and spread in the leaf tissue. Leaf structure plays an important role in preventing excessive leaf necrosis and large lesions. Martin and Alstatt (1940) found both *H. oryzae* and the small-spored (*Helminthosporium*) *Curvularia lunata* (Wakk.) Boed., associated with the black kernels.

9. Blast, *Piricularia oryzae* Cav.—The disease is serious in most of the humid rice-producing areas of the world. Rice grown under irrigation in the areas of low relative humidity, as in California, is not damaged by blast. Abe (1933) and Hemmi and Imura (1939) demonstrated that conidia are not produced below 88 per cent relative humidity.

Description and Effect.—The disease occurs on the leaves, culms, branches of the panicle, and the floral structures. Rotten neck, *i.e.*, lesions on the neck of the culm and on the panicle branches near the base of the panicle, is the most conspicuous symptom. Rotten neck also causes the greatest damage, as the lesions prevent kernel filling. The dark-brown lesions on the culms and branches of the panicles are indicative of severe necrosis of the tissues. The panicle or panicle branches

break over at the lesioned area. Brown lesions at the crown and nodes also occur. The leaf spots on young leaves are linear and on older leaves they are small and circular. The leaf spots caused by this fungus are somewhat similar in appearance to Helminthosporium leaf spots. The difference in conidiophores and conidia differentiate the two diseases. Leaf lesions occur on the seedlings and as the plants approach maturity. Severe infections result in the leaves and sheaths drying out and browning. The coalescing of lesions at the base of the leaf blade is a characteristic symptom of blast. Small circular brown lesions occur on the kernels.

The Fungus.—*Piricularia oryzae* Cav.
[*Piricularia grisea* (Cke.) Sacc.]

The conidiophores are simple, rarely branched, grayish, septate, and slender. Conidia are borne terminally. They are ovate, 2-septate when mature, the apex is pointed to blunt depending upon race, according to Tochinai and Shimamura (1932).

Etiology.—The fungus develops on rice, and a form morphologically similar occurs on the crab grass *Digitaria sanguinalis* (L.) Scop. Apparently the two are distinct races; therefore, the grass does not function as a source of inocolum for rice. Infection occurs from conidia whenever conditions are favorable, as the fungus mycelium and conidia are present on crop residues. Terui (1940) described the formation of conidia on the surface and in internal cavities of the leaves and culms. Spore germination, infection, and conidial production are dependent upon high relative humidity. The physiological condition of the rice plant is associated with disease development, according to Abe (1936), Hemmi (1933), Sakamoto (1940), and others. Specialization of the fungus is summarized by Aoki (1935) and Inoue (1939). The latter found a high correlation between cellulose decomposition in culture and pathogenicity.

Control.—Injury from early infections is reduced by flooding as soon as leaf spots appear, according to Tullis (1940). Hemmi (1933) and others report that maintaining the irrigation water helps reduce disease damage as the crop matures. Proper balance of fertilizers, avoiding excess nitrate supply, is desirable in controlling rotten neck damage. According to Nakatomi (1927), Ramiah and Ramaswami (1936), Suzuki (1937), and others moderately resistant varieties are available and resistance is associated with cell-wall composition and tissue anatomy. In the United States, the varieties Zenith, Rexoro, and Texas Patna are resistant to the disease.

10. Cercospora Spot, *Cercospora oryzae* Miyake.—The disease is widely distributed with the rice crop, according to Tullis (1937). The narrow linear spots are reddish-brown to dark reddish-brown, depending upon the rice variety. On susceptible varieties, the lesion fades to a lighter

brown along the margins, and it is light gray brown in the older center. On the more resistant varieties, the lesions are smaller and uniform in color. The lesions are generally more abundant on the leaves, although

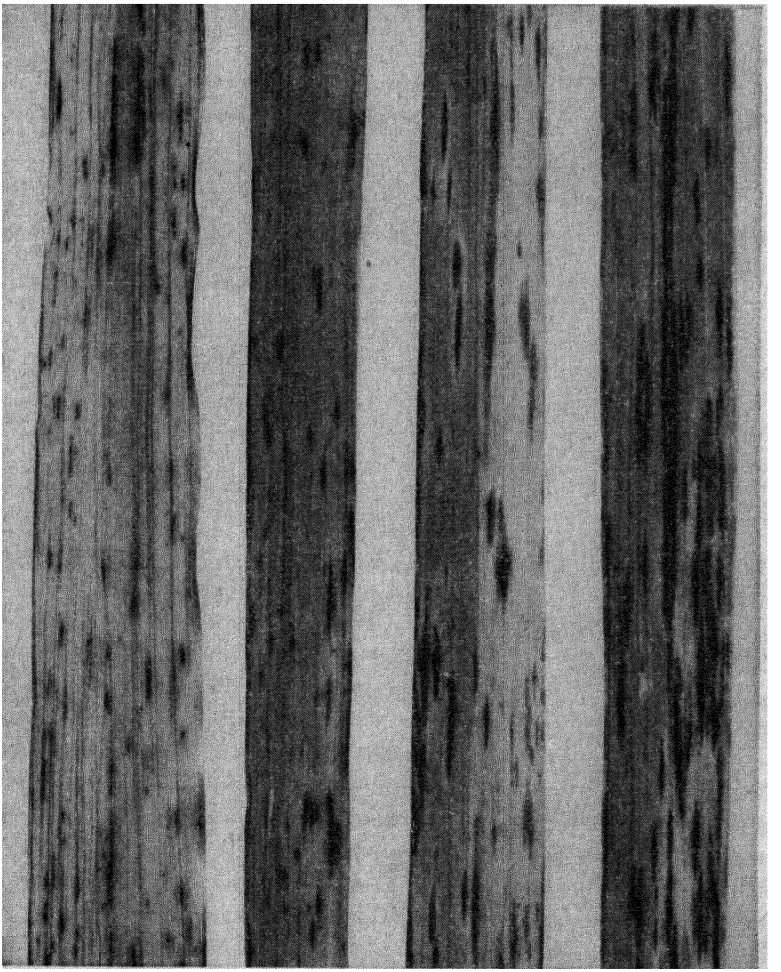

FIG. 38.—**Leaf spot of rice** caused by *Cercospora oryzae*, showing resistant reaction and susceptible.

spots on the sheath, culm, and floral bracts are present in heavy infections. Spots on the floral bracts spread laterally to form oblong lesions. (Fig. 38).

The Fungus.—*Cercospora oryzae* Miyake

The brown conidiophores emerge from the stomata singly or two to three and range from 88–140 microns long by 4–5 microns thick. The conidia are cylindrical to tapering toward the apex and vary in length and septation; septation is 3 to 10, length

20–60 microns by 5 microns wide, as reported in the original description by Miyake (1910).

According to Adair (1941), Jodon (1944), and Tullis (1937), a number of varieties and selections are resistant to the disease. The inheritance of resistance is due to several factors, according to Adair (1941) and Ryker and Jodon (1940).

At present six races of the parasite have been identified based on the reaction of six rice varieties, as shown in the following table:

Physiologic race	Leaf spot reaction on:					
	Blue Rose (C.I. 1962)	Blue Rose 41	Calora (C.I. 1561)	Fortuna (C.I. 1344)	Red Rice	Rexoro (C.I. 1779)
1	S	R	S	R	R	R
2	MR	S	R	R	R	R
3	R	R	S	R	R	R
4	R	R	R	S	R	R
5	R	R	R	R	S	R
6	R	R	R	R	R	S

11. Sheath and Culm Blight, *Rhizoctonia* Spp.—The Rhizoctonia sheath blight occurs on many crop plants in addition to rice. The symptoms vary somewhat on the cereals and grasses. On rice, the lesions are large, irregular, elliptical with reddish-brown margin, straw color, and light ochre-yellow or greenish-yellow center (Fig. 39). The lesions occur more commonly just below the ligule. The lesions on the culms are smaller than those on the sheath tissues. Ryker and Gooch (1938) reported that culms are not infected. White or light-tan mycelial strands develop under moist conditions. Salmon or tan irregular-shaped sclerotia frequently develop on the surface mycelium and within the leaf sheath under and adjacent to the lesion. Seedlings and mature plants are blighted under favorable conditions for disease development.

The Fungi.—1. *Rhizoctonia oryzae* Ryker and Gooch

Main mycelial strands are 6 to 10 microns in width, branching at an acute angle, with a slight constriction at the point of branching and a septation a short distance from the point of constriction. Later, short-celled much-branched hypae emerge at right angles from the main branches; certain of these form thickened short hyphae. These anastomose, forming masses of sclerotial cells of various shape and size. The sclerotia are salmon colored. Sclerotial masses are not formed on rice plants. No basidial stage is found.

Ryker and Gooch (1938) have reviewed the literature and discussed

the other species found on rice. *Rhizoctonia oryzae* is not pathogenic on the wide range of plants as occurs in *R. solani* Kuehn.

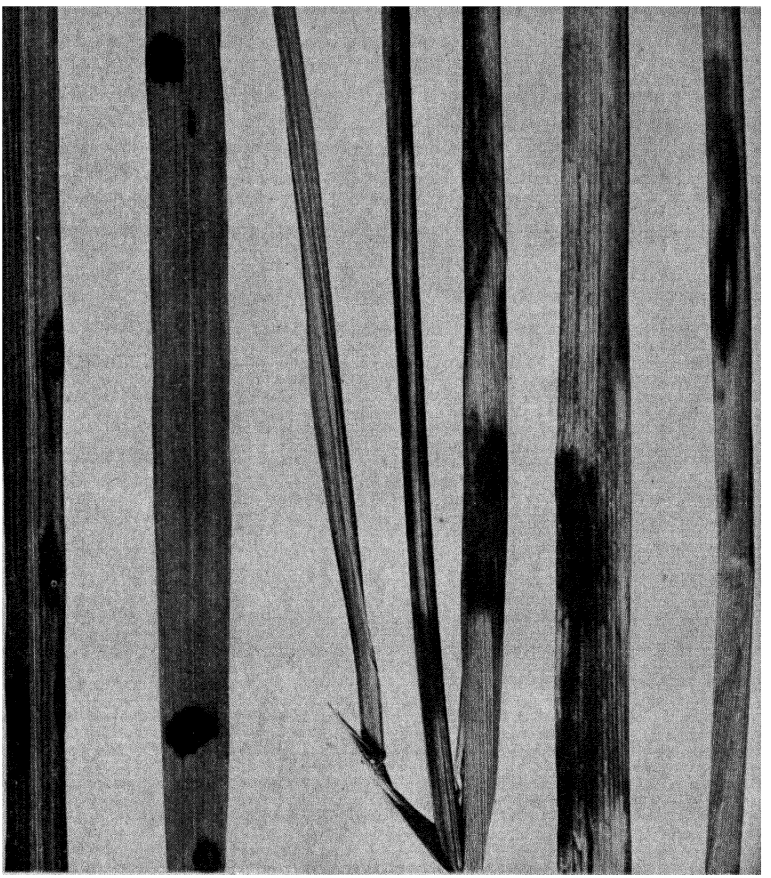

FIG. 39.—Leaf and sheath spots on rice caused by *Rhizoctonia oryzae*.

2. *Pellicularia filamentosa* (Pat.) Rogers or *Corticium solani* (Prill. and Del.) Bourd. and Galz.

(*Corticium vagum* Berk. and Curt.)
(*Corticium vagum* var. *solani* Burt.)
(*Hypochnus filamentosa* Pat.)
(*Hypochnus solani* Prill. and Del.)
(*Corticium solani* Prill. and Del.)
(*Corticium botryosum* Bresa.)
(*Rhizoctonia solani* Kuehn)

This species occurs on rice and many other crop plants. The fungus strains on rice in Asia and the Pacific Islands apparently produce larger sclerotia than is given in the species description, as discussed by Matsumoto (1934), Palo (1926), Park and Bertus (1934), Ryker and Gooch (1938), Wei (1934), and others.

The mycelium and branching are similar to the former species. Brown to black sclerotial structures form from intertwined anastomosed thickened cells rather than the separately branched filaments as in the above species. The sclerotia are tan, brown, or black and irregular in shape and size. The hymenophore consists of a dark loose weft of hyphae, turning grayish white when sporulating. The outer branches function as basidia and form two to six apical sterigmata bearing ovate to ovate-oblong sporidia. The sporidia germinate to form the *Rhizoctonia* mycelium. Rogers (1943) transferred the parasitic forms of *Hypochnus filamentosa* Pat., H. *solani* Prill. and Del., *Corticium vagum* Bert. and Curt., and *C. solani* Prill. and Del. to the genus *Pellicularia.*

Matsumota (1934) compared *Hypochnus sasakii* Shirai with *Corticium vagum* or probably *C. solani.* The hyphal cells in the former are smaller in diameter, mostly 6 to 8 microns, than in the latter, mostly 8 to 12 microns. The sclerotial cells near the periphery of the sclerotium show a corresponding difference in size. Hyphal fusions do not occur between the two fungi. The basidial stages of the two are similar. Growth of the mycelium on different media and temperatures differ in the two. He concludes, "It would be better to name our fungus *Corticium sasakii* (Shira) T. Matsum. instead of *Hypochnus sasakii* until further alteration is needed."

Voorhees (1934) reported a stalk rot of corn caused by *Rhizoctonia zeae* Voorh. Ryker and Gooch (1938) reported this fungus on rice in Louisiana.

Etiology.—The mycelium and sclerotia, where they are formed, persist on straw and stubble of rice and grasses. Infection occurs on the rice seedlings and plants when conditions are favorable. Thick stands of plants are conducive to infection. Proper balance of fertility is desirable to prevent excessive vegetative growth. The disease is of minor importance in the United States.

12. Kernel Smut, *Neovossia horrida* (Takahashi) Padwick and Azmatullah Khan.—The kernel smut of rice is distributed widely. The disease ordinarily causes little damage as few kernels in a panicle are smutted and the smutty grain can be removed in part by cleaning equipment, for when completely smutted they are lighter in weight than the healthy rice kernels. Anderson (1899) reported as high as 25 per cent smutted grains in rice fields in a limited area in South Carolina. Samples of threshed rice showed as high as 3.7 per cent smutty grains. These apparently were unusual cases.

The smutted kernels are not conspicuous in the field or in the threshed grain unless the smutty grains are broken. The hulls of the smutted

kernels appear dull gray. The smut sorus is enclosed almost completely by the lemma and palea (Fig. 40).

The Fungus.—*Neovossia horrida* (Takahashi) Padwick and Azmatullah Kahn
(*Tilletia horrida* Takahashi)

Anderson (1899) incorrectly reported this fungus as *Tilletia corona* Scribn. which occurs on several species of grasses.

The sori replace the ovaries and are concealed by the floral bracts. Chlamydospores

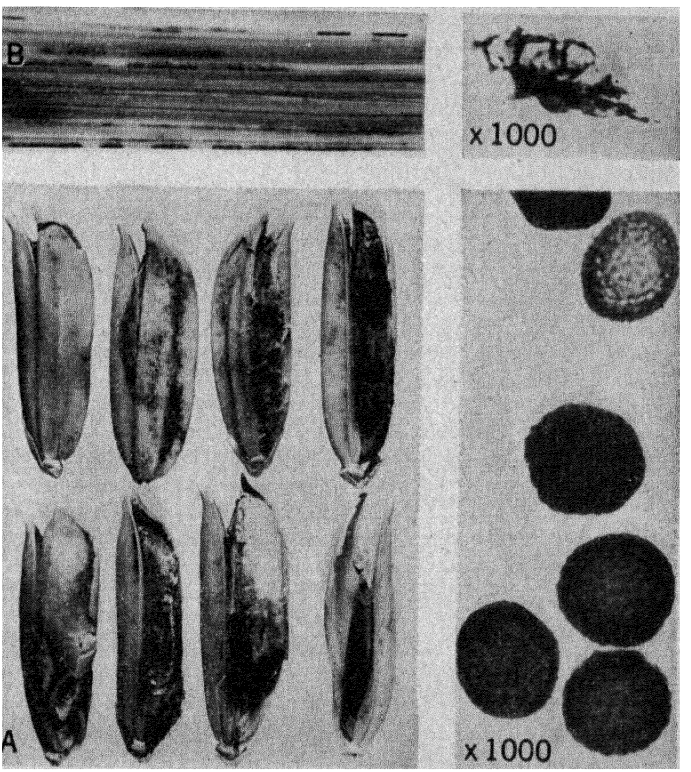

FIG. 40.—Kernel smut of rice (*A*) caused by *Neovossia horrida* and leaf smut (*B*) caused by *Entyloma lineatum*, with chlamydospores of the two fungi at same magnification.

are subspherical to spherical, opaque, dark olive brown with blunt peg-like thickenings or coarse scales, hyaline or slightly tinted around the exterior, 22 to 33 microns in longest diameter. The spores germinate by the formation of basidia and apical whorls of non-fusing sporidia (Padwick and Azmatullah Khan, 1944).

The etiology is different from that of the *Tilletia* spp. on the cereals and grasses. Takahashi (1896) and Teng (1931) described the germination after soaking in water. Tullis (1940) reported that the embryo

is not invaded and will germinate even when the entire endosperm is replaced by the spore mass. Floral infection by sporidia apparently accounts for the few scattered infections in the rice panicles. Varieties appear to differ in susceptibility and in the number of smutted kernels in a panicle.

13. Leaf Smut, *Entyloma lineatum* (Cooke) J. J. Davis or *E. oryzae* Syd.—The disease occurs in many of the rice-growing sections on rice and wild rice, *Zizania aquatica* L. According to Tullis (1934), the smut is of minor importance in the United States. The small black spots on the leaves are scattered and angular to elongate, with the epidermis persisting over the spot. The chlamydospores are angular to globose, closely packed, smooth walled, light brown, and 8 to 10 microns in diameter (Fig. 40). The spores germinate, usually in place, to form a promycelium with apical sporidia.

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CHAPTER VIII

RYE DISEASES

Cultivated rye, *Secale cereale* L., is chiefly a winter cereal grown extensively in Europe and Asia and on the lighter soils of North and South America. Spring varieties of rye occur; however, their use is limited as the extreme winter hardiness of rye permits the growing of the higher yielding winter varieties under most conditions. Cultivated rye has the basic chromosome number of seven pairs.

Rye is cross-pollinated and, therefore, heterozygous and relatively variable in the expression of characters. Inbreeding is possible, although progress in stabilizing a large number of characters is slow owing to general self-sterility. Self-fertile lines are obtainable, and considerable progress is possible in securing stable disease-resistant lines. Such lines are not in general use commercially in the production of hybrid rye seed, although their use is possible.

Rye diseases, in general, are of less importance economically than in the other cereal crops. The average annual loss from 1930 to 1939 in the United States is reported at 1.8 per cent of the crop, or 497,000 bushels (Plant Disease Survey).

1. Mosaic, Virus.—The mosaic on rye is found sparingly in the United States and in Asia. The virus is perhaps one of several that occurs on wheat (see wheat Mosaic, Chap. XI).

2. Bacterial Blight, *Xanthomonas translucens* f.sp. *secalis* (Reddy, Godkin, A. G. Johnson) Hagb.—The disease occurs in North America and Australia, according to Noble (1935) and Reddy and Johnson (1924). The lesions on rye are less stripe-like than on barley, and they generally develop an irregular margin (see Bacterial Blight of barley, Chap. III).

3. Powdery Mildew, *Erysiphe graminis secalis* El. Marchal.—Light infections of powdery mildew are common on rye although of no economic importance. Specialization of the physiologic variety on rye is not pronounced, owing largely to the heterozygous condition of the plant. According to Germar (1934) and Mains (1926), mildew-resistant rye selections are not difficult to secure.

The symptoms and etiology are similar to those on barley (Chap. III).

4. Fusarium Blight or Scab, *Gibberella* and *Fusarium* Spp.—The disease is common on rye in the humid and semihumid rye areas. The losses are frequently high in both reduced yields and in the damaged

quality. The "drunken bread" malady described as formerly common in the humid regions of Europe and Asia is attributable largely to *Gibberella* and *Fusarium* infected grain. The appearance of the disease on rye is similar to that on wheat (Chap. XI).

5. Snow Mold, Foot Rot, and Head Blight, *Calonectria graminicola* (Berk. and Br.) Wr.—The disease is common on rye, wheat, and many grasses. Its occurrence is associated with heavy snow covering in many areas where the disease is severe, as in the Northern sections of North America and Europe and Asia. The symptoms of the disease are similar to those on wheat (Chap. XI).

6. Ergot, *Claviceps purpurea* (Fr.) Tul.—Ergot is common on species of many genera of the Gramineae. The disease is world wide in the temperate, humid, and semihumid areas. Ergot is usually more severe on rye, although certain varieties of barley and wheat are infected heavily. *Bromus, Agropyron, Poa* spp., and other cultivated grasses are damaged, especially where grown for seed production.

Grain marketed through the Federal grading system of the United States is designated "ergoty" when it contains more than 0.3 per cent of ergot sclerotia by weight. In the past such grain was discounted heavily. The perfection of the gravity-type separator and other grain-cleaning equipment greatly facilitates the removal of the sclerotia to within the tolerance of the Federal grades and pure food laws. Many other cereal-producing countries apply a similar low tolerance for grains used for feed or food purposes.

The low tolerances of ergot sclerotia are necessary because these fungus bodies contain compounds harmful to the circulatory system of animals. The sclerotia contain varying amounts of compounds known as ergosterol, ergotoxin, ergotamin, ergostetrine, ergoclarin, etc. Some of these when properly purified are valuable medicinals. According to Barger (1931), Dudley and Moir (1935), Krebs (1936), Kussner (1934), Thompson (1935), Trabucchi (1934), and others, specific compounds or groups of these compounds cause constriction of the capillary blood vessels in specific tissues or generally. Large dosages or continuous smaller amounts of ergot result in constriction of the capillaries of the placental tissue and abortion. The lactation of animals is reduced or prevented by continuous small amounts of ergot. The effect is cumulative and frequently causes reduced circulation and breakdown of tissues especially in the extremities, such as fingers, toes, hoofs, and ears. Extract of ergot is used extensively in obstetrical medicine. Sound sclerotia of suitable composition demand high prices, especially during periods when the regular supply from the Mediterranean area is cut off from commerce. Many rye growers and processors receive high prices for ergot sclerotia during these periods of shortage. Environmental

conditions, especially excess moisture, influence the composition of the sclerotia; therefore, all sclerotia are not suitable for this use.

FIG. 41.—Rye spikes showing the conidial (*A*) and sclerotial (*B*) stages of ergot caused by *Claviceps purpurea*.

Description and Effect.—The ergot infection is conspicuous from blossoming of the cereals and grasses to maturity of the crop. Infection is evident first in the conidial "honeydew" stage when the conidia of the fungus are produced on a folded stromatic surface and released in a

sugary exudate. The exudate and conidia accumulate in droplets or adhere to the surface of the floral structures, depending upon the concentration of the exudate which is influenced by moisture. Insects feed upon the exudate and are conspicuous around infected spikes. The sclerotial stage soon follows the conidial. The blue-black sclerotial body replaces the kernel in the infected flowers. As the sclerotia develop, the floral bracts spread apart, and the bodies when fully developed are evident as they usually protrude beyond the floral bracts. Few to many sclerotia occur on a spike or panicle (Fig. 41). These sclerotia are conspicuous in the threshed grain or seed, and they can be detected by specific color tests in milled-grain products.

The Fungus.—*Claviceps purpurea* (Fr.) Tul.

Synonyms arranged under the three stages in the development of the fungus.

Sclerotial Stage	Conidial Stage
(*Clavaria solida* Munch.)	(*Spermoedia clavis* Fries)
(*Clavaria clavus* Schr.)	(*Sphacelia segetum* Lév.)
(*Clavaria secalina* Paul.)	(*Fusarium heterosporum* Nees)
[*Sclerotium clavus* (Tode) DC.]	(*Oidium abortifaciens* Berk. and Bro.)

Ascigerous Stage

(*Sphaeropus fungorum* Paul.)
(*Sphaeria purpurea* Fries)
(*Kentrosporium purpurea* Wallr.)
(*Cordyceps purpurea* Fries)
(*Cordiliceps purpurea* Tul.)

The morphology of the fungus comprises a series of developmental stages involving specific types of mycelium and spores. A mass of tightly compacted large hyphae form an absorbing structure in the vascular bundle of the rachilla in the base of the flower. The hyphal cells above this foot-like structure are shorter and remain in an active state of division, or they constitute an embryonic or generative hyphal mass situated in the position of the young ovary and are frequently associated with the residual tissues of the ovary wall of the grass flower. The latter structure is replaced completely or in part by this compact mass of actively dividing hyphae. The conidial-bearing mycelial stroma is differentiated first on the top of the generative hyphae. It is composed of a thin layer of compacted hyphae producing a stromatal surface of palisade-like single-celled conidiophores on the undulating and folding outer surface of the Sphacelial stroma. Small spheroid hyaline conidia are produced successively from the conidiophores, and they are held in a surgary exudate secreted by the stroma. The generative mass of hyphae soon differentiate sclerotial hyphae below the conidial stroma, and they in turn supply the contact with the conidial stroma during the remaining period of activity of the conidial stage. The sclerotium continues to elongate from the base by the differentiation of sclerotial cells from the generative mass of hyphae. As the sclerotium elongates, the hyphae included differentiate into the hard outer surface layer, the fertile hyphal mass, and the central larger storage cells. At the maturity of the suscept cereal

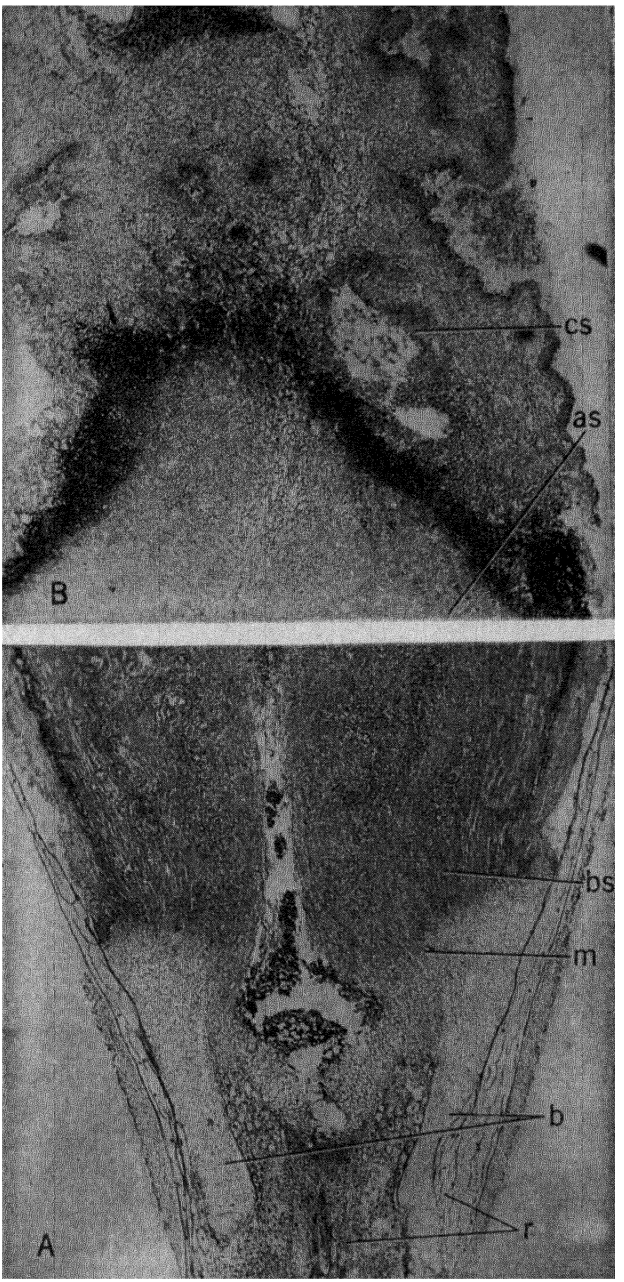

FIG. 42.—Median longisection of rye flower, showing young sclerotium (*A*) and conidial stroma (*B*) of *Claviceps purpurea*. (*r*) Rachilla and flower tissue, (*b*) attaching and absorbing mycelium, (*m*) region of mycelial differentiation, (*bs*) basal and (*as*) apical sections of sclerotium, and (*cs*) conidial stroma. × 100.

or grass, the fungus development consists of the foot-like attachment in the rachilla, the sclerotium similar in shape to the caryopsis of the suscept it replaces and enclosed by the floral bracts, and the dry conidial stroma still attached to the apex of the sclerotium (Fig. 42). The size of the sclerotia vary depending upon the development of the suscept and the number of infections in each spike.

The fertile hyphae within the sclerotium develop into stromata and stipes (Fig. 43). Sclerotial germination occurs only after a suitable conditioning period, such as overwintering in the soil. The stipes are cylindrical, the length and thickness varying depending upon the depth of the sclerotium below the soil surface. The stromatal head (sphaeridium) is spherical, pale fawn colored, and the upper surface is covered with minute elevations, the projecting ostioles of the submerged peripheral perithecia. Perithecia are flask-shaped, sunken below the surface with the pronounced ostiole protruding. The asci are long, somewhat curved, and surrounded by numerous peraphyses similar in form to the asci. Each ascus contains a bundle of eight slender filiform hyaline ascospores. The ascospores are ejected from the apex of the ascus through the ostiole of the perithecium.

Etiology.—Infection occurs through the young flowers, from wind-borne ascospores first and later from conidia. Penetration of the young ovary tissues occurs, and an absorbing organ of mycelium forms in the conductive tissue at the base of the ovary or in the rachilla. All or portions of the ovary are destroyed. The conidial and sclerotial mycelial masses develop within or above the remaining ovary tissues. The specialized fungus structures are attached to the flower by the absorbing mycelium until the sclerotium is mature. The sclerotia fall to the ground or are harvested with the grain. The sclerotia overwintering in the soil form perithecial stromatal heads that emerge from the soil by elongation of the stipe (Fig. 43). The stroma emerge from the soil shortly before blossoming of the grain or grass plants. Ascospores are produced in large numbers and are extruded from the asci at the period the suscepts are blossoming. The primary infection occurs from the wind-borne ascospores. In the spring of 1944, 10 to 15 germinating sclerotia per square foot were found under the plants in a nursery of *Bromus inermis* L. at Madison, Wis. Secondary infections from meteoric water or insect-borne conidia are extensive over a period of 1 to 2 weeks, depending upon environmental conditions.

Lewis (1945) discussed methods of preserving conidia in sugar solutions and using them in large-scale inoculations in the commercial production of ergot. Atanasoff (1920) and McFarland (1921) have summarized the literature and data on infection.

Control.—Crop rotations and removal or cutting of weed grasses prevent the formation and spread of sclerotia and reduce the amount of primary inoculum. Heavy infection of spring barley in the North Central United States is usually associated with weed grasses in or adjacent to the field. According to McFarland (1921), the sclerotia formed on *Agropyron*, *Arrhenatherum*, *Bromus*, *Glyceria*, *Phleum*, and

Poa spp. infect the cereal crops. The strain on *Lolium* spp. tested did not infect the cereals (see Chap. XII). Modern seed-cleaning equipment

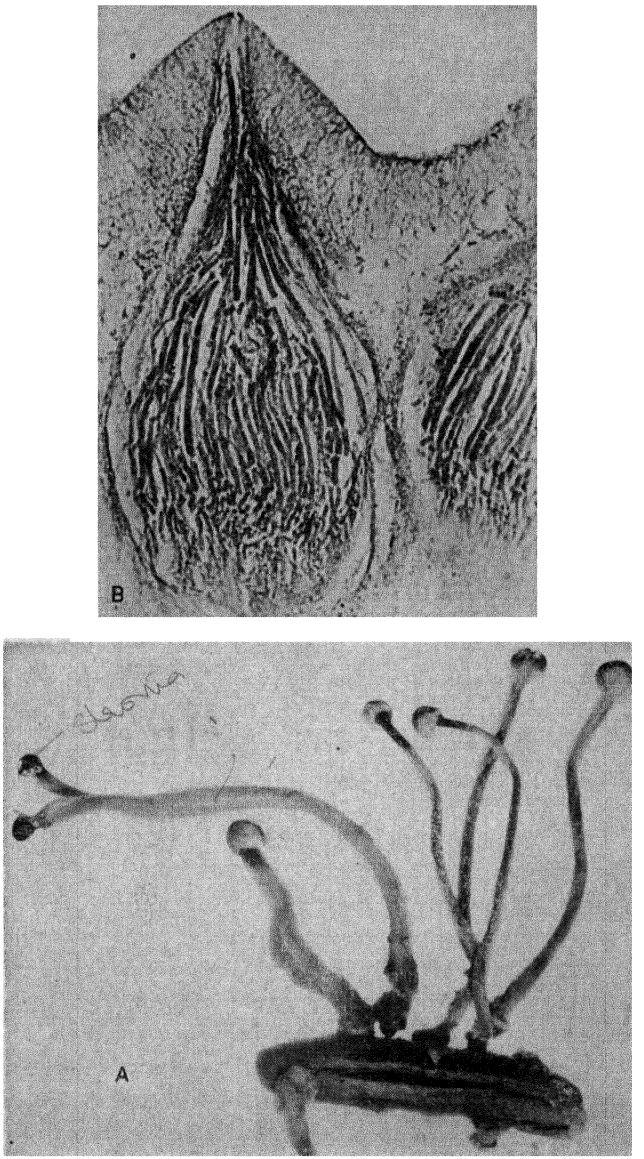

FIG. 13. Sclerotium and stromatal heads containing the perithecia of *Claviceps purpurea* (*A*); and section through perithecium (*B*).

removes the sclerotia from the seed grain. Sclerotia do not remain viable when stored over 1 year.

Some varieties of the cereals and grasses are less susceptible to ergot than others. Investigations on ergot resistance, however, indicate to date no resistant selections in rye or the cultivated grasses.

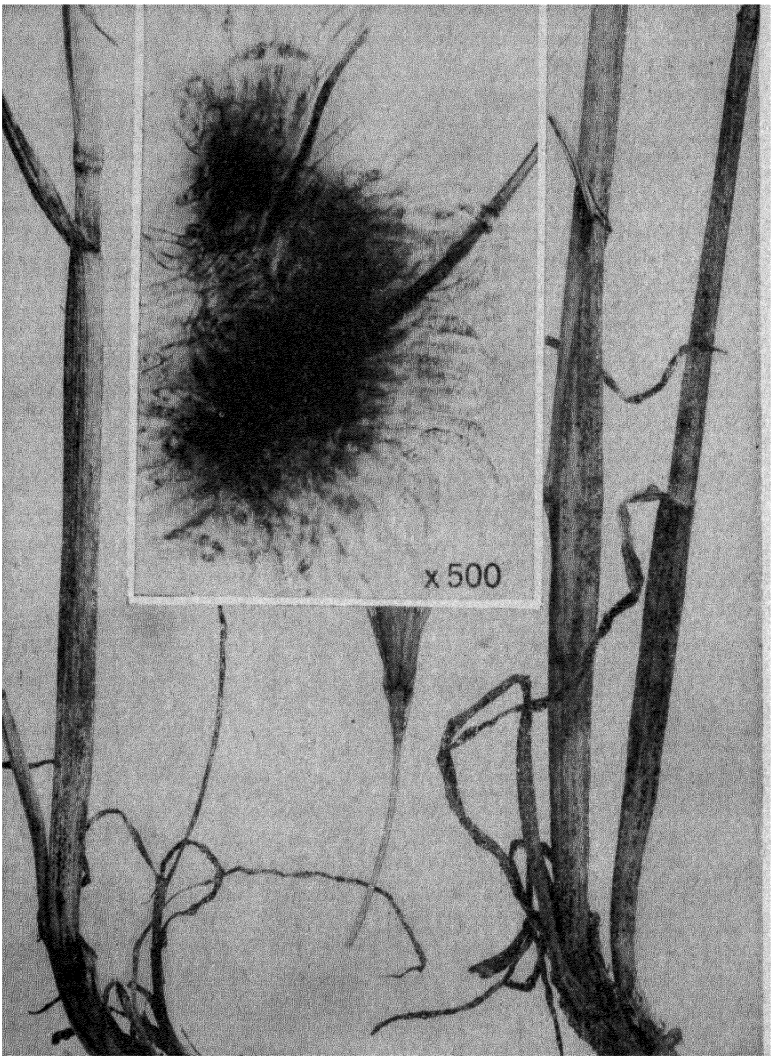

FIG. 44.—Numerous acervuli, bearing black setae and conidia of *Colletotrichum graminicolum*, formed on the basal leaf sheath and culm tissues of rye. Acervulus from Sudan grass, highly magnified, is shown in the insert.

7. Anthracnose, *Colletotrichum graminicolum* (Ces.) G. W. Wils.—The disease is distributed widely on the cereals and grasses. In North America according to Sanford (1935), Selby and Manns (1909), and

Wilson (1914), the disease is important especially in the soft red winter wheat area and in the North Central rye area. Barley, oats, and many grasses are damaged in this same general area. The disease is associated with conditions of low or unbalanced soil fertility, open coarse soils, and continuous grass-cereal culture.

Description and Effect.—The disease is apparent toward maturity of the crop. General reduction in vigor of plant development and premature ripening or dying are the gross symptoms of the malady. The presence of the mycelium and bleaching followed by browning of the culm bases and crown tissues appear first. This is followed by the development of the black acervuli on the surface of the lower leaf sheaths and culms especially (Fig. 44). Acervuli develop on the leaf blades of the dead plants when moisture is plentiful. Pronounced round to oblong lesions bearing acervuli occur on the green leaves of Sudan grass. The grain is shriveled when the attack occurs early. Spike infection and superficial grain infection occurs less commonly. Seedling and crown infection occur where the disease is severe.

The Fungus.—*Colletotrichum graminicolum* (Ces.) G. W. Wils.
(*Colletotrichum cereale* Manns)

The acervuli are superficial, circular to oval, with dark mycelium forming the basal stroma. The black to dark-brown setae form through or surrounding the conidial-forming stroma of the mycelium. Setae are septate and tapering at the apex. Conidia are spindle shaped, slightly curved, hyaline, and one-celled.

Etiology.—The fungus is saprophytic on crop residues. Primary infection is commonly from the mycelium and conidia on the crop residue. Infected seed is a possible source of seedling root and crown infection. Secondary spread late in the growing season is general from conidia as well as mycelium on the crop residue.

Control.—The disease is reduced by the rotation of crops and improved soil fertility. The use of legumes and other dicotyledonous crops in the rotation, plowing under cover crops, and proper balance of phosphate and potash greatly reduce infection and damage.

8. Rhynchosporium Leaf Scald, *Rhynchosporium secalis* (Oud.) J. J. Davis.—The disease is distributed widely on rye, but it causes less damage on this crop than on barley and smooth brome grass. See Chap. III, for a detailed discussion of the disease.

9. Septoria Leaf Blotch, *Septoria secalis* Prill. and Del.—The Septoria leaf blotch occurs generally in the rye-producing areas. The lesions are characteristic of this group of fungi on the cereals and grasses. The light-brown irregular blotches are differentiated readily by the development of the pycnidia so typical of this genus (Fig. 45).

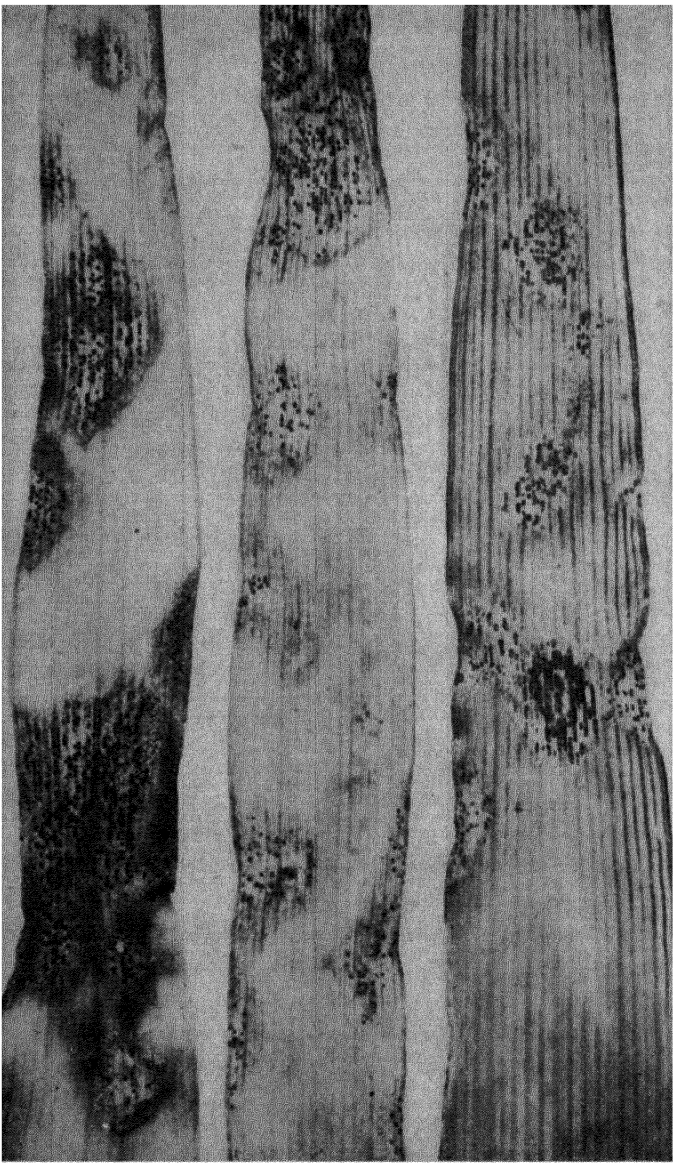

FIG. 45.—Leaf blotch of rye caused by *Septoria secalis*, showing the abundant dark brown pycnidia on the blotches.

The Fungus.—*Septoria secalis* Prill. and Del.

The pycnidia are subepidermal, black, globose, and smooth. The hyaline conidia are slender, cylindrical, slightly curved, rounded at the ends, and 3-septate. They average 2.7 microns wide by 35 microns long. See Chap. XI for the full discussion of the disease.

10. Stalk Smut, *Urocystis occulta* (Wallr.) Rab.—This fungus apparently is restricted to rye, and it is widely distributed throughout the world with the crop. The general distribution of this smut is in striking contrast to the rather limited distribution of the flag smut of wheat. The symptoms of the disease on rye and wheat are different, as indicated by the common names. In rye, the sori form in the parenchymatous tissue between the bundles of the culm tissue and less commonly in the leaf tissue. The sori form in the flowers and usually prevent the spike from emerging. Sori occur in the mesophyll of the leaf sheaths and less commonly in the leaf blade. The sori are covered by the epidermis in the early stages of development. The epidermis ruptures and the tissues shred, releasing the dark-brown spore mass (Fig. 46).

The Fungus.— *Urocystis occulta* (Wallr.) Rab.
(*Erysibe occulta* Wallr.)

The spore balls are oblong to spherical, 16 to 32 microns in diameter, and generally incompletely covered by the sterile cells. The sterile cells are light yellow to tan with irregular margins. The spores are reddish brown, oblong to subspherical, with flattened sides, and occur 1 to 2, rarely 3 to 4, in a smut ball. The spores germinate in place to form a promycelium (basidium) with usually four sporidia borne in an apical whorl and promycelium with irregular branching. Stakman *et al.* (1934) studied the cycle of development of the fungus. Fischer (1943) suggested the combination of this species with *Urocystis tritici* Koern. and *U. agropyri* (Preuss) Schroet. on the basis of similar morphology.

Etiology.—Seedling infection occurs commonly from seed-borne spores. The fungus develops with the culm primordia of the seedlings, and sori are produced in the parenchymatous and mesophyll tissues. In the Northern United States and Canada, soil infestation is uncommon. The source of inoculum is largely from seed-borne spores. The spores remain viable in dry soil to infect the fall-sown rye, as reported by Tisdale and Tapke (1927). Seed treatment controls this smut in most areas. According to Ling and Moore (1937) and Stakman and Levine (1916), temperature and moisture influence smut development and control of the disease. See Chap. XI, for a detailed discussion.

11. Head and Kernel Smuts, *Ustilago* and *Tilletia* Spp.—Other smuts are reported occasionally on rye. Several of the smuts have been designated by specific binomials, probably without adequate study of the disease. According to Humphrey and Tapke (1925), *Ustilago tritici* (Pers.) Rostr. is capable of infecting rye under favorable conditions, and it has been reported on rye several times. Bunt caused by the species of *Tilletia* common on wheat is found occasionally on rye, and certain races of these parasites are capable of infecting rye, as reported by Gaines and Stevenson (1923), Lobik (1930), Nieves (1935), and others.

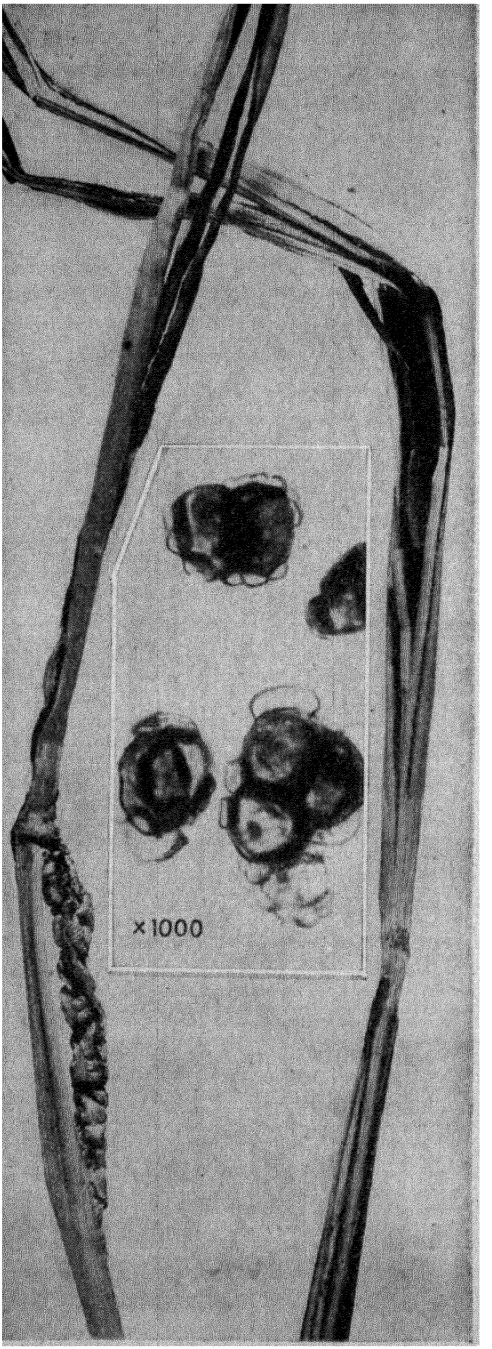

FIG. 46.—Stalk smut of rye caused by *Urocystis occulta* and chlamydospores of the fungus highly magnified.

12. Stem Rust, *Puccinia graminis secalis* Eriks.—Stem rust on rye is distributed more generally, and the physiologic races of this variety of the parasite occur more commonly on the weed grasses than those on wheat or oats, as discussed by Stakman *et al.* (1934). Stem rust resistant varieties and self-pollinated lines of rye occur, as reported by Cotter and Levine (1932), Mains (1926), Stakman *et al.* (1934, 1935), and Waterhouse (1936). Over 14 physiologic races occur on rye in the United States, according to Stakman *et al.* (1935); however, the heterozygous condition of the cross-pollinated rye makes determination of races unreliable. The detailed discussion of the disease is given in Chap. XI.

13. Stripe Rust, *Puccinia glumarum* (Schm.) Eriks. and Henn. —The stripe rust is less common on rye in both the Pacific and Intermountain areas of North America, Europe, and Asia (see Chap. XI).

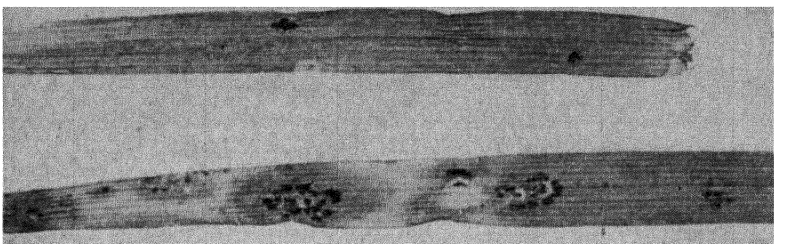

FIG. 47.—Leaf blade of rye showing the new uredia of *Puccinia rubigo-vera secalis* formed early in the spring from mycelium overwintering in the tissues adjacent to the center uredium, which was formed the previous autumn. Contrasted with spring infections, upper leaf.

14. Leaf Rust, *Puccinia rubigo-vera secalis* (Eriks. and Henn.) Carleton. —The leaf rust of rye is widely distributed on rye and many wild species of the genus *Secale*. The aecial stage of the fungus occurs on species of *Anchusa*, although the natural aecial infections are rare in the United States, according to Arthur (1934), Mains (1933), and Mains and Jackson (1924).

The leaf rust causes a reduction in tillering and lower yields when heavy infection occurs early. Losses occur more commonly in the southern range of rye culture where the fungus overwinters in greater abundance.

Description.—The round to ovate orange-brown uredia develop on the leaves and leaf sheaths. The telia are covered by the epidermis. The uredial development in the spring furnishes an excellent demonstration of overwintering of the uredial mycelium in the leaf tissue and the development of new uredia early in the spring (Fig. 47).

The Fungus.—*Puccinia rubigo-vera secalis* (Eriks. and Henn.) Carleton
[*Puccinia rubigo-vera* (DC.) Wint.]
(*Puccinia dispersa secalis* Eriks. and Henn.)
(*Puccinia secalina* Grove)
(*Puccinia dispersa* Eriks. and Henn.)

The morphology of the fungus and etiology are similar to the leaf rust of wheat (Chap. XI). Specialization and resistance are reported by Gassner and Kirchhoff (1934), Mains (1926), and Vavilov (1919).

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CHAPTER IX

SORGHUMS, SUDAN GRASS, AND JOHNSON GRASS DISEASES

The cultivated sorghums are largely annuals of the species *Sorghum vulgare* Pers. Martin (1936) included the perennial Johnson grass, *S. halepense* (L.) Pers., in the sorghum group. The following key is from Martin's grouping (1936, page 528), illustrating the relationship of the different sorghum types.

1. Annual sorghums, *Sorghum vulgare*
   - A. Sorgo (sweet or saccharine sorghum)
   - B. Grain sorghum
     1. Milo.
     2. Kaffir.
     3. Feterita.
     4. Durra.
     5. Msc. (hegari, darso, shallu, kaoliang, etc.).
   - C. Broom corn
   - D. Grass sorghum; Sudan grass, *S. vulgare* var. *sudanense* (Piper) Hitchc.
2. Perennial sorghum or Johnson grass, *S. halepense* (L.) Pers.

The crop is cultivated for grain, forage, and juice in the drier climates and on limited acreage in the humid sections of the United States. Like corn, the sorghum is sufficiently diverse so that varieties are grown in all locations in the United States.

The basic chromosome number of the genus is 5 chromosome pairs. The annual sorghums included in the above key all have 10 pairs; the perennial Johnson grass has 20 pairs of chromosomes. Certain of the sorghum varieties have been crossed with sugar cane hybrids. So far as known, such hybrids are pollen sterile and only a few fertile embryos develop.

The physiological anatomy of the sorghums is similar to corn. The seedling development and susceptibility to disease is very similar in the two. The development of the inflorescence, however, is different, as the sorghums develop panicles or heads of perfect flowers. The caryopsis of the sorghums threshes free from the floral bracts in most commercial varieties. The development of prussic acid, especially in the leaves, and the formation of hydrocyanic acid under some conditions is characteristic of the group.

Diseases cause relatively large losses in this crop. Seedling blights and root rots are important in reducing stands and plant vigor. The sorghum smuts are severe where control methods are neglected.

1. Nonparasitic Leaf Spotting and Weak Neck.—*Leaf spotting* and other types of pigmentation of the leaves and floral bracts as well as chlorophyll deficiencies occur in this group of plants. Purple, brown, and yellow spots and blotches are characteristic of the sorghums and Sudan grass. The color of the spots is determined in part by the genetic composition of the variety or selection. The nonparasitic spots are differentiated from the parasitic maladies as follows: (1) general absence of water-soaked areas in any portion of the pigmented spot, (2) uniform pigmentation over the spot and rather regular margin, (3) absence of necrosis especially in the early stages of spot development, and (4) the absence of fungi and bacteria associated with the spots. These pigmented areas on mature leaves, however, frequently show necrosis followed by secondary organisms invading the tissues. Environmental conditions influence the expression of the spotting. Pigmentation of this type is associated especially with progenies from hybrids or inbreds. Freed's sorgo is one of the very few sorghums that has never been observed to develop leaf spots. The various types of chlorophyll deficiencies—albino, veriscent, and other chlorophyll deficient manifestations—are associated frequently with material in the segregating progenies growing under environmental conditions favorable for expression of the defects. Chlorosis due to soil conditions, temperatures, etc., is present in some areas and varieties.

Weak neck is common in the major grain sorghum areas of the United States where the malady causes reduction in yield and plumpness of grain. The damage is pronounced in relation to combine harvesting, as the grain must stand in the field until completely dry. The culm breaks below the head, resulting in poorly developed heads with lightweight seed. Apparently the tissues of the peduncle and rachis do not develop sufficient thick-walled tissue, and these tissues ripen too early to support the developing head. The early maturation of these tissues is accompanied by physiological decline of the tissues; the upper culm tissues dry out and bleach to straw color; a soft spongy condition of the upper culm develops; and frequently the decline is accompanied by water-soaking and the accumulation of a sticky exudate during wet weather. Secondary organisms such as *Fusarium*, *Helminthosporium*, and *Alternaria* spp. later are associated with the dead tissues (Swanson, 1938, and Leukel *et al.*, 1943). The dwarf varieties developed for combining are derived largely from the milo types. These and the feterita types are early maturing, develop a dry stem, and a relatively thin rind, conditions conducive to weak neck. The development of dwarf varieties with culms more like

the sorgo and kaffir types in which the culms remain green longer apparently is the best means of control.

2. Mosaics, Infectious Viruses.—Certain of the grass virus maladies affect the sorghums. These are discussed in detail in Chap. X.

3. Bacterial Blights.—The bacterial stripe and streak diseases are common on the sorghums. Symptoms are more pronounced and defoliation is more severe than with the similar diseases on sugar cane. Burrill (1887, 1889) in Illinois was probably the first to describe the bacterial blights of sorghums in his early studies of bacterial diseases of plants. He described an organism that he considered to be the cause of the complex and named it *Bacillus sorghi* Burr. Kellerman (1888) and Kellerman and Swingle (1889) conducted a parallel study of this disease complex on the sorghums of Kansas. The red lesions on the sorghums were described and attributed to several distinct organisms by other investigators during this same period, as reviewed by Elliott and Smith (1929). Smith and his associates (1905, 1911) redescribed the symptoms of "Burrill's bacterial disease of broom corn" but attributed it to another bacterium, nonsporiferous, white on culture media, and with one to three polar flagella. They named the organism *Bacterium andropogoni* Smith and Hedges. Elliott (1929, 1930) continued the study of these bacterial diseases and differentiated the bacterial stripe and streak diseases of the sorghums.

Bacterial Stripe, *Pseudomonas andropogoni* (E. F. Sm.) Stapp [*Phytomonas andropogoni* (E. F. Sm.) Bergey *et al.*].—The disease occurs on many sorghums including Sudan grass and Johnson grass, and it is distributed chiefly in the grain sorghum areas of the United States and similar areas in other countries. Inoculations on corn and sugar cane result in lesions on some varieties. The disease is widely distributed and under favorable conditions causes leaf killing.

The lesions are linear stripes with the color continuous throughout the lesion. The stripes are narrow, water-soaked, and bounded by the veins when young. Later the stripes fuse and cover a large part of the leaf surface and extend into the leaf sheath. Stalks and floral structures show similar but more restricted lesions. The color ranges from dark purplish red to brown, depending upon the sorghum variety and type of pigments present. Abundant exudate, pigmented similar to the color of the stripe, forms in droplets and scales over the lesions (Fig. 48).

Bacterial Streak, *Xanthomonas holcicola* (Elliott) Starr and Burk. [*Phytomonas holcicola* (Ell.) Bergey *et al.*].—The disease is widely distributed in the United States and abroad on the sorghums, including Sudan grass and Johnson grass. Apparently this disease occurs more commonly on the sorghums, especially in the cooler climates, than the stripe. Streak causes considerable defoliation in the grain and grass sorghums.

The young lesions are narrow water-soaked streaks with red to brown margins and irregular blotches of color interrupting the continuity of the streak. Irregular oval blotches with tan centers and red margins, intermingled with the streaks, develop as the disease advances and lesions

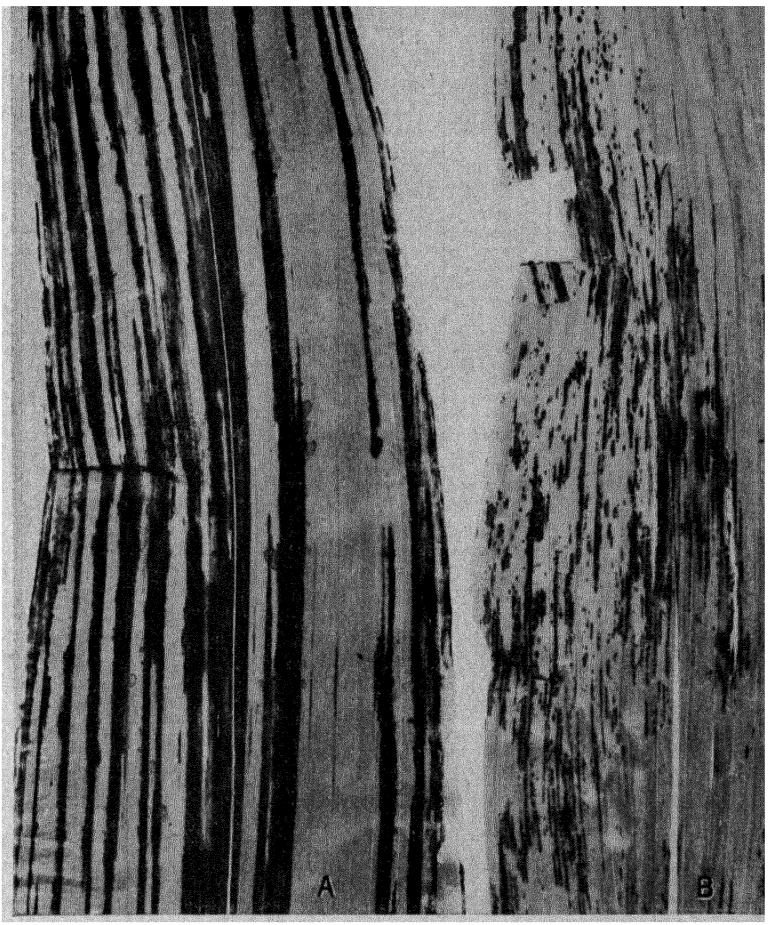

FIG. 48.—Bacterial stripe (*A*) and bacterial streak (*B*) of sorghum, caused by *Pseudomonas andropogoni* and *Xanthomonas holcicola* respectively.

coalesce (Fig. 48). Exudate is abundant as yellow to cream-colored droplets or scales.

Holcus Spot, *Pseudomonas syringae* v. Hall [*Phytomonas syringae* (v. Hall) Bergey *et al.*], [*P. holci* (Kend.) Bergey *et al.*].—Kendrick (1926) described this leaf spot of corn and sorghum. The holcus spot is characterized by tan red-bordered round to elliptical lesions on the leaves.

The spots coalesce to form irregular blotches, but do not elongate to form streaks or stripes. Exudate is not present on the lesion.

The various bacteria associated with the sorghum blights are similar in gross morphology (rods with polar flagella) but vary in physiology. All are seed-borne to some extent and are carried over on crop residue. Seed treatment, crop rotation, and the use of resistant varieties are the principal means of control. The prevalence of these diseases and the degree of control are dependent on seasonal conditions.

4. Milo Disease and Pythium Root and Crown Rot, *Pythium arrhenomanes* Drechs. and Other Fungi.—The root rot and crown rot of certain

FIG. 49.—The milo disease. This is a destructive disease controlled by resistant varieties as shown by the reaction of the resistant (left) and susceptible (center) varieties and the F1 (right) growing in infested soil. (*Courtesy of L. E. Melchers.*)

milo and darso varieties of the sorghums, more commonly known as the "milo disease," is widespread, especially in the southern plains area of the United States and apparently not common elsewhere. However the root rot caused by *Pythium arrhenomanes* and closely related species is world wide in distribution on sorghums and other members of the Gramineae (Pythium root rot of corn, sugar cane, wheat, see Chaps. IV, X, XI). The milo disease on the susceptible grain sorghums is an especially severe manifestation of a disease complex in which *Pythium*, *Fusarium*, and *Rhizoctonia* spp. frequently are associated (Melchers, 1942). The roots first show a brown to red discoloration and water-soaking. The rotting progresses from the rootlets into the larger roots and into the base of the crown. The leaves become yellow and dry out, the plants are stunted, and they fail to head. Under favorable conditions for disease

development and heavily infested soils, the plants are killed prior to heading (Fig. 49). Elliott *et al.* (1937) and Melchers and Lowe (1943) described the symptoms and etiology of the disease on the milos. Recent investigations by Melchers (1942) show that the disease involves other causal factors than *Pythium arrhenomanes* alone, as indicated in the earlier literature.

Most of the sorghums are resistant to this severe type of disease manifestation, according to Wagner (1936). Melchers and Lowe[1] list the reactions of commercial varieties to the milo disease. Bowman *et al.* (1937) and Heyne *et al.* (1944) reported that reaction to the disease is determined by a single major factor difference with susceptibility to the disease partly dominant. Melchers and Lowe (1943) describe the techniques used in developing resistant varieties. Susceptible milos and one resistant milo selection were crossed with kaffir, kaoliang and sorgo varieties, and the segregates were investigated.

This disease is controlled in the heavily infested areas by the use of resistant selections (Fig. 49). Resistant varieties are grown at present on about 4 million acres. Soil infestation spreads rapidly and persists for several years in the areas when susceptible varieties are grown. Recently seed transmission has been established. The morphology and etiology of the *Pythium* spp. are given in Chap. IV.

5. Downy Mildews, *Sclerospora sorghi* (Kulk.) Weston and Uppal and Other Species.—The sorghums are not damaged extensively by the downy mildews, although several species of *Sclerospora* occur on them in Asia and Africa (see table, page 75, The Fungi Causing Downy Mildew on the Gramineae). *Sclerospora sorghi* is limited apparently to southern Asia, chiefly India, where it is destructive on this crop. The symptoms on sorghum are similar to those of the oriental downy mildews on corn and sugar cane, according to Butler (1917), Melchers (1931), Uppal and Desai (1932), and Weston and Uppal (1932).

The Fungus.—*Sclerospora sorghi* (Kulk.) Weston and Uppal
(*Sclerospora graminicola* var. *andropogonis-sorghi* Kulk.)

Weston and Uppal (1932) described the conidial and oögonial stages of the fungus and compared them with *Sclerospora graminicola* as the basis for the new species. The conidia of *S. sorghi* lack an apical dehiscence papilla and germinate by the formation of a germ tube. The conidiophore has a definite basal cell, an extensive branching, and consequent arrangement of the conidia in a hemispherical plane on the long sterigmata of the conidiophores. The oöspores are similar in morphology to *S. graminicola* and germinate by the formation of a germ tube.

6. Gibberella Seedling Blight and Stalk Rot, *Gibberella zeae* (Schw.) Petch [*G. saubinetii* (Mont.) Sacc.] and *G. fujikuroi* (Saw.) Wr.—

[1] *U. S. Dept. Agr., Plant Disease Reporter Sup.* 126, 1940.

Seedling blight in cooler soils is frequently an important factor in reducing stands. Seed treatment with the mercury dusts increases stands, according to Leukel (1943) and Leukel and Martin (1943). The symptoms are similar to those on corn (Chap. IV).

7. Helminthosporium Leaf Blight, *Helminthosporium turcicum* Pass.—The corn leaf blight occurs sparingly on the sorghums and very extensively on Sudan and Johnson grasses. Considerable defoliation results on these two grass sorghums (Fig. 50). The lesions are similar to those on corn except for the development of more pigmentation around the margin of the lesions in the sorghum group. According to Lefebvre and Sherwin (1945) and Mitra (1923) as well as inoculation experiments by Allison,[1] specialization occurs within the species. Certain of the physiologic races of the parasite on corn infect the sorghum group. The sorghum races of the fungus apparently do not infect corn naturally. The extensive severe development of the disease on Sudan grass in the Northern United States and the relatively sparse occurrence of the leaf blight on corn are explained in part on the basis of specialization of the parasite.

The morphology of the fungus and etiology and control of the disease are discussed in Chap. IV. According to Chilton (1940), the fungus is seed-borne on Sudan grass as well as carried over on crop residue.

8. Anthracnose, *Colletotrichum* Spp.—The anthracnose is distributed widely on the sorghums, especially on Sudan and Johnson grasses. The disease is of little economic importance on the sweet and grain sorghums, but causes damage on Sudan grass (Allison and Chamberlain, 1946) and broom corn. *Colletotrichum graminicolum* (Ces.) G. W. Wils. causes root and crown lesions and small ovate to irregular zonate spots on the leaves of Sudan grass. The central area of the leaf spot is tan; the border is red to brown. Acervuli develop on the older portion of the spot (Fig. 50). Root and crown rot occurs on Sudan grass and broom corn, causing lodging and reduced yields. The interior of the base of the stalk is rotted, and numerous acervuli develop on the surface of the rotted portion. *C. lineola* Cordà is listed also as the species on the sorghums, although Wilson (1914) includes this under *C. graminicolum*.

The disease is abundant from mid-summer to maturity of the crop. Resistant inbreds and hybrids are the best means of control on Sudan grass. The disease is discussed in detail in Chaps. VIII and X.

9. Gloeocercospora Leaf Spot, *Gloeocercospora sorghi* Bain and Edg.—Bain and Edgerton (1943) described a zonate leaf spot on sorghums, corn, and sugar cane. The fungus was placed in a new genus in the Tuberculareaceae, as the conidia are borne in a sporodochium-like structure and

[1] Unpublished data on cross inoculations with *Helminthosporium turcicum* on corn and Sudan grass.

in a slimy matrix on short conidiophores. The spots appear first as small red to brown water-soaked lesions. As the spots enlarge they become

FIG. 50.—Leaf spots on Sudan grass produced by *Colletotrichum graminicolum* (*A*) compared with the leaf lesions caused by ***Helminthosporium turcicum*** (*B*). These are two serious diseases of this crop.

darker colored and elongate and form large seimcircular or irregular lesions frequently extending across the leaf blade. Both leaves and floral bracts are infected.

The Fungus.—*Gloeocercospora sorghi* Bain and Edg.

Sporodochia bearing hyaline, septate, short, single or branched conidiophores form on the lesion. Conidia are hyaline, elongate to filiform, variable in length, the longer ones tapering at the apex, and measure 20–195 by 1.4–3.2 microns. The conidia are borne in a slimy matrix and are salmon pink in mass. Sclerotia are 0.1 to 0.2 mm. in diameter, lenticular to spherical, black, and borne inside the necrotic tissue.

10. Sooty Stripe, *Ramulispora sorghi* (Ell. and Ev.) Olive and Lefebvre.—A fungus, somewhat similar in conidial morphology to the above, produces sooty stripe on the sorghums in the Southern United States and Asia. The lesions are elongate elliptic, regular in outline, first gray to tan in the center with a red margin, later sooty colored as the black loosely attached sclerotia develop on the lesions.

Ramulispora sorghi (Ell. and Ev.) Olive and Lefebvre in the Tuberculareaceae with synonyms (*Septorella sorghi* Ell. and Ev.), (*Ramulispora andropogonis* Miura), [*Titaeospora andropogonis* (Miura) Tai] apparently is the accepted binomial for this parasite.

The sclerotia are amphigenous, scattered, superficial, subglobose, coarsely tuberculate, and black. Sporodochia are amphigenous, erumpent from subepidermal stromata. Fasiculate conidiophores form from the sporodochia and the sclerotia. Conidia are filiform, one- to three-branched, hyaline, curved, tapering toward the apex, 3- to 8-septate, and measure 38–86 by 1.9–3 microns (Olive *et al.*, 1946).

Cercospora sorghi Ell. and Ev. occurs on the sorghums in the Southern United States, according to Seymour (1929) and Stevenson (1926).

11. Charcoal Rot, *Sclerotium bataticola* Taub.—The disease occurs as a cortical root rot and internal stalk rot of corn and sorghums as well as many other crops. The disease is distributed widely in the warmer climates and is prevalent in the drier Central United States, according to Livingston (1945) and others. The black cortical rot of the larger roots is conspicuous on seedlings and mature plants. The internal stalk rot and the development of numerous small black sclerotia on the infected tissues occurs later in the season. The stalks break over at the soil line or a few inches above.

The Fungus.—*Sclerotium bataticola* Taub.

Macrophomina phaseoli (Maubl.) Ashby is the pycnidial stage of the fungus. *Rhizoctonia bataticola* (Taub.) Butler is considered the same as *Sclerotium bataticola* by some mycologists, although the evidence is not conclusive (Henson and Valleau, 1937).

The pycnidial stage of the fungus is not common on sorghum under field conditions. All varieties are relatively susceptible, as shown by

Hoffmaster and Tullis,[1] and rank in about the following order: milos, darso, Sudan grass, feterita, hegari, kaffir, and the sweet sorghums.

Tullis[2] described a stalk rot of sorghum in Texas with symptons similar to charcoal rot on sorghums which was caused by *Spicaria elegans* (Cda.) Harz. and *Fusarium moniliforme.*

12. Loose Kernel Smut, *Sphacelotheca cruenta* (Kuehn) Potter.—The loose kernel smut of the sorghums is not distributed so widely in the United States as the covered kernel smut. The disease is common in Asia and Africa, according to Butler (1918). Apparently the disease is not common on Sudan grass. A similar loose kernel smut occurs on Johnson grass in South Central United States. Both grain and fodder yields are reduced by the smut.

Symptoms and Effects.—The smut is apparent by the early appearance of the smutted heads and dwarfing of the plants in most varieties. Generally all the flowers in a head are smutted. The floral bracts tend to elongate and proliferate. Frequently, the lemma and palea as well as the ovary contain smut sori. The membrane ruptures early, releasing the powdery black spore mass. The elongate central columella of the sorus persists after the spores are discharged. The symptoms range from a loose to covered type kernel smut, depending upon the sorghum variety (Fig. 51).

The Fungus.—*Sphacelotheca cruenta* (Kuehn) Potter
(*Ustilago cruenta* Kuehn)

The sori are formed in the ovaries and floral bracts. The chlamydospores are enclosed in a fungal membrane (peridium) composed of loosely joined rounded gray cells about twice the diameter of the spores. In many sorghum varieties the membrane is fragile and ruptures early. The chlamydospores are formed in elongated irregular clumps, not spore balls, that separate as they mature. The spores are round to elliptical, dark brown, with indistinct pits to reticulations on the surface, and 5 to 10 microns in diameter. They germinate to form characteristically a four-celled basidium (promycelium) with laterally borne sporidia. Secondary sporidia are produced on media.

Etiology.—Infection occurs in the early stages of seedling development. The fungus becomes established in the primordial tissues of the developing shoot, and spores are produced in the kernels and adjacent floral tissues. This systemic type of infection is characteristic of many of the smut fungi. As shown by Melchers and Hansing (1943), Reed (1923), and others, the development of *Sphacelotheca cruenta* in the sorghum plant causes dwarfing in most varieties and the early development of the head. Smut infection occurs over the range of environmental conditions favorable for development of sorghum. The chlamydospores are carried over on the seed and in the soil in the drier climates.

[1] *U. S. Dept. Agr., Plant Disease Reporter* **28**: 1175–1184. 1944.
[2] *U. S. Dept. Agr., Plant Disease Reporter* **28**: 1100. 1944.

Seed treatment and resistant varieties constitute the best means of control. Leukel (1943) reported the mercury dusts and copper carbonate

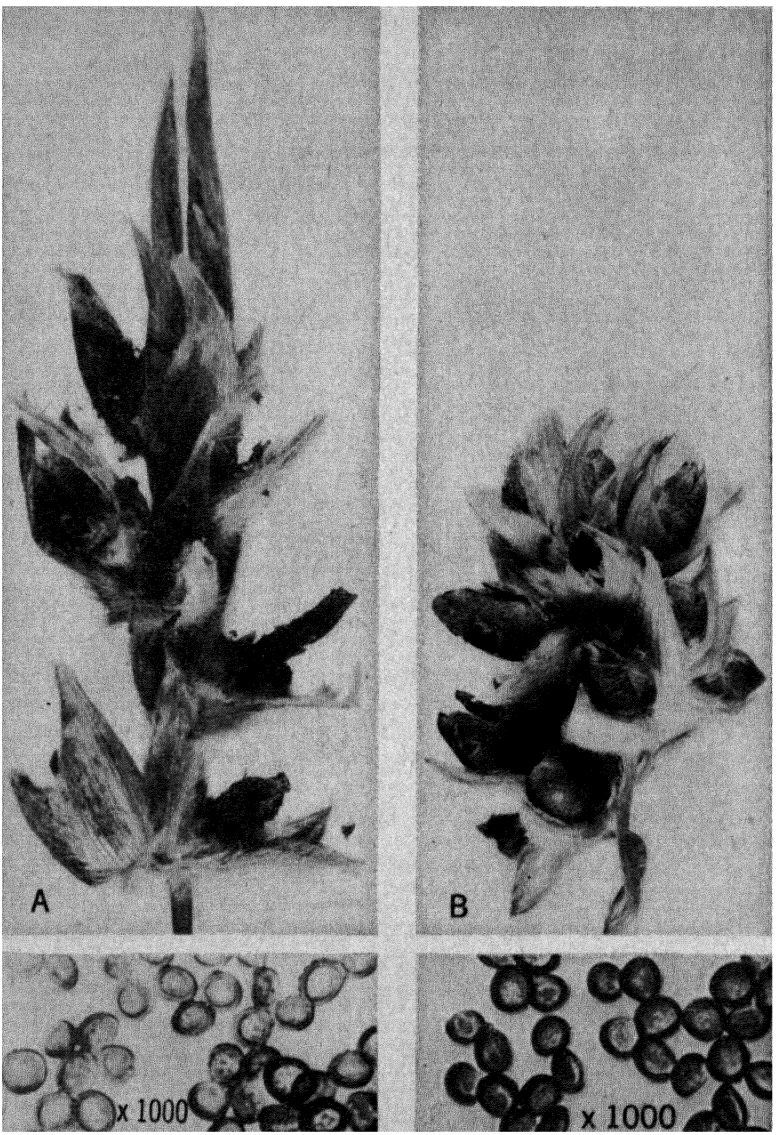

FIG. 51.—Sorghum spikelets, magnified, showing the loose (*A*) and covered (*B*) kernel smuts of sorghum caused by *Sphacelotheca cruenta* and *S. sorghi* respectively. Chlamydospores, highly magnified, are shown in the inserts.

as suitable for the control of both loose and covered kernel smuts. Stands were improved also in Pythium and Fusarium infested soils. Melchers

(1933) listed the milos, feteritas, hegari, and Dwarf Shantung kaoliang as resistant to moderately resistant to the two physiologic races of *Sphacelotheca cruenta*. Race 1, originally from India, attacks kaffir × feterita and Pierce kaferita, while race 2 infection on these varieties is very light. Race 2, found in Kansas, infects Red Amber × feterita and White Yolo, which are resistant to race 1. Rodenhiser (1934) reported on crosses between races of *S. sorghi* and interspecific crosses between *S. sorghi* and race 1 of *S. cruenta*. His results suggest (1) the origin of races by hybridization and (2) on the basis of sterility in the first generation interspecific hybrids, the two species are distinct rather than representing the extremes in a series of variants, as suggested by Tisdale *et al.* (1927). According to Melchers (1940), Spur feterita (C.I. 623) is the most resistant to the known races of both loose and covered kernel smut parasites.

The loose kernel smut occurring on Johnson grass in the South Central United States is similar to the loose smut of sorghum. Rodenhiser (1937) listed it as a third race of *Sphacelotheca cruenta*. Johnston *et al.* (1938) suggests the possibility of a different species.

13. Covered Kernel Smut, *Sphacelotheca sorghi* (Lk.) Clint.—The covered kernel smut is one of the most common diseases of sorghums in the United States and frequently reduces yield of grain. The disease is widely distributed in other countries on the sorghums and Sudan grass, as reviewed by Reed (1923) and Reed and Melchers (1925).

Symptoms and Effects.—Typically a kernel smut, the ovaries of the flowers are converted into smut balls. Sori rarely develop in the floral bracts. The outer membrane (peridium) of the sorus is tough and usually persists as the grain ripens. It varies in color with the different physiologic races of the fungus and with the sorghum varieties. The smut balls are elongated beyond the floral bracts and resemble somewhat the shape of the kernel (Fig. 51). The smutted plants are not reduced greatly in height and vegetative development as in the case of the loose smut, as shown by Melchers and Hansing (1943).

The Fungus.—*Sphacelotheca sorghi* (Lk.) Clint.
(*Sorosporium sorghi* Lk.)
(*Tilletia sorghi* Tul.)
(*Ustilago sorghi* Lk.)

The sori are formed chiefly in the ovaries. The chlamydospores are enclosed in a persistent fungal membrane that ruptures at the apex, exposing the dark spores and columella. The membrane is composed of round to elongate cells about the same diameter as the spores. Chlamydospores are globose to angular, olivaceous, brown, apparently smooth, but actually finely punctate to minutely echinulate, and 5 to 7 microns in diameter. They germinate to form characteristically a four-celled promycelium bearing lateral sporidia, although great variation occurs.

The etiology and control of the disease are essentially similar to the

loose kernel smut. Standard and new chemical dust seed treatments are described by Hansing and Melchers (1944). Environmental conditions influence infection, and some varieties, although infected, do not develop sori in the primary heads, according to Melchers (1933) and Melchers amd Hansing (1938). Resistant varieties occur in most of the sorghum groups, as reported by Melchers *et al.* (1932), Reed and Melchers (1925, 1940), Tisdale *et al.* (1927), and others. Spur feterita (C.I. 623) is resistant to all known races, and dwarf yellow milo (K.B. 2515) and Red Amber × feterita (K.B. 33308) are resistant to one or more of the physiological races of *Sphacelotheca sorghi*. Specialization and variation in the fungus is reported by the above authors, as well as Ficke and Johnston (1930), Melchers *et al.* (1932), and Tyler (1938). Five physiologic races of the parasite are differentiated, as shown in the following table:

Physiologic race	Reaction on sorghum varieties					
	Dwarf yellow milo (C.I. 332)	White Yolo (K.B. 2525)	Pierce kaferita (K.B. 2547)	Feterita × kaffir (F.C.I. 8917)	Feterita (S.P.I. 51989)	Kaffir × feterita (H.C. 2423)
1	R	IR	R	R	R	R
2	S	S	R	R	R	R
3	R	R	S	IR	IR	S
4	R	S	R	R	R	R
5	R	R	R	R	R	S

14. Head Smut, *Sphacelotheca reiliana* (Kuehn) Clint.—The disease occurs occasionally primarily on sorghums in the Central United States where it is of minor importance. In the Western United States and southeastern Europe and Asia, this smut is more generally distributed on corn. The smut is not evident until the panicle emerges. All or part of the floral structures are replaced by the smut sorus. The fragile membrane ruptures early, releasing the dark-brown dusty spore mass and exposing the vascular tissues of the inflorescence. According to Reed (1923) the feterita, milo, kaffir, kaoliang, and broom corn groups of sorghums are resistant. Physiologic races are distinct on sorghum and corn. See Chap. IV for the detailed discussion of the disease.

15. Long Smut, *Tolyposporium ehrenbergii* (Kuehn) Pat.—This smut is not reported in the United States and according to Kamat (1933), Kulkarni (1918), and McAlpine (1910) causes little damage to this crop in other countries. The sori develop in the ovaries, frequently only a few in each panicle. The sori are long, cylindrical, slightly curved, and rupture at the apex to release the brownish-green spore balls. The spores

remain united, more or less permanently in large groups. The exposed surfaces of the spores in the spore ball are covered by flattened echinulations. The spores germinate in place by the formation of an elongated promycelium, frequently branching. Numerous sporidia are produced singly or in chains.

Little experimental data are available on the etiology and control of the disease. Continuous culture in the same areas tends to increase the amount of infection.

16. Rust, *Puccinia purpurea* Cke.—This rust occurs in limited amounts on all the sorghums, including many of the grass species of this genus. The disease is more common in the Southern United States, and it is distributed generally with the crop. Butler (1918) reported the rust as one of the most common rusts of the cultivated crops of India. The uredia develop on both surfaces of the leaves. Pigmentation appears around the uredium. Telia develop in and adjacent to the uredia as in the corn rust. The aecial host is unknown. Resistant varieties are reported by Johnston and Mains (1931).

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CHAPTER X

SUGARCANE DISEASES

The sugarcane varieties in cultivation are largely a group of interspecific hybrids propagated vegetatively by stem cuttings. According to Brandes and Sartoris (1936), disease epidemics played an important role in the development and propagation of the interspecific hybrids. Nobilizing or resorting to natural crossing between the cultivated varieties and primitive hardy disease-resistant forms was started during the sereh epidemic. However, hybrids superior to the former commercial varieties were not produced until much later. Hybridization really became important in sugarcane improvement after J. Jeswiet in Java produced the selection P.O.J. 2878. The sugarcane mosaic epidemics and better parental material and techniques stimulated further, more systematic crossing and testing of larger populations of seedlings. Economic pressures and the better sugar yields of the latter hybrids also stimulated the breeding improvement. The following table gives the principal species used in crossing, their chromosome number and disease reaction.

Saccharum spp.	Number chromosome pairs	Adaptability	Reactions to diseases			
			Mosaic	Sereh	Downy mildew	Smut
S. officinarum L.	40	Tropics	Susc.	Susc.	Susc.	Mod. susc.
S. sinense Rovb.	58–60	Wide	Some susc.	Immune	.....	Susc.
S. barberi Jesw.	42–46	Temp. and subtropics	Susc. tolerant	Immune	.....	Mod. susc.
S. spontaneum L.	56	Wide	Immune*	Immune	Susc.	Mod. susc.
S. robustum.....	42	Wide	Susc.	.....	.....	

* *S. spontaneum* var. Koelawa A. is susceptible to mosaic.

Disease-resistant hybrids are used in the control of most commercially important diseases. The propagation by clones, chiefly culm bud cuttings, facilitates the use of good-quality disease-resistant selections. Quarantines are effective in preventing the further distribution of diseases carried in the cuttings.

1. Sugarcane-Grass Mosaics, Virus Transmitted by *Aphis maidis* (Fitch.) and others.—The grass mosaics occur on a relatively large

number of species. Sugarcane, corn, sorghum, certain millets, and a relatively large number of tropical grasses are susceptible in varying

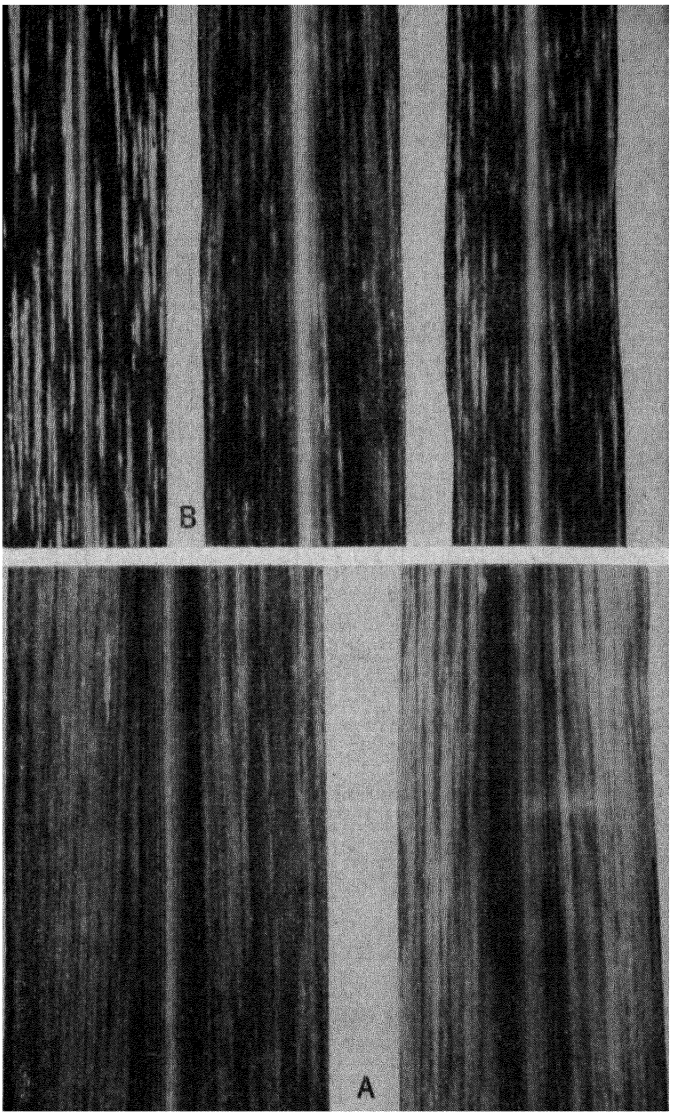

FIG. 52.—The green (*A*) and yellow (*B*) mosaics of sugarcane caused by viruses transmitted by aphids. (*Courtesy of C. W. Edgerton.*)

degrees to this mosaic complex. The diseases cause heavy losses in susceptible varieties, and they are widespread in distribution. Brandes (1919), Brandes and Klaphaak (1923), Kunkel (1924), Lyon (1921), and

other early investigators did not differentiate between the mosaics and the streak. Stahl (1927), Story (1925), and Story and McClean (1930) described the stripe and streak diseases on corn and sugarcane transmitted by leaf hoppers (*Peregrinus* and *Cicadulina* spp.). The symptoms of these diseases are similar in certain characters to those of the mosaics.

The mosaic disease complex is manifest by the light-green or yellow streaking and irregularly mottled appearance of the leaves. According to Tims *et al.* (1935) and Forbes *et al.* (1937), the green or mild and yellow or severe mosaics occur commonly on sugarcane in Louisiana. The symptoms of the two strains are similar in young plants, but the stripes are predominantly light green in the one and yellow in the other in the older leaves (Fig. 52). Plants carrying one virus strain are not infected with the other. Yellowing and browning are later symptoms on some cane varieties. Infected plants are lighter green in color than the healthy plants. Mottling or marling of the culm is common in older susceptible material. Culm cankers and internal necrosis occur in some varieties. Numerous strains of mosaic viruses occur on sugarcane with variations in symptoms. The symptoms of the mosaics also vary with the different sugarcane selections. In resistant varieties, the mosaic pattern on the leaves frequently becomes masked or disappears.

The principal insect vector is *Aphis maidis* (Fitch.). Three other aphids are reported by Ingram and Summers (1936, 1938) and Tate and Vandenberg (1939) as capable of transmitting the diseases to sugarcane. Apparently the aphids persist on the sugarcane to reinfect the annual crops such as corn and sorghum. Transmission of the viruses by mechanical means is very easy on sugarcane and on corn, as reported by Matz (1933), Sein (1930), Wilbrink (1929), and others. The mosaics on sugarcane are controlled by the use of resistant or tolerant varieties.

2. Streak, Stripe Diseases, and Chlorotic Streak, Viruses Transmitted by *Cicadulina mbila* (Naude), *Peregrinus maidis* (Ashm.), and *Draeculacephala portola* Ball, Respectively.—Storey (1925, 1926) reported a streak disease on corn and sugarcane in South Africa transmitted by the leaf hoppers *Cicadulina mbila* (Naude), *C. zeae* China, and *C. storeyi* China. The disease occurs in many sugarcane sections of Africa, Asia, and the Pacific area. Stahl (1927) described a stripe disease of corn in Cuba transmitted by the leaf hopper *Peregrinus maidis* (Ashm.) and differentiated this disease from sugarcane mosaic. The leaf stripes in streak are white instead of yellow as in mosaic. The stripes are uniformly distributed over the leaf as in mosaic, but usually the mottling is very conspicuous in the mosaic-infected leaves. There is little stunting of the plants in streak. Storey (1930, 1933) demonstrated, by means of leaf hoppers, at least a strain difference in the sugarcane and corn viruses causing streak. The virus of sugarcane streak caused mild transitory

symptoms on corn, and the virus of corn produced a mild streak on sugarcane. He concluded also that stripe and streak on corn were not the same. Resistant sugarcane varieties are reported by Storey and McClean (1930).

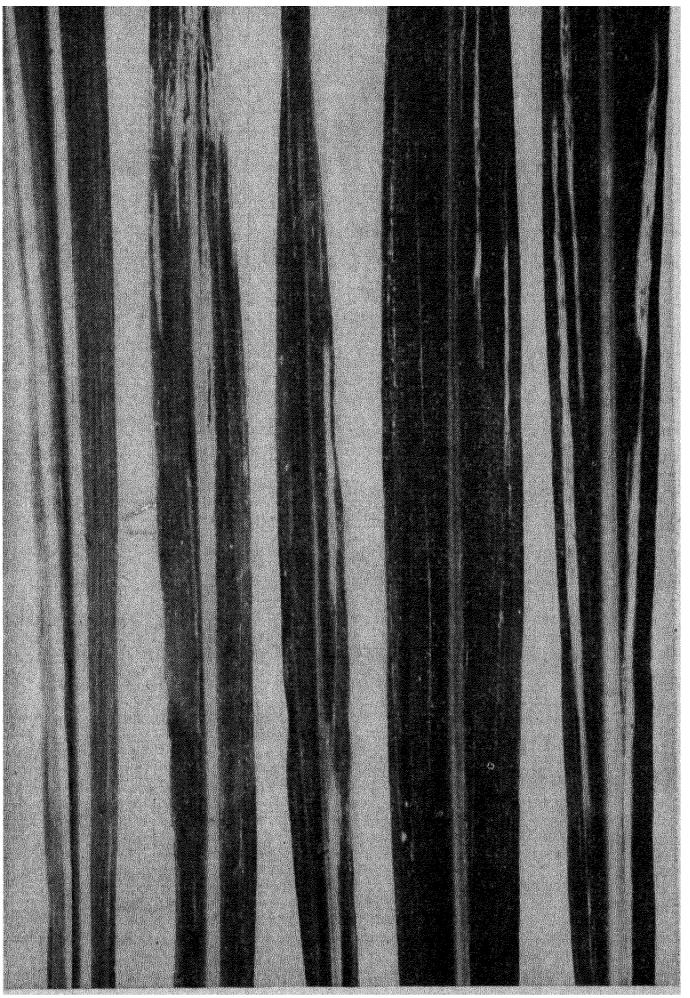

FIG. 53.—Chlorotic streak of sugarcane caused by a virus transmitted by leaf hoppers. (*Courtesy of C. W. Edgerton.*)

Chlorotic streak was first differentiated in Java and Hawaii in 1928 and 1929. The disease occurs also in Australia, the West Indies, Mauritius, and the Americas. As reported by Abbott and Ingram (1942) and Abbott and Sass (1945), the disease occurs in the Southern United States

and it is characterized by the wavy irregular margins of the white streaks and by cellular modifications and necrosis of tissue (Fig. 53). Intracellular bodies occur in the stalk tissues. The malady is transmitted by the leaf hopper *Draeculacephala portola* Ball. Edgerton and associates (1942) report its control by treating seed 20 to 30 minutes in water at 52°C.

Bell (1932) reported a dwarf disease different from mosaic or streak.

3. Fiji Disease, Virus Transmitted by *Perkinsiella vastatrix* (Bred.) and *P. saccharicida* Kirk.—The fiji disease, like mosaic, is of major importance to the sugarcane industry. Apparently, in contrast to the mosaic, the fiji disease is restricted in its distribution. The disease, insofar as is known, occurs only in the South Pacific area. The presence of narrow linear, smooth, white to brown galls on the undersurface of the leaf veins and the absence of striping and mottling form the outstanding differentiating symptoms. The leaves are shorter, stiffer, and darker green than in the healthy plants. Internodal elongation of the canes is checked later, and the canes split. Lower buds develop, forming shortened, distorted clumps. Intracellular bodies are present, as reported by Kunkle (1924) and others. Ocfemia *et al.* (1933, 1934) transmitted the disease by means of the leaf hopper *Perkinsiella vastatrix* (Bred.). Mungomery and Bell (1933) transmitted the disease using *P. saccharicida* Kirk. They state that probably several species of leaf hoppers are capable of transmitting the virus. The malady is carried in diseased cuttings. Some varieties are moderately resistant, but immune varieties are not reported. Quarantine, removing diseased plants, restricting ratooning, and disease-free cuttings are recommended.

4. Sereh, Cause Unknown.—Lyon (1921, 1923) discussed the sereh complex of Java, including its history, description, and the early literature pertaining to the disease. The symptoms include most of those for the virus diseases discussed above and, in addition, the red gum and staining of the vascular tissues and the development of adventitious roots from the aerial nodes. The upper buds of infected canes fail to develop after topping. The etiology of the disease is still vague and incomplete. The presence of bacteria in association with the diseased plants is suggested as a cause by some. Sereh was controlled in Java for many years by growing seed canes at high altitudes. The disease now is controlled by the use of resistant varieties and cuttings from healthy canes. Wilbrink (1923) reported the control of the condition by treating cuttings 30 minutes in water at 52°C.

5. Gumming Disease, or Cobb's Bacterial Wilt, *Xanthomonas vasculorum* (Cobb) Dows.—The disease causes serious loss in susceptible varieties in the Southern Pacific area, in the West Indies, in South America, and in other countries. Apparently, the further general spread

of the disease and introduction into the United States is preventable by quarantines and the use of resistant varieties.

In the early stages of development, the disease is primarily a leaf disease. In this respect it is similar to the bacterial wilt of corn. The pale-green to yellow stripes flecked with reddish dots, regular in outline when young and becoming diffused in outline as they become older, form along the margin and apex of the leaf blade. The longitudinal streaks enlarge and turn red to brown as the leaves mature and tissue necrosis advances. In the older canes, showing advanced stages of the disease, the inner leaves of the apical whorl develop linear stripes, while the older lower leaves show the red blotches and brown streaks. As the disease advances in susceptible varieties, the organism in the vascular tissues of the leaves moves down into the stalk tissues. The advanced leaf symptoms are associated with dwarfing of the plants and necrotic pockets in the stalk tissues. The presence of the honey-yellow bacterial exudate in the conductive tissues of the stalk and veins constitute the most characteristic symptom. Hughes (1939), Matz (1922), North (1935), and Smith (1914) described the symptoms, the organism, and the etiology in detail.

The Bacterium.—*Xanthomonas vasculorum* (Cobb) Dows.
[*Phytomonas vasculorum* (Cobb) Bergey *et al.*]
(*Bacillus vasculorum* Cobb)
(*Bacillus sacchari* Speg.)
[*Bacterium vasculorum* (Cobb) Mig.]
[*Bacterium vasculorum* (Cobb) E. F. Sm.]
[*Pseudomonas vasculorum* (Cobb) E. F. Sm.]

Etiology.—The disease is confined largely to sugarcane in nature, although Orian (1939) reported it spreading to corn and a species of palm in Mauritius. Inoculations into sorghums and corn produce symptoms similar to bacterial wilt of corn. Infected cuttings spread the disease both locally and long distances. In wet weather the exudate forms a slime on the surface of the infected plants. The bacteria are spread by splashing rain and flies, according to reports by North (1935) and Leach (1940). Some other insects, as in the case of Stewart's wilt of corn, may be associated with the spread of the disease. Resistant varieties, elimination, and quarantine are the best means of control.

6. Leaf Scald, *Bacterium albilineans* Ashby.—The disease is distributed throughout the Southern Pacific sugarcane area, Hawaii, and Brazil. The scald so far is confined to these areas, and it is not so widespread in distribution as the similar gumming disease. Neither disease is reported in the continental United States. The two vascular bacterial diseases are about equally important in causing losses.

The leaf scald symptoms differentiate this disease from the gumming disease. The narrow definitely margined leaf streaks are creamy or grayish white in color. The streaks usually extend the full length of the leaf blade and the leaf sheath and later broaden from the tip downward; followed by withering of the leaf blade in the same direction (Arruda and Amaral, 1945). The streaks are associated with the infected vascular bundles. The older lesions turn brown or redden and finally wither and dry out. The severe infection results in rather sudden wilting and drying out of individual culms or the entire plant. In both cases, weakened shoots develop from the basal axillary buds. These show the characteristic white leaf streak symptoms. The vascular bundles of the stalk show red staining, especially in the nodes. The absence of the yellow exudate in scald also differentiates this disease from the gumming type. Ashby (1929), Bell (1929), and North (1926) discussed the disease and compared the symptoms of the two somewhat similar diseases.

The Bacterium.—*Bacterium albilineans* Ashby

[*Phytomonas albilineans* (Ashby) Bergey *et al.*]

Motile with a single polar flagellum, the rods are more slender than *Xanthomonas vasculorum*, and growth on most media is slow.

The distribution and development of the disease in fields varies from scattered plants with mild symptoms to rapid wilting of most plants in large areas. New infections appear suddenly in fields some distance from diseased areas. This behavior suggests the importance of environmental conditions in the development of scald and the possibility of insects playing an important role in both dissemination and introduction of the parasite into the cane tissues. The disease is controlled by the use of resistant varieties.

7. Bacterial Blights of Sugarcane.—Two bacterial blights occur on sugarcane that are similar in symptoms to the two more common bacterial blights on sorghum and Sudan grass. The two diseases are widely distributed.

Red Stripe and Top Rot, *Xanthomonas rubrilineans* (Lee *et al.*) Starr and Burk. (*Phytomonas rubrilineans* Lee *et al.*).—The disease is common through the sugarcane areas including the Southern United States, although it is of minor importance according to Christopher and Edgerton (1930). Cottrell-Dormer (1932) demonstrated that sorghums, corn, Sudan grass, and Johnson grass are infected.

The leaf stripes are at first water-soaked narrow lesions. They elongate rapidly and turn red to maroon in color. Usually the stripe is bordered by a water-soaked or yellow zone. The stripes are continuous with a uniformly necrotic and colored area. Later the stripes coalesce,

forming alternate red and chlorotic stripes. Exudate is formed on the necrotic areas. Red staining occurs in the vascular bundles, progressing from the apex downward. Usually only portions of the culm interior are discolored by the bacteria. Later the apical culm tissues are rotted and the upper leaves are killed. Cottrell-Dormer (1932) and Wood (1927) associated the top rot and leaf stripe as the same disease complex. In the various studies in the Pacific area and in the United States, the disease is attributed to *Xanthomonas rubrilineans* (1924, 1925).

Streak or Mottled Stripe, *Bacterium rubrisubalbicans* (Christopher and Edg.) Burgw. (*Phytomonas rubrisubalbicans* Christopher and Edg.).—The symptoms of the disease on sugarcane are similar to the streak disease of sorghum. Christopher and Edgerton (1930) compared the disease with the streak on Johnson grass. They produced the disease by inoculations on Johnson grass and sorghum but not on corn. Cottrell-Dormer (1932) reported the same disease in Queensland. The disease is of minor importance.

The leaf stripes are linear with less regular margins and centers. The color is predominantly red, although frequently white areas or white margins occur. Where the streaks coalesce, mottled red and white bands are formed across the leaf blade. Top rot is not associated with the disease. Moderately resistant varieties are reported by Christopher and Edgerton (1930).

8. Pythium Root Rot, *Pythium arrhenomanes* Drechs., *P. graminicolum* Subr., and Others.—The root rot complex is common on sugarcane in practically all areas. The practice of ratooning or growing several crops of cane on the same root system tends to increase the damage in comparison with an annual crop like corn. Soil type, drainage, fertility, and root damage from nematodes and insects influence the incidence and damage from this disease complex. Carpenter (1934) summarized the literature and experimental data on these predisposing factors. The rootlets and roots show a water-soaked brown to gray rot resulting in a depleted root system.

The Fungi.—Essentially the same species of Pythium causing the root rots of corn, sorghums, and other grasses are associated with the sugarcane root rots. Edgerton *et al.* (1929) reported upon numerous isolations in Louisiana; *Pythium, Rhizoctonia,* and *Marasmius* spp. were the principal pathogenic fungi. Rands and Dopp (1934) reported on the variability of *Pythium arrhenomanes* and included nine of the species reported by Sideris (1931) in this species. The morphology of the two common graminicolous species of Pythium is given in Chap. IV.

Resistant varieties, soil drainage, and balanced fertility are essential in reducing losses from root rot.

9. Downy Mildew, *Sclerospora sacchari* Miy. and Other Species.—The disease on sugarcane caused by *Sclerospora sacchari* is the more common and destructive species on this crop. Downy mildew is restricted to a few countries in the southwestern Pacific area. Other species of *Sclerospora* occur to a limited extent on this crop (see table on the Downy Mildews of the Gramineae, Chap. IV). According to Leece (1941), Lyon (1915), and Miyake (1911) conidia and oöspores of *S. sacchari* occur on sugarcane. Symptoms on sugarcane are similar to those on corn.

The etiology and control of the disease on sugarcane, a perennial, varies somewhat from that on corn. Infection occurs on young tissues, especially those associated with the buds. Bud infection is probably more general in sugarcane than in corn. Disease-free cuttings and the use of resistant varieties constitute the best means of control. Quarantine apparently has prevented the entry of the disease into Cuba and the Southern United States.

10. Pokkah-bong, *Gibberella fujikuroi* (Saw.) Wr. and var. *subglutinans* Edwards (*Fusarium moniliforme* Sheldon and var. *subglutinans* Wr. and Reinking) and Other Organisms.—The disease is common on sugarcane and is widely distributed. Similar symptoms appear occasionally on corn. Damage is not severe except in very susceptible varieties of sugarcane.

The first symptoms of the disease are the light-colored twisted leaves as they come out of the terminal leaf whorl. Chlorotic areas especially on the base of the leaf and leaf sheath persist on plants with mild symptoms. Few to several leaves are affected. The chlorotic areas frequently are malformed, either narrow and twisted or stiff. In later symptoms on susceptible varieties, the top of the stalk is rotted and the growing point is killed. Pink necrotic areas are found in the leaves and culm.

Apparently *Gibberella fujikuroi* (Saw.) Wr. and the variety *subglutinans* Edwards are associated with the disease, as reported by Priode (1929) and others. Fawcett (1922) associated *Erwinia flavida* (G. Fawc.) Magrou (*Bacillus flavidus* G. Fawc.) with the disease. The cause of the disease is still indefinite. Resistant varieties apparently are reducing the severity of the disease.

11. Red Rot, *Physalospora tucumanensis* Speg. (*Colletotrichum falcatum* Went.).—The red rot is prevalent in the major sugarcane areas. The disease apparently is more severe, especially in reducing stands, in the cooler climates. Perithecia were collected on the five species of *Saccharum* and the grass *Leptochloa filiformis* (Lam.) Beauv., common in some sugarcane areas, as reported by Carvajal and Edgerton (1944). It is possible to inoculate the sorghums including Sudan grass and Johnson grass with *Physalospora tucumanensis*, but the *Colletotrichum* found naturally on the sorghums is different, according to Edgerton (1911) and recent reports to

the author. Abbott (1938) considered the same species on both groups of plants, based on morphology of the cultures isolated. *C. lineola* Cda.,

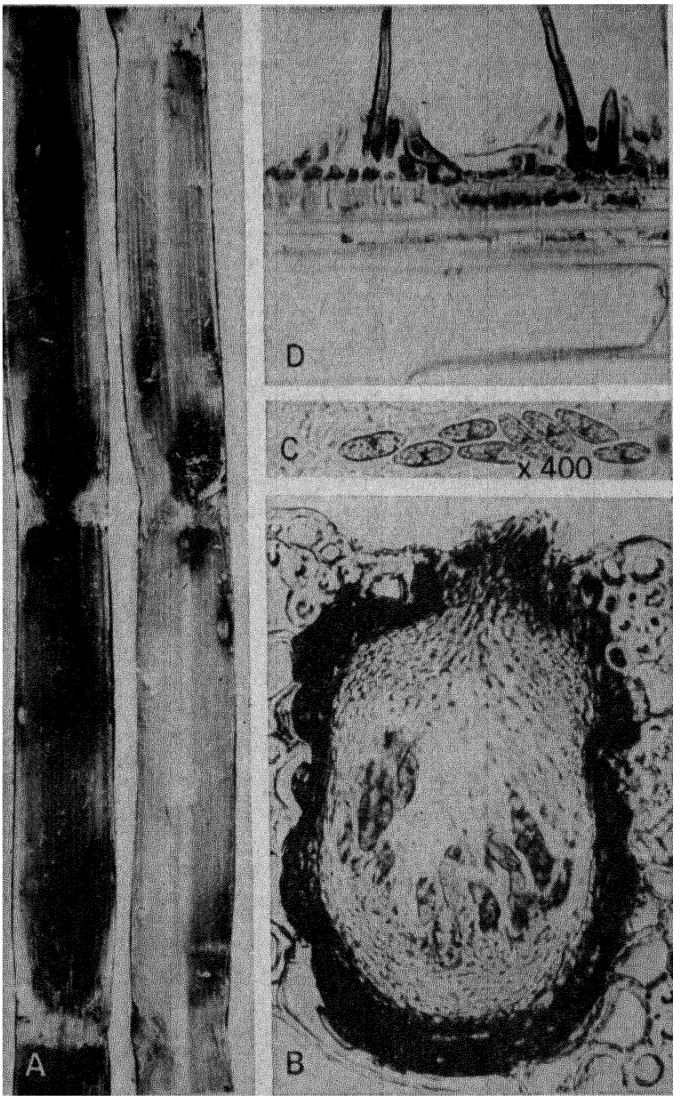

FIG. 54.—Red rot of sugarcane caused by *Physalospora tucumanensis* (*Colletotrichum falcatum*) showing the stalk rot (*A*). The perithecium (*B*), ascospores (*C*), and acervulus (*D*) of the fungus are shown highly magnified. (*Courtesy of C. W. Edgerton.*)

now considered a synonym of *C. graminicolum* (Ces.) G. W. Wils., is common on the sorghums, especially Sudan grass in the temperate zones.

Symptoms and Effects.—The disease is apparent as a red rot on the leaves, stalks, and stubble of the sugarcane (Fig. 54). The conspicuous red linear lesions are common on the midrib, especially during the latter part of the growing season. The stalk rot is less conspicuous from the exterior. In the split stalk, the longitudinal reddening of the internodal tissues interrupted by occasional white areas extending across the stalk tissue is the typical symptom. Stalk rotting is common in the cane after cutting, according to Edgerton and Carvajal (1944). Rotting of the young shoots on cuttings and reduction in stand is common during periods of cool weather. Edgerton *et al.* (1937) reported reduction in stands and yields ranging from negligible in resistant to severe in susceptible varieties. Sugar yields are lowered by the stalk rot because of the inversion of sucrose.

The Fungus.—*Physalospora tucumanensis* Speg.
(*Colletotrichum falcatum* Went.)

Mycelium in culture is white to dark gray with a cottony texture in the light type and dark gray with compact texture in the dark type. Thick-walled vegetative resting cells (chlamydospores) are more abundant in the latter type. The conidia are borne in stromata or singly in culture. On the midrib lesions, they are formed in poorly defined acervuli intermingled with dark setae. Conidia are one-celled, mostly falcate, 17 to 33 microns long (16–48 by 4–8 microns, range reported by Abbott 1938), and hyaline to pinkish in mass. Perithecia are submerged, scattered, and irregular in shape, 100 - 260 microns wide by 85 - 250 high, with a small portion of the ostiole protruding. Asci are clavate, thickened at the apex. Ascospores are irregularly biseriate, single celled, straight or fusoid, elliptical to ovate at maturity, and usually measure 18–22 by 7–8 microns. Paraphyses are abundant, septate, delicate, and extend to the ostiole.

The present evidence indicates two species common on the Gramineae: *Physalospora tucumanensis* (*Colletotrichum falcatum*) on the sugarcane and *C. graminicolum* on the small grains, Sudan grass, and many other grasses (see Anthracnose on rye, Chap. VIII).

Control.—Resistant varieties reduce the loss from red rot. Soil preparation and fertilization assist in producing a strong young plant growth, especially during the cooler season. Edgerton and associates (1942) described a hot-water treatment useful in controlling the infection in cuttings, but apparently predisposing to infection and rotting when seed canes were inoculated after treatment.

12. Helminthosporium Eye Spot and Brown Stripe, *Helminthosporium sacchari* (Breda de Haan) Butl. and *Cochliobolus stenospilus* (Carpenter) Matsu. and Yamamoto (*H. stenospilum* Drechs.).—The eye spot is widely distributed on sugarcane, lemon grass, *Cymbopogon citratus* (DC.) Stapf., and Napier grass, *Pennisetum purpureum* Schum. This is probably the common leaf spot of the group described on sugarcane. The brown stripe caused by *Helminthosporium stenospilum* Drechs. is

apparently less widely distributed and according to McRae (1933) is closely allied with the eye spot in both symptoms and morphology of the fungus, although the symptoms and the reporting of the ascigerous stage of the latter fungus by Carpenter differentiate the two fungi. Priode (1931) described a target blotch on sugarcane in Cuba differing somewhat in symptoms from the other leaf blights. Faris (1928), Lee and associates (1926), McRae (1933), Mitra (1930, 1931), Parris (1942), and others have reported on the symptomology of the leaf spots and the morphology of the fungi.

The initial spots of eye spot are yellow to brown. As the lesions spread, they become oblong to linear, reddish brown to red, and show gray centers when the fungus is sporulating. Streaks or runners extend from the spots toward the leaf tip. The lesions spread rapidly under favorable conditions, causing death of the leaf blade and sheath. Stem cankers occur in Napier grass. The brown stripe apparently differs in less zonation and more restricted widening of the lesions and the absence of streaks or runners.

The Fungi.—*Helminthosporium sacchari* (Breda de Haan) Butl.
(*Cercospora sacchari* Breda de Haan)
(*Helminthosporium ocellum* Faris)

The conidia are gray to brown, tapering toward the ends, straight or slightly curved, vary considerably in size, and germinate from the end cells. Specialization occurs within varieties and between species of the suscepts.

Cochliobolus stenospilus (Carpenter) Matsu. and Yamamoto
(*Helminthosporium stenospilum* Drechs.)

The fungus differs from *Helminthosporium sacchari* somewhat in conidial shape and is apparently restricted to sugarcane. The ascigerous stage with the coiled ascospores is similar to the other species of this genus.

Resistant varieties or hybrid selections offer the best means of control of this disease complex.

13. Smut, *Ustilago scitaminea* (Rabh.) Syd.—The sugarcane smut is present in the Pacific area, Asia, and South America. The smutted plants are stunted and develop a whip-like smut sorus from the apex of the cane. The elongate sorus consisting of tissues of the apical bud or the inflorescence and the black spore mass is the typical symptom. The slender canes of the *Saccharum sinense* or slender Asiatic type are the most susceptible. The disease causes severe damage to sugarcane in the Tucuman district of Argentina.

The Fungus.—*Ustilago scitaminea* Syd.
(*Ustilago sacchari* Rabh.)

The morphology of the fungus and the description of similar species on *Erianthus* sp. are given by Hirschhorn (1941). Chlamydospores are olivaceous to brown, globose or irregular, 4 to 9 microns in diameter, smooth or with fine papilla on the surface. Many hyaline or brown single or joined sterile cells are present with the spores.

The disease is controlled by the use of smut-resistant strains of sugar-cane.

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CHAPTER XI

WHEAT DISEASES

The cultivated wheats of the world comprise several species. However, only three species are of major importance commercially: *i.e.*, common wheat, *Triticum aestivum* L. or *T. vulgare* Vill.; club wheat, *T. compactum* Host., both hexaploids; and the durum wheat, *T. durum* Desf., a tetraploid. Varieties of the common wheat represent the major acreages in most countries. In the genus *Triticum*, as in the other small grains, the basic chromosome number is seven pairs. The genus represents a polyploid series with the important cultivated species in the 14 and 21 chromosome pair groups. The following table includes the *Triticum* spp. divided into the three chromosome groups.

Diploid Series, 7 Chromosome Pairs	Tetraploid Series, 14 Chromosome Pairs	Hexaploid Series, 21 Chromosome Pairs
T. aegilopoides Bal.	*T. dicoccoides* Korn.	*T. spelta* L.
T. monococcum L.	*T. dicoccum* Schubl.	*T. vulgare* Vill. or *T. aestivum* L.
T. thaoudar Reu.	*T. durum* Desf.	*T. compactum* Host.
	T. turgidum L.	*T. sphaerococcum* Perc.
	T. persicum Vav.	*T. macha* Decap.
	T. orientale Perc.	*T. vavilovi* Jackub.
	T. pyramidale Perc.	
	T. timopheevi Zhuk.	

The species highly resistant to the major wheat diseases are found in the diploid and tetraploid series. Vavilov (1914) attempted to show that the *Triticum monococcum* wheats were the most resistant to the rusts and powdery mildew, the *T. durum* groups somewhat less resistant, and the *T. vulgare* group most susceptible. The emmer group (*T. dicoccum*) is resistant to many races of the fungi causing leaf rust, stem rust, powdery mildew, and bunt. *T. timopheevi*, an emmer-like wheat, is resistant to races of these same fungi and apparently possesses a genom not present in other tetraploid wheats or as yet detected in the hexaploid species. Interspecific crosses are made to transfer these resistance factors to the *T. vulgare* wheats. The earlier literature was summarized by Clark (1936). Ausemus (1943) and Roemer *et al.* (1938) have discussed the resistance of wheat to the various important parasites.

During the early years, breeding for resistance to some of the major diseases was difficult. The common wheats were not resistant to a

sufficient number of physiological races, and only resistant durum and emmer parental material was available. The hybrids between these latter resistant wheats and the susceptible common wheats resulted in high sterility and linkage of undesirable characters. McFadden (1930), in the development of Hope and H-44, accomplished the transfer of many of the factors for disease resistance into a hard red spring wheat from the cross Yaroslav emmer (*T. dicoccum*) × Marquis (*T. vulgare*). Hope and H-44 are used widely in producing disease-resistant wheats. The cross *T. timopheevi* × *T. vulgare* is difficult to use, owing to high sterility and failure in transferring the disease-resistance factors to the common wheats. The direct cross has been made by several breeders, but elimination of undesirable characters and the maintenance of all the factors for disease resistance have not been accomplished to date (Shands, 1941). Kostoff (1936, 1937) reported the production of an amphidiploid (*T. timococcum*) with the major disease-resistance factors of *T. timopheevi* and *T. monococcum* and with 21 chromosome pairs. The cytology and genetics of interspecific hybrids in the genus *Triticum* and intergeneric hybrids with *Aegilops*, *Haynaldia*, *Secale*, and *Agropyron* spp. have been studied extensively (Aase, 1930, Kihara, 1937, Percival, 1921, Sando, 1935, Thompson, 1934, and Watkins, 1930).

The wheats are grouped into three classes based on the character of growth: winter, intermediate, and spring. The winter and spring varieties comprise the major economic groups (Clark and Bayles, 1935). The winter wheats, while more hardy than the other cereals excepting rye, are limited in their tolerance of winter conditions. The winter wheats are grown in North America south of approximately 45° latitude. Spring-wheat culture starts about 42° latitude and extends northward to the northern cultivated areas of Canada. The intermediate types occur in localized areas in the Southern United States and Mexico. World distribution is on a somewhat similar basis, although more intermediate types are grown in the wheat areas of the Southern Hemisphere. The wheats of the world are grouped commercially on the basis of their general usage.

The wheats are recognized in commerce under five general classes. The official wheat standards established in the United States and operating in world commerce under the "North American contract" are recognized as general wheat classes throughout most of the world. The five commercial classes in the United States are as follows: (1) hard red spring wheat grown in the North Central States and Canada; (2) durum wheat grown in limited areas in the drier sections of the same area as hard red spring wheat; (3) hard red winter wheat grown principally in the South Central states; (4) soft red winter wheats grown in the more humid Eastern states; and (5) white wheats, chiefly intermediate types, grown

in the Western states. The distribution of these general commercial classes throughout the world falls into similar ecological areas, but it is modified considerably by commercial demands, as, for example, the extensive durum wheat area in the Mediterranian region, the white wheats in Chile and Peru, etc.

Wheat is grown under a wide range of environmental conditions, principally for grain. The crop extends into the drier agricultural areas of the world as well as the humid sections (Carlton, 1920, Klages, 1942). Winter hardiness, drought resistance, and quality in addition to disease resistance are important in the breeding of wheats.

Diseases cause large losses in yield and quality. In the United States and Canada, reductions in yield and quality from stem rust, especially in 1904 and 1916, and again in 1935 and 1937, brought a demand for resistant varieties. The loss in North Dakota alone in 1935 was estimated at 100 million dollars. The aggregate loss in the United States and Canada caused by these stem rust epiphytotics was in the millions of dollars. Estimated average annual losses in the United States due to all wheat diseases for the 10-year period, 1930 to 1939, amounted to 10.5 per cent of the crop, or over 80 million bushels annually (Plant Disease Survey). Disease-resistant varieties are reducing these losses, stabilizing annual production, and materially reducing the cost of production.

1. Frost Injury and Winter Killing.—Frost injury occurs in both winter and spring wheat. The damage usually is localized, although occasionally seasonal conditions develop that cause damage over extensive areas. The damage occurs more frequently in the northern sections of the wheat belts or at high altitudes, and it is usually during the spring growing period and again before the wheat is mature. Leaf, crown, and young crown roots are injured from spring frosts. The entire leaf blade, or bands of tissue are frosted, resulting in loss of chlorophyll and necrosis. The frosted crown buds and young roots are first water-soaked, and later they show necrosis and browning. Frequently these frosted areas furnish avenues of entrance for parasitic fungi, especially of the crown-rot type, such as *Helminthosporium sativum* Pamm., King, and Bakke. Frost injury during the late summer and autumn is manifest by the shriveling and the green color of the pericarp. This type of injury affects not only the yield of grain, but also the quality of the crop. Fungi, especially *Alternaria* and *Penicillium* spp., invade the frosted tissues to cause further damage. Frosted grain is graded as damaged, and it is discounted on the markets. Frosted kernels are lower in germination and seedling vigor. Geddes *et al.* (1932), Johnson and Whitcomb (1927), Newton and McCalla (1934, 1935), and others have discussed the types of damage and the effect on composition and baking quality.

Winter killing consists of two kinds of injury, each associated with different environmental complexes. (1) The freezing and desiccation of the seedling tissues during periods of low temperature and low relative humidity in the absence of snow covering is the more common type of winter-wheat injury. (2) The depletion of reserves by high physiological activity of the dormant plant tissues and the invasion of the weakened tissues by fungi occur in periods of wet, cloudy warm weather or under heavy snow covering when the temperature remains above freezing for long periods. Wheat varieties respond differently to these two types of winter conditions, as shown by Akerman (1927), Salmon (1933), Tumanov (1931), and others. Winter wheats developed for winter survival under conditions of very low temperatures, dry air, and limited snow covering are generally very susceptible to winter injury of the second type when the temperatures are around freezing and the snow covering is heavy for long periods. The relation of storage reserves, bound water, and other physiological and chemical factors to cell killing, tissue rupturing, and tissue desiccation are discussed by Harvey (1935), Levitt (1941), Maximov (1929), Newton and associates (1924, 1931), and others.

Winter killing is due to a complex of factors that vary in different regions. The prevention of winter killing is associated with proper soil conditions and adapted varieties. The development of winter-resistant varieties is the best means of controlling this loss, as discussed by Clark (1936), Quisenberry (1931), Quisenberry and Clark (1929), Salmon (1933), and others.

2. Gray Speck, Nonparasitic.—The gray speck or yellow-gray leaf spotting of wheat due to soil mineral deficiencies in alkaline organic soils is common in both winter and spring wheats. The disease is manifest frequently on wheat and oats in the United States, although it is of minor importance. The symptoms and control are discussed in Chap. VI.

A leaf necrosis with similar symptoms was described by Straib (1935) as due to gene mutations in the Kolben wheat lines and in a number of the tetraploid wheats. Environment influenced the expression and severity of the necrosis.

3. Mosaics, Infectious Viruses, Soil-borne, Vector Uncertain.—The wheat mosaic is reported in the United States, Egypt, Russia, and Japan. Apparently it is scattered widely in limited areas and is associated with certain soil types and wheat varieties. The disease is of minor importance in the United States due to the high resistance of most important commercial varieties. Certain varieties of the other cereal crops and related grasses are mildy susceptible. Zazhurilo and Sitnikova (1939, 1940) reported the yellow mosaic as severe in southern Russian wheat fields.

Description.—The yellow and green types of mosaic are described by

McKinney (1937) and Wada and Fukano (1937). The former author listed seven mosaics on wheat based on symptoms on different varieties. The symptoms in both the yellow and green mosaics vary greatly with varieties. The yellow mosaic is characterized by the light yellowish-green mottling and striping, with dwarfing and excessive tillering in some varieties. The green mosaic is evidenced by the dark bluish-green color, with less distinct white mottling and striping and rosetting in some varieties. Intracellular bodies are present in the cells of the mosaic tissues (McKinney *et al.*, 1923, and Wada and Fukano, 1934, 1937). The latter authors described a difference in the intracellular bodies in the two mosaics. These mosaics are transmissable.

The method of transmission of the viruses in nature is uncertain. McKinney (1923, 1930) and Webb (1927, 1928) have shown transmission through the soil. Soils from mosaic-infested areas are capable of inducing the diseases even when enclosed in insect-proof cages. Soil, treated with heat or chemical disinfectants, does not induce the disease. Soil environmental conditions are important in the expression of the disease. The mosaics are transmitted by juices from diseased plants introduced mechanically into the crown and root tissues. Sukhov and Petlyuk (1940) and Zazhurilo and Sitnikova (1939, 1940) reported transmission of the oat mosaic by *Delphacodes straitellus* (Fall.) and the wheat mosaic by *Deltocephalus striatus* (Linn.). The latter leaf hopper is very similar to the vector of the rice mosaic, and it was reported by the latter authors as capable of transmitting the wheat mosaic to rice among other grasses. No insect vector has been associated with the diseases in the United States.

Control of the wheat mosaics in the United States is accomplished by the use of resistant wheat varieties, according to McKinney *et al.* (1925) and Webb *et al.* (1923). The diseases develop aggressively only on certain soil types, especially those high in organic matter and in areas of moderate winters.

4. Black Chaff, *Xanthomonas translucens* f. sp. *undulosa* (E. F. Sm., L. R. Jones, and Reddy) Hagb.—The disease is distributed widely on wheat. Damage is usually very light, as the disease rarely occurs in epiphytotics over extensive areas. Black chaff is frequently a complex of diseases when it occurs in association with pseudo-black chaff and brown necrosis on Hope and H-44 and derivatives from hybrids with these wheats. Hart and Zaleski (1935), McFadden (1939), and others have shown that the brown necrosis or melanistic reaction of these latter wheats and hybrid selections from these wheats carrying the mature plant factor for stem rust resistance is in some instances a type of reaction to stem rust. Melanism is induced in high temperature and humidity in the Hope and H-44 wheats, as shown by Broadfoot and Robertson (1933),

Hagborg (1936), and Johnson and Hagborg (1944). Apparently this type of necrosis as well as black chaff was considered by Goulden and Neatby (1929), Hayes *et al.* (1934), and perhaps some of the reports from other countries as the Hope and H-44 wheats are used widely in breeding for resistance to stem rust.

The bacterial black chaff occurs on the floral bracts, culms, and leaves of wheat, rye, and some grasses (Hagborg, 1942, Wallin, 1946). On the lemma, including the awn, the lesions appear as small, linear to striated, brown to black spots frequently coalescing to blacken the lemma. On the rachis and culm the lesions are longer and striated after they coalesce. The leaf lesions are light brown, translucent in the centers, irregularly linear, and frequently coalesce to form blotches. The young lesions on all tissues are water-soaked, and as they mature, pigmentation occurs and droplets or scales of exudate appear on the surface of the lesions, as described by Smith (1917), Smith *et al.* (1939), and Wallin (1946).

The Bacterium.—*Xanthomonas translucens* f. sp. *undulosa* (E. F. Sm., L. R. Jones, and Reddy) Hagb.
[*Phytomonas translucens* var. *undulosa* (E. F. Sm., L. R. Jones, and Reddy) Hagb.]
(*Bacterium translucens* var. *undulosum* E. F. Sm., L. R. Jones, and Reddy)

The short rods usually with one polar flagellum are similar in morphology to the species described on barley. The physiology of the variety differs somewhat and the variety shows specialization on wheat.

The etiology and control are similar to the bacterial blight of barley (Chap. III).

5. Basal Glume or Spikelet Rot, *Pseudomonas atrofaciens* (McCull.) Stapp.—The disease occurs in most of the wheat-growing sections of the world, especially where moisture is prevalent during the heading of the crop. The disease is of minor importance, as reported by McCulloch (1920) and Noble (1933). The light-brown bacterial rot at the base of the spikelet is not conspicuous unless examined closely. When the disease is severe, the rot extends into the rachis and into the base of the kernels. The lesions appear depressed and the tissues partly eroded with an inconspicuous gray bacterial exudate present in the sunken areas. These symptoms are distinctly different from those of "black point" caused by *Helminthosporium sativum* and other fungi, although the latter frequently occur in association with the bacteria to produce atypical symptoms.

The Bacterium.—*Pseudomonas atrofaciens* (McCull.) Stapp
[*Phytomonas atrofaciens* (McCull.) Bergey *et al.*]
(*Bacterium atrofaciens* McCull.)

The colonies are white in culture and consist of short cylindrical rods with one to four polar flagella.

The etiology and control of the disease is similar to that of the bacterial blight of barley (Chap. III).

6. Pythium Root Rot, *Pythium* Spp.—The Pythium root rots are distributed throughout the world on wheat, the other cereals, and grasses and especially on the fine prairie and loess soil types of North America. Under moist soil conditions and continuous cereal and grass culture, these browning root rots cause considerable damage (Ho and Melhus, 1941, Sprague, 1944, Subramanian, 1928, Vanterpool and associates, 1930, 1932, 1935, 1938, 1940, 1942). The characteristic symptom of the disease is the light-brown soft rot of the rootlets and roots and the pale-green stunted growth of the tillers. The sporangia and oöspores are abundant in the freshly rotted roots. A severe development of the disease results in a soft rot of the leaf sheaths and cortical tissues of the crown below the soil surface and the browning of the leaves.

The Fungi.—A number of morphologically similar species of *Pythium* occur as parasites on the Gramineae. The more prevalent are the following:

Pythium arrhenomanes Drechs.
Pythium tardicrescens Vanterpool
Pythium graminicolum Subr.
Pythium aristosporium Vanterpool
Pythium volutum Vanterpool and Truscott

These closely related species are differentiated by Drechsler (1936), Middleton (1943), and Vanterpool (1938) by minor differences in morphology. The general morphology is given in Chap. IV.

Etiology.—These *Pythium* spp. develop in the soil in association with roots and crop residues of the cereals and grasses. The aggressive parasitism of these fungi apparently is conditioned in part by their predominance in the soil microflora, or conversely by the absence of microorganisms that function dominantly in utilizing the nutritive constituents in the crop residues or that produce antibiotic substances inhibiting the development of the *Pythium* spp. The physiological condition of the cereal and grass plants especially as it affects root development and composition is important in determining both root invasion and the extent of root regeneration. The development of root rot is increased by a number of environmental factors, such as tightly compacted fine soils, high nitrogen in relation to phosphate, continuous cropping of cereals, or wheat and summer fallow. Garrett (1944), Simmonds (1941), and others have reviewed the literature and discussed the physiology and pathology of this type of parasitism. Further investigations are necessary on the etiology of this group of fungi and their control.

Control.—The methods of control of the Pythium root rot are inadequate at present. Wheat varieties show differences in reaction to the disease, but resistance under conditions favorable for the disease has not been demonstrated. Legumes in the crop-rotation system help reduce the damage. Balanced soil fertility is important in the control of the disease.

7. Downy Mildew, *Sclerospora macrospora* Sacc.—This downy mildew occurs in occasional locally restricted outbreaks on the cereals and grasses throughout the world. While it exists as a potential menace, especially to wheat and oats, the restricted and sporadic development of the disease in the Southern, Central, and Eastern United States probably indicates a continued minor relationship in cereal and grass production. The destructive potentialities of the disease under favorable environmental conditions are sufficiently great, however, to warrant careful watch for its occurrence and spread.

Symptoms.—The infected plants are erect, yellowish green, somewhat dwarfed, and they tiller excessively. The leaves are thickened, remain erect, and develop in a close whorl around the culm due to reduced internodal elongation and the stiff thickened conditions of the leaf blade. Many of the infected tillers turn brown and die. Disintegration of the parenchymatous tissue and shredding of the leaves is uncommon. The large brown oöspores held rather permanently in the mesophyll tissue between the veins of the leaf blade and sheath constitute the important diagnostic symptom.

The Fungus.—*Sclerospora macrospora* Sacc.

The conidial stage of the fungus is rare. The oöspores are imbedded firmly in the mesophyll and parenchyma of leaf and culm. The oöspores are light yellow, large, about 60 microns in diameter, globose, and smooth-walled. Peglion (1930) described the germination of the oöspores by the formation of a large papillate conidium or sporangium containing zoospores. The free oöspores as well as those imbedded in the leaf tissues apparently germinate in the same manner.

Etiology.—The oöspores remain viable for long periods within the dead tissue, and they are capable of causing infection under favorable conditions. The localized sporadic occurrence of the disease and the type of oöspore germination indicate the high moisture requirements of the fungus in relation to infection. The differences between the general distribution of *Sclerospora graminicola* in which the oöspores form a germ tube and the limited occurrence of *S. macrospora* in which the oöspores produce motile zoospores is explained in part by this difference in germination and the requirement of abundant free moisture for zoospore distribution and infection in the latter parasite. Good surface drainage, soil preparation, and crop rotation are the obvious means of control.

Temporary elimination of susceptible crops, wheat, oats, and grasses, in locally infested areas is recommended.

8. Powdery Mildew, *Erysiphe graminis tritici* El. Marchal.—Wheat and barley are the chief cereals damaged appreciably by powdery mildew. See Chap. III for the complete discussion. The disease is prevalent on winter and spring wheats during periods of cool, cloudy weather. Heavy mildew infection, especially during the period of tillering and internodal elongation of the plant, reduces the size of kernels and yield of grain. Allen and Goddard (1938), Allen (1942), and the Trelcases (1929) have studied the influence of powdery mildew on the metabolism of the wheat plant. Apparently a low ratio of carbohydrate supply to nitrogenous compounds in the wheat tissues increases mildew development and damage. The respiration activity of infected tissues is increased, which further depletes the total available carbohydrates in the plant, although there is a localized accumulation of carbohydrates in the infected cells. Root development and size of grain are reduced, owing largely to the constant deficit of available carbohydrates. Cherewick (1944) has shown that the effect of light and temperature upon the physiological condition of the wheat plant influences the initial stages of fungus infection more than disease development after infection.

Physiologic specialization of the parasite is developed highly on the cereals and grasses (Graf-Marin, 1934, Mains, 1933, Marchal, 1902 and 1903, and Vallega and Cenoz, 1941). Two physiologic races of *Erysiphe graminis tritici* occur commonly on wheat in the United States and one race in Canada. Norka (C.I. 4377) and Axminister (C.I. 1839) are resistant to race 1 and susceptible to race 2. Vallega and Cenoz (1941) described three races in Argentina differing from those in the United States. Chul (C.I. 2277) is susceptible to two, and Dixon (C.I. 6295) is susceptible to all three races. Schlichtling (1939) listed six races in Germany. Chul (C.I. 2277), Dixon (C.I. 6295), Hope (C.I. 8178), Huron (C.I. 3315), Sonora (C.I. 4293) are resistant to the two races in the United States. The newer soft red winter-wheat varieties produced in the United States are mostly resistant to intermediate in reaction to powdery mildew. The recent spring-wheat varieties are largely resistant to intermediate, although Thatcher (C.I. 10003), Mida (C.I. 12008), Rival (C.I. 11708), and Henry (C.I. 12265) are susceptible.

Varieties of *Triticum monococcum* and *T. timopheevi* are resistant to the races occurring in the United States and Argentina. Mains (1934) has shown that at least two independent factor pairs are associated with powdery mildew resistance in wheat. The disease is discussed in detail in Chap. III.

9. Ergot, *Claviceps purpurea* (Fr.) Tul.—Light infections are common in wheat in most humid and semihumid sections. Some varieties of

Triticum vulgare and a larger number of *T. durum* are susceptible, and occasionally these are damaged by ergot, especially in the durum wheat areas of North America. The detailed discussion of the disease is given in Chap. VIII.

10. Gibberella and Fusarium Blight or Scab, *Gibberella* and *Fusarium* Spp.—The disease occurs on the cereals and grasses in the temperate humid and semihumid areas of the world. Fusarium head blight causes severe damage to wheat, especially where temperature and relative humidity are high during the heading and blossoming periods. Stand, yield, and quality are affected by the disease. The scabbed wheat kernels are removed by modern cleaning equipment; therefore, the diseased kernels do not occur in large quantities in commercially milled wheat products. The scabbed kernels separated out are used chiefly for poultry feeds. The growth of the *Gibberella* and *Fusarium* spp. in the developing kernel results in the formation of compounds that act as strong emetics in man, pigs, dogs, and animals with similar digestive systems. The exact chemical nature of the substances is not known, although the specific biological action on the nerve center controlling the stomach muscles is ascertained. The physiological effects are manifest by violent nausea, dizziness, and temporary irritation and soreness of the stomach and intestinal membranes. Continued intake results in loss of appetite and general digestive disturbances. Pigs and dogs fed 10 per cent or more of badly scabbed grain vomit and then refuse the grain mixture. In Russia where the infected grain was used extensively for bread, the reaction was known as "intoxicating bread" (Agronomoff, 1934, Gabrilovitch, 1906, Naumov, 1916, Shapovalov, 1917, and others). Cattle, sheep, and poultry, with the exception of the pigeon, do not react to the infected grain.

Symptoms and Effect.—The disease occurs as a seedling blight, foot rot, and head blight. The blighted seedlings are characterized by a light-brown to reddish-brown water-soaked cortical rot and blight either before or after emergence (Fig. 55). The crown- and culm-rot phase of the disease occurs as the plants approach maturity. The head blight is conspicuous before the spikes mature. The infected spikelets first appear water-soaked, followed by the loss of chlorophyll, and a final bleached straw color (Fig. 55). During warm, humid weather, conidial development is abundant and the infected spikelets show a pink or salmon-pink cast, especially at the base and in the crease of the kernel. The infection frequently spreads to adjacent spikelets or through the entire spike. Perithecia develop on the infected floral bracts in some varieties of wheat under conditions of continued warm, wet weather (Fig. 56). The infected kernels are more or less shriveled, with a scabby appearance due to the tufty mycelial outgrowths from the pericarp, and they range in

color from white, pink, to light brown, depending upon the time of infection and environmental conditions during disease development.

The Fungi.—Several species of *Gibberella* and *Fusarium* are associated with the disease. Only the more important are listed below:

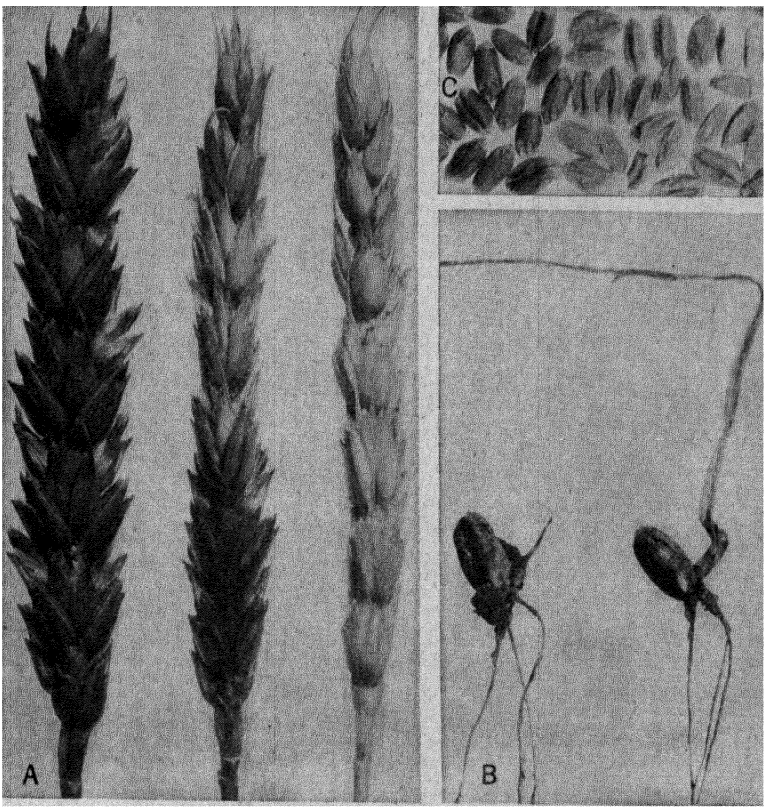

FIG. 55.—Head blight or scab of wheat caused by *Gibberella zeae* showing the blighted spikes (*A*), seedlings (*B*), and scabbed kernels (*C*).

1. *Gibberella zeae* (Schw.) Petch.
   Fusarium graminearum Schw. Conidial stage.
   [*Gibberella saubinetii* (Mont.) Sacc.]
   (*Pionnotes flavicans* Sacc. and Sacc.)
   (*Fusarium graminearum* Schw.)
   (*Fusarium roseum* Lk.)
   [*Fusarium bufonicola* (Speg.) Sacc. and Trott.]
   (*Fusarium rostratum* App. and Wr.)
   (*Fusarium discolor* var. *majus* Wr.)

(*Fusarium funicolum* Tassi.)
[*Fusarium insidiosum* (Berk.) Sacc.]

FIG. 56.—Spikelets of wheat showing the initial contact infection from the anther tissue followed by mycelial and conidial development on the lemma and palea (*B*); perithecia on the same structures of Turkey winter wheat (*A*).

(*Sphaeria zea* Schw.)
[*Botryosphaeria saubinetii* (Mont.) Niessl.]

(*Botryosphaeria dispersa* Ntrs.)
[*Gibbera pulicaris* (Fr.) Sacc.]
(*Gibberella tritici* Henn.)
(*Gibbera saubinetii* Mont.)

The mycelium is white to pink within and on the tissues. The conidia are borne in sporodochia or pionnotes, sickle-shaped, gradually tapering toward the apex, not constricted appreciably toward the base, have thin hyaline walls and septations, are generally 5-septate, and measure 41–60 by 4.3–5.5 microns. Thickened vegetative resting spores (chlamydospores) are absent. The perithecia are scattered on the surface, somewhat imbedded in the mycelium, smooth at the base with protuberant projections near the apex, purplish black to dark blue, and ovoid to subconical varying in size and shape. Asci are numerous, cylindrical, tapering to the base and hyaline. Ascospores are regularly eight, borne in one or two irregular rows, fusiform, slightly curved, largely three-celled, and measure 20–30 by 3.4–5.0 microns.

2. *Fusarium culmorum* (W. G. Sm.) Sacc.
(*Fusisporium culmorum* W. G. Sm.)
(*Fusarium culmorum* var. *leteius* Sher.)
(*Fusarium culmorum* var. *majus* Wr.)
(*Fusarium heidelbergense* Sacc.)
(*Fusarium mucronatum* Faut.)
(*Fusarium neglectum* Jacz.)
(*Fusarium roseum* var. *rhei* Karst.)
(*Fusarium rubiginosum* App. and Wr.)
(*Fusoma ochraceum* Corda.)
(*Fusarium sambucinum* Fuckl.)
(*Fusarium schribauxii* Del.)
(*Fusoma tenue* Grove)
(*Fusarium versicolor* Sacc.)

The conidia are borne the same as in the previous species. The conidia are somewhat wider and longer than in the previous species, slightly constricted toward the base, 5-septate when mature, and measure 30–50 by 4.8–7.5 microns. Thick-walled vegetative resting spores (chlamydospores) are common. No ascigerous stage is known.

3. *Fusarium culmorum* var. *cereale* (Cke.) Wr.
(*Fusarium cereale* Cke.)
[*Fusarium cerealis* (Cke.) Sacc.]
(*Fusarium equiseti* var. *crassum* Wr.)

The variety differs from the species by longer conidia, frequently with seven to nine septations.

4. *Fusarium avenaceum* (Fr.) Sacc.

The synonymy of this species is lengthy and complicated, as reported by Wollenweber and Reinking (1935).

The conidia are borne similarly to the previous two species. The conidia are much narrower and more tapering toward the apex than in the previous species, usually 3- to 5-septate, and measure 45–66 by 3.1–4.1 microns. Vegetative resting spores (chlamydospores) are uncommon.

Snyder and Hansen (1945) combine these species and others under *Fusarium roseum* (Lk.) Snyder and Hansen and the forms pathogenic on the cereals under forma *cerealis*. Both long usage of the former binomials and the distinct differences in morphology of these several species of *Fusarium* and *Gibberella* argue against the acceptance of the species combination in this case.

Etiology.—*Gibberella zeae* (*G. saubinetii*) is the common species on wheat in the corn belt of North America, with the other species more prevalent northward into Canada. A similar distribution occurs in Europe. In Asia, *G. zeae* is common in the Pacific coastal area and the other species in the northern interior.

This group of fungi are associated closely with crop residues of the cereals. The mycelium develops abundantly on the residues following maturity of the crop until the tissues are disintegrated the following season. Conidial development is profuse when conditions are favorable. Perithecial development in the corn belt occurs sparingly in the autumn but is abundant on the corn and small-grain straw and stubble the following late spring and summer. In warmer climates, the development of perithecia is abundant during the autumn and early spring. Seedling infection is from seed-borne inoculum and from the crop residue. Crown tissues are invaded largely from the mycelium in the crop residue. Head blight occurs during moist, warm seasons from conidia and ascospores produced on the crop residues, especially when such refuse is on the soil surface. The initial invasions of the developing kernels frequently are from contact infection of the mycelium growing saprophytically on dehisced anthers (Fig. 56). The spores are carried considerable distances by wind and air currents. Secondary infection is from conidia and mycelium, as reported by Dickson (1941), Koehler *et al.* (1924), Pugh *et al.* (1933), and others. Warm, moist conditions are important in relation to perithecial development and in influencing seedling- and head-blight development (Bayles, 1936, Dickson, 1923, Garrett, 1944).

Control.—Crop rotation, sanitation, soil preparation, and seed treatment are important control measures. Covering the crop residue completely when plowing and treating the seed with the organic mercury compounds aid in the control of head blight and seedling blight, respectively. Wheat varieties vary greatly in susceptibility; however, no highly resistant wheats are known. Christensen *et al.* (1929), Scott (1927), and others have listed the scab reaction of spring and winter wheats.

11. Snow Mold, Foot Rot, and Head Blight, *Calonectria graminicola* (Berk. and Br.) Wr.—The disease occurs uncommonly in the Northern United States and Canada; it is common in northern Europe and Asia where it causes considerable damage. Winter wheat and rye and the

grasses are damaged in areas where the snow covering is heavy and the soil temperatures are mild.

Symptoms and Effects.—The fungus is conspicuous on the leaf and crown tissues of the winter cereals and grasses as the snow is melting in the spring. The white superficial mycelium is abundant under moist conditions. Penetration and killing of the leaf and bud tissues result in bleaching and drying out of these plant parts. Frequently local areas or spots show the killing with healthy plants adjoining. Conidia of the Fusarium stage of the fungus are present on the dead tissues. Several other fungi cause similar symptoms but are differentiated by types of mycelium, conidia, or sclerotia. The crown rot stage of the disease is inconspicuous and frequently associated with other fungi. The head blight also is less noticeable, as frequently individual kernels are infected without extensive blighting of the floral bracts. The kernels are shriveled and light brown in color.

The Fungi.—1. *Calonectria graminicola* (Berk. and Br.) Wr.
Fusarium nivale (Fr.) Ces. Conidial stage
(*Nectria graminicola* Berk. and Br.)
(*Calonectria nivalis* Schaf.)
[*Fusarium nivale* (Fr.) Ces.]
(*Lanosa nivalis* Fr.)
(*Fusarium hibernans* Lind.)
(*Fusarium minimum* Fuckl.)
(*Fusarium loliaceum* Duc.)
(*Fusarium miniatulum* Sacc.)
(*Fusarium miniatum* Prill. and Del.)
(*Fusarium oxysporum* Klot.)
(*Fusarium secalis* Jacz.)
[*Fusarium tritici* (Lieb.) Eriks.]
(*Fusarium ustiliginis* Rostr.)

The mycelium is abundant, white to gray in superficial mass. The conidia are borne in sporodochia or pionnotes, light-salmon color, thickly sickle-shaped, tapering at the apex, usually 3-septate, and average about 23 by 3 microns. Perithecia are scattered on the surface or within the mycelial mat, frequently associated with clumps of sterile mycelium, round to oval, nearly smooth, and dark red or reddish brown in color. Asci are numerous, cylindrical, tapering gradually to the base, and hyaline. The eight ascospores are borne in one or two irregular rows in the ascus. They are spindle shaped, 1- to 3-septate, and average about 15 by 3 microns. Vegetative resting spores and sclerotia are absent.

2. *Calonectria graminicola* var. *neglecta* Krampe.
(*Fusarium nivale* var. *majus* Wr.)

The conidia are larger and thicker than in the species, without the pronounced tapering at the apex. The asci are much shorter than in the species.

Snyder and Hansen (1945) have combined all species of Section Arachnites of the genus *Fusarium* under the binomial *Calonectria nivale* Fr. [*F. nivale* (Fr.) Ces.] and used the trinomial *C. nivale* f. *graminicola* (Berk. and Br.) Snyder and Hansen for the cereal pathogenes.

Under the same environmental conditions, other species of *Fusarium* frequently produce similar symptoms, as described by Wollenweber and Reinking (1935).

The etiology is similar to the other Fusarium diseases of the cereal crops. Mycelium, conidia, and perithecia develop on crop residues. These fungi are aggressive as parasites only on weakened plants or, more specifically, plants depleted in cellular reserves and inactive in vegetative development, a condition frequently associated with excessive respiration rates and low light intensity. The fungus develops parasitically under somewhat lower temperature conditions than the other *Fusarium* spp. occurring on the cereals, with the possible exception of *Fusarium culmorum*.

Control measures are similar to the other Fusarium diseases. Soil drainage is especially important. In turf grasses, the disease is held in check by the use of fungicides. Mercurous chloride is used extensively as well as the organic mercury compounds (Dahl, 1934, Sampson, 1931, Schaffnit and Meyer, 1930, and others).

12. Take-all, *Ophiobolus graminis* Sacc.—The disease is common on the cereals and grasses in rather specific areas throughout the world (Kirby, 1925). Take-all occurs more commonly in the drier winter-wheat sections of Southwestern and Northwestern North America. The greatest losses from the disease occur in the porous alkaline soils where winter-wheat culture is continuous or associated with the culture of grasses and with the breaking of the native grass sod. This is one of a complex of diseases attacking the roots, crown, and basal culm tissues of the cereals and grasses. The plant parts invaded, the geographical distribution, the environmental conditions under which the diseases develop, and the fungi concerned are somewhat different for each disease. The terms "take-all," "Pietin," and "Fusskrankheit" through common usage are associated more generally with the disease caused by *Ophiobolus graminis* and similar species of this genus, although as shown by Garrett (1944) this is not indicated too clearly in the literature.

Symptoms and Effects.—The symptoms vary greatly under different environmental conditions. Under relatively moist conditions, the conspicuous symptoms of take-all appear about the time wheat is heading. Localized areas occur in which growth is checked, the green color fades, and rapid bleaching of the leaves, culms, and heads follows. This sequence of symptoms is characteristic for the disease under very favorable conditions, although not entirely specific for take-all. The main roots,

crown, and basal culm tissues show a dry rot accompanied by a dark-brown to black surface mat of thick-walled coarse mycelium. This fungal

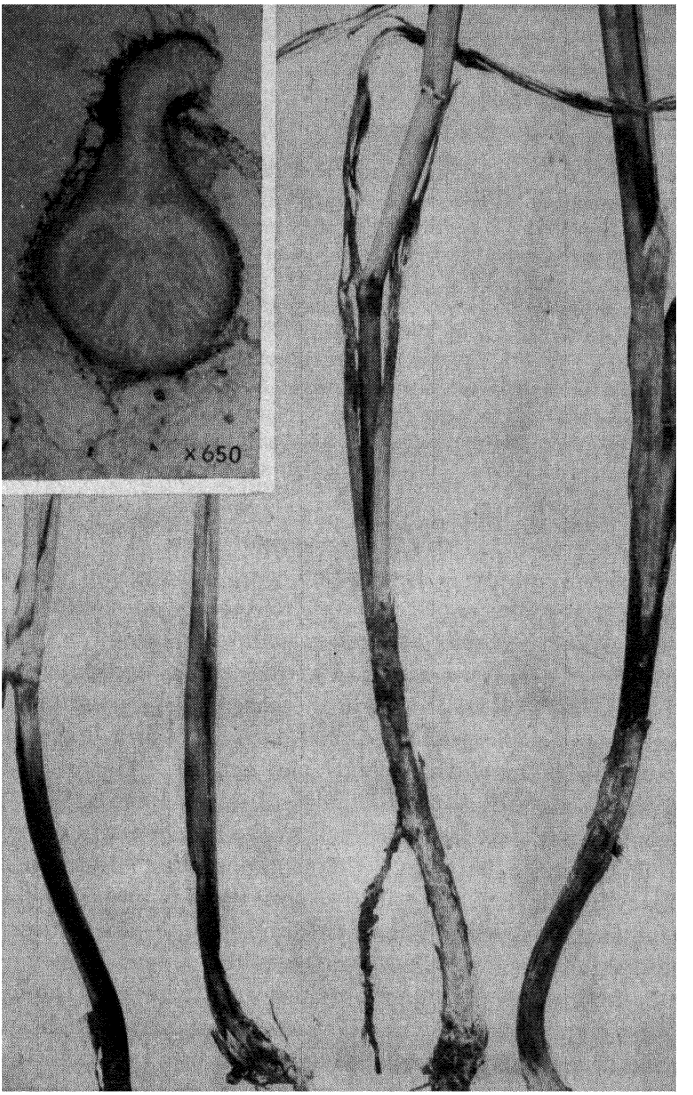

FIG. 57. The base of wheat culms showing the mycelial mat of *Ophiobolus graminis* beneath the leaf sheath. A section through the perithecium of *O. graminis*, highly magnified, is shown in the insert.

mat is conspicuous especially on the culm base under the leaf sheaths (Fig. 57).

Under drier conditions the symptoms are less conspicuous. The plant tillering is reduced, the plants are short, few plants show the dead bleached condition, and the mycelial mat is less pronounced. The presence of the fungus on the diseased tissues is the only sure criterion (Carne and Campbell, 1924, Fellows, 1928, 1938, Hynes, 1937, McKinney, 1925, Samuel, 1937).

The Fungus.—*Ophiobolus graminis* Sacc.
[*Ophiobolus cariceti* (Berk. and Br.) Sacc.]

The mycelium comprises a limited growth of fine hyphae, grayish in color, and an abundant development of coarse, thick-walled, brown to black, irregular hyphae. The perithecia are formed in or beneath the leaf sheath with strands of mycelium associated with the base and the cylindrical curved beaks extending through the sheath tissues. They are round to oblong, black, and about 400 microns in diameter (Fig. 57). The asci are numerous, elongate, clavate, straight or curved, and numerous thread-like paraphyses are present in the young perithecium. The mature asci are ejected from the perithecium during periods of abundant moisture. The ascus wall disintegrates in contact with free water to liberate the eight spores. The ascospores are hyaline, slender, tapering toward the ends, measure 3 by 70–80 microns, and are 5- to 7-septate when mature. The production of minute falcate conidia in the germination of the ascospores occurs under some conditions. *Ophiobolus graminis* var. *avenae* Turner is reported on oats.

Wojinowicia graminis (McAlp.) Sacc. and Sacc., weakly parasitic on wheat, occurs commonly in association with the take-all disease. The smooth-walled pycnidia of *W. graminis* frequently are confused with the perithecia of *Ophiobolus graminis* unless examined carefully. The conidia are long, slender, slightly curved, tapering at the ends, many septate, and are borne singly on short cells in the base of the pycnidium.

Etiology.—*Ophiobolus graminis* is soil-borne in rather direct association with straw and roots of the cereals and grasses. Garrett (1944) has reviewed the literature on the influence of environmental conditions on the development of the disease as well as the antagonistic phenomena of the microflora on the parasite. Undecomposed cereal straw and roots are necessary for survival and parasitic activity of *O. graminis*. Infection occurs from the active mycelium in the crop residue penetrating the root, crown, and culm tissues. The damage is dependent largely upon the presence or absence of soil microorganisms that suppress the activity of *O. graminis*; alkaline sandy soils, low phosphate and potash levels, and the absence of residues of crops other than wheat apparently suppress the soil microflora that inhibit the development and parasitic potentiality of *O. graminis*.

Control.—Crop rotations, involving legumes, other dicotyledonous crops, oats, or corn are important in reducing damage. A balanced fertility with a good supply of available phosphate and potash reduces losses. Wheat varieties show only small differences in susceptibility to

the disease that is associated apparently with adaptability of the varieties rather than resistance to the disease.

13. Foot Rot or Culm Rot, *Cercosporella herpotrichoides* Fron. and *Leptosphaeria herpotrichoides* DeNot.—The disease caused by these two fungi is reported in limited areas in northern Europe and the Northwestern United States and western Canada. The two fungi are found frequently under the same conditions, but Sprague (1934) and Sprague and Fellows (1934) have shown that, in the United States, Cercosporella is responsible for most of the damage rather than Leptosphaeria, as reported in the European literature. Other *Cercosporella* spp. cause leaf spots and scald on the grasses.

The disease is conspicuous near the end of the growing season by the lodging of the diseased plants. The lesions are evident first on the leaf sheath as elliptical to ovate spots with light straw-colored centers and brown margins. Similar spots occur on the culm beneath the lesions in the sheath. Necrosis also occurs around the roots in the upper crown nodes. Under moist conditions the lesions enlarge and a black stroma-like mycelium develops over the surface of the crown and base of the culms, giving the tissues a charred appearance. When infection occurs early, individual culms and weaker plants are killed before the grain is mature.

The Fungi.—1. *Cercosporella herpotrichoides* Fron.

The conidiophores on the lesions are simple or branched, short, erect, and originate from the stromal cells of the macrohyphae. The large ends of the obclavate conidia are attached terminally or subterminally. The conidia are curved slightly, chiefly 5- to 7-septate, and measure 30–80 by 1.5–3.5 microns. No perithecial stage is known.

2. *Leptosphaeria herpotrichoides* DeNot.

The perithecia are formed beneath the sheath and extend through the sheath tissues. They are subspherical with a smooth leathery surface and usually fine papillae around the ostiole. The asci are subcylindrical with eight spores. Ascospores are 6- to 8-septate, tapering toward both ends, and measure 25–40 by 4–6 microns. No conidial stage is known.

Etiology.—The organisms persist in the diseased tissues and other crop residue. The fungi become established in the fall-sown wheat, especially when sown early, and they develop whenever environmental conditions are favorable. Conidia of *Cercosporella* are abundant on the lesions in the spring and early summer, and they apparently are responsible as well as the mycelium for secondary spread.

Crop rotation, especially legumes, the use of spring grains, and delayed fall sowing aid in control of the disease. The wheats and grasses show differences in susceptibility, according to Sprague (1934, 1936).

14. Crown Rot and Root Rot, *Helminthosporium sativum* Pamm., King, and Bakke and Other Species.—The disease complex caused by this

group of fungi is distributed widely on wheat. Frequently, the *Helminthosporium* is associated with species of *Fusarium*, especially *F. culmorum*, further to complicate the relationship of this group of fungi to the root rot and foot rot of wheat. Henry (1924), Hynes (1935, 1937), Simmonds (1941), and Stakman (1920) have described the malady and reviewed the literature. *H. sativum*, under favorable conditions, causes severe damage to wheat, barley, and grasses, especially as the plants approach maturity. According to Sallans (1940), wheat recovers from the malady when growing conditions are unfavorable for the continued development of the disease. The disease complex on wheat was discussed by Bolley (1909, 1913) who associated the increase in prevalence and severity of the malady to continuous wheat culture. During the 35 years since this warning, the disease has been studied in most of the wheat-producing sections of the world. The kernel infection caused by *Helminthosporium* spp. and *Alternaria* spp. is severe on barley and the durum wheats, and certain of the stem rust resistant wheats are relatively susceptible, notably Apex (C.I. 11636) and Thatcher (C.I. 10003), according to Brentzel (1944) and Greaney and Wallace (1943).

The symptoms and development of the disease are similar on the several cereals. They are discussed specifically for *Helminthosporium sativum* in Chap. III.

The Fungi.—The species of *Helminthosporium* associated with the disease in wheat and barley apparently are diverse. Much confusion exists in the earlier literature regarding the species described as occurring on wheat, as reviewed by Drechsler (1923).

1. *Helminthosporium sativum* Pamm., King, and Bakke.

This predominant species is widely variable. The morphology is discussed in Chap. III. Dastur (1942) described *Cochliobolus tritici* Dast. on wheat culms, which likely is synonymous with *C. sativus* (Ito and Kuribay.) Drechs. although differing somewhat in morphology. The other large conidial types of the genus with dark conidial walls and germ tubes developing from the apical cells of the conidia that have been described on wheat probably differ sufficiently from *H. sativum* to be retained as species.

2. *Helminthosporium bicolor* Mitra.

The conidia are dark grayish brown, heavy walled with the two end cells light colored, typically cylindrical with abruptly rounded ends, although sometimes bilobed, straight, or slightly curved, 1- to 9-septate (average 5), measure 16.5–79 by 10–20 microns, and germinate from the apical cells (Mitra, 1930, 1930).

3. *Helminthosporium halodes* var. *tritici* Mitra.

Mitra (1930) based the variety on the difference in spore size (23–73 by 13–20 microns) and septation (2 to 9) from the species described by Drechsler (1923) on *Distichlis spicata* (L.) Greene, who discussed the similarity to *H. sativum*.

4. *Helminthosporium pedicellatum* Henry (1924).

The conidia are broadly fusiform, typically straight, widest near the middle, and tapering to both ends with a pedicel-like elongation at the base, olive-brown, 1- to 9-septate, but normally 7-septate, measure 31–91 by 14.7–29.4 microns, and germinate from the apical cells.

Some of the small-spored types transferred to the genus *Curvularia* by Boedijn (1933) occur on wheat and need further systematic study.

5. *Curvularia specifera* (Bainier) Boed. (*Helminthosporium tetramera* McK.)

The conidia are borne in clusters of varying numbers on irregular brown conidiophores. The conidia are oblong, symmetrical with rounded ends, light olivaceous to brown, predominantly 4-septate, and average 30 by 13.6 microns in size. Hynes (1937) reported smaller conidia (23–25 by 8.5–9 microns). Small dark-colored stipes remain attached to the conidia instead of the scar on the large-spored types. Germination is usually apical. Sclerotia are produced in culture; however, Hynes (1937) was unable to secure sclerotial production.

6. *Curvularia ramosa* (Bainier) Boed. [*Helminthosporium M* of Henry (1924)]

The conidia are borne as in the previous species. The conidia are irregularly oblong, curved to a greater extent on one side than on the other, dark brown. The center cells are usually darker than the apical cells, predominantly 4-septate, measure 12–36 by 8–16 microns, and usually germinate from the apical cells. Other species of this genus and small-spored Helminthosporium types are described by Sprague (1944).

7. *Helminthosporium tritici-vulgaris* Nishikado

This species of the thin-walled conidial type somewhat similar to *Pyrenophora tritici-repentis* Died. (*Helminthosporium tritici-repentis* Died.) has been reported causing a leaf spot on wheat in Japan by Nishikado (1929) and occurs in local areas in the United States.

The conidiophores develop singly or in pairs from the lesion surface and are simple with a swollen basal cell. Conidia are thin-walled, cylindrical, with rounded apical cells, generally straight or slightly curved, yellowish brown, 0 to 10-septate, measure 28.5–183 by 8.9–21.7 microns, and germinate from all cells.

The etiology and control of the diseases caused by these species is similar to that described for *Helminthosporium sativum* in Chap. III. The literature on the root rots is summarized by Garrett (1944) and Simmonds (1941).

15. Anthracnose, *Colletotrichum graminicolum* (Ces.) G. W. Wils.—The disease occurs on wheat in light soils of low fertility throughout the world. Wheat is damaged less than most of the other cereals. The disease is discussed in detail in Chap. VIII.

16. Septoria Leaf Blotch and Glume Blotch, *Septoria tritici* Rob. and *S. nodorum* Berk.—Two Septoria blotches occur on wheat throughout the world. The leaf blotch is generally the more important disease of the two as it is distributed over a wider area in both the hard red winter

and soft red winter wheat sections of the United States and occurs more consistently than glume blotch. Epiphytotics of the glume blotch, however, cause severe shriveling of the kernels in occasional seasons in the soft red winter wheat section. In Argentina especially, the diseases cause kernel shriveling and reduced yields in susceptible varieties.

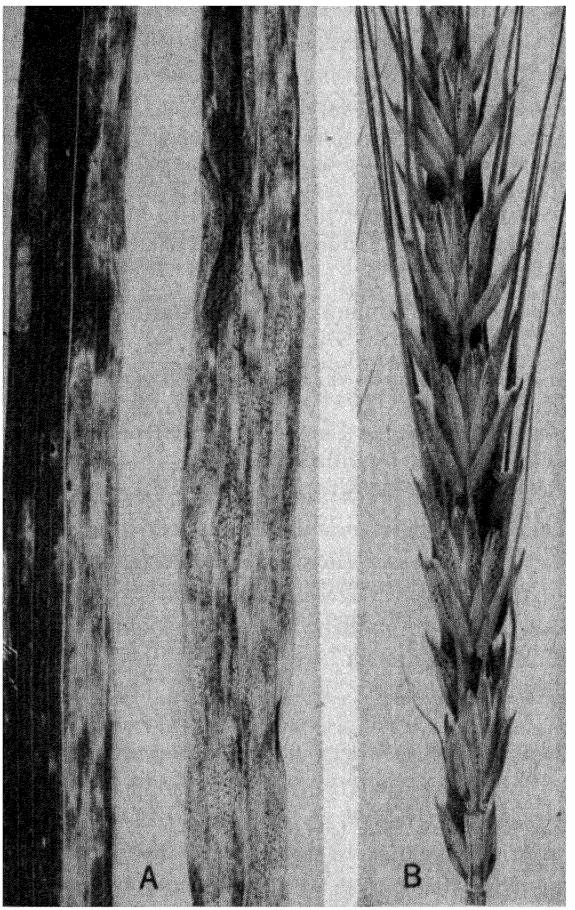

FIG. 58.—Leaf blotch (*A*) and glume blotch (*B*) infection of winter wheat caused by *Septoria tritici* and *S. nodorum* respectively.

According to Sprague (1934, 1938, 1944) and Weber (1922), physiologic races of these two species occur on some other cereals and grasses.

Symptoms and Effects.—The leaf blotch appears first as light-green to yellow spots between the veins of the leaves. The lesions spread rapidly to form light-brown irregular blotches with a speckled appearance as the pycnidia develop. The small submerged brown pycnidia in the

blotches are the final diagnostic symptom of the disease. Under favorable conditions, especially in the late autumn and early spring, defoliation and invasion of the crown tissues occur, resulting in weakened or dead plants (Fig. 58). Lesions on the culms, floral bracts, and pericarp of the kernels are less conspicuous and much smaller, with sparse pycnidial development.

The glume blotch caused by *Septeria nodorum* occurs more generally on the floral bracts and nodal tissues of the culm. The lesions are small, linear to oblong, light brown to dark brown in color, and the submerged pycnidia less conspicuous due to the darker color of the blotch than in the leaf blotch. Examination of the conidia is the only sure means of distinguishing between the two diseases.

The Fungi.—1. *Septoria tritici* Rob.
(*Septoria graminum* var. *tritici* Desm.)
(*Septoria cerealis* f. *tritici-vulgaris* Thuem)
(*Septoria tritici* Thuem)
(*Septoria triticina* Unam.)
(*Septoria tritici* Desm.)

According to Sprague (1944), Roberge, to whom Desmazieres credits the description, is the correct authority for the binomial. Two types of mycelium develop; a thin hyaline intercellular mycelium and a coarser olivaceous sterile superficial type. The pycnidia are subepidermal, usually in the stomatal cavity and subglobose, smooth walled, brown to black with slightly raised ostioles. Two types of spores are produced; macrospores, which predominate, and microspores. The macroconidia are slender, cylindrical with rounded ends, usually straight, hyaline, 3- to 7-septate, and vary considerably in size, averaging 50 by 2.2 microns for the spores produced in summer and slightly longer during the winter and spring. The microconidia are curved, aseptate, hyaline, and measure 5–9 by 1.0–1.3 microns. These occur in association with the macroconidia or alone and are not common.

2. *Septoria nodorum* Berk.
(*Septoria glumarum* Pass.)
(*Phoma hennebergii* Kühn)
(*Macrophoma hennebergii* Ber. and Vog.)

The mycelium is branched, irregular, hyaline at first to dark olivaceous when mature. Pycnidia are subepidermal, usually in the stomatal cavity, irregular in shape with the ostioles stomatal, brown to black, and larger than in the former species. Spores are oblong to cylindrical with rounded ends, hyaline, usually 3-septate, and measure on the average 26 by 3 microns. Perithecia of *Leptosphaeria* were described by Weber (1922) as associated with the fungus, although the relationship of this ascigerous stage has not been demonstrated.

Etiology.—Spores within the pycnidia and mycelium within the tissues persist for long periods of unfavorable conditions. The mycelium in infected kernels produces seedling infection, as reported by Machacek (1945). The spores are produced, germinate, and cause infection under a wide range of conditions. Infection by *Septoria tritici* is general in

the autumn on the leaves of winter wheat, and the mycelium remains active even at temperatures near freezing. Early spring spread of the disease is rather general. Infection in the spring wheat occurs from spores within pycnidia formed the previous season, spores produced on dead wheat refuse during the spring, and from infected seedlings. The etiology of the two species is similar. Evidence indicates that *S. nodorum* is less resistant to severe winter conditions and develops at higher temperatures than *S. tritici*. In North America, *S. nodorum* is prevalent in the more southern winter-wheat regions.

Control.—Crop rotation, sanitation, and plowing under volunteer wheat plants in the fall are important control measures. Mackie (1929) and others have reported many of the commercial wheat varieties of the United States as moderately resistant. Many of the important commercial varieties of Argentina are susceptible to *Septoria tritici*. Seed treatment with the organic mercury compounds kills the fungus borne in or on the grain.

17. Rhizoctonia Blight, *Rhizoctonia solani* Kuehn and Other Species.—The disease occurs on wheat, oats, and many grasses, and it is distributed widely throughout the world. While the malady is important in local areas, as reported by Goeffrey and Garrett (1932), Hynes (1937), and Subramaniam (1928), the disease is of minor importance on the wheat crop as a whole.

Symptoms.—The disease appears in patches in which the plants are stunted, and the leaves in many varieties show a purple cast. Plants are weakened and killed or more generally recover, in which case maturity is delayed and yield is low. The tan-colored cortical rot of the root system and tan zonate lesions on the basal leaf sheaths are the conspicuous symptoms. The characteristic mycelium of the fungus is present in the rotted tissues and root stubs near the crown

The Fungus.—*Rhizoctonia solani* Kuehn

The *Corticium* or *Pellicularia* stage is not common on the cereals and grasses.

The mycelium is white and brown intermixed. The young mycelium branches characteristically at acute angles with a constriction of the mycelium at the union and a septum in the mycelium at the constriction. The dark-gray to black irregularly shaped sclerotia are uncommon on the diseased roots, but they occur more frequently on the sheath tissues. They usually germinate to form mycelium. The Corticium stage consists of the club-shaped basidia with four apical sterigmata bearing sporidia that are hyaline and oval with tapering base. Rogers (1943) suggested the binomial *Pellicularia filamentosa* (Pat.) Rogers for the basidial stage of *Rhizoctonia solani*.

Peltier (1916) has described the fungus on numerous plants. Dickinson (1930), Monteith (1926), and others have described the disease and its control on the grasses used for golf greens. As in most other crops, the disease develops best at low temperatures.

The rotation of crops, as the graminicolous strains of the fungus are rather specific in parasitism, and balanced soil fertility are important control measures. The use of chemicals, such as mercurous chloride, is practical as a control measure on lawn and golf grasses.

18. Typhula Blight, *Typhula itoana* Imai and *T. idahoensis* Remsberg. —This disease develops under rather special environmental conditions and, therefore, is restricted in its occurrence. The blight occurs on the winter cereals and grasses during periods of heavy snow covering or cloudy weather with temperatures remaining slightly above freezing. In this respect the disease is similar to the snow mold caused by *Fusarium* spp., but apparently the two diseases are not found commonly in the same areas. The Typhula disease on wheat, especially, occurs in the intermountain valleys at relatively high altitudes. The disease is distributed more generally on the grasses and is world wide in occurrence.

Symptoms.—The Typhula blight is conspicuous as the snow disappears as a felty white mycelial mat over the plants and adjacent soil. The presence of numerous light- to dark-brown spherical sclerotia as the mycelium matures is the characteristic symptom. The plants overrun by the mycelium gradually lose the deep green color, wither, and turn brown. The disease occurs in spots varying in size and shape with less definite symptoms toward the margins of the areas. The killing of the plants ranges from dead leaf tissue to invasion and rotting of the culm, crown, and root tissues. The disease disappears as the temperature rises, moisture decreases, and sunlight increases.

The Fungi.—1. *Typhula itoana* Imai
(*Typhula graminum* Karst.)
(*Typhula elegantula* Karst.)
(*Sclerotium fulvum* Fr.)

The sclerotia are tawny to hazel brown, spherical to slightly flattened and superficial or imbedded in the diseased plant tissues. The sclerotia commonly germinate to form mycelium or thickened branching sterile structures with clamp connections apparent. The sclerotia, in soil or sand and in the presence of light high in ultraviolet rays, produce one to four thickened basidia, sometimes branched. The basidia are clavate, flesh colored, nonseptate with four apical sterigmata bearing sporidia. The sporidia are hyaline, ovate, slightly curved, and average 11.1 by 6.0 microns in size. This is probably the more common and widely distributed species on the cereals and grasses, according to Imai (1936), Remsberg (1940), Tasugi (1929, 1930, 1935), and Volk (1937).

2. *Typhula idahoensis* Remsberg

This species is similar in general morphology to the former. The sclerotia are chestnut brown, and the basidia are fawn to wood brown in color. This species on wheat is restricted apparently to the Intermountain areas of the United States and Canada, although it is distributed more widely on the grasses.

3. *Typhula graminum* Karst. is retained as a species by Remsberg (1940), but its pathogenicity on the cereals and grasses is questioned.

Cormack has found still another, at present unnamed, Basidiomycete developing at very low temperatures in Alberta, Canada. The mycelium develops parasitically on wheat among other crops at temperatures slightly above freezing.

Sclerotium rolfsii Sacc. occurs occasionally on wheat and other cereals and grasses in the warmer areas of the United States and other countries, according to Godfrey (1918), Tisdale (1921), and others. The small spherical brown sclerotia occur on the rotted culm, crown, and root tissues.

19. Loose Smut, *Ustilago tritici* (Pers.) Rostr.—The loose smut of wheat is distributed generally with the crop in the humid and semihumid wheat-producing areas. The disease is severe in the soft red winter and hard red spring wheat areas of Central and Eastern North America. The loose spore mass replaces the floral bracts and ovaries and is conspicuous from the heading to blossom period of the crop. The symptoms and etiology are similar to the loose smut of barley discussed in Chap. III.

The Fungus.— *Ustilago tritici* (Pers.) Rostr.
(*Uredo segetum* b. *tritici* Pers.)
(*Ustilago segetum* b. *tritici* Pers.)
(*Uredo carbo* b. *tritici* DC.)
(*Caeoma segetum* Link)
(*Erysibe vera* b. *tritici* Wallr.)
(*Ustilago carbo* a. *vulgaris* a. *triticea* Tul. and Tul.)
(*Ustilago segetum* var. *tritici* Jens.)
(*Ustilagidum tritici* Herzb.)
[*Ustilago tritici* (Jens.) Kell. and Swing.]

The morphology is the same as *Ustilago nuda*, causing the loose smut of barley (Chap. III). This is another case of where long usage of a binomial appears to warrant retaining it, although the two species, *U. tritici* and *U. nuda*, cannot be differentiated morphologically. Fischer (1943, 1945) and others have suggested the combination of the two as specialized races of the same parasite.

Control.—The loose smut infection within the kernel tissues is controlled by the modified hot-water treatment and more recently by the use of resistant varieties. The hot-water treatment is expensive and difficult to use except on a limited amount of foundation seed. The commercial varieties and selections show a wide range in loose smut infection. The club wheats and spelt are relatively susceptible. The durum wheats show more resistance than most of the common bread wheats. Some of the strains of *Triticum dicoccum* and *T. timopheevi* are highly resistant to the known physiological races. According to Roemer *et al.* (1938), Rudorf and Rosentiel (1934), Tapke (1929), Wingard and

Fromme (1941), and others, a number of varieties of the common wheats are highly resistant. Some of the more resistant spring wheats are Hope (C.I. 8178), a number of selections from Hope hybrids, and Preston (C.I. 3328). In the soft red winter wheats, Kawvale (C.I. 8180), Leap (C.I. 4823), and newer hybrid selections are the more resistant.

With the development of techniques for floral inoculation with loose smuts, more reliable data on varietal resistance and specialization of the parasites are available. Moore (1936) reported results using the partial vacuum method of inoculation. The introduction of the dry chlamydospores or the spores suspended in a dilute malt extract directly into the flowers by means of an hypodermic needle and rubber bulb from a dropper pipette also give reliable results. Tavcar (1934) and Tingey and Tolman (1934) reported morphological types of resistance associated with the closed flowers preventing entrance of the inoculum in the varieties Banat and Federation (C.I. 4734).

Physiologic specialization occurs in *Ustilago tritici*, as reported by Grevel (1930), Hanna (1937), and Radulescu (1935). Bever[1] using Forward (C.I. 6691), Hussar (C.I. 4843), Nabob (C.I. 8869), Purdue 1 (C.I. 11380), Trumbull (C.I. 5657), Wabash (C.I. 11384), Kanred-Gipsy selection, and Dawson (C.I. 3342) (American Banner) has shown a differential response for nine collections of the fungus.

The investigations of physiological races are not extensive enough to prepare a table showing the differential varieties for all classes of wheat. Stakman *et al.* (1935) have given a key based on Grevel's (1930) investigations.

20. Bunt or Stinking Smut, *Tilletia caries* (DC.) Tul. and *T. foetida* (Wallr.) Liro.—The stinking smut or bunt of wheat is associated closely with the historical development of plant pathology. It was among the first smuts to receive attention. The early literature on bunt was descriptive. Tillet in 1755 differentiated between la Carie (bunt) and le Charbon (loose smut) of wheat and established the infective principle of bunt dust; later, Tessier in 1783 and others considered it a degeneration of the grain. Perhaps the symptoms and fishy odor of this smut functioned unduly in the formulation of the concept that disease manifestations were morbid eruptions of vegetable matter: a concept that dominated the thinking of European mycologists for a time and reached its climax in Unger's book "Exantheme der Pflanzen" in 1833. The bunt disease also, during this same period, was the basis for the formulation of the experimental concept of parasitism in fungi by Prevost in 1807. The noted mycologists of the period, Bulliard, DeCandolle, Link, Tulas-

[1] Unpublished data supplied by Dr. W. M. Bever from cooperative investigations. Division of Cereal Crops and Diseases, B.P.I.S.A.E. U.S. Department of Agriculture and the department of agronomy, Illinois Agricultural Exper. Station.

nes, and Léveillé, were concerned with the disease and the two fungi. DeBarry in 1853 reconfirmed phases of the parasitic nature of the bunt fungus, and Kühn in 1858 summarized the information on practical smut control. The disease has received increasing attention through the years, especially since the detailed experiments of Brefeld in 1883.

The bunt of wheat is world wide in distribution. Whereas, loose smut occurs in the more humid wheat sections, bunt is more prevalent in the drier sections as well as occurring in the areas with high summer moistures.

The two species of *Tilletia* on wheat occasionally infect rye and *Agropyron cristatum* (L.) Gaertn. According to Fischer and Hirschhorn (1945), inoculation experiments have shown the susceptibility of *Aegilops ventricosa* Tausch, *Agropyron subsecundum* (Link) Hitchc., *A. trachycaulum* (Link) Malte, *Lolium perenne* L., *L. multiflorum* Lam., and *Hordeum nodosum* L. to one or both species. A distinct variety of *T. caries* that causes a dwarfing of the wheat plant occurs in North America (Rodenhiser and Holton, 1945). Several additional *Tilletia* spp. occur on the grasses, as discussed in Chap. XII. The bunt or stinking smut has been discussed in detail, including the important literature citations by Holton and Heald (1941).

The disease causes losses in yield, produces difficulties in threshing, and lowers quality. Prior to the general use of seed treatments and resistant varieties, bunt caused large reductions in yield of wheat. The presence of the spores in quantities caused explosions in separators and fire losses. The spores adhere to the threshed grain, and they are removed only by washing before the wheat is milled (Bates *et al.*, 1929). The Federal grain standards designate wheat that has an unmistakable odor of smut or that contains smut balls, portions of balls, or spores of smut in excess of a quantity equal to 14 balls of average size in 250 grams of wheat as "light smutty" and wheat with smut balls and spores in excess of 30 balls in 250 grams as "smutty". These descriptive terms are included with the regular grade designation. Smutty wheat is discounted, frequently more than the cost of cleaning the grain.

Symptoms —The symptoms of bunt usually are not apparent until the wheat is headed. However, some varieties of wheat infected with certain races of the parasites show dwarfing of the plants, small light-colored spots on the leaves, and a grayish cast of the foliage and culms during the period of tillering and internodal elongation (Angell, 1934, Churchward, 1934, Holton and Rodenhiser, 1942). According to Helyet *et al.* (1938), root development in smutted plants is reduced from shortly after heading to maturity. The smutted plants of many varieties appear bluish green to grayish green in color, and the heads frequently show characters unlike the healthy spikes of the variety (Fig. 59). The odor of trimethylamine is characteristically present from the period of spore

formation to maturity and in the threshed grain. The smut balls that replace the kernels frequently are conspicuous in the smutted spike. The

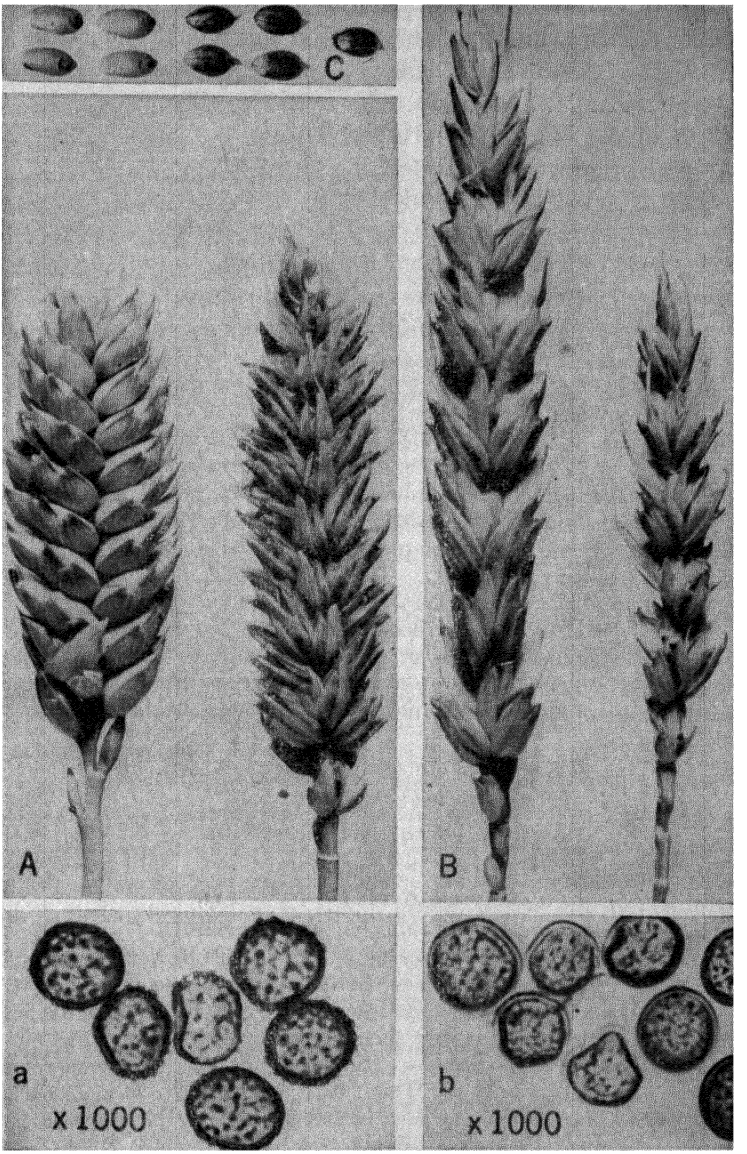

FIG. 59.—Bunt or stinking smut of wheat showing healthy (left) and infected (right) spikes of two wheat varieties (*A*) and (*B*) and the modification of the spikes by the smut. Healthy kernels and smut balls are shown (*C*). The chlamydospores of *Tilletia caries* (*a*) and *T. foetida* (*b*) are shown highly magnified in the insert.

smut balls, somewhat the shape of the wheat kernels, are grayish green changing to brown as the grain ripens. The presence of the smut balls

and the dark spores adhering to the kernels are evident in the threshed grain. The effect of bunt on the morphology and physiology of the wheat plant is reviewed by Holton and Heald (1941). In the dwarf bunt, the symptoms are evident by the reduced internodal elongation of the infected plants, and the spore balls are nearly round instead of the shape of the kernels.

The Fungi.—1. *Tilletia caries* (DC.) Tul.
(*Uredo caries* DC.)
(*Uredo sitophila* Dit.)
(*Lycoperdon tritici* Bjerk.)
[*Tilletia sitophila* (Dit.) Schr.]
(*Tilletia secalis* (Cda.) Kühn)
[*Tilletia tritici* (Bjerk.) Winter]

The latter synonym (*T. tritici*) was used extensively as the binomial for this fungus until recently (see the table p. 46, List of Common Names of the Diseases and Binomials of the Cereal Smut Fungi, Chap. III.)

The sori are formed in the ovaries, the pericarp of the caryopsis persists, and the sori are more or less the same shape as the kernel. The chlamydospores are brownish black, globose to subglobose, light to dark brown, reticulate with reticulations ranging from minute shallow meshes to deep indentations, and 15 to 23 microns in diameter (Fig. 59). The sterile cells are globose to subglobose, thin walled, hyaline, smooth to faintly reticulate, and 12 to 18 microns in diameter. The spores germinate to form a promycelium and an apical crown of long filiform sporidia, variable in number, usually 8 to 16, which fuse in pairs while still attached. The spores of the variety of this species causing dwarf bunt are difficult to germinate, and even when presoaked in water at low temperatures the germination percentages are low. The branched promycelium and irregular type of sporidial formation in this strain is not typical for the species, as discussed by Holton (1943).

2. *Tilletia foetida* (Wallr.) Liro
(*Erysibe foetida* Wallr.)
(*Ustilago foetens* Berk. and Curt.)
[*Tilletia foetens* (Berk. and Curt.) Schr.]
(*Tilletia levis* Kühn)

Tilletia levis was used extensively for this fungus until recently.

The sori are formed as in the previous species. The chlamydospores are globose to ovoid or elongate, light grayish-brown to olivaceous brown, smooth, usually 17 to 18 microns in diameter or 17-20 by 18-22 microns in size. Sterile cells are hyaline, thin-walled, smaller than the spores, mostly about 14 microns in diameter (Fig. 59). Germination is similar to the former species. Holton (1944) described the morphology of hybrids between these two species. Mitra (1931) reported *Neovossia indica* (Mitra) Mundkur (*Tilletia indica* Mitra) on wheat in India, with only a few kernels per spike infected. The chlamydospores are reticulate, larger than those of *T. caries* (average 35 microns in diameter), and germinate to produce a whorl of apical sporidia that do not fuse.

The geographic distribution of the two species differs somewhat. *Tilletia foetida*, the smooth-spored species, is more common in the North Central and Eastern North America than *T. caries*, while both species occur in the western areas, although the latter species is more prevalent

(Rodenhiser and Holton, 1945). Similar variations in distribution are reported from other countries, as reviewed by Churchward (1932).

Etiology.—The etiology of the two species is similar. Seedling infection occurs, resulting in a systemic invasion of the seedling primordium, and spores develop in the ovaries. The inoculum is from seed-borne chlamydospores or spores in the soil in the drier winter-wheat areas. Churchward (1940) has investigated penetration and establishment of the fungus in the wheat seedling. Penetration and the early phases of systemic infection apparently occur in the resistant wheats. Environmental conditions influence the infection and establishment of the parasite in the wheat seedlings, as reviewed by Holton and Heald (1941).

Control.—Seed treatment and the use of bunt-resistant varieties are the combined methods of control. The use of seed treatment even with bunt-resistant varieties is recommended to prevent the occurrence and propagation of specialized races of the parasites to which the resistant variety is susceptible. This recommendation is based on field experience with resistant varieties and on investigations of the occurrence of new physiologic races in nature or by hybridization. Seed treatment with copper carbonate dust or the mercury dusts, especially Ceresan, controls bunt under most conditions. The dwarf bunt is not amenable to control by these seed treatments, according to Bamberg (1941) and Fischer and Hirschhorn (1945). Many wheat varieties resistant to certain groups of the physiologic races of one or both species of *Tilletia* are known. Certain of these, such as Ridit (C.I. 6703), Oro (C.I. 8220), Hohenheimer (C.I. 11458), Hussar (C.I. 4843), Hope (C.I. 8178), and Florence (C.I. 4170), the latter two are spring wheats, are differentially resistant to all but a few known races. According to Rodenhiser and Holton (1945), the four selections of hard red winter wheat from Oro × Turkey-Florence, Rex × Oro, Rex × Rio, and Rio × Rex are more generally resistant to all races of the two *Tilletia* spp. In many areas where the physiologic races are limited, the disease is controlled by the use of varieties resistant to a limited number of races. In areas such as the Pacific Northwest, varieties combining resistance to a large number of races are essential for bunt control. The bunt-resistance varieties used and the inheritance of resistance or genetic analysis are summarized by Holton and Heald (1941) and discussed by Roemer *et al.* (1938). Briggs and coworkers (1933, 1940, 1945) have explained the intermediate percentages of bunt in homozygous resistant lines on the basis of multiple factors, with certain factors permitting specific amounts of bunt.

Specialization in the the two species is clearly defined and apparently quite stable, as reported by Holton and Rodenhiser (1942) and Rodenhiser and Holton (1945). Certain races produce specific morphological or physiological responses on particular wheat varieties. However, pathogenicity is the important criterion in differentiating the races and

applying the information to breeding for bunt resistance. The reaction of physiologic races of the two species to the differential wheat varieties, as summarized by the authors cited above, is given in the following table.

REACTION OF WHEAT VARIETIES TO PHYSIOLOGIC RACES OF *Tilletia caries* AND *T. foetida*

Physiologic race no.	Ridit (C.I. 6703)	Oro (C.I. 8220)	Hohenheimer (C.I. 11458)	Hussar (C.I. 4843)	Albit (C.I. 8275)	Martin (C.I. 4463)	White Odessa (C.I. 4655)	Ulka (C.I. 11478)	Marquis (C.I. 3641)	Canus (C.I. 11637)	Mindum (C.I. 5296)
T. caries											
T- 1	R	R	R	R	R	R	R	S	I	R	R
2	R	R	R	R	R	R	R	S	R	R	S
3	R	R	R	R	R	R	R	S	S	S	I
4	R	R	R	R	I	S	S	S	S	R	I
5	R	R	R	R	I	S	S	S	S	S	I
6	R	R	R	R	S	S	S	S	S	R	I
7	R	R	R	I	S	S	S	S	S	I	I
8	R	R	R	S	S	S	S	S	S	S	I
9	R	R	I	R	R	R	R	S	I	R	I
10	R	R	S	R	R	R	R	R	I	R	R
11	S	R	R	R	R	R	R	I	S	S	I
12	R	R	S	R	S	I	S	S	R	R	
13	S	R	R	S	S	R	S	-	I	I	
14	R	R	R	R	S	R	S	S	R	R	
15	R	R	S	S	S	S	S	S	S	S	
16	R	S	S	R	R	R	R	S	I	I	
T. foetida											
L- 1	R	R	R	R	R	R	R	S	I	R	I
2	R	R	R	R	R	R	R	S	S	R	I
3	R	R	R	R	R	R	R	S	S	S	I
4	R	R	R	R	S	S	S	S	I	R	I
5	R	R	R	R	S	S	S	S	S	S	I
6	R	R	R	I	S	S	S	S	S	S	I
7	R	R	R	I	S	S	S	S	S	S	I
8	R	S	R	R	R	R	R	S	S	S	I
9	S	R	R	I	S	R	S	S	I	S	
10	I	R	R	R	R	R	R	S	I	S	
11	R	R	R	R	R	R	R	S	I	I	
12	R	R	R	R	S	R	R	S	I	R	
13	R	R	S	R	R	R	R	S	S	R	
14	R	R	R	R	S	I	S	S	S	R	
15	R	R	R	S	S	S	S	S	S	I	

T-1 etc., race numbers for *T. caries*, L-1 etc., race numbers for *T. foetida*.
R—resistant (0 to 10 per cent infection); I—intermediate (11 to 40 per cent); and S— susceptible (41 to 100 per cent).

The race of *Tilletia caries* causing dwarf bunt is not included in the table.

21. Flag Smut, *Urocystis tritici* Koern.—The disease is distributed widely thorughout the world on wheat, but apparently it is of major importance only in a limited number of areas. Flag smut on wheat was found first in the United States in 1919 (Humphrey and Johnson, 1919), and it has appeared since in scattered localized areas in the South Central winter-wheat region and in the Pacific Northwest. The disease was eliminated partly in these local areas by a shift in crops and by the use of resistant varieties of wheat in the adjacent territory. Flag smut is of major importance only in the winter-wheat sections of mild winter climates and with the general use of susceptible varieties and continuous wheat culture. According to Miller and Millikan (1934), the flag smut of wheat in South Australia in 1931 was damaging 3 to 4 per cent of the crop, owing to the general use of susceptible varieties and continuous wheat culture. The smut damage started decreasing with the wider use of the resistant variety Nabawa. Noble (1937) reported for New South Wales as follows: "Flag smut has ceased to be the serious disease it was some years ago before resistant varieties were grown . . . ."

Symptoms and Effects.—The disease is evident from the late seedling stage until maturity of the crop. The early symptoms of the smut are the gray to grayish-black linear sori in the older leaf blades and leaf sheaths (Fig. 60). The sori form in the mesophyl tissue between the veins, and they are covered by the epidermis of the leaf during the earlier stages of development. Later the epidermis ruptures, releasing the black spore mass, and finally the leaf tissue freys along the linear sori. The symptoms appear on the leaves as they unfold and in the culm tissues as the culms elongate. In most susceptible varieties, the plants are dwarfed by reduced internodal elongation. Usually spike development is stopped prior to its emergence from the leaf whorl. The disease in wheat is characteristically a leaf smut in contrast to the stalk smut produced by the species on rye.

The Fungus.— *Urocystis tritici* Koern.

[*Urocystis occulta* (Wallr.) Rab.]

Fischer (1943) suggested the combination of the morphologically similar species *Urocystis tritici*, *U. occulta*, and *U. agropyri* (Preuss.) Schroet. on wheat, rye, and grasses, respectively, under *U. agropyri*, recognizing the specialized varieties of the fungus on the different cereals and grasses. Long usage argues against this change as in the case of the generic name, Urocystis (Zundel *et al.*, 1940).

The sori are linear, black, at first covered by the epidermis, chiefly in the leaves and upper culm tissues and frequently stunting plant development and aborting the spike (Fig. 60). The sporeballs are globose to oblong, measure 18–35 by 35–40 microns, are composed of one to four dark chlamydospores, and are hyaline to brown with somewhat smaller sterile cells often incompletely surrounding the fertile cells. The chlamydospores are angular to globose, dark reddish brown, smooth, and 14 to 20 microns in

diameter. The spores germinate in place to form a short promycelium, with or without septations, and three to four hyaline cylindrical sporidia are borne near the apex.

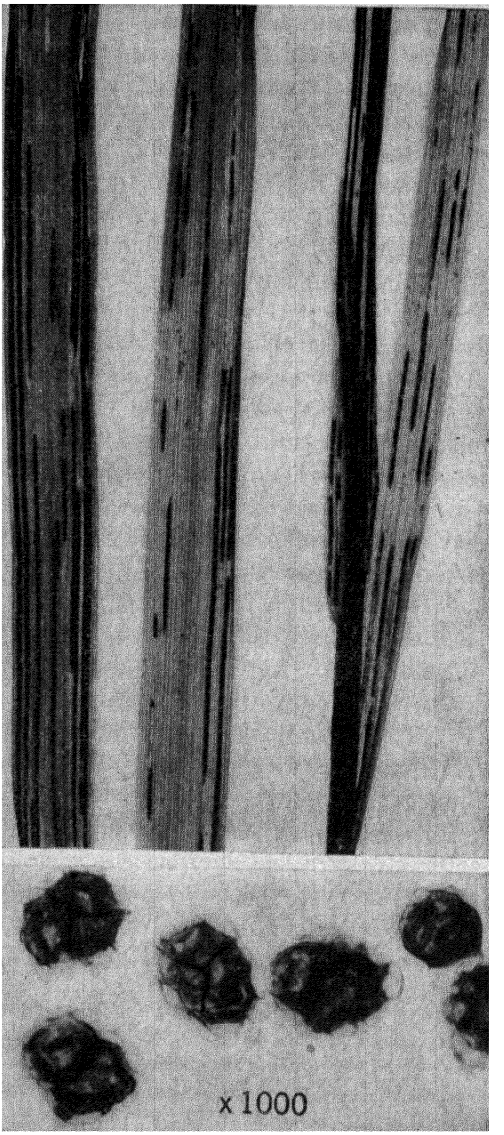

FIG. 60.—Flag smut of wheat caused by *Urocystis tritici*, and chlamydospores highly magnified.

Etiology.—Seedling infection from soil or seed-borne chlamydospores is followed by the systemic invasion of the primordia and the development of spores in the unfolding leaf tissues. The spores are in part soil-

borne in the regions where the disease is severe. Environmental conditions influence both the infection and the complete establishment of the mycelium in the primordial tissues, as reported by Angell (1934), Angell *et al.* (1937, 1938), Faris (1933), Jones and Seif-El-Nasr (1940), Noble (1923, 1924, 1934), and others.

Control.—Seed treatment, crop rotation, and the use of resistant varieties are important control measures. Seed treatment is partly effective under conditions of soil infestation. Resistant varieties, as cited earlier, offer the most practical means of control. The lists of the important resistant wheats and the genetics of inheritance of resistance are reported by Carne and Limbourn (1927), Pridham and Dwyer (1930), Shelton (1924), Shen (1934), Tisdale *et al.* (1923, 1927), Yu *et al.* (1931, 1934, 1936, 1945), and others.

Physiologic specialization occurs in *Urocystis tritici*. According to Yu *et al.* (1936, 1945), 12 races differentiated on five wheats occur in China. However, Verwoerd (1929), in Africa, found no evidence of specialization in collections he tested. Holton and Johnson (1943) have differentiated two races in the United States that are separated widely geographically: race 1 from Kansas and probably representative of the Illinois, Missouri, Kansas area and race 2 from Washington. Oro × Federation-38 and -40 are resistant to race 1 and susceptible to race 2. Oro (C.I. 8220) apparently gives the same differential reaction as the two hybrid selections.

22. Stem Rust, *Puccinia graminis tritici* Eriks. and Henn.—Stem rust is distributed generally with the wheat crop. In the drier areas, the disease develops in epiphytotic form only in moist seasons. This disease probably has caused greater and more spectacular damage than any other disease of the wheat crop. Losses are usually higher in the spring-wheat sections of North America than in either the soft or hard winter-wheat areas, as summarized by Craigie (1944, 1945). This is due apparently to two main factors: (1) the relatively high summer precipitation in the spring-wheat areas and (2) the plant growth occurring over a longer period of favorable summer conditions. Greaney *et al.* (1941) have reviewed the earlier literature on the effect of stem rust on composition and have shown that nitrogen is decreased and ash constituents are modified. Yield of grain is reduced, and composition is modified, varying considerably with the stage of plant growth when the rust develops. In addition to wheats and other small grains, a large number of grasses are susceptible to this and other physiologic varieties of the parasite.

Symptoms and Effects.—Two stages of the rust occur on the wheats and grasses. The red rust or uredial stage is evident on the leaves and culms at any stage of plant growth. The uredia are reddish brown in color, usually oblong in shape, and the epidermis of the leaves and culms

is ruptured and pushed back around the pustule. The uredia of this rust are distinguished readily from those of the leaf rust, except perhaps in the very early spring or late autumn when the uredia of the two rusts appear somewhat similar. The differences in morphology of the urediospores of the two are always distinguishing characters. In resistant varieties, development of the uredia is reduced or prevented, and yellow flecking or brown necrosis are the characteristic symptoms (Stakman *et al.* 1944). The black rust or telial stage develops more abundantly on the leaf sheathes and culms of the rusted plants, especially during and just prior to maturation of the wheat tissues. The telia are oblong to linear, dark brown to black, and the teliospores are exposed. Severe stem rust development results in numerous uredia and telia on the leaves, culms, and spikes and the drying out and early maturity of the wheat plant.

The aecial stage of this rust occurs on *Berberis* and *Mahonia* spp. early in the spring. The orange-yellow lesions are common on the leaves, petioles, and blossoms of several *Berberis* spp. The pycnial (spermagonial) lesions occur first, usually on the upper surface of the leaf. The lesion is slightly elevated, orange yellow, and produces an exudate when mature that attracts insects. The aecia develop on the undersurface of the leaf immediately below the spermagonial lesions or surrounding them where the infections develop on the petioles or blossoms. The elongated aecial cups with serrate marginal peridia are conspicuous over the surface of the lesion (Fig. 61).

The Fungus.—*Puccinia graminis tritici* Eriks. and Henn.
(*Uredo linearis* Pers. and others)
(*Uredo graminis* Eriks. and Henn.)
(*Uredo frumenti* Mart.)
(*Puccinia cerealis* Mart.)
(*Puccinia linearis* Roehl.)
(*Puccinia agropyri* Otth.)
(*Lycoperdon lineare* Schrk. and others)
(*Erysibe linearis* Wallr.)
(*Caeoma lineare* Schlecht)
(*Caeoma berberidis* Schlecht)
(*Aecidium elongatum* Lk.)
(*Aecidium lineare* Gmel.)
(*Aecidium berberidis* Gmel.)

The synonomy and history of this fungus is given in more detail by Sydow.[1] Excellent color plates of the spore forms and symptoms are included with a detailed discussion of the disease by Eriksson and Henning.[2]

[1] Monographia Uredinearum, Vol. 1, pp. 692–698, 1904.
[2] Die Getreideroste, 1896.

The spermagonia (pycnia) and aecia develop on some of the *Berberis* and *Mahonia* spp., chiefly *B. vulgaris* L. Prior to aecial development the flask-shaped pycnia of the

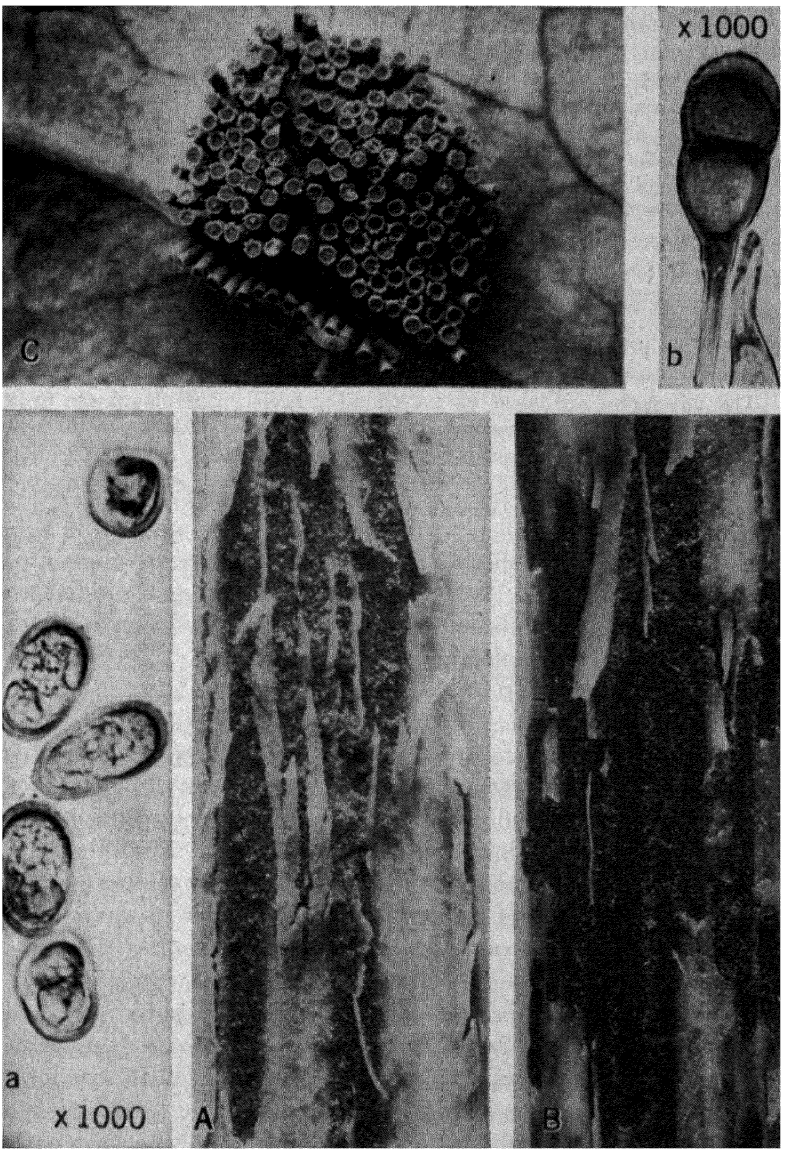

FIG. 61.—*Puccinia graminis* The aecium on the leaf of *Berberis vulgaris* (*C*) and the uredia (*A*) and telia (*B*) on wheat are shown magnified by 20. The urediospores (*a*) and teliospores (*b*) are shown magnified by approximately 1000.

upper surface of the leaf bear numerous hyaline thread-like spermatia and flexuous paraphyses-like hyphae in a slightly viscous exudate. Allen (1930, 1933), Buller (1938), Craigie (1927, 1928, 1931), and others have demonstrated the role of the spermatia and

flexuous hyphae in the initiation of the binucleate or dikaryotic stage of the fungus. The aecial stage develops on the undersurface of the leaf below the spermagonium in which the fusion of spermatium and flexuous hypha occurs. The aecial lesion is elevated, orange yellow, and numerous shallow tube-shaped aecial cups develop on the surface of the lesion (Fig. 61). The length of the peridial tubes varies with environmental conditions. The margins of the peridia are turned out and serrated. Aeciospores are borne in chains within the cups. Aeciospores are subglobose to hexagonal, light orange yellow, smooth, and germinate by the formation of a hyaline germ tube.

The uredia develop on the cereals and grasses. The uredia are oblong to circular with the epidermis ruptured and turned back around the margin of the pustule, orange red to chestnut brown in color, and dusty in appearance due to the presence of the numerous urediospores (Fig. 61). The urediospores are elliptical or pyriform with four conspicuous germ pores arranged around the equator of the spores, dark orange yellow, echinulate, and free from the short pedicels when released (Fig. 61).

The telia form in the uredia and independent of the uredia as the cereal and grass plants approach physiological maturity. The telia are naked, oblong to linear, and dark brown to black, with the ruptured epidermis usually conspicuous around the margins. The teliospores are borne on persistent pedicels. The spores are fusiform to clavate, two-celled, slightly constricted at the septum with the apex thickened and rounded or slightly pointed, smooth, and chestnut brown in color. The teliospores germinate in place after a rest period to form a four-celled promycelium (basidium) and laterally borne sporidia on minute pointed sterigmata.

Specialized varieties of the fungus are restricted to the larger groups of the cereals and grasses. It is a common practice to indicate these varieties by the use of trinomials (Eriksson, 1894, Eriksson and Henning, 1896). Numerous physiologic races occur within these varieties, as summarized by Stakman *et al.* (1944).

Etiology.—The parasite is characteristically a heteroecious long-cycle rust fungus. The complete cycle occurs in the cooler climates where the barberry, cereals, and grasses occur in close proximity. The teliospores are formed as the infected cereal and grass tissues approach maturity. The teliospores remain dormant over winter and usually require low temperatures such as freezing and thawing to germinate. Nuclear fusion and the initiation of the true diploid phase of the parasite occur during the teliospore maturation or prior to germination. The teliospores germinate to produce basidia with characteristically four haploid cells and laterally borne sporidia. The sporidia are wind-borne and infect the susceptible *Berberis* spp. by direct penetration through the epidermis. The spermatia and flexuous hyphae form in the initial (spermagonial or pycnial) lesions on the barberry. The spermatia are transferred largely by insects or meteoric water, and fusions between the spermatia and flexuous hyphae apparently initiate the binucleate or dikaryotic phase of the fungus. The fungus is predominantly heterothallic in nature. Inbreeding has been reported by Johnson and Newton (1938) and Newton and Johnson (1937) as well as the abnormal production of uredia and telia on the barberry. The aecia develop following the sexual fusion,

and aeciospores are produced in great abundance early in the spring. The aeciospores are wind-borne to the gramineous plants, where infection occurs through the stomata. The mycelium in the cereal and grass tissues produces the urediospores which cause numerous secondary infections on the susceptible cereals and grasses. The urediospores frequently furnish the inoculum for the progressive spread of the rust over an extensive area, as in the spread northward in North America and Europe. Later as these plant tissues approach physiological maturity the mycelium produces teliospores. As in most of the heteroecious rust fungi, two widely different suscepts support the aecial and the uredial and telial stages in the complete life cycle of *Puccinia graminis.*

The barberry supports the sexual stage of this economically important fungus. The discovery of the role of the spermatia and the flexuous hyphae borne in the spermagonia on the barberry reemphasized the importance of the aecial host in inducing genetic variability in the parasite. The barberry species are susceptible to infection by sporidia of many of the different specialized varieties and numerous physiologic races of the parasite. Therefore, cross fertilization between these numerous races is possible and affords the mechanism for the production of new potentially dangerous races. Investigations by Johnson *et al.* (1932, 1934), Newton *et al.* (1930, 1931, 1932), Stakman *et al.* (1930, 1934), and others have demonstrated experimentally the role of the barberry in the production of hybrids among the various specialized varieties and physiologic races. The significance of the barberry, therefore, becomes twofold: (1) the completion of the life cycle of the parasite in the cooler temperate zones and early spring spread of the rust to the cereals, and (2) the production of new potentially dangerous races of the parasite through hybridization occurring in the aecial stage on the barberry.

Environmental conditions influence greatly the etiology of stem rust. Infection and development of the rust on the cereals and grasses and the barberry are determined in large part by temperature and moisture conditions. In nature, low temperatures or freezing and thawing are necessary before the teliospores will germinate. The reaction of many resistant varieties is influenced also by high temperatures. The persistence of the parasite in the uredial stage either as perennial mycelium or freshly produced viable urediospores is determined by mild winters and moist summers. The spread of urediospores from regions of mild climate to those of less favorable environment is important in the production of stem rust epiphytotics. Craigie (1945) has reviewed the mass of literature on the epidemiology and the control of stem rust.

Control.—The control of stem rust is largely by means of barberry eradication and by the use of resistant varieties. Barberry eradication

is important in the temperate zones where the aecial infection occurs naturally. The breaking of the cycle of the parasite, in the regions where the uredial stage does not survive the winter, prevents the early spring spread of the rust to the cereals and grasses. In areas where the barberry is abundant and the climate favorable for the rust as in the Transcaucasian Mountains, small-grain production is impractical and corn is the chief grain produced. The elimination of the susceptible barberries prevents further genetic variation of the parasite through hybridization and recombination of characters for broader parasitic potentialities. Many countries have passed laws preventing the sale, distribution, and growing of the susceptible barberry species. Early-maturing varieties of the grains and early planting aid in escaping stem rust damage.

Varieties resistant to stem rust offer the most practical means of control. Earlier varieties of *Triticum vulgare* resistant to groups of physiologic races common in the areas were used, as discussed by Aamodt (1927). Frequently such varieties, when grown more or less exclusively in areas, tend to increase and spread inoculum of races of the parasite to which they are susceptible. No species of *Triticum*, practical for use in commercial wheat production, is resistant to all the physiologic races of *Puccinia graminis*. Therefore, it becomes necessary to make recombinations to incorporate the characters controlling resistance to groups of races into new wheat varieties of suitable agronomic type and milling and baking quality. Numerous varieties gradually approaching these requirements are being developed. McFadden (1930) transferred the Yaroslav emmer characters for stem rust resistance in the seedling stage and mature plant stage to the hexaploid varieties Hope (C.I. 8178) and H-44. These varieties under field conditions in the United States and Canada are resistant to most races of the parasites causing stem rust, leaf rust, loose smut, bunt, and powdery mildew. These wheats are used extensively in breeding programs, and Hope germ plasm occurs in many of the recent stem rust resistant wheats. According to Hart (1944) and others, Hope is moderately susceptible to race 21 in the seedling stage and to races 15B and 34 in the mature plant stage when grown at high temperatures or when sown late. The same reaction occurs in the selections from Hope and H-44 crosses. Vernal emmer has been crossed with the durums to produce stem rust resistant durum varieties. These also are suceptible to both biotypes of race 15. The durum wheats Acme (C.I. 5284) and Iumillo (C.I. 1736) react similarly. McMurachy's selection (C.I. 11876), resistant to stem rust and Helminthosporium root rot, is moderately susceptible in late plantings to races 11, 15, 21, 34, 56, and 147. The Kenya wheat selections are more or less susceptible to races 15B and 34 when grown at high temperatures or sown late. *Tri-*

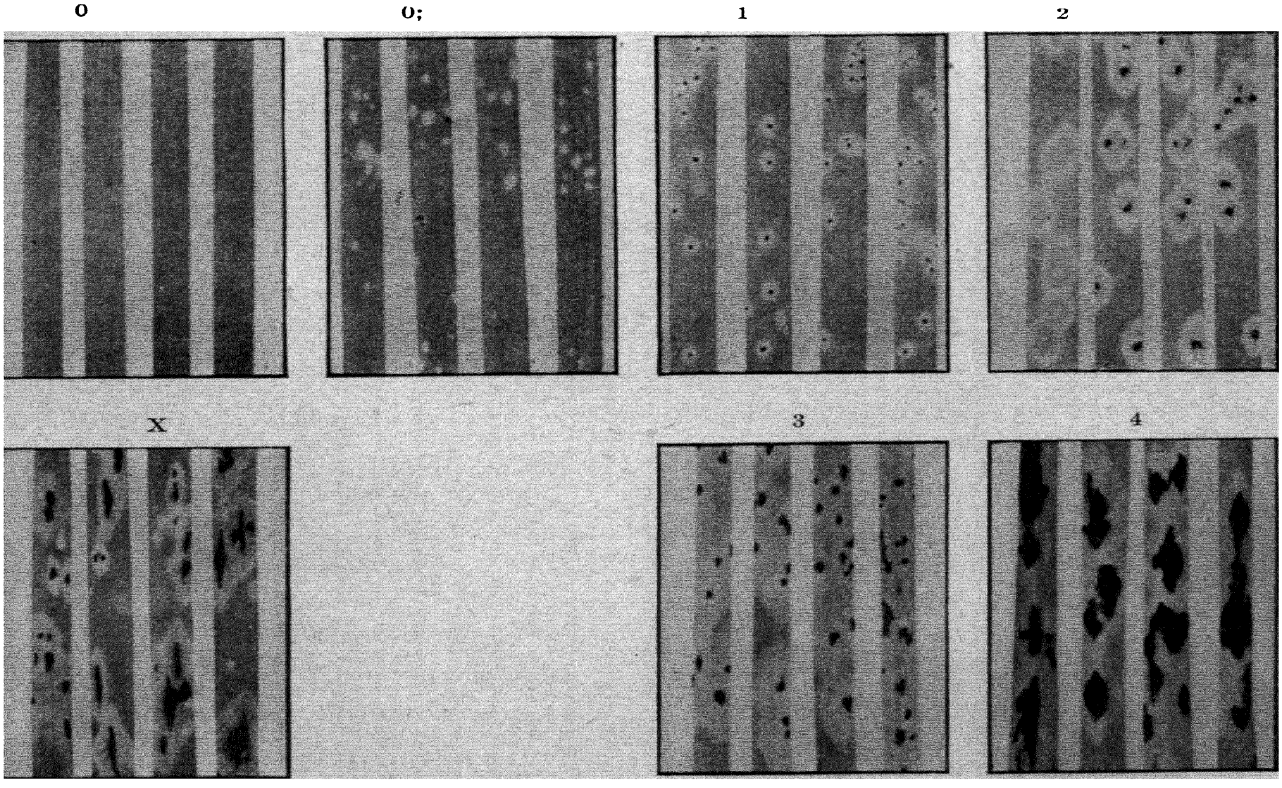

FIG. 62.—Uredial infection types produced by physiologic races of *Puccinia graminis tritici* on the differential varieties of *Triticum* spp. 0, 0;, 1, and 2 are resistant types of reaction; X is mesothetic (resistant and susceptible on the same plant); and 3, 4 are susceptible reactions. (*Courtesy of Conference for the Prevention of Grain Rust.*)

ticum timopheevi, resistant to most physiologic races of the parasites causing stem rust, leaf rust, loose smut, bunt, and powdery mildew, is moderately susceptible to the biotype 15B and to race 19 in the greenhouse. It is also susceptible to race 189 in Peru, according to Garcia-Rada *et al.* (1942). The five important newer sources of resistance to stem rust are moderately susceptible to one or two physiologic races of *Puccinia graminis tritici*, therefore, still other sources of resistance to stem rust are needed.

Resistance to stem rust in the different varieties is controlled by a single factor or at most a few factor pairs. These differ for several of the different types and sources of resistance. In some instances, minor factors modify the type and extent of the reaction, as suggested by Clark (1936) and others. According to Watson (1941), resistance in McMurachy's selection and in certain of the Kenya selections is in each controlled by a simple factor pair with the factor for resistance in the former allelomorphic to that of the latter.

The morphological and physiological nature of stem rust resistance has been investigated extensively. The relationship of stomatal openings to infection and sclerenchyma tissue to rust damage has been reviewed by Hart (1931) and Peterson (1931). Dickson (1934), Hart (1931), Stakman (1915), Thatcher (1943), and others have discussed the type of resistance associated with the incompatible physiological relationship of the protoplasts of the cells of the wheat plant and those of the rust parasite (see also Chap. II). The flecking type of rust reaction evident in several types of rust resistance and the brown necrosis in the mature plant resistance in Hope and its derivatives (McFadden, 1939, Thatcher, 1943) are external manifestations of this incompatibility between the cells of the wheat plant and those of the parasite.

Physiologic specialization is another expression of the rather specific compatibility between the rust parasite and its suscept. According to Stakman *et al.* (1944), biotypes form the basic concept for physiological races. By using the uredial infection types produced on 12 varieties and species of *Triticum* (Fig. 62), 189 physiologic races of *Puccinia graminis tritici* are differentiated. Within certain of these races, specific biotypes appear which in epidemiology and breeding investigations must be considered more specifically, as in the case of 15A and 15B discussed above. The distribution and prevalence of these races from season to season is discussed by Craigie (1945) and Stakman *et al.* (1944). As pointed out earlier, the desirable types of resistance to stem rust include resistance to large groups of these races.

23. Stripe Rust, *Puccinia glumarum* (Schm.) Eriks. and Henn.—The stripe rust is restricted in distribution in contrast to both the stem rust and leaf rust of wheat. This rust is found along the Pacific Coast and

Intermountain areas of North America. It extends east of the Rocky Mountains in the northern, cooler sections of Canada. Essentially the same distribution occurs in South America with the rust spreading out

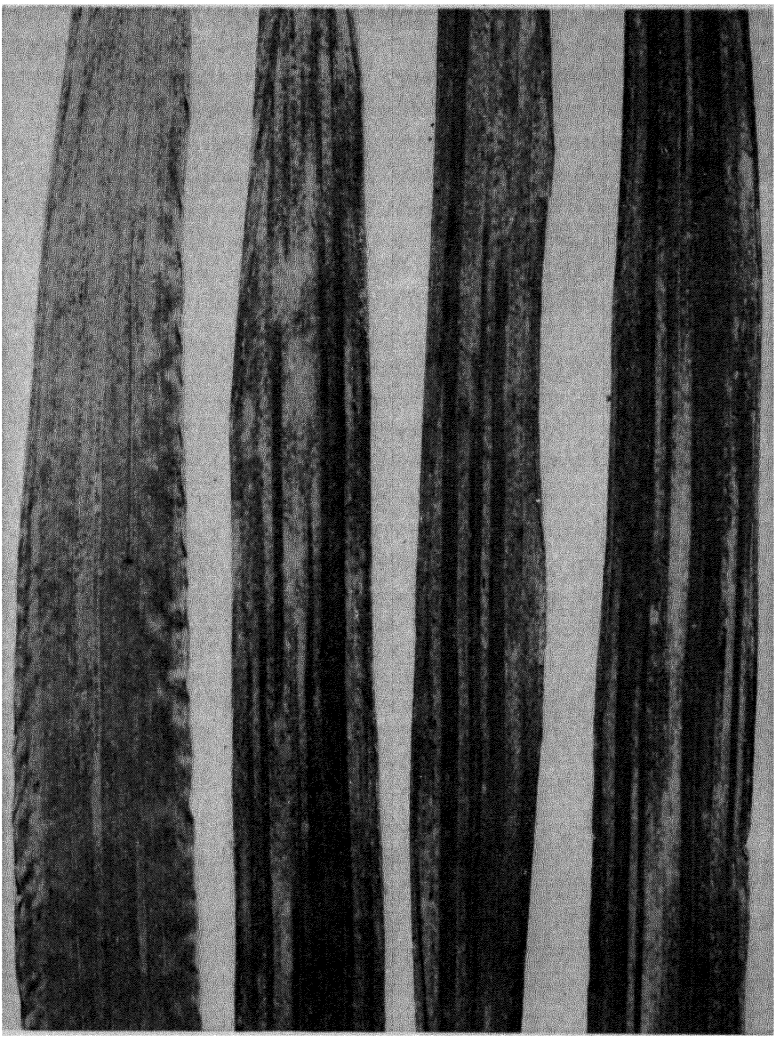

FIG. 63.—The uredial stage of stripe rust of wheat caused by *Puccinia glumarum*.

eastward over the cooler plains area of Southern Argentina. Again in Europe and Asia, the stripe rust is prevalent in the northern and eastern cooler regions and in the mountainous areas of Central Europe and Asia. Apparently this rust is limited in its distribution to the areas of relatively cool summer temperatures and humid winters. The stripe rust is com-

mon on wheat, barley, rye, and many grasses. In areas where the disease is severe, it causes reduction in yield of the grains and grasses, as shown by Bever (1937). The rust is known as glume rust and yellow rust as well as stripe rust.

Symptoms.—The stripe rust appears early in the spring before the other rusts are abundant. This is especially evident in sections of mild winters and heavy snow covering. The linear, narrow, citron-yellow uredia appear on the autumn foliage and on new growth early in the spring. The uredia coalesce to produce long stripes between the veins on the leaf blade and sheath and small linear lesions on the floral bracts (Fig. 63). The pustules frequently break through the epidermis on the inner surface of the leaf sheath and lemma and palea. Under favorable conditions, uredia form on the culms, especially inside the leaf sheaths. The telia develop as narrow linear pustules covered permanently by the epidermis.

The Fungus.—*Puccinia glumarum* (Schm.) Eriks. and Henn.
(*Uredo glumarum* Schm.)
(*Trichobasis glumarum* Lév.)
(*Puccinia tritici* Oerst.)
(*Puccinia neglecta* West.)
(*Puccinia rubigo-vera* Aut.)

The aecial stage of this rust is unknown. The uredia are linear, citron to orange yellow, usually narrow, with a marked tendency to form stripes on the leaves and culms. Spatulate paraphyses sometimes occur around the margin of the uredium. The urediospores are round to ovate, finely echinulate, with three or four germ pores. The telia are linear and covered by the epidermis. Brown paraphyses border the telia and are intermingled with the teliospores along the margin of the telium. The teliospores are oblong to cuneiform, smooth, slightly constricted at the septum, with the apex less thickened and pointed than in *Puccinia graminis*. The spore morphology is similar to *P. rubigo-vera* (DC.) Wint.

Etiology.—The mycelium and urediospores develop abundantly in the late autumn on the cereals and grasses, and they are important in the persistence of the fungus. The mycelium and, less frequently, the spores remain viable over winter and develop early the following spring. The late-autumn and early-spring development of this rust is characteristic. Secondary infection and aggressive development of the parasite occurs during the early growth period of the susceptible cereals and grasses. In the cooler areas, especially where light intensity is low, the rust develops aggressively until maturity of the crop. In less favorable areas, the rust development is checked in late spring, and summer survival becomes the critical period in the life cycle of the parasite. The summer environment apparently functions more specifically than the winter period in restricting the distribution of this rust.

Control.—Many of the commercial varieties of wheat and the other cereals are resistant to this rust. Resistant strains are found in many of the grasses common in the areas where this rust is prevalent, as shown by Hassebrauk (1932), Hungerford and Owen (1923), Newton and Johnson (1936), Sanford and Broadfoot (1933), and others. The inheritance of resistance to this rust was among the first studied, as reported by Armstrong (1922), Biffen (1905), and Nilsson-Ehle (1908, 1909, 1911) and summarized by Roemer *et al.* (1938). A single factor or a few factor pairs function in the inheritance of resistance.

Physiologic specialization occurs, although the differentiation of races is complicated by the response of the parasite to environmental conditions. Bever (1934) and Newton and Johnson (1936) differentiated three races in the United States and Canada. Eriksson (1894) included this parasite in the early studies on specialization. Extensive investigations on specialization of this parasite are reported from Europe, with considerable confusion in the number of races and their response on different varieties, as reported by Gassner and Straub (1932, 1934), Roemer *et al.* (1938), Rudorf (1929), Straub (1937), and others.

24. Leaf Rust, *Puccinia rubigo-vera tritici* (Eriks.) Carleton.—The leaf rust of wheat is generally distributed through the humid and semihumid wheat-producing areas of the world. The leaf rust is distributed more uniformly and occurs more regularly than either of the other rusts of wheat. Until recently, leaf rust has caused damage in the Central and Eastern United States on the winter wheats. In recent years damage has been severe in the Central United States and Canada on the hard red spring wheats. This rust also occurs on barley and a number of grasses, as shown by Johnston (1936, 1940) and Mains (1933). Damage is severe especially when the rust infection occurs early and continues during the growing season, as reported by Caldwell *et al.* (1934), Johnston and Miller (1934), Waldron (1937), and others in the United States; Greaney *et al.* (1941) and Peterson and Newton (1939) in Canada; Neill (1931) in New Zealand; Phipps (1938) in Australia; and Butler and Hayman (1906) in India. Total yield of grain is reduced appreciably. Kernel volume is lowered without appreciable shriveling of the grain, and nitrogen content of the grain is decreased.

Symptoms.—The uredial stage of the rust is evident on the leaves from the seedling stage to maturity. In the early spring, new uredia frequently form in a circle around uredia of the previous autumn. The uredia are round to slightly oblong, orange yellow, and generally the ruptured epidermis is inconspicuous around the uredia. The covered telia form adjacent to the uredia and in new locations, especially on the leaf sheaths (Fig. 64).

The Fungus.—*Puccinia rubigo-vera tritici* (Eriks.) Carleton
(*Uredo rubigo-vera* DC.)
[*Puccinia rubigo-vera* (DC.) Wint.]
(*Puccinia dispersa* sp.f. *tritici* Eriks. and Henn.)
(*Puccinia triticina* Eriks.)

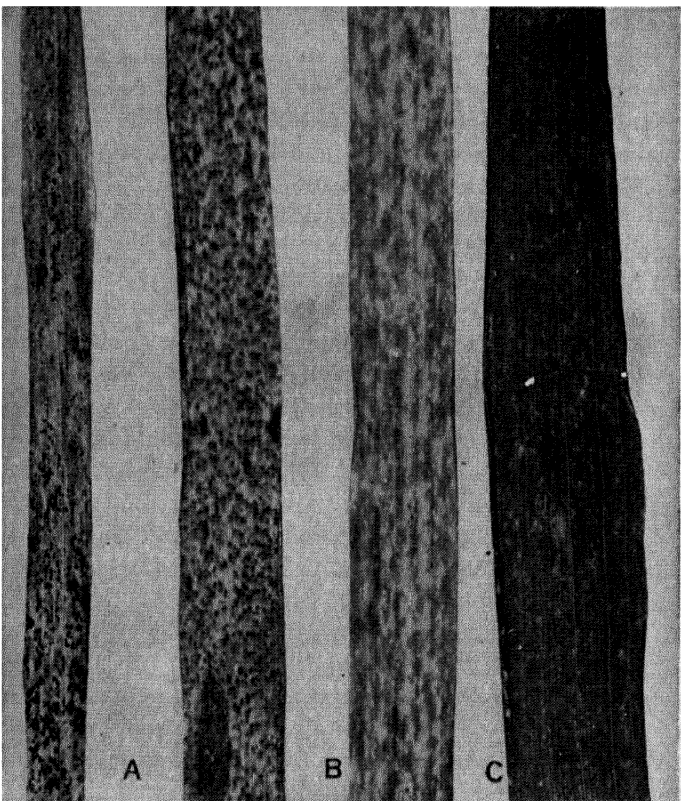

FIG. 64.—Reaction of wheat varieties to *Puccinia rubigo-vera*. (A) Two leaves fully susceptible showing telial and uredial development; (*B*) chlorosis and necrosis following infection; and (*C*) highly resistant reaction.

Puccinia triticina Eriks. was used generally following Eriksson's (1894) division of the morphological species *P. rubigo-vera* into the several species based on specialization. Mains (1933) investigated the accial and uredial suscepts and suggested the use of the trinomial, which has been generally accepted.

The accial stage occurs infrequently on several species of *Thalictrum* as citron-yellow swollen lesions. The spermagonia form on the upper surface of the leaflet, followed by the small aecia on the under surface. The uredia on wheat and some grasses are round to slightly oblong, orange yellow, and occur on the leaves and less commonly on the floral bracts. The urediospores are round to ovate, echinulate, with three to four germ pores distributed over the surface. Both the uredia and urediospores differ morphologi-

cally from the comparable structures in *Puccinia graminis*. The telia are small, oval to oblong, black, and covered by the epidermis. The teliospores are surrounded by a thin layer of brown paraphyses. The teliospores are oblong to cuneiform, smooth, brown, slightly constricted at the septum, and rounded at the apex without pronounced apical thickening of the wall. Germination of the different spore forms is similar to that of *P. graminis*.

Etiology.—This rust persists largely in the uredial stage. Infection of the more common aecial host *Thalictrum polygamum* Muhl. in North America is uncommon. Mains (1933) has listed 12 species of *Thalictrum* as susceptible to this rust. In both North America and Europe, the aecial stage is not important in the etiology of this rust parasite. The uredial mycelium and urediospores, to a lesser extent, survive the winter on winter-wheat plants as far north as winter wheat survives. New uredia develop early in the spring from this mycelium, and infection spreads from the new urediospores. There is some spring spread from urediospores produced the previous autumn. In the areas of moderate to plentiful moisture, the urediospores on the mature crop reinfect the volunteer wheat seedlings and the fall-sown wheat. In such areas the grasses are not important in the production or continuance of the fungus. It is questionable whether the grasses play an important part in the maintenance or distribution of this rust, although Johnston (1940), Mains (1933), and others have shown that a number of grasses including perennials are susceptible. The urediospores spread northward similar to those of *Puccinia graminis*, as discussed by Craigie (1945).

Control.—Leaf rust resistant varieties offer the chief means of control of this disease. Leaf rust resistant wheats are essential to economical wheat production, especially in the humid regions where winter and spring varieties overlap, as in the North Central United States. Most of the commercial winter varieties of this area are moderately to highly resistant to the races commonly occurring in this region. Many of the newer spring and winter wheats are resistant to the common races of both *Puccinia graminis tritici* and *P. rubigo-vera tritici*.

According to Guard (1938), Johnston (1940), Vallega (1942, 1944), and others, more varieties resistant to leaf rust occur in the wheat species with lower chromosome numbers. *Haynaldia villosa* (L.) Schur. and *Triticum monococcum* L. are highly resistant. Nearly all the durums are resistant, whereas the emmers range from susceptible to resistant. *T. timopheevi* Zhuk. is resistant in both North and South America. Resistance to relatively large groups of races of the parasite are found in the *T. vulgare* Vill. group of varieties. Hope is intermediate in reaction to some few races and resistant to most. Resistance is controlled by several single factor pairs in the different sources of resistance, as discussed by Mains *et al.* (1926), Roemer *et al.* (1938), and others.

Specialization is well developed in this parasite as over 128 physiologic races are differentiated on eight wheat varieties, as reported by Hassebrauk (1937), Johnston and Mains (1932), Newton and Johnson (1941), Vallega (1942, 1944), and others. Many races are localized geographically, while others are widely distributed, especially when urediospore dissemination occurs over wide areas.

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CHAPTER XII

GRASS DISEASES

The grasses are receiving increasing attention as farm crops in many agricultural areas of the world. The more intensive cultivation of selections obtained from the indigenous or introduced species and the reestablishment and maintenance of the more desirable natural ones are changing the concepts of grassland agriculture. Grasses are becoming specific crop plants in the rotation system on the farm, in the soil-conservation program, in pastures, and on the range. As such, they are evaluated not as grass associations, but rather as species, varieties, or strains which have distinct and widely varied propensities. The multiplication of individual strains, by seed or clonal propagation, results in large populations similar or alike. The comparisons of large numbers of strains usually demonstrate differences, including disease reaction, between those single strain units within a species. Under these conditions diseases become a more evident factor in the development and economy of certain selected strains. Fortunately, the variability within a species generally affords ample scope for selecting disease-resistant lines when a sufficient array of clonal or selfed strains are investigated under conditions favorable for disease development. Furthermore, the planting of an area with a single strain of grass without first evaluating it for disease reaction increases the chances for a disease normally of minor importance in a mixed-grass population to become one of major significance. Therefore, the investigation of the diseases of the grasses coincident with the breeding procedure is an essential phase of grass improvement.

The importance of the disease and the etiology of the parasite vary with the production of the grass in pure stands, in nursery rows, or in seed fields and its use in pastures or on the range. Frequently a disease is severe in nursery rows or seed fields and unimportant in the pasture mixtures. Control measures often differ greatly under these varied conditions of growth. Much of the information available concerning the diseases of grasses is based largely on observation under pasture conditions or where the plants are growing in their natural habitats. The chapter on grass diseases must deal largely with symptoms of the diseases, the morphology of the parasites, and only the more general phases of etiology and control. Information on the geographical distribution of the diseases frequently is inadequate, as the diseases have been

studied intensively in some regions and in others not at all. The wide variety of grass species involved and the still wider range of parasites make the task more difficult in the space allotted. Reference should be made to the diseases on the cereal crops for more detailed discussions of many of the diseases common to the Gramineae.[1]

1. Nonparasitic Maladies.—The grasses, like the cereal crops, are subject to external manifestations of unfavorable environmental conditions. The response to soil mineral deficiences is similar in manifestation to that in the different cereal groups. Chlorosis, leaf flecking, and spotting are the common symptoms.

Pigmented spots or blotches commonly occur on the leaves of many species, such as those of *Andropogon*, *Bromus*, *Dactylis*, *Panicum*, and *Sorghum*, especially when inbreeding is employed in the breeding program.

Bends and proliferation reported in many genera of the forage and pasture grasses are listed in this group, chiefly since no cause for either condition is known (Fischer, 1941, and Nielsen, 1941). The upper portion of the culm or the rachis bends downward usually in a hairpin-like turn. The inflorescences develop into proliferated leaf-like structures similar to "crazy top" in corn. Both conditions frequently occur on the same plant.

2. Mosaics, Viruses.—Two mosaic complexes occur on the grasses. The grass mosaic complex, prevalent on sugarcane, corn, and related grasses, is relatively common on many of the grasses of the warmer climates. The chlorosis, mottling, or striping and other symptoms generally found are similar to those existing on sugarcane. The wheat mosaic occurs on *Bromus inermis* Leyss in South Central United States and is reported in Southern Russia, according to McKinney *et al.* (1942) and others. Some of the *Avena* spp. are infected with the oat mosaic. Based upon the reaction of the cereals, strains of the grasses resistant to these mosaic complexes probably occur.

3. Bacterial Blights, *Pseudomonas coronafaciens* var. *atropurpureum* (Reddy and Godkin) Stapp, *Xanthomonas translucens* (L. R. Jones, A. G. Johnson, and Reddy) Dows., and Other Species.—Specialized varieties of the two common cereal bacterial pathogenes occur on the grasses. The bacterial blight caused by *Pseudomonas coronafaciens* var. *atropurpurea* (Reddy and Godkin) Stapp is distributed widely on *Bromus inermis* Leyss and *Agropyron repens* (L.) Beauv. The lesions are first circular to elliptical, water-soaked, later browning and coalescing to form purplish-brown areas on the leaf blade and sheath (Fig. 65). Exudate on the surface of the lesion is absent. Spots on the pedicels and panicles

[1] General reference to the *U. S. Dept. of Agr., Plant Disease Reporter and Supplements*, for the compilations and notes contained therein; those by G. W. Fischer, J. R. Hardison, C. O. Johnston, and R. Sprague were especially valuable.

are smaller and restricted. Many lines of these grasses are susceptible to bacterial blight. The disease occurs on *Agropyron*, *Bromus*, *Elymus*, *Lolium*, and *Phleum* spp. and other genera, according to Allison and

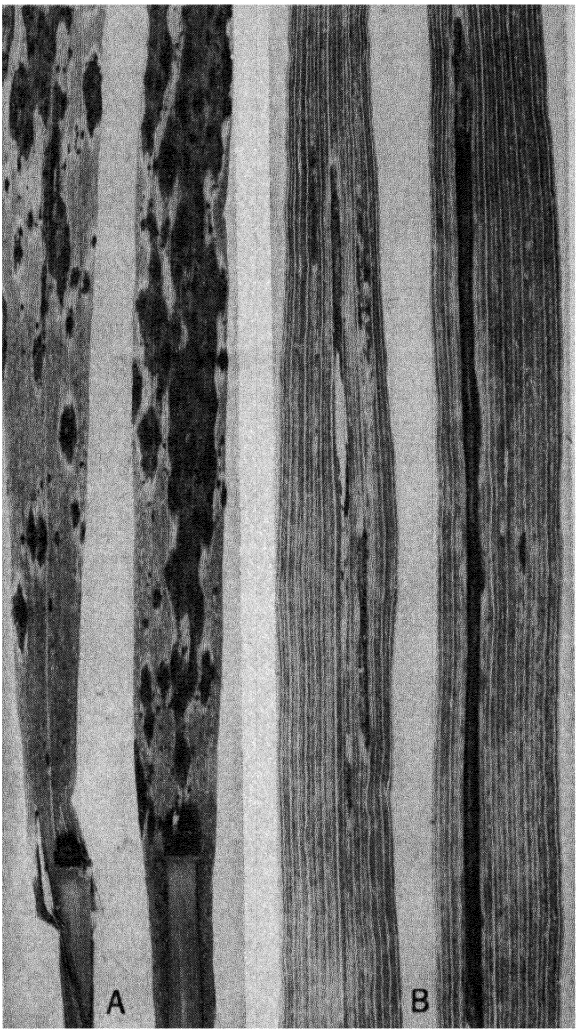

FIG. 65.—Bacterial blight caused by *Pseudomonas coronafaciens* var. *atropurpureum* (*A*) and *Xanthomonas translucens* f. sp. *cerealis*, (*B*) on *Bromus inermis*.

Chamberlain (1946), Reddy and Godkin (1923), and others. Certain races of *Xanthomonas translucens* f. sp. *cerealis* Hagb. cause an irregular brown leaf lesion or stripe with exudate on the surface of the leaves of *Agropyron*, *Bromus*, and *Hordeum* spp. (Wallin, 1946) (Fig. 65). Strains of the

Agropyron show differences in reaction to the disease (see Chap. III). Wallin and Reddy (1945) described *X. translucens* var. *phleipratensis* Wal. and Reddy on timothy. The head blight caused by *Bacterium agropyri* (O'Gara) Stapp, [*Phytomonas agropyri* (O'Gara) Bergey *et al.*], (*Aplanobacter agropyri* O'Gara), described by O'Gara (1916), occurs on *Agropyron* spp., *Sitanion jubatum* J. G. Smith, *Sporobolus airoides* (Torr.) Torr., and other grasses. The disease is similar to the European bacterial blight (Sampson and Western, 1941) of *Dactylis glomerata* L. caused by *Corynebacterium rathayi* (E. F. Sm.) Dows., [*Phytomonas rathayi* (E. F. Sm.) Bergey *et al.*], (*Aplanobacter rathayi* E. F. Sm.), which apparently occurs occasionally on this grass in North America.

The head blight caused by the former species is prevalent in Northern North America, where it causes some damage in seed production. The lesions occur sparsely on the leaves; they involve mainly the inflorescences. Severe infection results in erect leaves and distorted spikes, or panicles partly enclosed in the leaves, with an abundant yellow exudate over the diseased surfaces. Seed transmission of these two diseases is common. Exclusion of seed lots from areas where the disease occurs and seed treatment with solutions of formaldehyde or organic mercury compounds are recommended.

The sorghum and sugarcane bacterial blights occur on the related grasses. The more prevalent of this group are the bacterial stripe and streak of Johnson grass [*Sorghum halepense* (L.) Pers.] and Sudan grass [*S. vulgare* var. *sudanense* (Piper) Hitchc.] (see Chap. IX).

4. Pythium Seedling Blight and Root Rot, *Pythium* Spp.—The *Pythium* spp. common on the cereal crops cause severe damage to the grasses, as reviewed by Sprague (1944). This disease complex apparently is more severe in the heavy or fine soil areas of West Central North America, but it is general in occurrence in all areas. Preemergence seedling blight and damping-off of young seedlings more generally is caused by *Pythium debaryanum* Hesse. *P. graminicolum* Subr. causes a seedling blight and root rot of grasses. *P. arrhenomanes* Drechs., similar to the above, is distributed widely on the grasses, especially in association with the root browning and root rotting of seedlings and growing plants. The plants are weakened and yellowed or killed by the root rot, depending upon the environmental conditions. Differences in the tolerance of lines of the grasses are evident, and selection for resistance to this root-disease complex is important in grass breeding. Crop sequence, soil fertility, soil preparation, and time of seeding are important in control. Other species of *Pythium* are associated with the complex, as discussed in Chap. XI.

5. Downy Mildews, *Sclerospora* Spp.—The downy mildews are not serious diseases on the major forage and pasture grasses. *Sclerospora*

graminicola (Sacc.) Schroet. occurs especially on species of *Pennisetum* and *Setaria*, but the grasses of economic importance in this group are grown largely in tropical or in the Asiatic countries according to Hiura (1935). *S. macrospora* Sacc. occurs in localized areas on a number of economic grasses, as well as the cereals, in the more temperate zones. Apparently the disease is restricted in its spread, although its occurrence in increasing frequency in the Central United States warrants further consideration, especially for forage grasses (see Chaps. V and XI).

6. Powdery Mildew, *Erysiphe graminis* DC.—Powdery mildew is distributed widely on many genera of the grasses. However, the disease is of direct economic significance at present on a relatively few grasses, notably *Agropyron, Agrostis, Avena, Festuca,* and *Poa* spp. and strains. Very resistant and susceptible types occur in the nursery investigations with many of these grasses. Powdery mildew is potentially more dangerous in nurseries and seed fields than in pastures. The disease appears during cool somewhat cloudy seasons, especially on the leaves. The powdery superficial, white, gray, or buff mycelium and conidia develop in blotches or spread uniformly over the leaf surface. The leaves frequently brown and dry out, gradually reducing the leaf area and forage. The symptoms are less conspicuous where the grasses are grazed or cut closely.

Erysiphe graminis consists of many specialized varieties and races on the cereals and grasses. According to Hardison (1944, 1945) and others, specialization is less restricted in the grasses than in the cereals. Certain of the wild grasses furnish inoculum for the cereal crops as well as for some commercial grasses. Resistant varieties or strains offer the best means of control of the disease (see Chaps. III and XI).

7. Ergot, *Claviceps* Spp.—Several species of *Claviceps* somewhat similar morphologically occur on the cereals and grasses. According to Langdon (1941), *Claviceps pusilla* Ces. is the species causing ergot on 11 genera of grasses of Australia and the South Pacific. One genus of this group of grasses, *Digitaria,* is common in the United States, and several others are introduced. Apparently the disease is not reported on these grasses in the United States. *C. paspali* F. L. Stevens and Hall is severe on Dallis grass, *Paspalum dilatatum* Poir, and other species in the warmer areas (Lefebvre, 1939). The ergot sclerotia frequently are infected by *Fusarium* spp. in the humid Gulf area of the United States. The sclerotia and perhaps *Fusaria* in combination cause respiratory disturbances, partial paralysis, and poisoning of livestock feeding on the heavily infected Dallis grass in this area (Brown, 1916). This type of poisoning is more common than abortion and capillary constriction in the body extremities, which are the more usual pathological responses of animals consuming the sclerotia of *Claviceps purpurea* (Fr.) Tul. The composition

of the sclerotia of the two species is different in both quantity and type of alkaloids present (Gieger and Barrentine, 1939).

The common species, widely distributed on the grasses, is *Claviceps purpurea* (Fr.) Tul. This species occurs on the cultivated and wild grasses throughout the temperate zones. The disease reduces seed production in areas of high summer humidity and causes abortion, reduction in milk secretion, and constriction of the capillaries when eaten in quantities by livestock. The disease is particularly severe on *Agropyron* spp., *Agrostis gigantea* Roth., *Bromus inermis* Leyss, *Calamogrostis* spp., *Dactylis glomerata* L., *Elymus condensatus* Presl., *E. triticoides* Buckl., *Festuca elatior* L., and the *Poa* spp.

The fungus infects the young ovaries of the grass flowers, produces conidia, and finally produces sclerotia that replace the seed in the infected flowers. The conidial, or "honey-dew," stage is conspicuous on the spikelets as the grasses are in the late-bloom stage. Insects collect around the diseased florets as they feed on the sticky exudate. The sclerotia develop below the conidial stromata and grow into elongated brownish-black or purplish-black bodies somewhat the shape of the grass caryopsis (Fig. 66). When mature, the sclerotia usually extend beyond the floral bracts. The sclerotia become detached from the mature plant and fall on the ground, or part of them are harvested with the hay or seed crop. The sclerotia remain on the soil surface, overwinter, and produce the ascigerous stage, which is well synchronized with the blossoming of the grasses the following season. The description of the disease and morphology of the fungus is given in Chap. VIII.

Apparently the same race of the parasite occurs on most of the common grasses (McFarland, 1921, Stäger, 1903, 1905, 1922). Ascospores or conidia produce infection on many of the grasses and cereals commonly grown in the temperate zones. The parasite on *Lolium* is specialized to this genus and some few closely related grasses. There is some evidence also that the race on *Glyceria* is restricted to this genus. While there are marked differences in susceptibility among the grass species, the economic grasses show little indication of highly resistant strains. Cutting grasses in pastures and waste places before blossoming helps reduce the inoculum and resultant damage from the sclerotia.

The species *Claviceps microcephala* (Wallr.) Tul. is listed as occurring on grasses in both North America and Europe. Petch (1938) and others have shown that this species is not morphologically distinct from *C. purpurea*. The two species are reported on the same or similar groups of grasses and in approximately the same areas, although the former species is reported from somewhat more northerly regions in both North America and Europe. Other species differing somewhat in morphology and pathogenicity are reported.

FIG. 66.—Ergot sclerotia on *Bromus inermis*.

8. Epichloe Head Blight or Choke, *Epichloe typhina* (Fr.) Tul.—The disease is of minor importance in North America. It occurs sparingly on a large number of grasses but is sometimes abundant on *Poa* spp. in North Central North America. In Europe and Asia, however, the disease is a serious factor in seed production, especially in the cool, humid northern regions, as summarized by Sampson and Western (1941). The disease is restricted apparently to limited areas in North America where the seasons are cool and the winters are mild or where the plants are protected by snow. The fungus *Epichloe typhina* (Fr.) Tul. produces a perennial mycelium in the crown buds of infected plants and a systemic infection of the primordial tissues. In summer, the mycelium forms a white felt over the surface of late tillers or more characteristically a white stroma with minute conidia over the inflorescence as it emerges. The stroma usually encloses all the spike or part of the inflorescence in the case of the more open panicle type of grass species. The color of the stroma changes to orange as the plants approach maturity, and numerous perithecia with papillate ostioles develop submerged in the stroma. The asci contain eight hyaline filiform many-celled ascospores, which are forcibly discharged and wind-borne. The stroma disintegrates after the plants are ripened fully. Seed transmission occurs in *Festuca rubra* L. and probably in other grasses in which diseased plants produce seed.

9. Tar Spot, *Phyllachora* Spp.—Tar spots are distributed widely on a large number of grasses; however, they are localized and rarely become general in nursery, field, or pasture. The black sunken glossy spots on the leaves are conspicuous when the plants are infected heavily. *Phyllachora graminis* (Fr.) Fckl. is one of the more common species, although many others are described on the grasses. The perithecia are immersed in the black stroma with the ostioles opening on both surfaces of the leaves. The asci are cylindrical with short pedicels, intermixed with filiform paraphyses, and contain eight ovoid hyaline unicellular ascospores. Orton (1944) has summarized the literature concerning the species on the Gramineae. Over 40 species were described, the differentiation being based on the clypei and the size and shape of the ascospores.

10. Snow Mold and Foot Rot, *Calonectria graminicola* (Berk. and Br.) Wr. [*Fusarium nivale* (Fr.) Ces.].—This disease of the grasses is similar to that occurring on winter wheat. Damage is more severe on turf species, especially where they are grown under conditions of heavy nitrogen application to force late summer vegetative development (Fig. 67). The disease and its control is discussed by Broadfoot (1938), Bennett (1933), Dahl (1934), Wollenweber and Reinking (1935), and Sampson and Western (1941). The treatment of turf of golf greens and lawns with fungicides, especially the mercury compounds, is practical. This also

controls the snow scald and snow mold caused by *Typhula*, *Sclerotium*, and *Rhizoctonia* spp. (see Chap. XI).

11. Seedling Blight and Crown Rot, *Gibberella* and *Fusarium* Spp.—This type of seedling blight and crown rot, so common on certain of the cereal crops, is of minor importance on most of the grasses. *Fusarium culmorum* (W. G. Sm.) Sacc. causes some damage in the more northern

FIG. 67.—Snowmold damage on a golf green, photographed early in the spring.

sections of North America and Europe. The many species associated with decaying roots and crowns of the grasses apparently develop as secondary or saprophytic organisms on the weakened or dead tissues, according to Sprague (1944) and others. Spikelet and spike blight caused by *Gibberella zeae* (Schw.) Petch [*Gibberella saubinetii* (Mont.) Sacc.] occurs sparingly on some grasses in the humid, warm sections of the corn belt and other humid regions.

12. Take-all, *Ophiobolus graminis* Sacc.—Take-all sometimes occurs on native grasses in Canada and Western United States and in Australia.

Evidence has been secured that indicates that it was indigenous on the grasses in these regions, spreading to wheat later. The disease is discussed in detail in Chap. XI.

13. Helminthosporium Foot Rots and Leaf Blights.—Some 30 species of *Helminthosporium* occur on the grasses in various parts of the world, as reported by Drechsler (1923, 1929, 1930, 1935), Henry (1924), Hynes (1937), Nishikado (1929), and Sprague (1944). Some, such as *Helminthosporium sativum* Pamm., King, and Bakke, are of major importance in the economy of grass production, others are more restricted in distribution and in the grasses attacked and are potential hazards in the production of susceptible lines of the grasses only.

Helminthosporium sativum Pamm., King, and Bakke probably ranks as the species of first importance on the grasses. The general symptoms, morphology, and etiology of diseases caused by this cosmopolitan species are discussed in Chaps. III and XI. The parasite causes seedling blight and root rot on a wide range of grass species, especially in the central prairie area of North America. According to Andrews (1943), Christensen (1922), and others it is probably the most important seedling and root parasite on the grasses in the humid North Central area. This fungus is exceeded in importance only by *Pythium* spp. in the drier prairie areas of the United States and Canada, as summarized by Sprague (1944). The disease also occurs on grasses in other countries. Both Pythium and Helminthosporium root rots are important in reducing the stands in many of the economic grasses. The Poas are resistant to *H. sativum*, but stands in *Poa pratensis* L. are damaged, especially in the more southern range of this grass, by another species, *H. vagans* Drechs.

Helminthosporium vagans Drechs. causes a leaf spot and culm and crown rot of *Poa pratensis* and some other species of this genus. The organism is widely distributed in North America, and it is common in Europe. The bluish-brown leaf spots are numerous on the leaves. The diffuse brown lesions on the base of the culm and crown are abundant from midsummer to autumn (Fig. 68). Young plants, especially seedlings in old sod, are killed, and older plants are weakened. The conidia are cylindrical to slightly tapering toward the apex, rounded at the ends, measure 8-10 by 50-280 microns, and are dark olivaceous in color. They germinate by the formation of germ tubes from the end and middle segments. Blue grass strains show differences in susceptibility. *H. poae* Baudys occurs on *Poa secunda* Presl. and *P. trivialis* L. in North America.

Pyrenophora bromi Died. (*Helminthosporium bromi* Died.) is common on *Bromus inermis* Leyss and some other species of the genus. The disease appears as small dark-brown oblong spots on the first leaves to develop in the spring and continues until mid-summer (Fig. 68). The older lesions are surrounded by a yellow margin. Severely infected leaves

turn yellow and dry out. Conidia form sparsely on the lesions and withered leaves. Perithecia initials form in the lesions during the summer, and mature ascospores develop the following spring. The conidia are cylindrical with rounded ends, measure 14-26 by 45-265 microns, are 1- to 10-septate without pronounced constriction at the septations, and

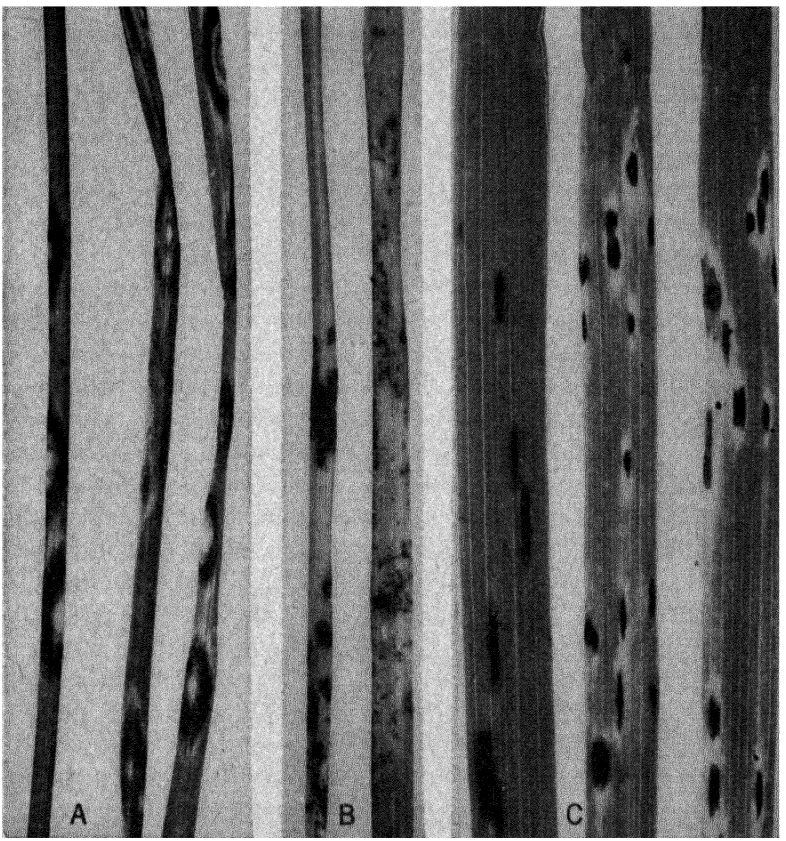

FIG. 68.—(*A*) Leaf spots caused by *Helminthosporium vagans* on *Poa pratensis*. (*B*) Leaf blotch caused by *H. dictyoides* on *Festuca elatior*. (C) Brown leaf spot caused by ***Pyrenophora bromi*** on ***Bromus inermis***.

germination occurs from the middle and end cells. The ascospores are light brown, measure 20-30 by 45-72 microns, are uniformly divided by three transverse septa, and longitudinal septa occur in one or both of the middle cells (Chamberlain and Allison, 1945, and Drechsler, 1923). The disease is generally more abundant in nursery or seed fields than in meadows. Variation occurs in the reaction of *B. inermis* strains to the organism.

Pyrenophora tritici-repentis Died. (*Helminthosporium tritici-repentis* Died.) is widely distributed on some *Agropyron* and *Elymus* spp. The disease is found more commonly in North America on *Agropyron repens* (L.) Beauv. The leaf spots are indefinite brown blotches with conidia on the older areas. The brown lesions around the base of the culms are not defined clearly. The conidia are cylindrical, straight, the basal cell tapering sharply to the hilum, and measure 7-9 by 80-220 microns. The ascospores are light brown, with three transverse and two longitudinal septa, and are 18-28 by 45-70 microns in size.

Three species of *Helminthosporium* occur on the creeping bent grasses and redtop (*Agrostis* spp.). *Helminthosporium stenacrum* Drechs. causes a mild leaf blighting without definite pigmented spots. The conidia are hyaline to yellow, cylindrical with rounded ends, and occasionally produce narrowed distal prolongations. They are 15-23 by 53-135 microns in size, and germinate from several or all segments. *H. erythrospilum* Drechs. is distributed widely in North America on *Agrostis* spp. on which it produces small reddish-brown or russet spots followed by the killing of the leaves. The conidia are yellow to light olivaceous in color, straight cylindrical with abruptly rounded ends, measure 8-16 by 25-105 microns, and germinate from any or all cells. *H. triseptatum* Drechs. causes straw-colored leaf spots and some wilting of leaves on the *Agrostis* spp. The disease occurs sparingly on velvet grass, *Holcus lanatus* L.; orchard grass, *Dactylis glomerata* L.; redtop, *Agrostis gigantea* Roth.; and timothy, *Phleum pratense* L. The conidia are dark olivaceous in color, ellipsoidal or short cylindrical, sometimes tapering toward the base, with hemispherical end cells, 2- to 3-septate, measure 15-21 by 35-50 microns, and germinate from the basal cell.

Helminthosporium giganteum Heald and Wolf produces a zonate eye spot with brown margins and tan centers on the foliage and inflorescence of a large number of grasses, especially in the warmer climates of North America. The huge conidia are pale brown, cylindrical, slightly tapering to both ends, with rounded apical cells, 5-septate, measure 15-21 by 300-315 microns, and germinate from the middle and end cells.

Helminthosporium siccans Drechs. occurs on several cultivated species of *Lolium* in North America and Europe. The dark-brown elongated spots coalesce, forming mottled discolored areas, drying of the leaf blades and finally of the sheaths. The inflorescences are spotted and reduced in development. The conidia are yellow to light brown, straight, slightly tapering toward the apex, with rounded end cells, measure 14-20 by 35-130 microns, and germinate from middle and end cells.

Helminthosporium cynodontis Marig. occurs abundantly on the same grasses as *H. giganteum* and causes leaf withering and bleaching. The conidia taper toward both ends, are generally curved and 3- to 9-septate,

measure 11-14 by 27-80 microns, and germinate from the end cells only.

Helminthosporium dictyoides Drechs. produces a brown net blotch on the leaves of *Festuca elatior* L. The disease is widely distributed in North America, and when severe it causes spotting, browning, and wilting of the leaves (Fig. 68). The conidia are yellow, straight, tapering gradually to the apex, typically 3- to 5-septate, 14 to 17 microns wide at the base by 23 to 115 microns long, and germinate from the end cells.

Helminthosporium turcicum Pass. causes leaf blight and defoliation of Sudan grass and Johnson grass. The races of the parasite on these grass sorghums do not infect corn readily. The morphology of the fungus is given in Chap. IV.

Helminthosporium ravenelii Curt. causes sooty spike on several species of *Eragrostis* and *Sporobolus* and probably on *Panicum*. The disease is widely distributed in the warmer, humid climates. The conidia are straight or sigmoid curved, tapering somewhat more at the base, rounded at the ends, usually 3- to 4-septate, measure 12-19 by 22-78 microns, and germinate from the apical cells. Nishikado (1929) listed *H. miyakei* Nishikado as an additional species causing sooty spike in Asia on *Eragrostis pilosa* (L.) Beauv.

A few of the small-spored types (*Curvularia*) occur commonly on the grasses in addition to those described on wheat (Chap. XI). *Curvularia geniculata* (Tracy and Earle) Boed. is common on the dead tissues of the grasses. Under favorable conditions, root and crown tissues are invaded. The conidia form in dense clusters on the conidiophores. They are curved more on one side than on the other, taper toward both ends, generally 4-septate with the center cell larger and darker colored, and measure 7-14 by 19-45 microns.

The *Helminthosporium* spp. on the grasses are differentiated into two groups, as discussed by Drechsler (1934), Ito and Kuribayashi (1931), and others. In the species with the light-colored epispore wall, generally cylindrical conidia, and germinating from the middle and end cells of the conidia, the ascigerous stages are in the genus *Pyrenophora* (*Pleospora*). In contrast, the species forming conidia with darker olivaceous epispore wall, conidia tapering toward the ends, and germination from the apical cells only, the perithecial stages are in *Cochliobolus* (*Ophiobolus*). A few species are associated with *Leptosphaeria*.

14. Anthracnose, *Colletotrichum graminicolum* (Ces.) G. W. Wils.—The disease on the grasses occurs primarily as a root, crown, and culm rot, with the exception of Sudan grass on which it causes a zonate leaf spot. Considerable damage results on old sod in areas of light soils and depleted fertility. Anthracnose is discussed in Chaps. VIII and IX.

15. Mastigosporium Leaf Fleck.—Leaf fleck occurs in spring and autumn on a large group of grasses. Numerous purplish-brown flecks

occur on the leaves. The lesions elongate and coalesce to form irregular blotches. The center of the mature lesion is lighter colored, and groups of glistening white to gray conidia are conspicuous on these areas. In Europe, *Mastigosporium album* Riess is the predominating species. The oblong conidia are 3- to 5-septate, with several long bristle-like append-

FIG. 69. Scald caused by *Rhynchosporium secalis* on *Bromus inermis*.

ages arising from the apical cell, measure 14-18 by 40-62 microns, and are borne on short thick conidiophores. The variety of this species on orchard grass in Europe forms smaller conidia without appendages (Sampson and Western, 1941). According to Sprague (1938, 1940), the muticate type without appendages, commonly occurring on *Agrostis*, *Calamagrostis*, and *Dactylis* spp. in North America, is referred to another species, *M. rubricosum* (Dearn. and Barth.) Sprague. *M. album* var. *calvum* Ell. and

Davis, *M. album* var. *muticum* Sacc., *M. calvum* (Ell. and Davis) Sprague, *Fusoma rubicosa* Dearn. and Barth., and *Amastigosporium graminicolum* Bond-Mart. are synonyms. The conidia are usually 3-septate without appendages. A third species, *M. cylindricum* Sprague, on *Bromus vulgaris* (Hook.) Shear occurs in North America. The conidia of this species are cylindrical with blunt rounded ends and are much smaller than in the others. The leaf fleck diseases, when severe, defoliate susceptible lines of the grasses.

16. Leaf Scald, *Rhynchosporium secalis* (Oud.) J. J. Davis and *R. orthosporum* Caldwell.—Scald is widely distributed on several genera of the grasses, as well as on barley and rye. Races of *Rhynchosporium secalis* (Oud.) J. J. Davis, the species causing scald on the cereals, are of major importance on *Bromus*, *Agropyron*, and *Elymus* in Northwest and North Central North America, Europe, and Asia. Specialized races occur on these grasses and cereals, as discussed by Caldwell (1937) (see Chap. III). The leaf scald on *Bromus inermis* Leyss is severe during the early spring and late autumn (Fig. 69). Lines vary greatly in their reaction to the disease. *R. orthosporum* Caldwell is differentiated from the former species by the longer conidia and the absence of the beak on the apical cell. This species occurs in localized areas on *Alopecurus*, *Calamagrostis*, and *Dactylis* spp.

17. Streak or Brown Leaf Blight, *Scolecotrichum graminis* Fckl.—Many grasses are susceptible to this disease, which is distributed widely in the temperate zones. *Bromus inermis* Leyss is among the few economic grasses resistant to the disease. The grayish-brown to dark-brown linear lesions occur on the leaf blade and extend into the leaf sheath. Defoliation results from the lateral spread of the infection, especially in the leaf sheath. In the mature lesions, the dark-gray masses of conidiophores, arranged in rows as they emerge through the stomata on the upper surface of the leaf, are the distinguishing symptom of this leaf blight (Fig. 70). The conidophores are fasciculate, olive-gray, unbranched, irregular in shape, and occur in dense clusters in the stomatal openings. The conidia are elongate with brown to olive-brown slightly tapering ends and are aseptate. Several species of *Scolecotrichum* have been described on the grasses, although most authors recognize the one species *S. graminis* Fckl. Von Hohnel transferred this species to the genus *Passalora* on the basis that the type species of the genus *Scolecotrichum* was founded on a misconception. Horsfall (1929), on the basis of the description of *Cercospora graminicola* Tracy and Earle (1895), included the species under this binomial.

18. Ascochyta Leaf Spot.—Several species of *Ascochyta* occur on the grasses (Grove, 1935, 1937, Sprague, 1943). The indefinite spots of variable color appear on the older leaves and culms. The light-brown

lens-shaped or globose pycnidia with the pores opening to the surface form in groups within the tissues on these spots. The pycnidial wall is usually thin and frequently poorly defined at the base. The spores are

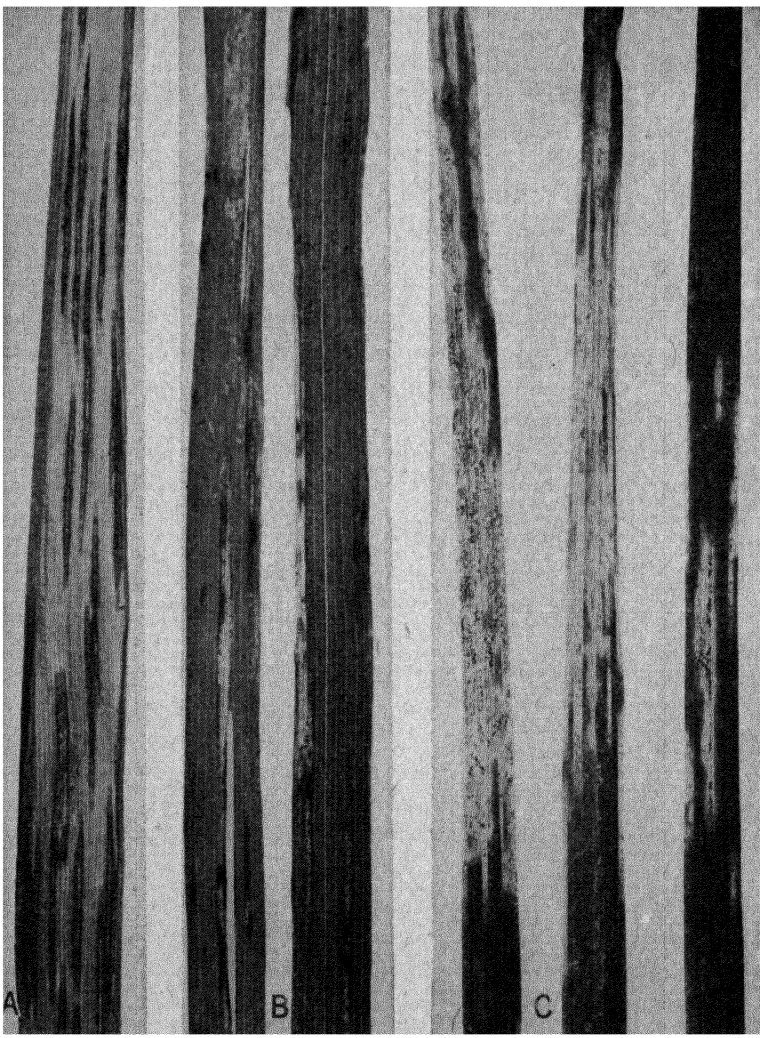

FIG. 70.—Streak or brown leaf spot caused by *Scolecotrichum graminis* on *Phleum pratense* (*A*), *Dactylis glomerata* (*B*), and *Festuca elatior* (*C*).

hyaline to yellow, 1-septate, cylindrical to fusoid, and relatively short. Several species occur on the grasses. *Ascochyta graminicola* Sacc. and *A. agropyrina* (Fairm.) Trott. are the more common species. This group of leaf spot diseases occurs chiefly on the mature tissues of many grasses, and most of them are of minor importance.

19. Stagonospora Leaf Spot.—The pycnidia form on the older leaves and culms with or without a conspicuous lesion. The pycnidia are globose to flattened, dark colored, and immersed or partly projecting through the epidermis often with a pore at the apex. The spores are hyaline, oblong, fusoid, or ellipsoid with two or more septa. *Stagonospora arenaria* Sacc. is distributed widely on the grasses.

20. Septoria Leaf Blotch, *Septoria* Spp.—Numerous species of *Septoria* occur on the grasses and cereals, especially in the temperate and subtropical zones. These leaf blotches occasionally cause defoliation and reduce seed yield. The irregular blotches are straw-colored to brown with dark-brown to black pycnidia on the older portions of the lesions. In many instances, the blotches are similar to those produced by *Stagonospora* or *Ascochyta*. Likewise the morphological distinctions between the three genera on the grasses are not clearly defined, as pointed out by Sprague (1944). *Septoria* spp. generally are more aggressive as parasites on the cereals and grasses than those of closely related genera.

In the genus *Septoria*, the pycnidia are subepidermal, slightly erumpent, and are formed in the older portions of the flecks or spots on leaf, culm, and inflorescence of the Gramineae (Frandsen, 1943, Grove, 1935, 1937, and Sprague, 1944). The pycnidia are globose to lens-shaped, brown to black, ostiolate, parenchymatous, comprising the outer several layers of brown polygonal cells and the inner layers of subhyaline to hyaline flattened or bulbous cells differentiating conidiophores. The conidia (pycnospores) are hyaline to chlorinous, nonseptate to multiseptate, predominantly at least ten times as long as broad, straight to curved, and fusiform, filiform, or scolecosporous. Some of the species occurring on the grasses are listed in addition to those given under the respective cereal crops. Sprague (1944) gives a more detailed description of these and other species. He (1943) has differentiated the wider multiseptate yellowish-brown to light-brown spore color of the Septoria-like types on the grasses and transferred these to the genus *Phaeoseptoria*.

Septoria tritici Rob. on *Triticum* spp. and the varieties *S. tritici* f. *avenae* (Desm.) Sprague on oats, *S. tritici* var. *lolicola* Sprague and A. G. Johnson on *Lolium* spp., and *S. tritici* f. *holci* Sprague on *Holcus lanatus* L. are long-spored types (see Chap. XI). *S. passerinii* Sacc. occurs on *Hordeum* spp. and *Sitanion hystrix* (Nutt.) J. G. Smith (see Chap. III). *S. secalis* var. *stipae* Sprague occurs on *Stipa* and *Agrostis* spp. *S. bromi* Sacc. is distributed widely on *Bromus* spp., and it occurs early in the spring, decreases in abundance during the hot summer, and reappears again in the autumn. The conidia are slender, whip-like, usually 2-septate and stouter in the late autumn. This leaf blotch is not so abundant as *Selenophoma bromigena* (Sacc.) Sprague and A. G. Johnson on *Bromus inermis* Leyss in Central North America (Fig. 71). *S. jaculella* Sprague

with straight, 2- to 5-septate conidia occurs on *Bromus* spp. in the Western United States. *S. macropoda* Pass. and varieties are distributed widely on *Poa* spp. The pycnidia are flattened to globose, light brown, and produce small 1- to 3-septate filiform conidia. A second species, *S.*

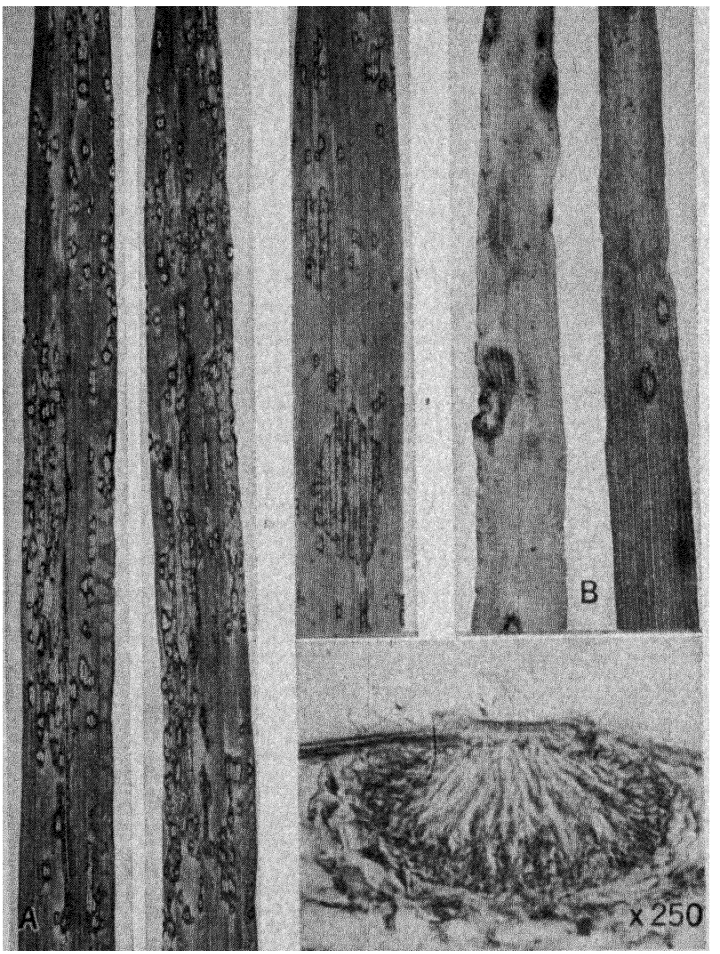

FIG. 71.—Typical leaf lesions of *Selenophoma bromigena* (*A*) and *Septoria bromi* (*B*) on *Bromus inermis* and pycnidium of *S. bromi*.

oudemansii Sacc., is common on *Poa* spp., and it is distinguished from the former by the lighter golden color and large closely packed cells of the pycnidia. The conidia are mostly cylindrical, wider, and shorter than in the former species, and usually 1-septate. *S. elymi* Ell. and Ev. (*S. agropyri* Ell. and Ev.) is common on *Elymus* and *Agropyron* spp. The pycnidia are flattened, thin-walled, and brown. The conidia are cylin-

drical to filiform and usually 3-septate. *S. infuscans* (Ell. and Ev.) Sprague (*Cylindrosporium infuscans* Ell. and Ev.) occurs on *Elymus* spp. in Western North America and possibly elsewhere; a long-spored species *Septoria pacifica* Sprague on *Elymus mollis* Trin. occurs in the western region. Other species occur on the grasses (Davis, 1942, Frandsen, 1943, Grove, 1935, 1937, and Sprague, 1944).

The etiology and control are similar to that given for the cereals. Strains of the cultivated grasses show differences in susceptibility to the specific fungi causing these diseases.

21. Selenophoma Leaf Blotch.—Sprague and Johnson (1940, 1945) transferred the species with falcate nonseptate spores to the amended genus *Selenophoma*. *Selenophoma bromigena* (Sacc.) Sprague and A. G. Johnson on *Bromus inermis* Leyss is one of the more common species of this group. The initial lesions appear as small brown flecks on the leaves early in the spring. The spots enlarge to form irregular blotches with translucent older portions in which the black pycnidia develop (Fig. 71). The mature pycnidia frequently drop out of the tissue, leaving small holes in the lesions. The spores are hyaline, narrow, tapering at the ends, slightly sickle-shaped, and nonseptate. Septa are formed during germination on nutrient media. Lines of smooth brome grass show some differences in reaction to the disease (Allison, 1945). *S. donocis* (Pass.) Sprague and A. G. Johnson and related forms occur on many species of grasses in North America.

22. Rhizoctonia Root and Crown Rot.—Root rot, crown rot, and eye spot lesions occur on the lower leaf sheath tissues and culms of many grasses. The disease appears in localized spots early in the growing season. Brown patch of turf grasses in which the crown and leaf tissues are killed in local spots is a severe manifestation of the disease. The snow mold type of injury also occurs occasionally. *Rhizoctonia solani* Kuehn and other species of this genus are associated with the disease (Monteith, 1926, Sprague, 1944). Control of the disease on turf grasses is obtained by fungicides and the use of resistant strains in the creeping bents (Broadfoot, 1936, Monteith and Dahl, 1932). The disease is discussed in more detail in Chap. XI.

23. Typhula Snow Mold or Snow Scald.—The snow mold caused by several species of *Typhula* is common on the grasses early in the spring, especially where the grass has been under a heavy snow covering (Fig. 67). Remsberg (1940) has summarized the literature on the genus and given the morphology of a number of species, including those associated with the disease on grasses. *Typhula itoiana* Imai is apparently the most prevalent and widely distributed species on the cereals and grasses. Wernham (1941) reported a difference in the reaction of bent strains to this disease. Several resistant to *Rhizoctonia* and *Fusarium* were sus-

ceptible to *Typhula*. Andrews (1944) and Broadfoot and Cormack (1941) reported high- and low-temperature Basidiomycetes, respectively, different from those above which cause severe damage to grass crowns of *Agropyron cristatum* (L.) Beauv. in Minnesota and westward during the summer in the former and to certain legumes and grasses early in the spring in Western Canada in the latter.

24. Leaf Smuts, *Ustilago and Urocystis* Spp.—Numerous smuts occur on the grasses (Clinton, 1906, Fischer and Hirschhorn, 1945, Liro, 1924, 1938, and Zundel, 1939, 1946). Like those present on the cereals, the spore-bearing sori are more or less specific to certain morphological parts of the grasses, such as, the leaves, culms, inflorescences, and caryopses; in some species the sori are formed in gall-like structures on the young tissues of any part of the plant. As an aid in recognition of the smuts and on the same basis used in the cereals, the grass smuts are grouped, primarily on the basis of symptoms, as presented by Fischer and Hirschhorn (1945).

The leaf smuts of the grasses appear chiefly in the leaf blades and leaf sheaths. These smuts are subdivided on the basis of symptoms into stripe smut, flag smut, and spot smut. While sori frequently develop in the inflorescences of the infected plants, the latter structures generally fail to develop and emerge from the leaf whorl, and therefore, they are inconspicuous on the smutted plants. The sori are linear, forming as long or short stripes between the veins of the leaves, depending upon the species involved. After the leaf epidermis is ruptured and the spores are discharged, the leaf tissues frequently present a brown shredded appearance. Chlamydospores in the shredded tissues aid in the identification of the fungus species. The sori in the leaf spot smuts are covered by the epidermis and are more permanent. The etiology of this group of smut fungi on the grasses is similar. Seedling or young crown-bud infection is followed by the systemic infection of the primordia. Spore formation occurs in the leaves as the latter become fully developed. In the perennial grasses the mycelium usually persists in the crown tissues and dormant buds over several years. In some strains of stripe smut, infection of bud primordia occurs in established perennial plants. Seed infection occurs in some grasses (Fischer, 1940). Two leaf smuts are distributed generally on a large number of the important pasture and turf grasses.

Stripe smut caused by *Ustilago striiformis* (West.) Niessl occurs commonly on redtop (*Agrostis gigantea* Roth), the creeping bents (*A. palustris* Huds. and *A. tenuis* Sibth.), timothy (*Phleum pratense* L.), and blue grass (*Poa pratensis* L.). It is less widely distributed on certain other *Agropyron*, *Agrostis*, *Beckmannia*, *Elymus*, *Festuca*, *Holcus*, *Lolium*, *Poa*, and *Sitanion* spp. and some other grasses. Physiologic races are

distinguished on certain of the grasses (Davis, 1930, Fischer, 1940). Resistant strains occur in some of the grasses. The sori form as long,

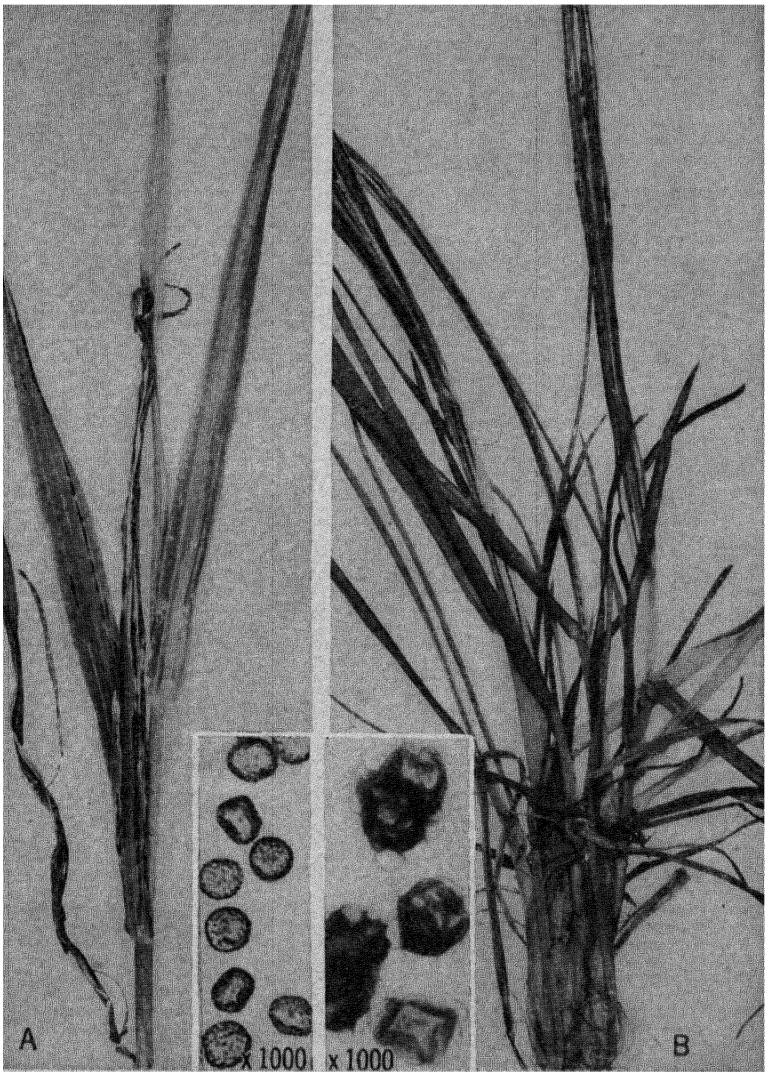

FIG. 72.—Stripe smut on *Phleum pratense* (*A*) and flag smut on ***Poa pratensis*** (*B*) caused by *Ustilago striiformis* and *Urocystis agropyri* respectively. The chlamydospores of the two species are shown highly magnified in the inserts.

narrow almost black stripes in the leaves and leaf sheaths (Fig. 72). The leaves are shredded after the dispersal of the spores. Internodal elongation and the development of the inflorescence is restricted in many grasses.

The spores are globose to ellipsoid, dark olive brown, prominently echinulate, and chiefly 9 to 12 microns in diameter. Spore germination occurs directly or after a resting period with the formation of a branched promycelium and less frequently sporidia (Davis, 1924, Fischer, 1940, Kreitlow, 1943, 1944).

Flag smut caused by *Urocystis agropyri* (Preuss) Schroet. is manifest especially in the upper leaves of a somewhat similar group of grasses as in the former species. This smut is widely distributed but occurs less abundantly than the former (Fig. 72). The spore balls are globose to elongate and are composed of one to four reddish-brown smooth fertile spores surrounded by smaller hyaline to light-brown sterile cells. The spore germination in this species is erratic, but is probably similar to that of the species occurring on wheat and rye. Fischer and Hirschhorn (1945) included the morphologically similar *U. tritici* Koern. and *U. occulta* (Wallr.) Rab. as races under *U. agropyri*.

Brown stripe smut caused by *Ustilago longissima* (Schlecht.) Meyen is generally distributed on species of *Glyceria*. The long numerous sori in the leaves are light brown in contrast to the darker lesions of the former smuts. Spores are globose to elongate, golden yellow to light olivaceous, minutely echinulate, and 7 to 9 microns in diameter. *U. aculeata* (Ule) Liro occurs occasionally on *Agropyron* and *Elymus* spp. and cannot be distinguished macroscopically from the common stripe smut. The spores are dark brown, ovoid to globose, 12 to 19 microns in diameter, coarsely verrucose, and germinate with a branched promycelium. *U. echinata* Schroet., occurring sparingly on Reed canary grass (*Phalaris arundinaceae* L.) is similar to the large-spored type previously described.

Leaf spot smuts caused by *Entyloma* spp. occur on many of the grasses throughout the world. The sori are formed in the leaves and less frequently in the floral bracts, and they appear as tar-like angular to oblong spots or blisters covered by the epidermis of the suscept. This group of *Entyloma* spp. is relatively similar in morphology, and confusion exists in the specific names used. The chlamydospores are angular to globose, 6 to 14 microns in diameter, closely packed, usually smooth walled, light brown to reddish brown in color, and germinate, frequently in place, by the formation of a promycelium with terminal sporidia (Chap. VII). *Entyloma lineatum* (Cke.) J. J. Davis is common on *Zizania aquatica* L. and rice in North America and Europe. *E. irregulare* Johans on *Poa* spp. is distributed widely in Europe and North America, especially in the Northwest. *E. crastophilum* Sacc., similar and perhaps synonymous with *E. irregulare*, occurs over the same area as the former on *Phleum* and *Agrostis* spp. and some other grasses.

25. Culm Smuts.—The culm smuts are found chiefly in Western North America and in somewhat similar habitats in South America, Europe,

North Africa and Asia. Four species are differentiated on the grasses by Fischer and Hirschhorn (1945).

Ustilago spegazzinii Hirsch. [*U. hypodytes* (Schlecht.) Fr.] and the

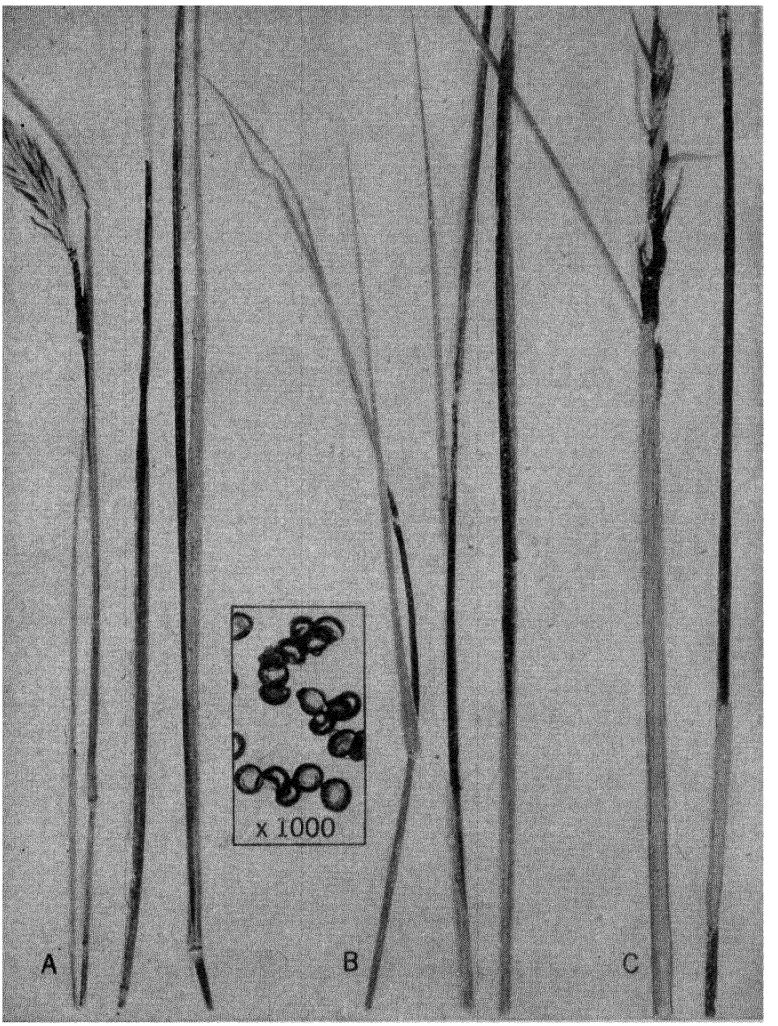

FIG. 73.—Culm smut caused by *Ustilago spegazzinii* on (*A*) *Agropyron cristatum*, (*B*) *A. trichophorum*, and (*C*) *A. elongatum*. The chlamydospores are shown highly magnified in the insert.

variety *U. spegazzinii* var. *agrestis* (Syd.) G. W. Fisch. and Hirsch. occur on *Agropyron, Elymus, Poa*, and some other genera. The sori are chiefly superficial on the internodes of the culms and sometimes on the aborted inflorescences. The naked linear dark-brown to black sori are covered

by the leaf sheaths (Fig. 73). The spores are yellowish brown to olivaceous, globose to angular, with bipolar subhyaline crests of epispore echinulations; the remainder of the epispore wall is minutely echinulate. The chlamydospores are 4 to 6 microns in diameter and germinate by the formation of a branched promycelium and aerial sporidia. According to Bond (1940) and Fischer (1945), the mycelium persists in the crown and stolon tissues of the perennial grasses. Infection occurs in crown-bud primordia, and sori appear from two to three seasons following infection. This may be associated with the period required for the grass primordia to develop into culms, as suggested by Bond (1940). The increase of smut in permanent stands of grasses makes this an important disease in areas where inoculum is present. Some selections of crested wheat grass [*Agropyron cristatum* (L.) Beauv.] apparently are resistant.

Ustilago williamsii (Griff.) Lavrov, formerly included under *U. hypodytes*, occurs on *Oryzopsis* and *Stipa* spp. in Western North America and Argentina. The symptoms are similar to those caused by *U. spegazzinii*. Spores are globose to subglobose, dark olivaceous brown, and the epispore is smooth, but deeply cracked and bearing two bipolar cap-like appendages. Germination occurs readily with the formation of a promycelium and sporidia. Culm smut caused by this species appears earlier after infection than in the former species.

Ustilago halophila Speg. produces a culm smut on *Distichlis* spp. in the drier sections of North and South America and Australia. The plants are dwarfed, and the sori are covered early by the epidermis of the culm. The spores are globose to irregular, yellow brown to olive brown, smooth, without bipolar areas or appendages, and 5 to 7 microns in diameter. Spore germination is typically by the formation of a promycelium and sporidia.

Ustilago nummularia Speg. occurs on *Ammophila*, *Oryzopsis*, and *Stipa* spp. in Western and Intermountain North America, Argentina, and Northern Europe. Sori are naked on the internodes and into the inflorescences. Spores are smaller than the former species and germinate by means of a branched promycelium without sporidia.

26. Head Smuts.—The smuts forming sori in grass inflorescences are numerous and include some of the species occurring on the cereal crops. These smuts on the grasses are more abundant as a group in Western North America, although certain species are widely distributed. The head smuts, as grouped, include those producing spores in the inflorescence as a whole or those in which the floral bracts of the spikelets as well as the ovaries are involved. These are differentiated from the kernel smuts in which the sori usually are formed only in the ovaries. Only the smuts of major economic importance are included.

Ustilago bullata Berk., causing the common head smut on a large number of grasses, results in major losses in many economic species, as reviewed by Fischer (1940). This smut is less common in the North Cen-

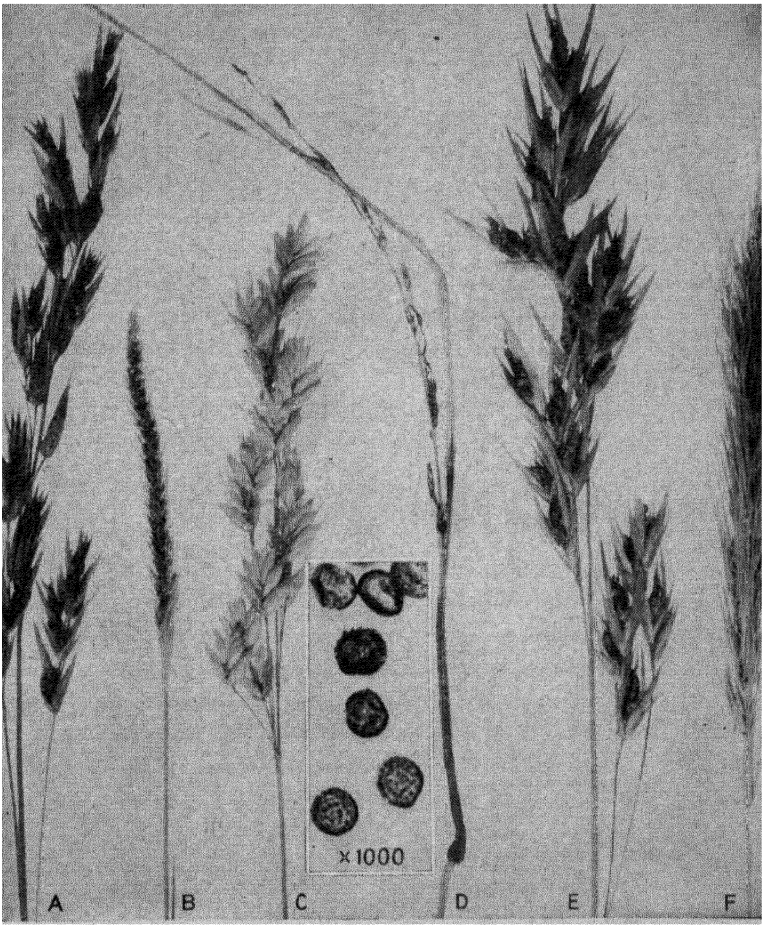

FIG. 74.—Head smut of grasses caused by *Ustilago bullata* showing the range in symptoms on (*A*) *Bromus carinatus*, (*B*) *Agropyron pauciflorum*, (*C*) *Bromus hordeaceus*, (*D*) *B. polyanthus*, (*E*) *B. catharticus*, and (*F*) *Elymus canadensis*. The chlamydospores, highly magnified, are shown in the insert.

tral and Eastern United States, and apparently it is restricted to the drier areas in other countries as well as in North America. The sori formed in the spikelets involve all or part of the floral bracts. They are enclosed in the epidermal membranes of the floral structures, which persist in varying degrees in the different grasses (Fig. 74). The spore mass is

dark brown to purple black and loose to semicovered in type, depending upon the grass species. The spores are globose to irregular, dark brown to olive brown, minutely echinulate to verrucose, 5 to 14 but more often 7 to 9 microns in diameter, and germinate to form a promycelium and sporidia. Seedling infection occurs, and in some of the perennial grasses the disease persists for several years. Fischer (1940) differentiated eight physiologic races and reported resistant lines in some grasses. Parker (1942) reported resistant lines in *Bromus catharticus* Vahl. (*B. unioloides* H.B.K.). This head smut is attributed to three species, similar morphologically, by Zundel (1939, 1946) and others: *U. bromivora* (Tul.) Fisch. de Waldh., *U. bullata*, and *U. lorentziana* Thuem. These are considered synonyms of *U. bullata*.

Ustilago mulfordiana Ell. and Ev. occurs on *Festuca* spp. in the Intermountain states and in Western North America. Sori form in the aborted inflorescences and are concealed partly by the enveloping leaves. Spores are dark olivaceous brown, globose to ovoid, coarsely echinulate, and 12 to 19 microns in diameter.

Ustilago sitanii G. W. Fisch. produces a brown loose smut of the inflorescences and linear sori in the upper leaves of *Sitanion* and *Hordeum* spp. Spores are globose to ovate, light brown, minutely echinulate, 4 to 5 microns in diameter, and germinate to form a promycelium without sporidia.

Sorosporium syntherismae (Pk.) Farl. is distributed widely on *Cenchrus* and *Panicum* spp. The sorus is covered by a fungus membrane when young, which later ruptures, releasing the dark spore mass. The entire inflorescence is involved in the sorus, and later only the vascular elements remain. Spore balls are globose to oblong, dark brown and persist until the sori are mature. Spores are globose to elongate, brown, minutely verrucose, 8 to 11 microns in diameter, and germinate to form a promycelium and sporidia.

27. Kernel Smuts.—The more important kernel smuts of the grasses are those caused by species of *Tilletia*. These smuts occur on the grasses of the Western and Intermountain areas of North America and similar areas in other countries (Fischer and Hirschhorn, 1945). They are of economic importance on some of the better range and forage grasses. The sori form in the ovaries and assume the general shape and color of the caryopses of the species. The *Tilletia* spp. on some of the more important grasses are described briefly. Some species of *Sphacelotheca* cause kernel smuts on the grasses.

Sphacelotheca sorghi (Lk.) Clint. is distributed widely on Sudan grass and other sorghum grasses (see Chap. IX).

Tilletia guyotiana Hariot on *Bromus* spp. Spores are yellow to chest-

nut, deeply reticulate, and 21 to 24 microns in diameter. The sterile cells are hyaline, smooth, and 14 to 18 microns in diameter.

Tilletia fusca Ell. and Ev. on *Festuca* spp. Spores are dark, reddish brown, deeply and prominently reticulate, and 21 to 27 microns in diameter. Sterile cells are grayish yellow, smooth, and 12 to 20 microns in diameter.

Tilletia cerebrina Ell. and Ev. on *Deschampsia* spp. Spores are grayish brown, reticulate with the reticulations varying in size and shape, and 21 to 25 microns in diameter. The sterile cells are hyaline to greenish yellow and 12 to 17 microns in diameter.

Tilletia elymi Diet. and Holw. on *Elymus glaucus* Buckl. Spores are light olive brown to dark violaceous brown, globose to subglobose, and deeply reticulate. Reticulations are variable in size and shape and the spores are 21 to 28 microns in diameter. Sterile cells are hyaline to greenish yellow, smooth, thin-walled, and 17 to 21 microns in diameter.

Tilletia holci (West.) Schroet. on *Holcus lanatus* L. is similar in spore morphology and size to *T. cerebrina*.

Tilletia asperifolia Ell. and Ev. on *Muhlenbergia asperifolia* (Nees and Mey.) Parodi. Spores are yellowish to dark brown, 17 to 21 microns in diameter, and are enveloped by a thin hyaline membrane, distinctly reticulate to cerebriform. The sterile cells, larger than the spores, are hyaline, and the walls are laminated.

T. caries (DC.) Tul. occurs on the wheat grasses (see Chap. XI).

Many other smuts occur less generally on the cultivated and wild grasses (Clinton, 1906, Fischer and Hirschhorn, 1945, Zundel, 1946) (see also Chaps. III, V to IX, and XI).

28. Stem Rust, *Puccinia graminis* Pers.—Stem rust is common on many of the grasses. The uredia develop on the leaves and culms, and the telia generally form on the leaf sheaths and culms. The symptoms and etiology are the same as on the cereal crops (see Chaps. VI and XI). The specialized varieties and physiologic races occurring on the cereals as well as *Puccinia graminis agrostidis* Eriks., *P. g. phlei-pratensis* (Eriks. and Henn.) Stakman and Piemeisel, *P. g. poae* Eriks. and Henn., and other varieties cause damage on the grasses. Both hay and seed production in many of the economic grasses are reduced by stem rust. Some species, *e. g.*, *Bromus inermis* Leyss, are resistant to stem rust. The reaction of certain of the grasses to *P. graminis*, *P. rubigo-vera* (DC.) Wint., *P. glumarum* (Schm.) Eriks. and Henn., and *P. coronata* Cda. is given in the following table.

29. Leaf Rusts.—The leaf rusts on the grasses are caused by several species, the most important being *Puccinia rubigo-vera* (DC.) Wint. Mains (1932) combined a large number of morphologically similar leaf

The RANGE in REACTION of SOME GENERA and SPECIES of the GRASSES to *Puccinia graminis* and SPECIALIZED VARIETIES, *P. rubigo-vera*, *P. glumarum*, and *P. coronata* in the UNITED STATES and CANADA*

Genera of grasses or species	Varieties of *P. graminis*						*P. graminis*†	*P. rubigo-vera*	*P. glumarum*	*P. coronata*
	agrostidis	*avenae*	*phlei-pratensis*	*poae*	*secalis*	*tritici*				
Aegilops (5 species)	...	...	...	...	...	...	S	R-S	S	
Agropyron (33 species)	...	R	R	...	R-S	R-S	R-S	R-S	R-S	R-S
Agrostis (19 species)	S	R-S	...	...	R	R	R-S	R-S	...	R-S
Alopecuris (6 species)	S	R-S	...	..	R	R	R-S	R-S	...	R-S
Ammophila arenaria (L.) Link	..	S	..	..	.	..	S	...	...	R-S
Andropogon (2 species)	..	R	...	...	...	...	R	...	...	R
Anthoxanthum (2 species)	R	R-S	..	.	R	R	R-S	....	R	R-S
Arctagrostis latifolia (R. Br.) Griseb.	...	...	...	.	.	..	...	...	....	S
Arrhenatherum elatius (L.) Morland Koch	...	R-S	R-S	....	R	R	R-S	R	R	R-S
Avena (11 species)	....	S	S	....	...	...	S	R-S	...	R-S
Beckmannia (2 species)	....	R	R	....	R	R	R-S	..	R	R-S
Bouteloua (2 species)	...	R	.	...	R	R	R-S	..	....	R
Brachypodium distachyon (L.) Beauv.	..	R	...	...	...	...	R	...	....	R
Briza (3 species)	..	...	...	...	...	...	S	R	...	R
Bromus (39 species)	R-S	R-S	R	....	R-S	R-S	R-S	R-S	R-S	R-S
Buchloë dactyloides (Nut.) Engelm.	...	...	...	...	...	...	S	...	...	
Calamagrostis (9 species)	R	S	..	..	..	R	R-S	.	.	R-S
Calamovilfa longifolia (Hook.) Scrib.	...	.	...	...	...	..	S	..	...	R-S
Catabrosa aquatica (L.) Beauv.	....	.	..	...	...	....	S	...	...	
Chloris (2 species)	....	...	...	...	...	...	...	....	S	R
Cinna (2 species)	....	..	..	...	...	....	S	S	....	R-S
Corynephorus canescens (L.) Beauv.	..	..	...	...	...	....	S	..	..	
Cynodon dactylon (L.) Pers.	....	R	...	...	...	R	R-S	...		R
Cynosurus cristatus L.	...	R	...	...	....	R	R	....	..	R
Dactylis glomerata L.	S	S	R-S	R	R	R-S	R-S	R	R-S	R-S
Dactyloctenium aegyptiacum (L.) Richt.	....	R	...	..	...	...	R	...	...	R
Danthonia (5 species)	...	R	...	...	...	...	R	...	...	R
Deschampsia (6 species)	....	R-S	...	...	...	S	R-S	R	....	R-S
Distichlis spicata (L.) Greene	....	...	...	...	...	...	S	...	....	R
Echinochloa crusgalli (L.) Beauv.	....	R	...	...	...	...	R-S	...	....	R
Elymus (26 species)	....	R	R	....	R-S	R-S	R-S	R-S	R-S	R-S
Eragrostis (2 species)	....	R	R	....	R	R	R	...	....	R
Festuca (25 species)	....	R-S	R-S	....	R	R-S	R-S	R-S	R-S	R-S
Glyceria (6 species)	....	R-S	....	...	...	...	R-S	R-S	....	R-S
Hierochloë (2 species)	....	S	....	...	...	...	R-S	...	....	R-S

The RANGE in REACTION of SOME GENERA and SPECIES of the GRASSES to *Puccinia graminis* and SPECIALIZED VARIETIES, *P. rubigo-vera*, *P. glumarum*, and *P. coronata* in the UNITED STATES and CANADA* (*Continued*)

Genera of grasses or species	Varieties of *P. graminis*						*P. graminis*†	*P. rubigo-vera*	*P. glumarum*	*P. coronata*
	agrostidis	*avenae*	*phlei-pratensis*	*poae*	*secalis*	*tritici*				
Holcus (2 species)	S	R-S	R-S	. . .	R	R	R-S	R-S	. . . .	R-S
Hordeum (10 species)	R	R	R	. . . .	R-S	R-S	R-S	R-S	R-S	R-S
Hystrix (2 species)	. . . .	R	. . .	. . . .	S	S	R-S	R-S	S	R-S
Koeleria cristata (L.) Pers.	S	R-S	S	. . . .	R	R-S	R-S	R-S	S	R
Lagurus ovatus L.	. . . .	. . . .	. . . .	. . . .	. . . .	. . . .	S	. . . .	. . . .	R-S
Lamarckia aurea (L.) Moench.	. . . .	. . . .	. . . .	. . . .	. . . .	. . . .	S	. . . .	. . .	R-S
Limnodea arkansana (Hutt.) Dewey	. . . .	S	. . .	. . . .	. . . .	. . . .	S	. . .	. . . .	S
Lolium (4 species)	. . . .	R	R-S	. . . .	R	R	R-S	R	R	R-S
Melica (6 species)	. . .	. . .	. . .	. . .	. . . .	R	R-S	R-S	. . . .	R
Milium effusum L.	. . . .	S	. . .	. . . .	. . .	. .	S	. . . .	. . . .	
Molinia caerulea (L.) Moench.	. . . .	. . .	. . .	. . .	. .	. . .	S	. . . .	. . . .	R
Muhlenbergia (3 species)	. . . .	R	R	. . . .	R	R	R-S	. . . .	. . . .	R
Panicum virgatum L.	. . .	. .	.		.	S	S	. . . .	. . . .	R
Paspalum setaceum Mich.	. . .	. .	.	.	.	.	.	. . . .	. . . .	S
Phalaris (6 species)	R	R-S	S	. . . .	R	R	R-S	. . . .	S	R-S
Phleum (4 species)	. .	R-S	S	R	R	R	R-S	. . . .	. . . .	R-S
Phragmites communis Trin.	. .	. . .	. . .	. . .	. .	. .	S	. . . .	. . . .	R
Poa (36 species)	. .	R-S	R-S	R-S	.	.	. . .	R-S	R-S	. . . .	R-S
Polypogon monspeliensis (L.) Desf.	. . . .	S	. . .	. . .	.	.	. .	S	. . . .	. . . .	R-S
Puccinellia (6 species)	. .	. . .	. . .	. .	. .	. .	. . . .	S	R-S	. . . .	R
Schedonnardus paniculatus (Nut.) Trel.	. . . .	R	. . .	. .	.	.	R	. .	. . . .	R
Secale montanum Gaus.	. . . .	. . .	. . .	. . .	. .	. .	. . . .	R-S	S	
Sitanion (3 species)	. . . .	R	. . .	. . .	. . . .	S	R-S	R-S	S	
Sorghastrum (2 species)	. . .	. . .	. . .	. . .	. . .	.	R-S	. . .	. . . .	R
Sphenopholis (3 species)	. . . .	R-S	. . .	. . .	. . .	. . . .	R-S	R	. . . .	R
Sporobolus (3 species)	. . . .	R	. . .	. . . .	S	R-S	R-S	R	. . . .	R
Stipa (5 species)	. . . .	R	R	. . . .	R	R-S	S	. .	. . . .	R
Triodia flava (L.) Smyth.	. . . .	R	. . .	. . .	. . .	. . .	R-S	. . . .	. . . .	R
Trisetum (6 species)	. . . .	S	. . .	. . .	. . .	. .	R-S	R-S	S	R-S

* Fischer, G. W. and M. N. Levine. Summary of the recorded data on the reaction of wild and cultivated grasses to stem rust (*Puccinia graminis*), leaf rust (*P. rubigo-vera*), stripe rust (*P glumarum*), and crown rust (*P. coronata*) in the United States and Canada. U. S. Dept. Agr. Pl. Dis. Reporter Supl. 130. 1941 (Mineographed).

† *Puccinia graminis* without designation of race or including all of the physiologic races.

R—resistant or failed to infect; S—susceptible, based on observation or inoculation.

rust species under this binomial. On the basis of the reaction of the grasses to the rusts and in most instances including the reaction of the plants known to be the aecial hosts of these rust parasites, Mains grouped them into specialized varieties of *P. rubigo-vera* using the trinomial system. He listed 56 trinomials for the varieties with uredia and telia on the cereals and grasses and with aecia on species or groups of species of *Thalictrum*, *Clematis*, *Anemone* and *Hepatica*, *Aquilegia*, *Delphinium*, *Ranunculus*, *Actaea*, *Aconitum*, *Anchusa*, *Onosmodium* and *Macrocalyx*, *Symphytum* and *Pulmonaria*, *Lithospermum* and *Myosotis*, *Phacelia*, *Hydrophyllum*, and *Impatiens*, as well as some few with aecia unknown. These specialized varieties of *P. rubigo-vera* cause the more important leaf rusts on the grasses. The urediospores are ovate to globose with germ pores distributed in contrast to the elliptical or pyriform urediospores with equatorial germ pores in *P. graminis*. The telia of *P. rubigo-vera* are covered by the epidermis in contrast to the naked telia in *P. graminis*. See Chap. XI for the detailed morphology. The reactions of some of the grasses to this species are given in the preceding table.

Crown rust caused by *Puccinia coronata* Cda. is distributed widely and is important economically on oats and many grasses (see the preceding table). Several specialized varieties of the fungus occur on the grasses as well as physiologic races. The aecial stages of these varieties are specialized somewhat on *Rhamnus* spp. and on *Berchemia scandens* (Hill) Trel., *Elaeagnus commutata* Bernh., and *Shepherdia canadensis* (L.) Nutt. The morphology and etiology of this species is given in Chap. VI. The digitate projections, forming a crown on the apex of the teliospore, differentiate the fungus from those causing other leaf rusts.

Puccinia poae-sudeticae (West.) Jørstad is distributed widely on many species of *Poa* and a few other grasses. It is prevalent on *Poa pratensis* L. in the humid temperate zones. The aecial stage of this fungus is unknown. The uredia are epiphyllous, orange yellow, with numerous peripheral paraphyses. The paraphyses around the urediospores differentiate this species from *P. rubigo-vera*. The urediospores are similar in the two species. Telia are covered rather permanently by the epidermis, and numerous subepidermal paraphyses are present, especially in the periphery of the telium. The teliospores are oblong or clavate, dark brown, and short-pedicled.

30. Stripe Rusts.—The brown stripe rust caused by *Puccinia montanensis* Ell. occurs on the leaves of *Agropyron* and *Elymus* spp., *Melica imperfecta* Trin., and some few other grasses. The aecia occur infrequently on *Berberis fendleri* Gray. This rust is distributed chiefly through Western North and South America. The uredia on the leaves form narrow long lines, dark reddish brown in color. Numerous clavate paraphyses surround the urediospores. The telia are oblong, grayish brown and

often form narrow lines below the leaf epidermis. This rust is differentiated from the yellow stripe rust by the differences in color, shape, and time of appearance of the uredia and the shape and abundance of paraphyses in the uredia.

The yellow stripe rust caused by *Puccinia glumarum* (Schm.) Eriks. and Henn. is common on many grasses in the Intermountain and Pacific Coast area of North and South America and northern Europe and Asia. The geographic distribution of this rust is limited to the areas of relatively cool climates. Many of the economically important grasses are damaged in early spring by this rust. (See the preceding table for the reaction of grasses.) The uredia form orange-yellow stripes on the leaves and floral bracts, especially during cool cloudy weather. The uredia are orange yellow, with occasional hyphoid paraphyses around the outer edges. Telia form less abundantly than the uredia, and they appear as fine dark lines below the epidermis. The morphology, etiology, and control are discussed in Chap. XI.

Many other rusts occur on the grasses, as listed by Arthur (1934) and others.

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SECTION III

DISEASES OF LEGUMES

CHAPTER XIII

ALFALFA AND SWEETCLOVER DISEASES

The common alfalfa or lucerne, *Medicago sativa* L., is grown extensively for hay and pasture. Varieties of this species constitute one of the most valuable hay and seed crops of the forage legumes. Some few additional species, notably *M. falcata* L., are used as forage crops in some areas (Tysdal and Westover, 1937). Many species, both perennial and annual, occur wild in central and western Asia, southern Europe, and northern Africa. Grossheim (1930) lists 25 species of *Medicago* occurring in the Transcaucasian area. Some of the annual species, introduced from Europe, occur wild in pastures and waste places in North and South America. Some of the diseases described occur on the annual as well as the perennial species.

The genus comprises a polyploid series with a basic chromosome number of 8 pairs (Wipf, 1939). One annual species, *Medicago hispide* Gaertn. is apparently the exception with 7 chromosome pairs. *M. sativa* and *M. falcata* are tetraploid species with 16 chromosome pairs. Polyploids of the former species are reported.

Alfalfa is one of the ancient perennial forage legumes adapted especially to semiarid regions. In this widely diverse cross-pollinated species, strains or varieties occur that are adapted to cold, relatively high summer humidity and other variations in climate. The generally high self-sterility of the plants make the isolation and stabilization of the desirable characters for plant growth, disease resistance, and seed production difficult. Selfing, sib-pollination, and clonal propagation are important in the breeding program and especially in the comparisons of disease reaction.

The perennial species develop a crown of stem branches, including axillary and secondary buds and a tap root. The stems and roots develop secondary thickenings. The annual stems develop from the crown buds. The crown and root tissues constitute the important storage tissues.

Several species of *Melilotus* are used for forage and soil improvement. Varieties of white sweetclover, *Melilotus alba* Desv., yellow sweetclover,

M. officinalis (L.) Lam., and *M. indica* All. are used more commonly. The species studied have eight chromosome pairs. The adaptation of the biennial and annual varieties is relatively wide. The plant structure and general anatomy is similar to alfalfa, and many of the diseases are similar in the two crops.

1. Cold and Winter Injury.—Winter injury is common in alfalfa, especially in the northern range of its cultivation. The crown and root tissues of alfalfa and clover plants are damaged by low temperatures and desiccation, smothering by ice sheets and tissue injury, and heaving by freezing and thawing. Frequently one or a combination of these types

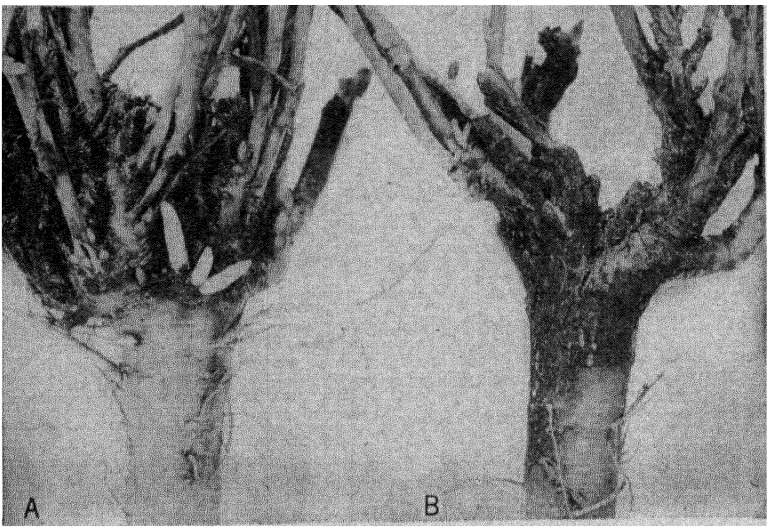

FIG. 75.—Winter injury in alfalfa root and crown. (*A*) Sound. (*B*) injured. (*Courtesy of F. R. Jones.*)

of injury results in winter killing of extensive acreages of these crops. Plants with less severe injury are damaged further by the entrance of fungi and bacteria into the tissues before the injured tissues recover or are repaired by cellular activity (Jones, 1928, Peltier and Tysdal, 1931, Weimer, 1929, 1930) (Fig. 75).

Damage in 1-year old plants is located usually in the phloem and phloem rays or is restricted to the large cells exterior to the phloem fibers. Injury in the exterior of the phloem results in a sheath of damaged tissues surrounding the root, which kills the phellogen or cork cambium exterior to it. Injury in the phloem usually is accompanied by necrosis of the large cells in the xylem rays in the center of the root. In more severe winter damage, portions of the root near the crown are killed completely

or disorganized sufficiently for fungi to enter and rot the tissues, including those of the base of the crown. Under such conditions, the plants fail to recover the following spring.

Damage in plants the second season or later is largely in the parenchymatous cells of the phloem and phloem rays. The location of the injured cells is different than in the first year, however, as they are located inside rather than outside the last group of fibers of the phloem. This injury usually extends inward through the cambium and xylem rays of the previous season's growth and results in breaks in the cambium cylinder, but usually the injury does not extend inward to the center of the root. Damage in the sheath of cells immediately beneath the phellogen occurs independently or in conjunction with phloem injury (Fig. 76).

Injury of the crown stems is similar to that in the roots. The large cells beneath the phellogen show damage first. The ray cells are damaged, and the bundles become separated by these dead cells. Phloem parenchyma is injured as in the root. Crown buds are damaged or killed in some instances. Jones (1945) using clonal populations from a large number of plants has shown differences in type and degree of injury in plant lines and in stem growth of injured plants.

The nature of the injury appears to be due in part to the separation of the cells along the middle lamellae. When extensive, this results in the physiological isolation and death of the tissue. The cells are killed and sometimes ruptured by the freezing. These injured cells and those adjacent respond by biochemical changes and the deposition of brown amorphous substances in and between the injured cells. The injured cells are isolated ultimately by the meristematic activity of the surrounding cells.

The response to injury is conditioned by the character of the cells adjoining the injured tissues. If these tissues are capable of rapid meristematic activity, repair is rapid and complete before decay of the injured tissues progresses far beyond the damaged cells. Under less favorable situations, fungal or bacterial development in the tissues is extensive. According to Jones (1928), Jones and Weimer (1928), Wiant and Starr (1936), bacterial wilt of alfalfa occurs in greater abundance in plants damaged by winter injury. The initial tissue damage and recovery in injured plants differs with varieties, the storage of reserves in the previous season, the environmental conditions and other factors (Albert, 1927, Graber *et al.*, 1927, Nelson, 1925, Steinmetz, 1926).

Control of winter injury depends largely upon the use of adapted varieties, resistant lines, and management of the crop. The varieties adapted to the more northern sections appear to go into dormancy in better physiological condition to withstand winter injury than those that continue vigorous vegetative growth until late autumn. Hardening and

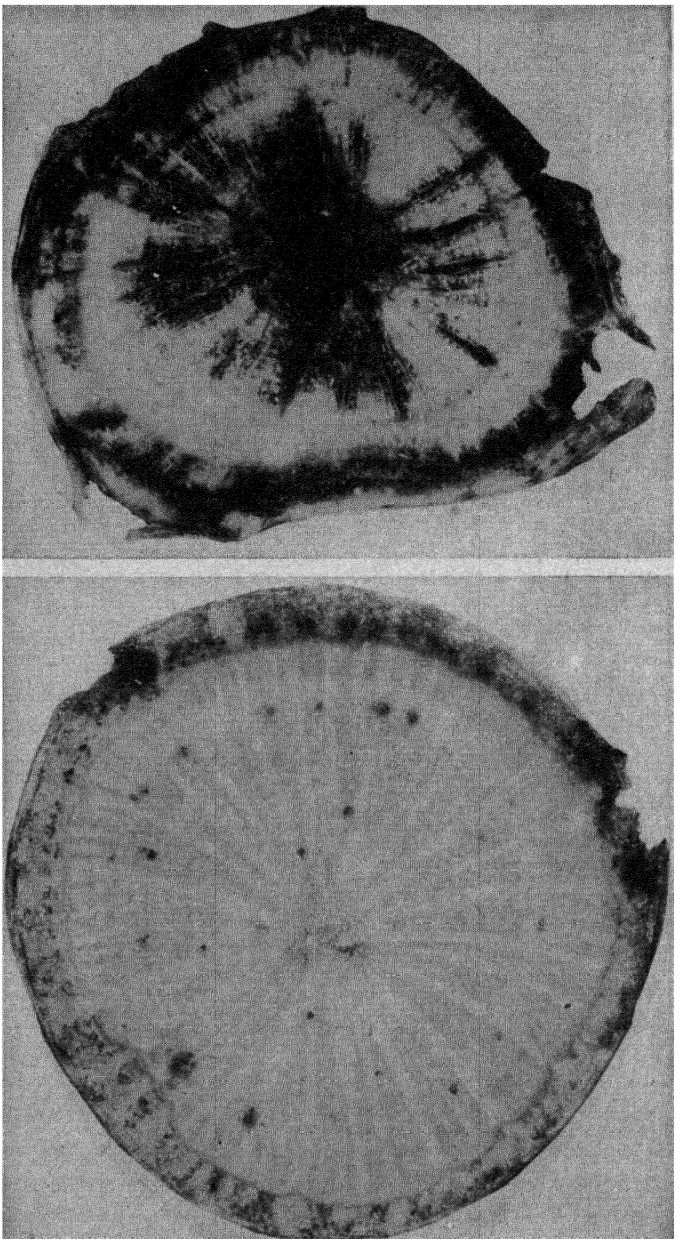

FIG. 76.—Transections of alfalfa roots showing the tissues damaged by low winter temperatures. (*Courtesy of F. R. Jones.*)

freezing tests on young plants under artificial conditions is not always a reliable means of measuring varietal response to this complex as it functions in the field. Selection of surviving plant lines growing under field conditions, as suggested by Jones (1945), and combining those lines in synthetic varieties is the most reliable method of control. Crop management designed to ensure adequate storage of reserves in the crown and root is essential to winter survival and recovery in the spring. Continuous pasturing and frequent cutting especially late into the autumn depletes the reserves essential to plant development and winter survival. The importance of growth and decay of transient or noncambial roots in these crops has been suggested by Jones (1943).

2. Yellowing, Leaf Discoloration, and Dwarfing. Nonparasitic.—Several types of yellowing and other discoloration of foliage and shortened stem growth are common in alfalfa and sweetclover. Frequently, the symptoms and plant response are similar for a number of unfavorable environmental conditions. These causal complexes cannot be determined without careful study of past and present environmental conditions. Deficiency of mineral nutrients is cumulative in effect, and frequently production is reduced materially before symptoms are expressed fully.

Boron deficiency in the soils of many agricultural areas results in dwarfed plants and yellow to bronze foliage, especially in alfalfa. Bauer *et al.* (1941), Colwell and Lincoln (1942), McLarty *et al.* (1937), Piland and Ireland (1941), and others have reviewed the symptoms and effects of boron deficiency in alfalfa. The symptoms are short terminal internodes, death of terminal buds, and yellow or red foliage. These symptoms differ from the marginal yellowing and white spotting of the leaflets due to potash and phosphate deficiencies and are somewhat less distinctly differentiated from leaf-hopper injury. White spots on the leaflets are characteristic of some plants at certain stages of growth, as shown by Jones (1945).

Heavy potato leaf-hopper infestation causes yellowing and dwarfing of plants. The leaflets yellow and frequently brown along the margins. Internodal elongation, plant vigor, crown bud development, and transient root survival are direct or indirect effects of the injury. The terminal buds and flower primordia appear grayish green and greatly retarded in development, or they dry out and fail to develop. The primary tissues of the stem show punctures and necrotic spots (Smith and Poos, 1931); however, the secondary growth from the cambium gives better evidence on the type of injury (Jones, 1945). The necrosis of cambium cells around the punctures and excess phloem production in adjacent portions of the cambium result in irregular thickening of the stunted stems. The presence of the leaf hoppers is further evidence of the cause (Granovsky, 1928, Hollowell *et al.*, 1927, Johnson, 1934, 1936, 1938, Jones, 1945).

Yellowing and stunting of the plants occurs as a response to other unfavorable environmental conditions, such as heat and drought. The abnormal appearance of the plants is localized or general in the field or area. The floral buds are retarded and under severe conditions yellow and dry out (Jones, 1937).

3. Alfalfa Mosaic, Witches' Broom, and Dwarf, Viruses Transmitted by Aphids and Leaf Hoppers.—Several mosaics occur on alfalfa and sweetclover. These plants and the clovers apparently harbor a large number of viruses occurring on annual legumes and other crop plants. The virus complex on these plants needs further investigation, especially as the perennial species of this group not only function as sources of virus infection, but also as winter habitat for the aphid vectors. The viruses occurring on alfalfa and sweetclover are numerous, and many of them are studied inadequately (Black and Price, 1940, Holmes, 1939, Pierce, 1934, Price, 1940, Snyder and Rich, 1942, Zaumeyer, 1938, Zaumeyer and Wade, 1935). Weiss[1] summarizes the viruses described, primarily on the leguminous crops.

Alfalfa Mosaic, Virus Transmitted Mechanically and by *Macrosiphum pisi* (Kltb.).—This mosaic is distributed widely in North America, although it is more common west of the Rocky Mountains and possibly occurs in Europe and Asia. The virus or strains occur on a wide range of crop plants, and it is important naturally on especially bean, pea, potato, celery, and paprika pepper.

The first symptoms on alfalfa are small greenish-yellow spots followed by more extensive and diffuse chlorosis. The leaflets become crinkled, irregular in shape, brittle, and there is no necrosis of tissues (Weimer, 1934). The symptoms are masked by high temperatures.

The virus and strains are transmitted mechanically to a wide range of plants in 27 families in addition to the Leguminosae. The pea aphid, *Macrosiphum pisi* (Kltb.) is the common vector in nature. The virus persists in the crown tissues of infected plants. Laufer and Ross (1940) and Ross (1941) have studied the virus protein.

Several other legume viruses occur on the alfalfa, and in some instances this perennial crop may function in carrying the viruses through the winter.

Witches' broom, Virus Transmitted by Grafting and Possibly *Scaphytopius acutus* (Say.).—Witches' broom of alfalfa occurs in the Northwestern and Intermountain areas of the United States and in Australia. The disease is serious in local areas in these locations. The disease is manifest by the production of numerous fine stems, shorter than on the healthy plants. The leaflets exhibit marginal chlorosis and crinkling, and the flower buds develop poorly. Infected plants show the disease

[1] *U. S. Dept. Agr. Plant Disease Reporter, Sup.* 154, 1945.

each season, although they are relatively short-lived. A similar disease possibly occurs on the clovers, according to Menzies (1946). The disease is transmitted by grafting (Edwards, 1936). The leaf hopper *Scaphytopius acutus* (Say.) is a possible vector (Menzies, 1946). Varieties react differently to the disease.

Alfalfa Dwarf, Virus Transmitted by Amblycephalinae Leaf Hoppers.—Alfalfa dwarf occurs in the Southwestern United States and possibly in New South Wales. The disease is severe in limited areas, especially where the virus disease, Pierce's disease of grapes, is prevalent. The plants are dark green, dwarfed, and gradually decline in vigor until they die after several seasons. The roots show the deposition of brown gum-like material in the xylem bundles (Weimer, 1936, 1937). The disease is prevalent under high moisture conditions and where alfalfa and vineyards are in close proximity.

The alfalfa dwarf virus is transmitted by grafting. According to Hewitt *et al.* (1946), the virus is transmitted from alfalfa to alfalfa, alfalfa to grape, and vice versa, by several species of leaf hoppers: (*Draeculacephala minerva* Ball, *Carneocephala fulgida* Nott., *C. triguttata* Nott., *Helochara delta* Oman, *Neokolla circellata* (Baker), *N. confluens* (Uhl.), *N. gothica* (Sign.), and *N. hieroglyphica* (Say.). Viruliferous leaf hoppers are common in the areas where the disease occurs.

4. Sweetclover Mosaic, Ring Spot, and Streak, Viruses.—*Mosaic on sweetclover* apparently may be caused by anyone of the following viruses: alfalfa mosaic, bean yellow mosaic, pea common mosaic, pea mottle, pea streak, pea wilt, and red clover vein mosaic. The symptoms produced by these various viruses are not differentiated on sweetclover. Leaf mottling, chlorosis, and other mild type symptoms usually develop.

Tobacco ring spot virus produces light-yellow irregular spots and mottling of the leaflets in nature. A strain of the virus produces veinal chlorosis, pronounced puckering of the leaflets and dwarfing.

Tobacco streak apparently may be harbored on the common white sweetclover on which it produces general chlorosis and chlorotic ring and line patterns.

5. Bacterial Wilt, *Corynebacterium insidiosum* (McCull.) H. L. Jens.—The bacterial wilt of alfalfa is distributed widely in North America, and it is apparently less common in South America, Europe, and Asia. The disease is probably the most important malady of the crop in the United States. During the past 20 years it has spread over most of the important alfalfa-producing areas of the country. Alfalfa plants are killed so rapidly that fields are unprofitable after 3 or 4 years.

Symptoms.—The plants are reduced in vigor, the leaves yellow and bleach, and the plants die in the late summer. The leaflets on infected plants are smaller and thicker prior to the loss of the green color. The

stems are smaller and more numerous in the earlier stages of disease development. The tap root shows a pale-brown discoloration of the outer woody tissue. This is evident when the outer bark is peeled off or when the stem is sectioned (Fig. 77). The bacteria are present in the xylem bundles of the new growth and spread tangentially into the parenchymatous tissue and adjacent bundles, resulting in distribution around the

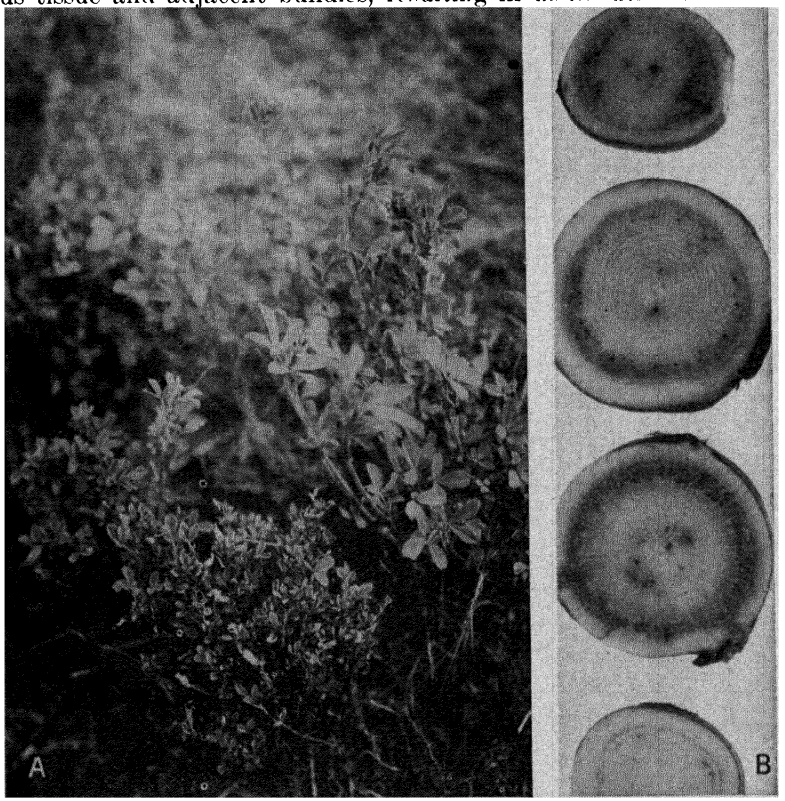

FIG. 77.—(*A*) Alfalfa plant, lower center, showing the symptoms of wilt caused by *Corynebacterium insidiosum*. (*B*) Sections through the root showing the brown discoloration of the outer woody tissues. (*Courtesy of F. R. Jones.*)

circumference of the root. The initially invaded areas are associated with wounds in the phloem rays caused especially by winter injury. The wilted plants occur first in the lower portions of the fields, either as scattered plants or more frequently in groups of plants.

The Bacterium.—*Corynebacterium insidiosum* (McCull.) H. L. Jens.
[*Phytomonas insidiosa* (McCull.) Bergey *et al.*]
(*Aplanobacter insidiosum* McCull.)

The colonies are pale yellow and consist of short rods with rounded ends without flagella.

Etiology.—The infection of the plants occurs during the spring and early summer. The bacteria in the older diseased plants are released by the breakdown of the infected tissues, and they are distributed in the soil water. Entrance into the plants occurs commonly through rifts in the root tissues caused by winter injury during the previous winter (Jones, 1928, Jones and Weimer, 1928). The rapid spread of the bacteria in the root tissues occurs in the xylem tissue formed during the spring and early summer. The tissues later in the summer are resistant to invasion. The infected plants usually die during the later summer of the second year.

Control.—The control of the disease is difficult, as indicated by the general spread in the alfalfa-producing areas. Proper management of the crop to prevent injury of the root tissues helps reduce the spread of the disease. Resistant varieties offer the only satisfactory means of controlling the disease. Resistance to bacterial wilt must be associated with resistance to winter injury to secure long life in plants in wilt-infested areas (Jones, 1945). Some lines or composite varieties are resistant to winter injury and capable of survival over long periods. (Brink *et al.*, 1934, Jones, 1934, 1940, Peltier, 1933, Peltier and Schroeder, 1932, Peltier and Tysdal, 1934). The Turkestan types and varieties contain more wilt tolerance than the common alfalfa.

6. Bacterial Stem Blight, *Pseudomonas medicaginis* Sackett.—The disease occurs locally in the Western part of North America, on alfalfa principally. The disease is of minor importance. The lesions occur on the stems and foliage of the younger plants and extend into the crown and roots in some older plants. The dark to light-brown spots are linear, with droplets or scales of bacterial exudate. The lesions commonly occur on the stems in association with frost cracks.

The Bacterium.—*Pseudomonas medicaginis* Sackett
[*Phytomonas medicaginis* (Sackett) Bergey *et al.*]
[*Bacterium medicaginis* (Sackett) E. F. Sm.]

Light-yellow colonies of short rods that are motile by means of 1 to 4 polar flagella develop on media.

The disease occurs in the early spring, especially when low temperatures cause injury or cracking of the young stem epidermis, and therefore, it is common only in the spring growth. Varieties react differently to the spread of the disease in the stem tissues (Richards, 1934, 1936-1937, Sackett, 1910).

7. Crown Wart, *Urophlyctis alfalfae* (Lagc.) Magn.—The crown wart on alfalfa occurs in the warmer areas of North and South America and Europe. The similar disease on white clover, common in Europe, occurs in the Gulf area of the United States. The disease is confined to local wet areas where some damage occurs.

The galls are swollen and modified stem bud primordia (Fig. 78). The primordial scales, leaves, and stipules in the infected buds thicken to form the scale-like galls around the central axis of the undeveloped stem. The galls are usually near the soil surface. Small leaf galls are formed occasionally. The galls are white when young, and gray to brown as they decay and dry out in mid-summer. The swollen cells of the gall tissue contain the resting spores of the fungus in various stages of development.

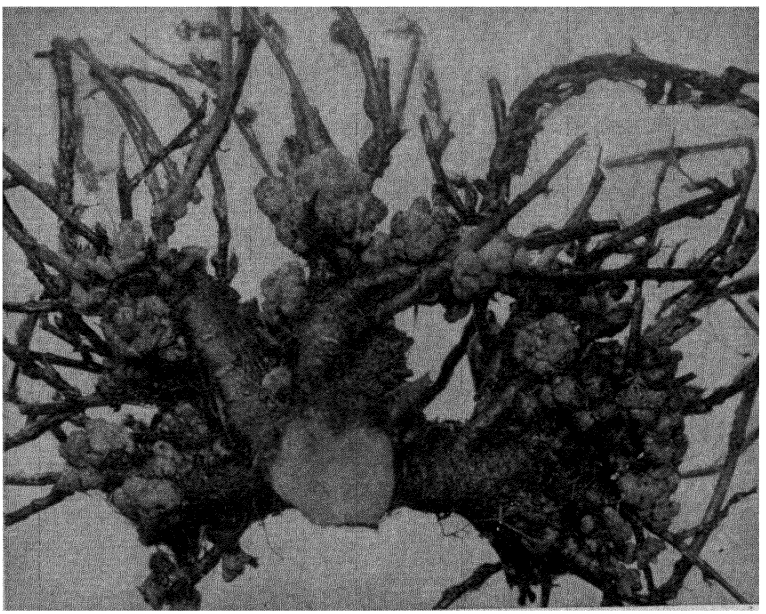

FIG. 78.—Crown wart of alfalfa caused by *Urophlyctis alfalfae*. (*Courtesy of F. R. Jones.*)

The Fungus.— *Urophlyctis alfalfae* (Lage.) Magn.
(*Cladochytrium alfalfae* Lage.)
(*Oedomyces alfalfae* Lage.)

The branched haustorial processes form in a well-defined zone near the apex of the vegetative cell and the resting spore. The resting spores are initiated from the apex of the vegetative cell by the enlargement of the axial haustorial element. The mature resting spores are thick-walled, light brown to golden yellow, with the zone of scars of the haustorial elements where the wall is thinner (Jones and Drechsler, 1920, Wilson, 1920).

Urophlyctis trifolii (Pass.) Magn., similar to the above species in morphology, occurs in Europe and Asia and is reported on white clover in the South Central United States. *Olpidium trifolii* (Pass.) Schroet., causing leaf curl on white clover and possibly the same as *U. trifolii*, occurs in

the United States as well as in Europe. *U. trifolii* is listed as synonymous with *O. trifolii* by Chilton *et al.* (1943), but distinguished morphologically by Atkinson (1940).

8. Pythium Damping-off and Root Rot, *Pythium debaryanum* Hesse and Other Species.—The *Pythium* spp. associated with the legumes occur throughout the world, especially in the finer texture acid soils (Buchholtz, 1942). The seed-rotting preemergence blighting and post-emergence damping-off reduce the stands in the small seeded legumes, especially alfalfa and sweetclover. The disease is primarily associated with the seedlings, although rootlet rot and stem rot occur in established plants, especially in wet locations. The soft rot of the tissues and presence of the sporangia and oöspores in the freshly rotted tissues are the most certain symptoms. Several species of *Pythium* are associated with the disease on the small seeded legumes. These species also occur on a wide range of cultivated and wild plants.

Middleton (1943) described this group of morphologically similar species with somewhat spherical nonproliferous sporangia. They differ primarily in the number and origin of the antheridia attached to the oögonia. *Pythium debaryanum* Hesse is distinguished by the terminal or intercalary sporangia and oögonia and by one to six monoclinous and declinous antheridia attached to each oögonium. The sporangia germinate by either zoospores or germ tubes. *P. ultimum* Trow has chiefly terminal sporangia and oögonia and usually one monoclinous antheridium attached to each oögonium. The sporangia germinate by germ tubes only. *P. splendens* Braun, *P. vexans* DBy., and several others of this group occur on these crops.

The etiology of the group is similar. The seed and seedling attack from the soil-borne mycelium is associated with the maturity and general condition of the seed and the soil environment in which the seed germinates. Fully matured seed, well-prepared seed bed, a balanced fertility, including liming of acid soils, and seed treatment under some conditions (Allison and Torrie, 1945) are the best control measures.

9. Phytophthora Root Rot of Sweetclover, *Phytophthora* Spp.—A soft rot of the root and crown of sweetclover plants occurs in the spring in the North Central United States. The tops bleach and wither due to the spongy soft rot of the root and lower portion of the crown. The disease is of minor importance. *Phytophthora cactorum* (Leb. and Cohn) Schroet. is associated with the disease. Resistant strains of sweetclover are the best means of control where the disease becomes severe (Jones, 1939). *P. megasperma* Drechs. occurs on sweetclover but is less pathogenic (Cormack, 1940).

10. Downy Mildew, *Peronospora trifoliorum* DBy.—The downy mildew is distributed widely on alfalfa in the temperate zones of the world. In

the Northern United States the disease causes considerable damage to the first cutting of hay and weakens the plants. The disease occurs

FIG. 79.—Downy mildew of alfalfa caused by *Peronospora trifoliorum* showing (*A*) malformation, chlorosis, and necrosis of tissues of a systemically infected plant and (*B*) downy mass on the leaflets.

rarely on sweetclover and not on the clovers in the United States, although the downy mildew is severe on clover in some sections of Europe. The characteristic symptoms are the light-green leaves especially at the apex

of the stem and the grayish-white mycelium on the surface of the leaves. Internodal elongation is reduced, the stems are smaller, and the leaflets are twisted and rolled in severe infections. The conidiophores and violet conidia are conspicuous on the undersurface of the leaflets. Where the infection is systemic, the stems are swollen and the foliage chlorotic, with conidial production abundant just prior to the collapse of the leaf tissue (Fig. 79). Oöspores are formed in the leaf tissue.

The Fungus.—*Peronospora trifoliorum* DBy.

The extensive synonymy is given by Chilton *et al.* (1943).

The mycelium is abundant in the leaf and stem tissues, and the grayish surface mycelium is extensive under humid conditions. The conidiophores are slender, dichotomously branched at acute angles, with the secondary branches curving downward in the older conidiophores. The conidia are globose to broadly elliptical, violet colored, measure 15–20 by 18–36 microns, and germinate by the formation of germ tubes. The oöspores are globose, smooth-walled, light brown, and 24 to 30 microns in diameter.

Etiology.—The oöspores in the dead tissues and the perennial mycelium in the crown buds enable the parasite to persist when once established. The mycelium is probably carried on the seed in some areas. Secondary spread occurs from the conidia whenever environmental conditions are favorable. Clonal lines of alfalfa show differences in susceptibility. Some varieties are damaged more than others. Specialization apparently occurs on the clovers and alfalfa.

11. Spring Black Stem, *Ascochyta imperfecta* Pk., *Mycosphaerella lethalis* Stone, and *Phoma trifolii* E. M. Johnson and Valleau.—This complex of similar diseases on alfalfa, sweetclover, and the clovers is common in North America and Europe. The disease on alfalfa causes appreciably more damage than the others. Disease development is severe during the cold wet conditions of early spring, and in cold seasons damage extends into the early summer. The infections spread extensively late in the autumn, but stem blackening is less pronounced, although pycnidial development is abundant.

Symptoms.—On alfalfa and sweetclover the disease appears as dark-brown to black lesions on the stems and petioles. When the disease is severe, young shoots are blackened and killed and stems are girdled by the lesions. The brown spots on the leaves are small, irregular in shape, and coalesce to form the blackened areas (Fig. 80). The infected leaves turn yellow and wither before they drop. The brown spots appear on the pods under cool growing conditions. Pycnidia are not common on the lesioned stems during the growing season. Pycnidia are numerous, however, on the old stems produced in the previous autumn and interspersed with the new spring growth of stems. Black stem symptoms occur on other legumes, but they are caused by other species of *Ascochyta*. Although the fungi on alfalfa and sweetclover infect the other crops, each

occurs predominantly on the one crop (Cormack, 1945, Johnson and Valleau, 1933, Sprague, 1929, Toovey *et al.*, 1936).

The Fungi.—1. *Ascochyta imperfecta* Pk.
(*Phoma medicaginis* Malbr. and Roum.)
(*Diplodina medicaginis* Oud.)

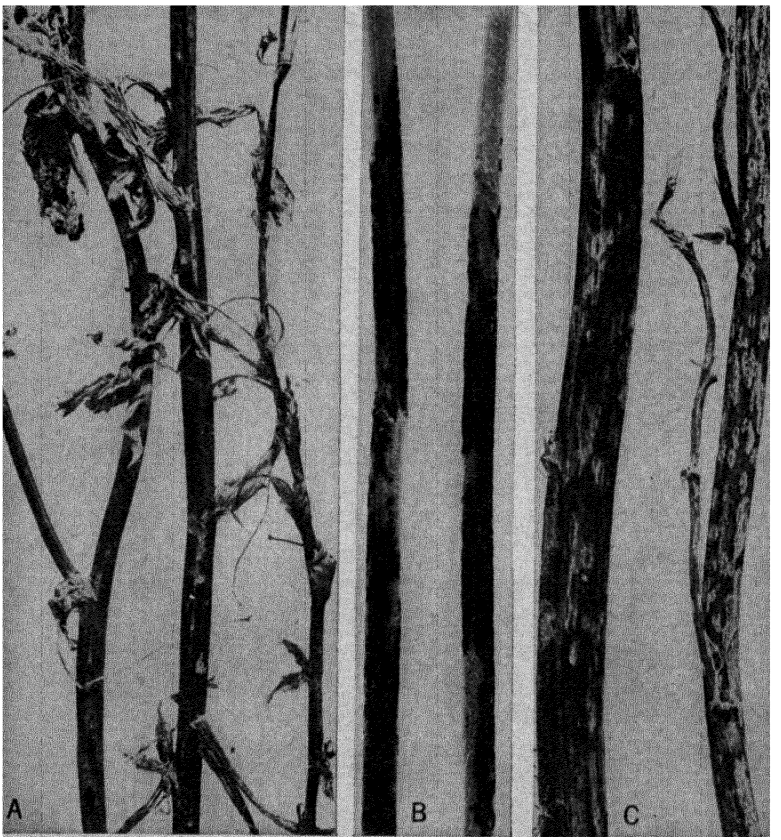

FIG. 80.—The spring black stem disease of alfalfa (*A*), red clover (*B*), and sweetclover (*C*) caused by *Ascochyta imperfecta*, *Phoma trifolii*, and *Mycosphaerella lethalis* respectively; and stem cankers of *A. caulicola* (*C*), right.

The pycnidia are globose, ostiolate, without a beak, and light to dark brown in color. The spores are hyaline, oval or cylindrical with rounded ends, straight or slightly curved, and uniseptate when mature. Spore septation and size are variable. No perfect stage is known. This fungus causes the spring black stem on alfalfa. Remsberg and Hungerford (1936) wrongly described *Pleospora rehmina* Staritz as the perfect stage of *Phoma medicaginis*. The fungus is seed-borne as well as persisting on crop residue (Cormack, 1945).

2. *Phoma trifolii* E. M. Johnson and Valleau

This species differs in morphology from that above by the nonseptate oval spores and is the cause of black stem on the clovers.

3. *Mycosphaerella lethalis* Stone
(*Ascochyta lethalis* Ell. and Barth.)

The conidial stage is *Ascochyta meliloti* (Trel.) J. J. Davis, as described by Jones (1944), although, *A. meliloti* Trusova has priority. The other synonyms are given by Chilton *et al.* (1943).

The perithecia are submerged, globose with the ostioles elongated into a beak. The asci are cylindrical to clavate with an outer and inner wall. The ascospores are hyaline, ellipsoidal, two-celled, and slightly constricted at the septum. The pycnidia are globose, ostiolate, and brown. The spores are hyaline oblong, slightly curved, one-septate when mature, and measure 5–6 by 13–20 microns. This fungus causes spring black stem on sweetclover.

4. *Ascochyta caulicola* Laub. causes a stem canker and hypertrophy of the stems of sweetclover without stem blackening as a characteristic symptom. This species is morphologically similar to the pycnidial stage of the former fungus (Jones, 1938).

Etiology.—The development of the disease caused by the several parasites is similar. Pycnidia develop abundantly on the stems in late autumn and the following early spring. The fungi persist in the crop refuse on the surface of the soil. They are seed-borne in areas where the environment is favorable for pod infection. The infection and development of the disease is favored by cool, wet weather (Cormack, 1945, Jones, 1939, and Peterson and Melchers, 1942). The disease appears the second year after seeding and thereafter. Local cortical infection occurs through natural openings or injuries. Crown and root infections followed by rotting occur in alfalfa.

The control of the disease is accomplished by crop management and adapted resistant varieties. The fungi apparently persist in crop residues for only one season; therefore, crop rotation is practical, especially in the biennials. Burning the old stems early in the spring before the plants start growth reduces the inoculum. Commercial varieties show differences in reaction to the disease, and resistant plants occur in alfalfa (Koepper, 1942). Selfed lines of sweetclover are resistant to both spring and summer black stem diseases (Jones, 1944). Seed treatment is advisable especially when infected seed is used in areas free from the disease.

12. Summer Black Stem and Leaf Spot, *Cercospora zebrina* Pass. and *Mycosphaerella davisii* F. R. Jones.—The summer black stem and leaf spot diseases develop on alfalfa, clover, and sweetclover during warm, moist weather. This disease complex frequently occurs in association with the spring black stem during early summer and again in the early autumn. The disease is distributed extensively on alfalfa, clover, sweet-

clover, and several wild legumes in Central and Eastern North America and in Europe. Damage to white clover pastures is severe during late summer in the Southern United States.

The symptoms vary somewhat on the different legumes. The leaf spots range from light-brown linear lesions on red clover to large circular ashy-gray to tawny sunken spots on sweetclover. The center of the lesions are gray to black when conidial production is abundant. The lesions on the stems, petioles, and inflorescences are reddish brown to dark brown, depending upon the tissues. The lesions are sunken, and necrosis of tissues is extensive. The lesions are conspicuous when the plants are in the blossom stage of development. Infected seed is shriveled and discolored with mycelium on the surface. The fungi are restricted closely in the legumes attacked (Hopkins, 1921, Horsfall, 1929, Jones, 1944, Nagel, 1934).

The Fungi.—1. *Cercospora zebrina* Pass.

(*Cercospora medicaginis* Ellis and Ev.)

The complete synonymy is given in Chilton *et al.* (1943) in which they include *C. davisii* Ell. and Ev.

The conidiophores are hyaline to brown, nonseptate, and rather long (35 to 45 microns). The conidia are cylindrical fusoid, hyaline to light yellow, 3- to 6-septate, and average 3 by 50 microns in size. No spermagonial or perithecial stage is known. This species occurs on alfalfa and the clovers.

2. *Mycosphaerella davisii* F. R. Jones

The conidial stage is *Cercospora davisii* Ell. and Ev., not *C. meliloti* Oud. according to Jones (1944), and he retains *C. davisii* distinct from *C. zebrina* on alfalfa and the clovers.

The perithecia are inconspicuous, often few and scattered on dead overwintered sweetclover stems. They develop beneath the epidermis through which the ostiole opens, and they are spherical and dark colored. Asci are cylindrical to clavate, grouped at the base of perithecia, and develop in succession throughout the summer. Ascospores are irregularly biseriate, hyaline, straight or slightly curved along one side, bluntly pointed, and measure 4–5 by 12–20 microns. Spermagonia develop at low temperatures and are thickly scattered, black, subepidermal, erumpent, often approaching an acervulus in form, and bear rod-shaped spermatia. Conidiophores are amphigenous, tufted, straight or subflexuous, pale brown, continuous or 1- to 2-septate. Conidia are first cylindrical, later acicular, hyaline to greenish yellow, 1- to 13-septate, and 2.2 to 4.5 microns wide at the base by 20 to 140 long. This fungus occurs on the several species of *Melilotus*, or sweetclovers.

Etiology.—The fungi persist in the old stems of the legumes and produce conidia in abundance under warm, moist conditions. They are seed-borne in areas where moisture is plentiful during the period of seed development. Secondary spread from conidia occurs during wet, warm weather.

Control.—Crop management and use of resistant or adapted varieties helps reduce the damage, especially on sweetclover. Burning or removal

of crop residue aids in reducing the summer inoculum. The disease is more severe in second year's growth that is cut or grazed than in stands that make complete growth without retarded development. Resistant

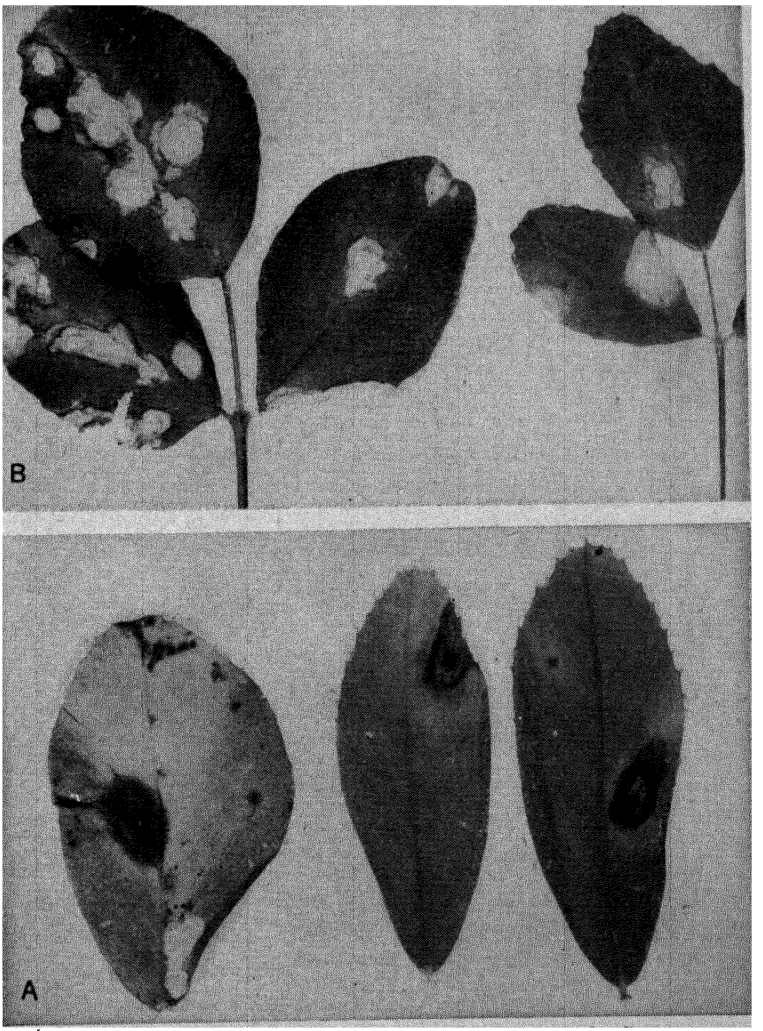

FIG. 81.—Leaf spots on alfalfa (*A*) and sweetclover (*B*) caused by the *Stagonospora* stage of *Leptosphaeria pratensis*.

lines of sweetclover suggest the use of resistant varieties (Jones, 1944).

13. Leaf Spot and Root Rot, *Leptosphaeria pratensis* Sacc. and Briard. —The disease occurs on alfalfa, sweetclover, and some other legumes, and it is distributed widely in humid areas. Albrecht (1942) reported

the disease causing severe damage during the spring in white clover pastures in the Southeastern United States.

On alfalfa the disease is primarily a root rot, although the leaf spot is common. The leaf spot is circular to angular, pale buff with light brown margins, and with numerous pycnidia in the central portion of the lesion (Fig. 81). The ashy-gray lesions on the annual stems are uncommon. The brown to black root and crown rot is the common symptom on alfalfa. Necrosis and dry rot frequently involves the upper portion of the tap root and crown. No new buds form above the lesion, and the plant eventually dies. Secondary organisms enter the rotted areas, especially under conditions of high moisture, to confuse the symptoms. On sweetclover the circular tan leaf spots are numerous in spring and late autumn with brown pycnidia abundant (Fig. 81). The inconspicuous stem lesions are prevalent during the growing season and spread rapidly as the plants mature. In the late autumn the stems are brown internally and golden brown on the surface as the pycnidia of the Phoma stage develop. The fungus develops three stages: Stagonospora, largely on the leaf spots; Phoma, on the stems in late fall; and the perfect stage on the stems in the late autumn and spring (Jones and Weimer, 1938).

The Fungus.—*Leptosphaeria pratensis* Sacc. and Briard.
Stagonospora meliloti (Lasch.) Petr. Conidial stage
(*Phoma meliloti* Allesch.)

The extensive synonomy is given by Chilton *et al.* (1943) and Jones and Weimer (1938).

The perithecia, formed in the old stems, are globose, dark brown, and the ostiole is usually papillate. The ascospores are oblong to fusoid, yellow, usually with three septa, and 25 to 30 microns long. The Stagonospora pycnidia are submerged in the tissues, with the neck or rostrum extending through the epidermis. The rostrum forms a central canal narrower at the base than at the apex. This funnel-shaped rostrum is a distinguishing morphological character and occurs also in the deeply submerged Phoma pycnidia. The spores vary greatly in size and the presence or absence of the septum.

Etiology.—Both the conidia and ascospores serve as primary inoculum for the early spring leaf infection. Secondary spread from conidia occurs during warm, wet-weather conditions. The stem and root infections apparently are associated with injuries of various types. The influence of environment, especially temperature, on the development of the different stages of the fungus is pronounced; the *Stagonospora* stage develops at summer temperature and the *Phoma* stage during low autumn temperatures. The disease is prevalent in the second and following years.

Control of the disease is largely by crop management. The removal or burning of old stems and crop residue reduces the inoculum for spring crown and leaf infection. Differences in susceptibility occur in both

alfalfa and sweetclover. The latter crop is relatively more susceptible, according to Jones *et al.* (1941).

14. Common Leaf Spot, *Pseudopeziza medicaginis* (Lib.) Sacc. and *P. meliloti* Syd.—This leaf spot is distributed rather generally on alfalfa throughout the world. It is probably one of the most common diseases

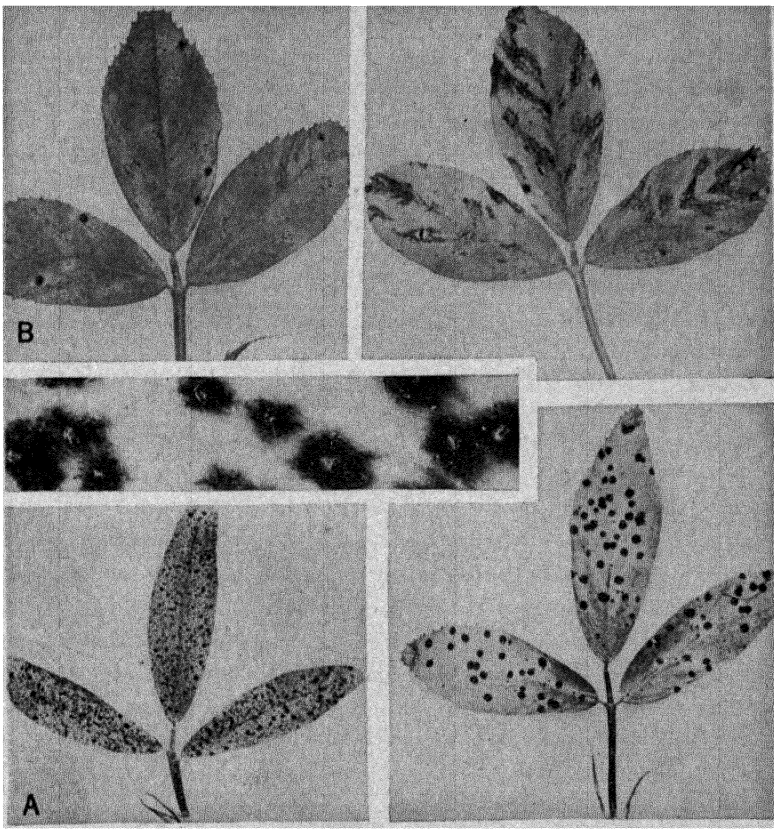

FIG. 82.—The common leaf spot caused by *Pseudopeziza medicaginis* (*A*) and the yellow leaf blotch caused by *P. jonesii* (*Pyrenopeziza medicaginis*) (*B*) on alfalfa. The apothecia of the former fungus are shown, slightly magnified, in the insert.

on alfalfa and yet of minor importance in most areas, as the infection is generally light. Severe infection causes defoliation (Jones, 1919).

Symptoms.—The circular small brown spots occur on the leaflets. The spots are restricted in size, usually do not coalesce, and generally do not cause discoloration of the surrounding leaf tissues. The small dark-brown to black raised disk (apothecium) in the center of the mature spot is a distinguishing characteristic (Fig. 82). Small elliptical spots occur on the succulent stems but rarely form apothecia. Heavy infections

cause defoliation, especially of the lower leaves. On sweetclover the spots are less pronounced.

The Fungi.—1. *Pseudopeziza medicaginis* (Lib.) Sacc.

The apothecia arise in a stroma beneath the epidermis, solitary or clustered, the epidermis is ruptured, and the hymenia are raised above the surface of the leaf. The perithecia are imbedded in the apothecium. Nonseptate paraphyses slightly longer than the asci are numerous. Asci are clavate with eight ascospores arranged in two rows. The spores are hyaline, ovate to oblong, without septations, and 8 to 14 microns long.

2. *Pseudopeziza meliloti* Syd.

This species is similar in morphology to the previous species and occurs less abundantly on *Melilotus alba*. *Pseudopeziza trifolii* (Biv.-Bern.) Fckl. occurs sparingly on the clovers. The synonymy of the species is given by Chilton *et al.* (1943).

Etiology.—The fungus persists in the leaf tissues until they are decomposed. Apothecia overwinter on the fallen leaflets, and new ones are formed on the undecomposed leaves in the spring. Ascosporic inoculum is abundant during the entire growing season. The disease develops whenever environmental conditions are favorable.

Varieties differ in their reaction to the disease. Resistant lines of alfalfa are reported by Jones *et al.* (1941).

15. Yellow Leaf Blotch, *Pseudopeziza jonesii* Nannf.—The disease apparently is distributed less generally than the common leaf spot although world wide in occurrence. Although leaf blotch occurs less frequently than the spot disease, it causes severe defoliation when present in any abundance. Alfalfa is the only legume damaged by the disease.

Symptoms.—The young lesions appear as yellow blotches elongated parallel to the leaf veins. The lesions enlarge, and the color changes to orange yellow, shading to yellow at the margins. Small orange to brown pycnidia develop, especially on the upper surface of the blotch (Fig. 82). Apothecia are scarce on the undersurface of the blotch until the leaflets drop. The stem lesions are elongate yellow blotches which soon turn dark brown. The stem lesions are not abundant (Jones, 1918, and Nannfeldt, 1932).

The Fungus.—*Pseudopeziza jonesii* Nannf.
(*Pyrenopeziza medicaginis* Fckl.)

Pseudopeziza jonesii Nannf. is used in the current literature, especially the European, although, *Pyrenopeziza medicaginis* Fckl. is preferred by some pathologists. The synonyms are given in Chilton *et al.* (1943).

Apothecia develop on the dead leaf tissue and are partly closed by mycelial strands until mature. The asci are borne among numerous nonseptate paraphyses. Asci are cylindrical to clavate with eight ascospores usually in two rows. The spores are ovoid, hyaline, nonseptate, and 8 to 11 microns long. The pycnidia are formed subepidermally and rupture the epidermis in irregularly lobed cavities. The bottle-shaped conidiophores are closely packed on the interior of the pycnidial cavities. The conidia vary greatly, usually they are cylindrical with rounded ends.

Etiology.—Ascospores are produced in the late spring from overwintered apothecia. Primary infection occurs from ascospores that are produced in decreasing numbers as the growing season advances, and they are produced again late in the autumn. The conidia apparently do not cause infection.

Cutting the crop before leaf drop is desirable in both leaf spot diseases. The leaves are saved for hay, and perithecial inoculum is reduced. Plants in waste areas also supply a source of inoculum. Burning the leaves and stubble in the early spring also reduces the spring inoculum. Apparently all the alfalfa varieties are moderately susceptible to the disease.

16. Root Rots and Wilt, *Fusarium, Cylindrocarpon* Spp. and Other Fungi.—The root rots included in this complex of diseases are common on the perennial and biennial leguminous crops. Apparently they are associated in many instances with plants weakened by unfavorable winter or summer conditions, such as winter injury of various types and summer dormancy due to drought. Usually the plants are attacked before they become active vegetatively after winter or summer dormancy (Cormack, 1934, 1937, Scott, 1926, and Weimer, 1928). The fungi associated with the rots vary greatly in areas and seasons. The root rots of this type are prevalent more generally in the colder and drier climates. The wilt type of disease occurs more generally in the warmer climates. Damage, especially to older stands of the perennial crops, is severe when environmental conditions and other diseases are weakening the plants.

Symptoms.—These root rots are conspicuous in the early spring. The mycelium is found around the roots and crowns and in ruptured and injured tissues. The lesions vary from irregular brown rotted areas to the complete disintegration of the root and lower crown. Such rots progress slowly in their development, with final weakening and death of the plants during the spring and summer.

The wilt of alfalfa caused by *Fusarium oxysporum* f. *medicaginis* (Weimer) Snyder and Hansen shows relatively little cortical rot during the early stages of disease development. The stems yellow, wilt, and dry out; the symptoms vary considerably depending upon environment. The vascular system shows the characteristic browning due to fungus invasion and root-tissue response.

The Fungi.—*Fusarium avenaceum* (Fr.) Sacc., F. *arthrosporoides* Sherb., *F. culmorum* (W. F. Sm.) Sacc., *F. poae* (Pk.) Wr. *F. scirpi* var. *acuminatum* (Ell. and Ev.) Wr., *Cylindrocarpon ehrenbergi* Wr., *C. obtusisporum* (Cke. and Hark.) Wr., *Plenodomus meliloti* Mark.-Let., and other species are reported associated with the root rot. *F. oxysporum* f. *medicaginis* (Weimer) Snyder and Hansen causes a wilt disease of alfalfa. The morphology of the wilt fungus is given in Chap. XVI.

17. Rhizoctonia Root Rots.—Two types of root rots caused by species of *Rhizoctonia* occur on the legumes. (1) The violet root rot caused by *Rhizoctonia violacea* Tul. or *Helicobasidium purpureum* (Tul.) Pat. (*R. crocorum* Fr.) is common in many European areas (Sampson and Western, 1941), and it occurs rarely in North America. The rotted areas are brown with mats of mycelium attached in the early stages. Later the roots are rotted and shredded with a brown to dark-violet discoloration. The rotted portion extends from the crown to as far as 6 inches below the soil line. The disease is associated with low areas subject to flooding and frequently follows root injuries. (2) Smith (1943) described a root cankering due to *R. solani* Kuehn in limited areas in the Southwestern United States. The dark sunken areas occur on the tap root and branches. The root is not rotted completely as in the former disease. This fungus is active on alfalfa during the high summer temperatures and relatively inactive in the cooler periods. Strains of the same species cause a seedling cortical rot and root rot of older sweetclover plants in the more northern areas.

Cormack (1941) reported damage to legumes in early spring by a Basidiomycete that killed the plants in a relatively short period. The disease apparently is northern in distribution in North America.

The *Sclerotium* rots occur on alfalfa but are more common on the clovers. Sanford and Cormack (1933) reported indications of resistance in alfalfa. Alfalfa stands are damaged by *Phymatotrichum omnivorum* (Shear) Dugg. in the Southwestern United States (see Chap. XV).

18. Rust, *Uromyces striatus medicaginis* (Pass.) Arth.—The rust is general on alfalfa in North America and Europe, and it is probably world wide in the humid temperate zones. The uredia and telia occur on alfalfa. In Europe the aecial stage occurs sparingly on *Euphorbia cyparissias* L. The reddish-brown uredia and telia develop on the leaves late in the season. The telia form in the same lesions and in independent sori.

The Fungus.— *Uromyces striatus medicaginis* (Pass.) Arth. or *U. striatus* Schroet.

The synonyms are given in Chilton *et al.* (1943).

The aecia are scattered somewhat elevated, and the infected *Euphorbia* plants are dwarfed. Peridia are white with irregular edges; spores are orange colored and finely verrucose. The uredia on the alfalfa are small, round, and mostly on the leaves. The urediospores are globose to elliptical and light brown. The telia, formed later in the uredia and independently, are naked, round to oblong, and dark brown. The teliospores are ovate to pyriform and dark brown in color.

In Europe the rust is heteroacious, with the aecial stage forming a perennial mycelium in the *Euphorbia* sp. In North America the rust persists in leaf tissue in the milder climates and apparently spreads northward during the summer. Koepper (1942) reported *Medicago*

ruthenica Trantr., with eight chromosome pairs, immune to rust; *M. falcata* L. variable in reaction; and varieties of *M. sativa* L. ranging from susceptible to resistant. The variety Ladak is the most resistant commercial variety. The Turkestan types in general are suceptible.

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CHAPTER XIV

CLOVER DISEASES

Varieties of four species of *Trifolium* constitute the major crops of the true clovers, although others are important in local areas. Red, *Trifolium pratense* L., crimson, *T. incarnatum* L., alsike, *T. hybridum* L., and white clover including Ladino, *T. repens* L., are the important species, widely used. There are some 250 described species for the genus. These apparently constitute two polyploid series with basic chromosome numbers of 7 and 8 pairs. *T. pratense* is a diploid with 7 chromosome pairs; *T. incarnatum* and *T. hybridum* are diploids with probably 8 pairs, although 7 pairs are reported for the former species by some investigators; and *T. repens* is a tetraploid with 16 chromosome pairs (Pieters and Hollowell, 1937, and Wipf, 1939).

The cultivated clovers are cross pollinated and highly variable. Local varieties vary greatly in winter hardiness, type of growth, certain morphological characters, and reaction to disease. The plants are self-sterile in the main, and lines are stabilized by sib-pollination and selection. Clonal propagation is useful in comparison of larger populations, especially in disease-reaction studies. Perennial and biennial types are used chiefly in the cultivated varieties. The North American pubescent types of red clover are better adapted to resist damage of insects than are the European types, which have glabrous stems. Many of the diseases are common to more than one species of the genus.

1. Cold and Winter Injury.—Varieties of the true clovers vary greatly in their resistance to cold and winter injury. Many of the winter hardy varieties are the result of survival of the hardy plants during the process of adaptation. The types of injury and relation of these injuries to invasion of the crown and root tissues especially by fungi are similar to those described for alfalfa (Chap. XIII).

2. Yellows, Leaf Discoloration, and Dwarfing.—The reaction of the clovers to deficiencies of the essential mineral elements is somewhat similar to alfalfa, although as a group these crops apparently are more tolerant than alfalfa. Leaf hopper injury in the smooth-stemmed red clovers is severe and prevents their economical production in areas where leaf hopper infestations are common. The symptoms are yellowing and graying of foliage, reduced internodal elongation, and blighted flower heads somewhat similar to the gross symptoms in alfalfa.

3. Mosaics, Viruses Transmitted by Aphids and Leaf Hoppers.—The virus diseases of the clovers transmitted especially by the pea aphid represent a complex of viruses occurring on legumes and other crop plants. As in alfalfa, the clover virus diseases are perhaps of greater importance on some of the annual leguminous crops than on the clovers themselves. As discussed by Johnson (1933), Johnson (1942), Osborn (1937), and others, two types of symptoms occur on the clovers: (1) the leaf mottling to chlorosis, with or without dwarfing of the plants, and (2) the vein yellowing without appreciable dwarfing of the stems. The literature pertaining to the viruses on clovers has been summarized by Weiss.[1] At least three virus diseases are definied as clover mosaics: red clover vein mosaic, alsike clover mosaic, and subterranean clover mosaic.

Red Clover Vein Mosaic, Virus Transmitted by *Macrosiphum pisi* (Kltb.).—The malady is common on a wide range of legumes both perennial and annual through temperate North America and probably Europe. This virus may be identical with pea streak virus 1. The symptoms on clover are yellow irregular to regular patterned discoloration along the veins of the leaflets of new growth without mottling and with little reduction in plant size or vigor. The symptoms are similar on the other clovers except *Trifolium incarnatum* in which the plants are stunted and killed (Osborn, 1937).

The virus is transmitted by expressed juice and the pea aphid *Macrosiphum pisi* (Kltb). Other viruses occur naturally on the red clover, some of which are undetermined. Red clover is a natural overwintering harbor for the potato yellow dwarf virus as well as a food plant of the vector; the clover leaf hopper, *Aceratagallia sanguinolenta* (Prov.).

Alsike Clover Mosaic, Virus.—The mosaic is distributed widely in the United States and Canada, although it is of relatively minor importance on this clover. The alsike clover apparently harbors mosaics of the annual legumes, notably the common pea mosaic (Murphey and Pierce, 1937).

The virus and strains, to date transmitted only by extracted juice, produce stippled interveinal chlorosis with irregular patterns of light and green. The leaflets are smaller but not misshapen. Several viruses occur naturally on white clover (Johnson, 1942, and Zaumeyer and Wade, 1935, 1936), notably those causing pea mottle and pea wilt, which in combination cause a severe mosaic in white clover.

Subterranean Clover Mosaic, Virus Transmitted by Aphids.—This disease is described from Australia where it causes systemic mottling, distortion of the leaflets, dwarfing of the plants, and a reduction in seed

[1] *U. S. Dept. Agr. Plant Disease Reporter, Supplement* 154, 1945.

set, according to Aitken and Grieve (1943). It apparently is transmitted by aphids, and probably it is seed-borne.

4. Bacterial Blight, *Pseudomonas syringae* v. Hall, [*Phytomonas trifoliora* (L. R. Jones *et al.*) Burk.].—The disease occurs sparingly in various parts of North America and Europe and is of little economic importance. The angular leaf spots occur on the leaves, stipules, petioles, and stems of red clover (Jones *et al.*, 1932). The spots are dark brown with exudate on limited portions of the lesions. Infections occur during cool, wet conditions at any time during the growing season.

5. Powdery Mildew, *Erysiphe polygoni* DC.—Powdery mildew is distributed widely on red clover in the temperate zones of the world. Prior to 1921, the disease was uncommon in the Central and Eastern United States; yet within approximately 3 years after this date, the malady was prevalent throughout all North America. Heavy epidemics cause some reduction in yield and quality of the forage and hay (Horsfall, 1930 and Yarwood, 1934).

The light-gray superficial powdery growth of mycelium and conidia is conspicuous on the leaves. Leaves yellow and turn brown when the infection is severe. The mycelium develops on the surface of the epidermal cells of the leaves with haustoria within the cells.

The Fungus.—*Erysiphe polygoni* DC.

Salmon (1900) listed the extensive synonymy for this fungus. Peterson (1938) described the perithecia of *Erysiphe polygoni* and *Microsphaera alni* (DC.) Wint. on *Trifolium pratense* in the Eastern United States.

Perithecia are formed commonly in Western North America and northern Europe and uncommonly in Central and Eastern North America. Perithecia are scattered with variable simple appendages. The asci are usually ovate with four or eight spores. The ascospores are hyaline, oblong with rounded ends, and are one-celled. The ovate hyaline to gray conidia are borne in chains from an apical generative cell of a short simple conidiophore. Production of conidia is on a diurnal cycle (Yarwood, 1936).

Etiology.—The obligate parasite persists on the foliage in the moderate climates and apparently spreads rapidly during late summer and autumn. Epiphytotics of the disease are favored by relatively dry summer weather. Frequent rains reduce the development and spread of the disease (Yarwood, 1936). The source of inoculum in areas where epiphytotics occur is largely from conidia.

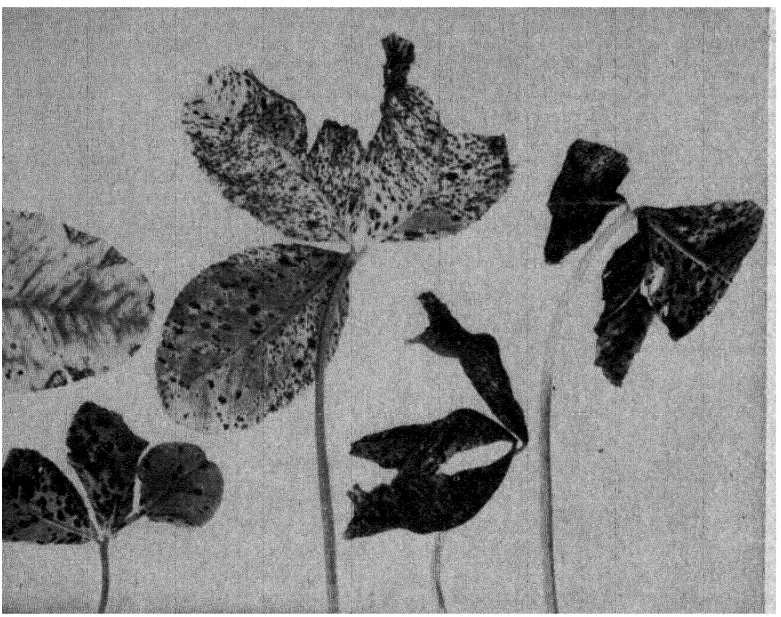
Control of the disease is chiefly by the use of resistant varieties. Jones *et al.* (1941) and Smith (1938) describe the range of resistance and reaction of resistant varieties. Resistant plants are found in most varieties. The European red clover varieties are generally more resistant than the American (Horsfall, 1930 and Mains, 1923, 1928).

Specialization of the parasite occurs on the clover species and probably

within the species, according to Hammarlund (1925), Neger (1902, 1923), Salmon (1900, 1903), and Yarwood (1936).

6. Spring Black Stem, *Phoma trifolii* E. M. Johnson and Valeau. (See Alfalfa.)

7. Sooty Blotch, *Cymadothea trifolii* (Fr.) Wolf.—The leaf blotch is distributed throughout the temperate zones on alsike and white clover and less commonly on the red and crimson, according to Horsfall (1930) and Elliott and Stansfield (1924). The dark-brown or black angular



blotches, more abundant on the lower surface, usually cause only minor defoliation. On the lower surface of the leaf the slightly elevated spots are covered with conidiophores bearing conidia of the Polythrincium stage. Later in the season the blotches appear black and warty due to the formation of the spermagonia and the perithecial stroma (Fig. 83). The leaves yellow, wither, and brown when the blotches are numerous.

The Fungus.—*Cymadothea trifolii* (Fr.) Wolf

[*Dothidella trifolii* (Fr.) Bayl.-Elliott and Stansf.]

(*Polythrincium trifolii* Schm. and Kunze)

(*Sphaeria trifolii* Pers.)

[*Placosphaeria trifolii* (Pers.) Feke.]

[*Plowrightia trifolii* (Pers.) Kill.]

The conidial stage develops during the spring and early summer. Conidiophores are borne on modified cells of the mycelium that form stromata on the undersurface of the leaf. The conidiophores are black, waxy, wavy, simple, and bear conidia followed by apical elongation around the attached conidia. Conidia are pale olivaceous, obovate, 1-septate, and slightly constricted at the septum. The spermagonial stage is formed during the autumn along with the perithecial bearing stroma. Spermagonia are globose to flask-shaped with abundant spermatia extruded in an exudate. Perithecia are imbedded in the stroma and are irregular in shape and opening. The asci are clavate with usually eight ascospores. Ascospores are oblong to ovate-oblong, hyaline, 1-septate, and constricted at the septum. Wolf (1935) reported on the cytology of the ascigerous stage and reviewed the literature on the morphology and etiology of the fungus. The fungus has not been grown on artificial media.

Etiology.—Primary infection occurs from conidia and ascospores. The ascospores develop in early spring, and they are responsible for the early infection. Conidial production is abundant during the summer, and it declines in the autumn as spermagonia and stroma develop. The disease is prevalent on low wet meadowland. Although red and crimson clover are infected in Europe (Killian, 1923), they are generally resistant in the United States.

8. Pseudoplea Leaf Spot, *Pseudoplea trifolii* (Rostr.) Petr.—This leaf spot is distributed generally in the humid, temperate climates on the clovers and alfalfa. Frequently the disease is severe enough on white clover to cause foliage browning. Apparently, this leaf spot occurs only on white clover in Britain (Sampson and Western, 1941).

Symptoms.—Numerous small black sunken spots occur predominantly on the leaves, although other young tissues show spots when infections are severe. The spots rarely enlarge, although a few larger lesions develop on white clover. Later the spots show a reddish-brown margin. The inconspicuous perithecia are imbedded in the necrotic tissues of the lesions late in the autumn.

The Fungus.—*Pseudoplea trifolii* (Rostr.) Petr.
[*Saccothecium trifolii* (Rostr.) Kirsch.]
(*Sphaerulina trifolii* Rostr.)
(*Pleosphaerulina briosiana* Poll.)
(*Pseudoplea briosiana* von Hohn)
(*Pseudoplea medicaginis* Miles)

Sampson and Western (1941) suggested *Saccothecium trifolii* as the preferable binomial.

The perithecia are flattened to round, imbedded in the tissues, with pronounced ostiole opening to the surface. Paraphyses are absent, and asci are few to a perithecium. The asci are broadly ovate with a thickened apex and contain eight closely packed spores. The ascospores are cylindrical, tapering at the ends, hyaline to light yellow, triseptate, and muriform late in their development.

Etiology.—The perithecia develop in the late autumn or in the following spring. Ascospores are the source of inoculum during the spring and

summer. The disease develops during cool, moist weather. No conidial stage is known (Hopkins, 1923, Horsfall, 1930, Jones, 1916, and Miller, 1925).

9. Sclerotinia Root Rot and Crown Rot, *Sclerotinia trifoliorum* Eriks.—The disease is distributed extensively, especially in the regions of mild winters or heavy snow cover. In the United States, the disease is of considerable economic importance in the southern clover belt. Damage is severe in the early spring when the plants are susceptible to the invasion of this and similar fungi, according to Pape (1937), Valleau *et al.* (1933), and others.

Symptoms and Effects.—The symptoms of the disease vary with season, weather conditions, and tissues invaded. Brown leaf spots occur in wet periods, especially late autumn. The heavily infected leaflets drop off and are overrun by the mass of white mycelium. Infection spreads downward into the roots and crown. The spring symptoms are largely a soft rot of the crown and roots and less frequently the basal portion of the annual stems. The new growth of the infected plants wilts, and the dead tissues are overgrown by the white mycelium under conditions of high moisture. The sclerotia develop following the mycelium and are numerous in the soil and on the dead tissues.

The Fungus.—*Sclerotinia trifoliorum* Eriks.

The sclerotia are black and vary in size and shape, ranging from smaller than clover seed to several times their diameter. Sclerotia germinate forming disk-shaped buff to pink apothecia 3 to 8 microns in diameter, borne on slender stalks. The hymenium is composed of closely packed eight-spored asci interspersed with slender simple paraphyses. The ascospores are usually arranged in one row in the narrow cylindrical ascus. The spores are oblong with rounded ends to elliptical and are one-celled. Spherical microconidia are formed in dense clusters on the aerial mycelium under some conditions.

Etiology.—The apothecia emerge from the soil in autumn, and the ascospores are discharged with force enough to carry them into the foliage above. Leaf and stem infections result under conditions of high moisture, and mycelium also develops on the dead leaves on the soil surface. Infection of the crown and root tissues is initiated in the autumn and spreads during the winter and early spring in mild climates or under heavy snow covering (Eriksson, 1880, Esmarch, 1925, Gussow, 1903, Pape, 1937, Wadham, 1925). In the United States clover belt, the aggressive development of the fungus occurs in the spring as the plants are recovering from winter injury and resuming vegetative activity after the winter dormant period (Valleau *et al.*, 1933, and Wolf and Cromwell, 1919). The sclerotia develop during the summer and remain viable for long periods. In Europe, considerable attention is given to the presence of sclerotia in seed lots. Doyer (1934) described the sclerotia of three

fungi occurring in clover seed lots: *Sclerotinia trifoliorum, Typhula trifolii* Rostr., and *Mitrula sclerotiorum* Rostr. Adapted varieties show some resistance to the disease.

Other sclerotium-forming fungi cause disease in the clovers and other legumes. *Sclerotium bataticola* Taub. is common as a root rot on red clover in the South Central United States, according to Henson and Valleau (1937). *S. rolfsii* Sacc. causes a root and crown rot in the warmer

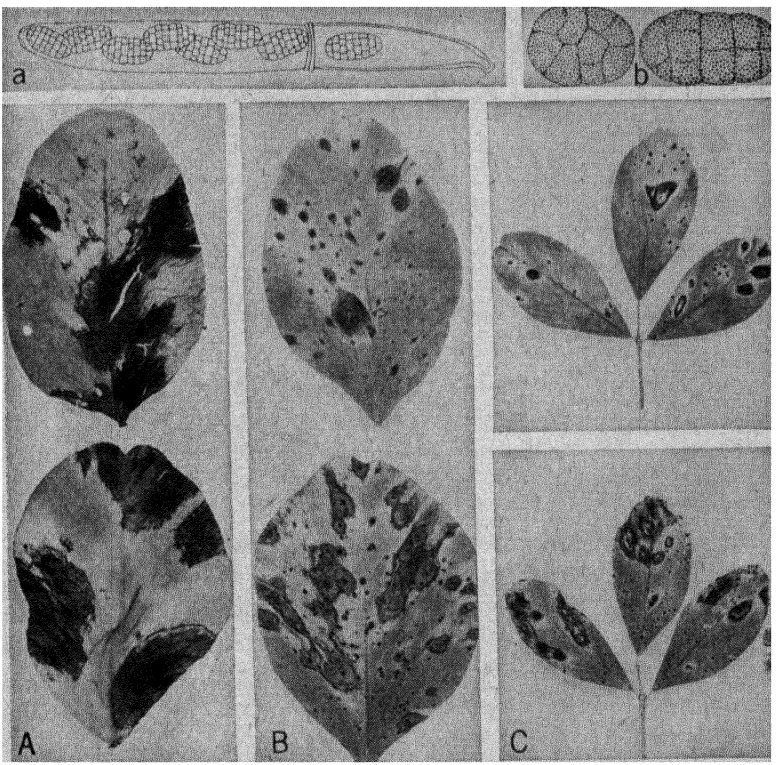


FIG. 81.—Leaf spots of red clover caused by *Pleospora herbarum* (*Stemphylium botryosum*) (*A*) and *S. sarcinaeforme* (*B*) and of alfalfa caused by *P. herbarum* (*C*). Drawings of the ascus with ascospores (*a*) and conidia (*b*) of the former species are shown. (*Courtesy of O. F. Smith.*)

areas of the United States. These fungi are associated with clover survival in pastures and meadows, especially in the Southern United States. Resistant strains of red and white clover are the best means of control (Albrecht, 1942). The clovers are relatively susceptible to the root rot caused by *Phymatotrichum omnivorum* (Shear) Dugg. (Taubenhaus and Ezekiel, 1936).

10. Stemphylium Leaf Spot, ***Pleospora herbarum*** (Fr.) Rab. (*Stemphylium botryosum* Wallr.) and *S. sarcinaeforme* (Cav.) Wiltshire.—These

leaf spots are common on the clovers and alfalfa in humid climates throughout the world. Some killing of the lower leaves occurs in red clover. The latter fungus species apparently is restricted to red clover, according to Smith (1940) and Wiltshire (1938).

Symptoms.—The lesions first are small, irregular dark-brown sunken spots; later development results in irregular concentric zonated light- and dark-brown lesions (Fig. 84). The wrinkled dark-brown to sooty leaves in the final stages of leaf blight remain attached to the plant. Stem and petiole lesions are restricted sunken brown spots.

The Fungi.—1. *Pleospora herbarum* (Fr.) Rab.
(*Stemphylium botryosum* Wallr.)
(*Macrosporium medicaginis* Cugini)

The perithecia are globose, membranous, and black. Asci are oblong to clavate with outer and inner walls. Ascospores are elongate to ovate, characteristically 7-septate, and muriform when mature. The conidiophores are short, arise singly or in groups, are occasionally branched, septate, and swollen at the apex. The conidia are ovate to elongate, 3- to 4-septate, somewhat constricted at the septa, muriform, and olivaceous, and echinulations to fine warts are numerous over the surface. Specialized races of this fungus occur on the clovers and alfalfa, according to Smith (1940).

2. *Stemphylium sarcinaeforme* (Cav.) Wiltshire
(*Macrosporium sarcinaeforme* Cav.)

The conidiophores and conidia are similar to the former species, except that the spores are smooth. This species apparently occurs on red clover only. Smith (1940) and Wiltshire (1938) reviewed the taxonomy and morphology of the several species on the legumes.

11. Southern Anthracnose, *Colletotrichum trifolii* Bain and Essary.—This anthracnose occurs in the warmer sections of North America, Europe, Kenya, and South Africa on the clovers and alfalfa. The disease is frequently severe on red clover in the southern clover belt of the United States, especially in unadapted susceptible varieties, and it occurs in mild form in mid-summer as far north as Canada, according to Monteith (1928) and Neal (1924).

Symptoms and Effects.—The light- to dark-brown lesions on the stems and petioles are the characteristic symptoms. The girdling of the stems results in the killing and browning of the foliage. The presence of the acervuli and numerous dark-brown setae on the mature sunken lesions differentiates this malady from the northern anthracnose and other stem diseases (Fig.85).

The Fungus.—*Colletotrichum trifolii* Bain and Essary

Acervuli develop in scattered erumpent form and vary considerably in diameter. Setae are few to numerous, uniseptate or continuous, dark brown to black, and much longer than the conidia. The conidiophores are cylindrical, varying in length with moisture, hyaline, and produce a succession of conidia apically. Conidia are hyaline to pink, straight, rounded at the ends, and without septations.

Etiology.—The fungus persists on the stems, crowns, and roots and spreads rapidly in warm, humid weather. The lesioning and killing of stems occurs largely on the new growth of the second crop. Resistant

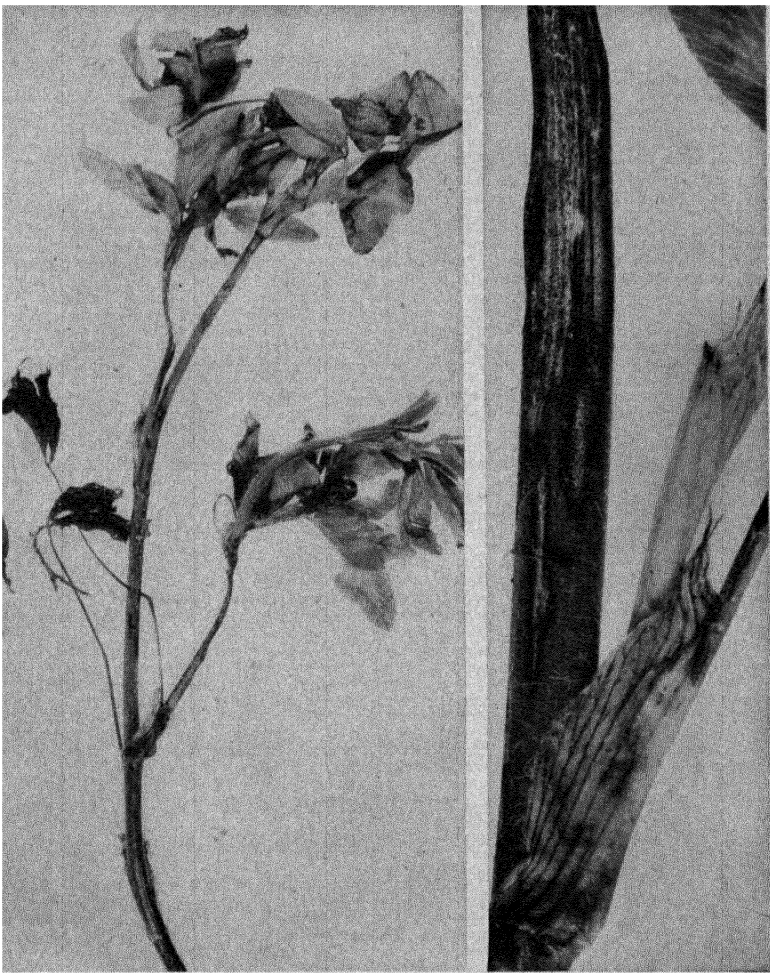


FIG. 85.—Red clover attacked by *Kabatiella caulivora:* stem lesions and petiole necrosis followed by death and browning of the leaves. The symptoms are relatively similar for both the northern and southern anthracnose.

varieties apparently keep the disease under control in the southern clover belt of the United States, according to Bain and Essary (1906), Monteith (1928), Pieters and Monteith (1926), and Wolf and Kipps (1926). Sampson (1928) studied comparatively the fungi causing the southern and northern anthracnoses.

Two other species, *Colletotrichum destructivum* O'Gara and *C. graminicolum* (Ces.) G. W. Wils., are reported on clovers. The former species occasionally causes some damage. These several species are similar morphologically.

12. Northern Anthracnose, *Kabatiella caulivora* (Kirch.) Karak.—The northern anthracnose is restricted largely to clovers, especially red and crimson, in the cooler sections of North America, Europe, and Asia. Damage is severe during periods of cool, wet weather, according to Sampson (1928).

Symptoms and Effects.—The brown lesions develop as sunken linear areas on the stems and petioles. Necrosis and cracking of the stem tissues are pronounced. Girdling of the stems and browning of the foliage symptoms are similar to the southern anthracnose. The white masses of conidiophores and conidia borne in irregular acervuli without setae are conspicuous in the deeper stem depressions and cracks (Fig. 85).

The Fungus.—*Kabatiella caulivora* (Kirch.) Karak.
(*Gloeosporium caulivorum* Kirch.)

The acervuli are small, irregular, hyaline, and without setae. The clusters of short cylindrical hyaline conidiophores bear one or more hyaline curved nonseptate conidia.

The etiology and control of the disease are similar to the former anthracnose. The fungus develops at low temperatures and invades the tissues during cool, humid conditions. Varieties resistant to the southern anthracnose are frequently susceptible to the northern anthracnose.

13. Rusts, *Uromyces trifolii* (Hedw. f.) Lév.—The clover rusts are distributed widely in the humid and semihumid areas. Usually these rusts cause little damage, although susceptible lines are damaged especially in the late autumn.

The varieties of *Uromyces trifolii* are all autoecious long-cycled rusts. The aecia occur as swollen light yellow to orange-yellow sori on the stems, petioles, and leaves. The aecial stage is more common on the white and alsike clovers than on the red clover. The uredia appear as small brown pustules on any portion of the green plant. The telia are similar in appearance to the uredia and occur in the old uredia or independently. The uredia and telia are not associated with hypertrophy of the tissues.

The Fungi.—1. *Uromyces trifolii* (Hedw. f.) Lév.

Arthur (1934) combined the morphologically similar species on the clovers and indicated their specialization by the use of the trinomials: U. *trifolii trifolii-repentis* (Liro) Arth. on *Trifolium incarnatum* L. and *T. repens* L.; *U. trifolii hybridi* (W. H. Davis) Arth. on *T. hybridum* L.; and *U. trifolii fallens* (Desm.) Arth. on *T. incarnatum* L., *T. medium* L., and *T. pratense* L. The synonymy is included by Arthur (1934).

The pycnia are in groups, chiefly on the leaves. Aecia with cup-shaped peridia are in groups on any tissues. The aeciospores are globoid, hyaline, and finely verrucose.

THE DISEASES OF ALFALFA, THE CLOVERS, AND SWEETCLOVER, WITH THE CAUSAL AGENT LISTED UNDER THE MORE COMMON SUSCEPT

Disease	Alfalfa	Medium red clover	White, alsike, and other perennial clovers	Sweetclover
Mosaic	Several viruses	Several viruses	Several viruses	Several viruses
Dwarf	Virus	None	None	None
Witches' broom	Virus	None	Possibly same as alfalfa	None
Bacterial wilt	*Corynebacterium insidiosum*	None	None	None
Bacterial blight	*Pseudomonas medicaginis*	*Pseudomonas syringae*	None	None
Crown wart	*Urophlyctis alfalfae*	*Urophlyctis trifolii* (Europe)	*Urophlyctis trifolii*	None
Leaf curl	None	*Olpidium trifolii* (Europe)	*Olpidium trifolii*	None
Pythium blight	*Pythium debaryanum* and other species	Same	Same	Same
Phytophthora root rot	None	None	None	*Phytophthora cactorum*
Downy mildew	*Peronospora trifoliorum*	Rare	Rare	Rare
Powdery mildew	Rare	*Erysiphe polygoni*	Rare	Rare
Spring black stem	*Ascochyta imperfecta*	*Phoma trifolii*	None	*Mycosphaerella lethalis*
Summer black stem	*Cercospora zebrina*	Less common	Less common	*Mycosphaerella davisii*
Stem hypertrophy	None	None	None	*Ascochyta caulicola*
Leaf spot and root rot	*Leptosphaeria pratensis*	None	Same	Same
Zonate leaf spot	*Pleospora herbarum*	*Stemphylium sarcinaeforme*	Same	Uncommon
Sooty blotch	None	Uncommon	*Cymadothea trifolii*	None
Pseudoplea leaf spot	Same	Same	*Pseudoplea trifolii*	Uncommon
Common leaf spot	*Pseudopeziza medicaginis*	*Pseudopeziza trifolii*	Same	Uncommon
Yellow leaf spot	*Pseudopeziza jonesii*	None	None	None
Sclerotinia root rot	Same	*Sclerotinia trifoliorum*	Same	Same
Fusarium, Cylindrocarpon root rots	Fusarium and Cylindrocarpon	Same	Same	Same
Southern anthracnose	Uncommon	*Colletotrichum trifolii*	Same	Rare
Northern anthracnose	Rare	*Kabatiella caulivora*	Uncommon	None
Rhizoctonia root rots	*Rhizoctonia spp.*	Uncommon	Uncommon	Rare
Rusts	*Uromyces striatus*	*Uromyces trifolii*	Same+*Uromyces nerviphilus*	Rare

Uredia are generally on the undersurface of the leaves and light brown. Urediospores are globoid or broadly ellipsoid, have a light brown wall, are thick, echinulate, and have 2 to 6 pores scattered or equatorial. Telia are in the uredia or are independent, brown, and not covered by the epidermis. Teliospores are one-celled, globoid or broadly ellipsoid, brown, and smooth or have a few scattered warts and short fragile pedicles.

2. *Uromyces nerviphilus* (Grog.) Hotson
(*U. flectens* Lagerh.)

This species is similar in morphology to the former and occurs on the veins and petioles of *Trifolium repens* L. and possibly *T. hybridum* L. Frequently it is in association with the former species. The uredial stage of this questionable species is omitted in the cycle.

Other diseases occurring on the clovers are discussed in Chap. XIII. The table on page 325 gives a summary of the more important diseases of alfalfa, sweetclover, and the clovers and the interrelationship of suscepts and parasites.

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CHAPTER XV

SOYBEAN DISEASES

The soybean, *Soja max* (L.) Piper [*Glycine max* (L.) Merrill], ranks high as an industrial and feed crop in the United States, especially during recent years. It is an ancient crop of eastern Asia. While the plant is largely self-pollinated, the species is represented by diverse types. Cold tolerance, date of maturity, disease reaction, as well as morphological characters, vary considerably in the collections introduced and in the varieties developed in the United States (Morse and Cartter, 1937). The cultivated species as well as the wild species of Asia, *G. ussuriensis* Regel and Maack, have 20 chromosome pairs.

Compared with other field crops, damage from diseases at present is comparatively light. However, the relatively large number of diseases occurring on the crop, the recent intensive breeding program in the United States, and the extensive acreage devoted to the crop all combine to increase losses from diseases unless special attention is given to the selection of disease-resistant varieties.

1. Leaf Spot and Leaf Discoloration, Nonparasitic.—Marginal leaf yellowing, firing, and browning are associated with mineral deficiencies, especially low potash, and other environmental disturbances (Kornfeld, 1933). Leaf scald occurs under conditions of high temperature and low humidity. Aphids and leaf hoppers cause damage when infestations are heavy (Gibson, 1922). According to Johnson and Hollowell (1935), the glabrous varieties are susceptible to injury by the potato leaf hopper, whereas the pubescent types that are grown commercially are relatively resistant.

2. Mosaics, Viruses.—The soybean mosaic is distributed widely on the crop and causes some damage in susceptible varieties. The leaves are dwarfed, margins are curled downward, the surface is puckered with dark-green areas between the veins and sometimes chlorotic spotting. The petioles and internodes are shortened, especially with early infections. The pods are stunted, flattened, and curved. Seed setting is delayed and reduced greatly. The virus apparently is limited to the soybean. It is transmitted in seed from infected plants, by mechanical means, and by aphids. *Myzus persicae* (Sulz.) and *M. convolvuli* (Kltb.) are the more common vectors, although six other species are reported as vectors (Gardner and Kendrick, 1921, Heinze and Kohler, 1940, Kendrick and

Gardner, 1924). Several other legume viruses produce symptoms on the soybean as well as the tobacco-ring spot virus that produces bud blight.

3. Bud Blight, Tobacco Ring-spot Virus.—The disease apparently is increasing in prevalence in the soybean area in the Central United States. The symptoms vary depending upon the age of the plant at the time infection occurs. Infections prior to the completion of terminal elon-

FIG. 86.—Bud blight of the soybean caused by the tobacco ring-spot virus. (*A*) Twisting and blighting of stem apex; (*B*) pod necrosis and browning.

gation of the stem result in unilateral elongation of the stem apex, a bronzed appearance of the young leaves, and ultimately necrosis and brittleness of the apical growing point (Fig. 86). Internal reddish-brown discoloration is evident, first in the pith near the nodes, and later spreading through the stems and accompanied by necrotic areas. The plants do not produce seed, and they remain green until frosted. Infections with the virus near the flowering period, which is generally more common, result in withering and dropping of young pod clusters and dark-brown blotches on the remaining poorly developed pods (Allington, 1946).

The tobacco ring-spot virus is associated with the bud blight of soybean. The virus is transferred mechanically, and insect transmission is indicated by the behavior of the disease in the field. Control measures and the reaction of varieties are unknown.

4. Bacterial Blight, *Pseudomonas glycinea* (Coerper) Stapp (*Bacterium glycineum* Coerper), (*B. sojae* Wolf).—The bacterial blight is distributed widely with the crop. The disease appears on the leaves, stems, and pods as light- to dark-brown irregular spots without a marginal halo. The lesions become confluent as they enlarge, the necrotic tissues dry out, and portions drop out. A limited amount of gray or brown exudate accumulates on the surface of the lesion (Coerper, 1919). Wolf (1920, 1921) retains the two species: *Pseudomonas sojae* Wolf causing the southern blight and *P. glysinea* (Coerper) Stapp, the northern.

The bacteria are seed-borne and persist in crop residue (Coerper, 1919, Kendrick and Gardner, 1921). Lesions on the cotyledons of infected seed and crop residue are a probable source of primary infection. Secondary spread occurs under favorable environmental conditions throughout the growing season. Defoliation and injury to the pods is minor under most conditions. Varieties vary widely in reaction to the disease.

5. Wild Fire, *Pseudomonas tabaci* (Wolf and Foster) Stapp [*Phytomonas tabacae* (Wolf and Foster) Bergey *et al.*], (*Bacterium tabacum* Wolf and Foster).—The disease occurs on soybeans as well as on tobacco in most of the intensive producing areas of North America. The lesions on the leaves are variable in size, and they are surrounded by a wide yellow halo under most environments. The restricted type of spot with indistinct halo is usually dark brown in contrast to the light-brown more extensive type of lesion. Exudate does not occur on the surface. When beating rains are frequent, the lesions spread rapidly and coalesce to involve the entire leaflets. Defoliation occurs around the base of the plant under such conditions. The infection usually occurs in definite areas rather than spread uniformly over the field, and it occurs relatively late in the season. The organism persists on crop refuse and in the soil (Allington, 1945).

6. Bacterial Pustule, *Xanthomonas phaseoli* var. *sojense* (Hedges) Starr and Burk.—The disease is more common in the southern soybean area, although it occurs in northern sections of the United States. Apparently the disease occurs in the Asiatic soybean areas. The lesions are common on the leaves as yellow pustular outgrowths on both surfaces. The spots later change to reddish brown with marginal yellowing under some conditions. The bacteria enter the stomatal cavity where they develop between the cells. Cell enlargement occurs without water-soaking of the tissues. The slightly elevated pustule, usually without exudate, differ-

entiates this disease from the bacterial blights. Small reddish-brown spots occur on the pods of susceptible varieties.

The Bacterium.—*Xanthomonas phaseoli* var. *sojense* (Hedges) Starr and Burk.
[*Phytomonas phaseoli* var. *sojense* (Hedges) Burk.]
(*Bacterium phaseoli* var. *sojense* Hedges)
(*Pseudomonas glycines* Nak.)
(*Bacterium glycines* Elliott)
(*Phytomonas glycines* Mag.)

The rod-shaped organism with single polar flagellum, capsules, and no spores appears yellow in culture (Hedges, 1924).

The organism persists in crop residues, especially in the southern areas. Secondary spread is rapid on susceptible varieties, especially during periods of beating rains. Resistant varieties are reported (Lehman and Woodside, 1929, Wolf, 1924).

7. Pythium Root Rot, *Pythium debaryanum* Hesse.—The light-brown soft rot of the roots and basal portions of the young stems is caused by the group of *Pythium* spp. common on legumes. The disease described by Lehman and Wolf (1926) is general throughout the soybean areas of the world, although relatively less damage occurs on this crop than on the small-seeded legumes (Chap. XIII).

8. Downy Mildew, *Peronospora manshurica* (Naum.) Syd.—The disease occurs rather generally with the culture of the soybean. Damage is confined to the regions of relatively cooler climates and susceptible varieties, where both foliage and beans are infected.

Symptoms.—The lesions on the leaves appear first as large or small chlorotic spots on the upper surface of the leaves. The mature lesions are grayish to dark brown with the downy mass of gray conidiophores and gray to violaceous conidia on the lower surface of the lesions. In susceptible varieties, the lesions spread rapidly over the leaf surface, causing yellowing and finally browning of the leaf. Oöspores are abundant in the brown leaf tissue. Pod infections occur at any of the nodes, but they are not evident from the exterior. In infected pods, the interior of the pod and the seed coat of the beans are incrusted by the grayish mass of mycelium and oöspores (Fig. 87). These symptoms are similar to those described by Snyder (1934) for the downy mildew on the pea, *Pisum sativum* L.

The Fungus.—*Peronospora manshurica* (Naum.) Syd.
(*P. trifoliorum* var. *manshurica* Naum.)
(*P. sojae* Lehman and Wolf)

The conidiophores are slender, branched, gray to pale violet, 5 to 9 microns wide by 240 to 500 long, and occur in groups from the stomata. The conidia are oblong

to nearly round, pale gray violet, and 18 to 21 microns wide by 24 to 27 long. The oospores are globose, smooth, hyaline to pale yellow, and 20 to 28 microns in diameter (Gaumann, 1923, Lehman and Wolf, 1924, Naoumoff, 1914).

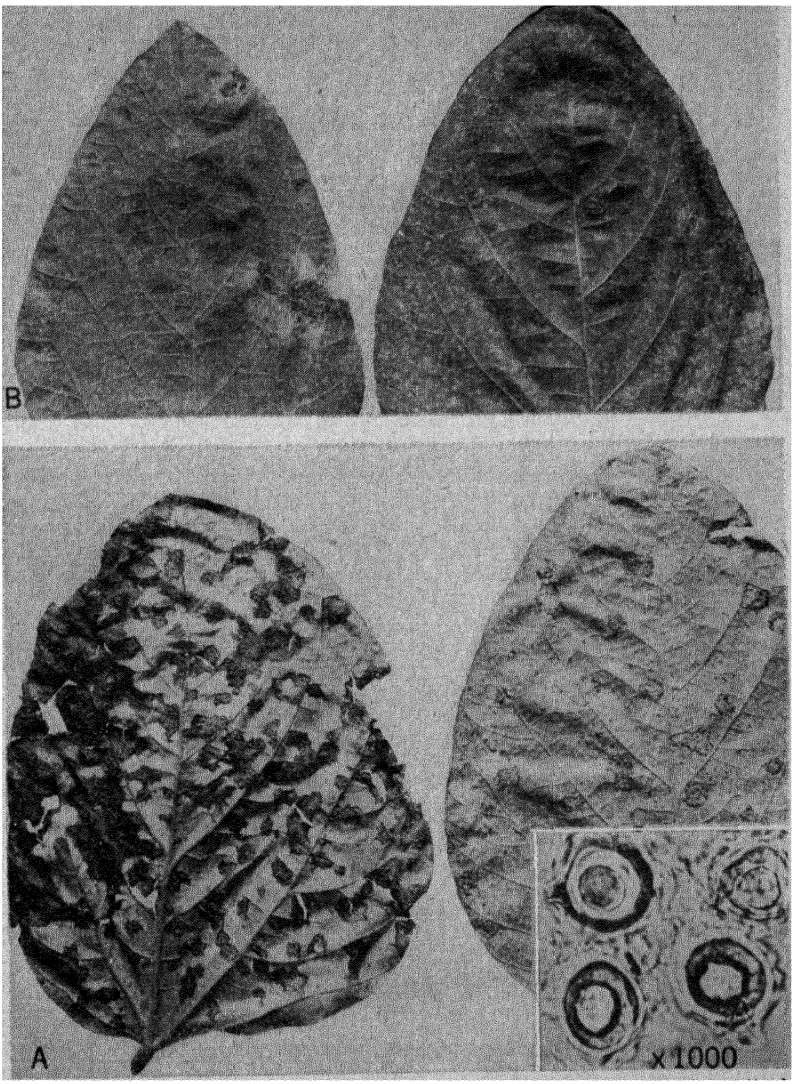

FIG. 87.—The downy mildew of soybean caused by *Peronospora manshurica:* (*A*) characteristic leaf lesions on susceptible varieties; (*B*) contrast between large and restricted leaf lesions of different varieties. Oöspores from infected beans are shown in the insert.

Etiology.—The oöspores persist in crop residue and on the seed. The oöspores on infected seed apparently produce both a local and a systemic infection in the young seedlings. Probably infection occurs in the

hypocotyl from the oöspores on the seed coat or in the crop residue. The mycelium develops through the hypocotyl into the first several trifoliate leaves and into some nodal buds (Jones and Torrie, 1946). Resistant varieties are the best means of control. Some of the high-yielding yellow selections adapted to the soybean belt of the United States are very susceptible. Seed treatment is effective in reducing seedling infection.

9. Powdery Mildew, *Erysiphe polygoni* DC.—The powdery mildew occurs sparingly on the soybean in the Southern United States and in Europe (Lehman, 1931). Apparently this is a specialized variety of *Erysiphe polygoni* (see Chap. XIV).

10. Anthracnose, *Glomerella glycines* Lehman and Wolf.—The disease is prevalent in the warmer areas in Asia, Europe and North America. In the United States it extends northward into the principal soybean belt. The lesions appear as dark-brown cankers on the cotyledons and hypocotyls of the seedlings. Seedling blight occurs before or after emergence, according to Ling (1940). The stem and pod lesions are indefinite brown areas causing premature death of the plants or plant parts. Seed infection is evident in some instances by the brown lesions. Numerous black setose acervuli develop on the infected parts during wet weather (Lehman and Wolf, 1926, Wolf and Lehman, 1926).

The Fungus.—*Glomerella glycines* Lehman and Wolf
(*Colletotrichum glycines* Hori)

The perithecia, submerged in the mycelial tufts, are membraneous, globose, rostrate, and 220 to 340 microns in diameter. The asci are oblong to bluntly clavate, 9.5 to 13.5 microns wide by 70 to 106 long, and interspersed with oblong paraphyses. Ascospores are hyaline, unicellular, bluntly pointed, slightly curved, and 4 to 6 microns wide by 18 to 28 long. Ascervuli are black with numerous brown setae. Conidia are hyaline, unicellular, bluntly tapered, curved, 4 microns wide by 20 to 22 long, and remain viable for relatively short periods.

Etiology.—The mycelium persists for relatively long periods in the crop residue and infected beans. Seedling stands are reduced by seed-borne mycelium and soil infestation on crop residue in the milder climates. Secondary stem and pod infections occur during wet weather. Ascosporic inoculum is probably of secondary importance in the areas where the disease is of importance, although it is a factor in the more northern areas. Apparently many of the commercial varieties are relatively resistant. Seed treatment with the organic mercury compounds reduces the loss in seedling stand.

11. Pod and Stem Blight, *Diaporthe sojae* Lehman.—The disease occurs in North America, Europe, and Asia. Damage usually occurs late as the plants are maturing, although premature killing of the plants and

reduced yields of beans are common in the Central United States. The dark-brown lesions with indefinite margins occur on the stems and pods and less commonly on the leaves. The infection usually starts at the junction of stem and branch and girdles the stem or branch. Pycnidia occur, scattered or arranged in rows, in the older dead portions of the lesions (Lehman, 1923, Johnson and Koehler, 1943).

The Fungus.—*Diaporthe sojae* Lehman

The perithecia, formed in the black mycelial stroma, are spherical with a slender tapering beak. Asci are sessile, clavate, hyaline, with eight spores borne in one or two rows. The ascospores are hyaline, elongate elliptical, aseptate, and 2 to 4 microns wide by 10 to 12 long. The Phomopsis stage consists of the subglobose osteolate pycnidia with short beaks or frequently without beaks. The conidiophores are slender, tapering, simple, and hyaline. The conidia are oblong to fusiform, straight, continuous, hyaline, and 2 to 3 microns wide by 6 to 7 long.

The fungus persists in crop residue as mycelium or pycnidia of the imperfect stage. The mycelium is seed-borne when conditions are favorable for late pod infection.

12. Fusarium Wilt or Blight, *Fusarium oxysporum* f. *tracheiphilum* (E. F. Sm.) Snyder and Hansen.—The Fusarium wilt of the cowpea and soybean occurs in the southern soybean belt of the United States and extends into the Western plains area. The roots and base of the stem show browning as well as the browning or blackening of the vascular bundles. The fungus is a vascular parasite, but true wilting does not occur in the woody plants. The leaves yellow and drop, and the pods are poorly developed. Apparently specialized strains of the parasite occur on the soybean and cowpea (Cromwell, 1919). Resistant varieties are the best means of control, as in the cowpea (Johnson and Koehler, 1943, Orton, 1902). Liu (1940) reported a pod lesioning and seedling blighting caused by a fungus resembling (*Fusarium tracheiphilum* E. F. Sm.) *F. oxysporum* f. *tracheiphilum*. Other *Fusarium* spp. cause seedling blight and root rot. The morphology of the fungus is given in Chap. XVI.

13. Cercospora Leaf Spot, *Cercospora daizu* Miura.—The frog eye spot occurs in Asia, Europe, and North America. The disease is distributed in the southern section of the soybean belt of the United States, and it is prevalent on late maturing varieties. The typical zonate gray spots with purplish-brown margins are numerous on the leaves. The stem and pod spotting occur as the plants mature, especially on late varieties. The brown to gray spots on the pods are smaller and less zonate than on the leaves. The fungus invades the pods, and mycelium covers the beans, but is not deep seated in the seed coat, according to Lehman (1928, 1934).

The Fungi.—*Cercospora daizu* Miura

The conidiophores, formed from thin stroma, are pale sooty black, nonseptate to rarely septate. The conidia are hyaline, cylindrical or fusiform, have a rounded apex, somewhat acute base, are nonseptate to as many as six septa, not constricted at the septa, and 5 to 7 microns wide by 39 to 70 long.

Another species, *Cercosporina kikuchii* Matsu. and Tomoyasu, probably a *Cercospora*, is reported from Eastern Asia (Matsumoto, 1928, Matsumoto and Tomoyasu, 1925).

The parasite persists in leaves and stems as well as in infected seed. Secondary spread from conidia occurs, especially late in the growing season. Early maturing varieties escape severe injury, although they yield less than the later varieties when free from disease. Seed treatment apparently does not control the disease in the area where damage is severe.

14. Septoria Brown Spot, *Septoria glycines* Hemmi.—The disease is distributed widely in North America, Europe, and Asia. The heavy infections are usually early and scattered, and defoliation occurs on the heavily infected leaves. Heavy early infections of the primary leaves is common; subsequent develoment of the leaf spotting is less common, except during wet seasons. The angular spots are brown turning to reddish brown as they become older. The spots range from small to large, especially where the smaller lesions coalesce. The small spots on the stems and pods occur late in the season. Pycnidia develop in the mature lesions (Hemmi, 1915, 1940). Seed infection is not conspicuous (Wolf and Lehman, 1926).

The Fungi.—*Septoria glycines* Hemmi

The pycnidia are submerged, flattened to globose, and open to the surface with a large pore. Conidia are hyaline, filiform, curved, have one to three indistinct septa, and are mostly 1.4 to 2.1 microns wide by 35 to 40 microns long.

A second species, *Septoria sojina* Thuem, causes light-brown indistinct spots on soybean leaves in Europe and Asia.

A third species, *Septoria sojae* Syd. and Butl., is reported on soybeans. The three species need further comparative study.

15. Sclerotial Root, Stem, and Crown Blights.—Several cosmopolitan parasites cause premature killing of the soybean plants. The general symptoms are similar in that the roots, crown, and basal portion of the stem are invaded, resulting in yellowing or wilting and the ultimate weakening or death of the plant. The various fungi are identified by the type of mycelium and the size, shape, and color of the sclerotia.

Sclerotium rolfsii Sacc. occurs in the sandy soils of the southern area of the United States. The large globose brown sclerotia on the base of the stem and upper portion of the tap root differentiate the southern blight. This organism attacks a wide range of crop plants in the southern states.

Sclerotium bataticola Taub. causes the charcoal rot of crop plants. The disease extends into the corn-belt section of the soybean area and probably causes more total damage to the soybean crop than the former species. Small round to irregular black sclerotia are produced below the epidermis on the dead stems and roots, and the *Macrophomina* pycnidia occur in the same tissues.

Rhizoctonia solani Kuehn and other species occur on the soybean, especially in the northern half of the area in the United States and in Asia. The white mycelial mats in association with the lesioned tissues during wet weather and the characteristic branching of the mycelium are the important distinguishing characters.

Phymatotrichum omnivorum (Shear) Dugg. causes a root rot of the soybean. The dead root and stem base show the characteristic brown discoloration below the bark.

16. Brown Stem Rot, Unidentified Fungus.—This disease is general in some years in the Midwestern United States. Damage is caused by premature killing in the autumn followed by extensive lodging. Symptoms consist of browning of the pith and xylem of the stem, starting at or below the soil level and progressing slowly upward with only slight external symptoms. Leaf symptoms are occasional early blighting of the lower leaves followed by a rapid interveinal chlorosis of the upper leaves and subsequent necrosis. In advanced stages of the disease, the outside of the stem appears brown and the weakened stems lodge badly. Low temperatures are necessary for disease development. The fungus appears to be soil-borne in crop residue and common in the commercial soybean area of the United States. The dense putty-colored nonsporulating mycelium grows slowly in culture. A few fields have indicated that rotation may alleviate the disease (Allington, 1946).

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SECTION IV

DISEASES OF FIBER AND OTHER FIELD CROPS

CHAPTER XVI

COTTON DISEASES

Cotton is one of the major fiber crops of the world. The several *Gossypium* spp. cultivated for fiber and seeds and the wild species were placed by Webber (1939) and others in four groups, as follows: (1) the wild Australian species (*Gossypium sturtii* F. Muell.) with 13 chromosome pairs, (2) the wild and cultivated Asiatic species with 13 chromosome pairs, (3) the wild American species with 13 chromosome pairs, and (4) the American semiwild and cultivated species with 26 chromosome pairs. Hybridization between the Asiatic and American cultivated species is difficult due to sterility and incompatibility in chromosome pairing (Harland and Atteck, 1941, Silow, 1941, Stephens, 1942). Crossing within the Asiatic and within the American cultivated species is general. Three types of American cotton are grown: (1) the sea island, (2) the American-Egyptian, and (3) the upland. Commercial varieties of all three types are probably of hybrid origin, and all three are adapted to somewhat different environments and industrial uses.

The cultivated species and varieties are grown over a wide range of environments within the warmer climatic zones of the world. In the tropical regions, many of the species are perennial. Adaptation of the hybrids to a shorter growing season results in annuals with indeterminate growth habit.

Cotton breeding received attention early in the history of the crop in North America. Probably John Griffin was the first to employ what is essentially the back-cross method 50 years before the geneticists fully established the method as a sound procedure in plant breeding (Ware, 1936).

The general spread of the boll weevil and Fusarium wilt over the cotton belt at the beginning of the present century caused a change in types of cotton grown. The earlier maturing varieties with determinate growth and fruiting habit as well as boll types better adapted to withstanding boll weevil injury replaced many of the older varieties, according to Ware (1936). By 1895, the Fusarium wilt disease had spread over the south-

eastern cotton belt of the United States and breeding for wilt resistance was started (Orton, 1900, Orton and Gilbert, 1912, Ware, 1936). Orton, in 1899, probably was the first man in the history of agriculture to submit plant populations to an epidemic of a disease in order to obtain resistant selections. However, selections on naturally infested soil were made by E. F. Smith and E. L. Rivers in 1895. Insects, diseases, and foreign economic competition all have played an important part in the changing of varietal types and the regions where cotton was grown.

Plant diseases are estimated as causing an average annual loss of 17.1 per cent of the crop, or 1,737 bales, between the years 1930 to 1939 (Plant Disease Survey).

1. Leaf Discoloration or Rust, Nonparasitic.—Mineral deficiencies cause leaf discoloration and dropping in cotton. Potash is the most common mineral deficiency in cotton production. The malady is called "rust" because the leaves develop a rusty-brown color. Potash deficiency is manifest by a reduction in plant growth, by the tip and margin of the leaves developing a yellow to brown coloration, and by curling of the leaf margins. The leaves finally turn reddish brown and shed prematurely, and the bolls are small and fail to open (Cooper, 1939, Neal and Gilbert, 1935). The potash deficiency also accentuates the development of the wilt disease (Young and Tharp, 1941).

Not only the vegetative development and fruiting of the cotton plant are effected by unfavorable environmental conditions, but also the retention or conversely the shedding of the bolls after they have formed. Dunlap (1945), Wadleigh (1944), and others have investigated the influence of environmental factors, such as light intensity, temperature, moisture, and mineral nutrients, on plant development and boll shedding.

2. Leaf Curl, Virus Transmitted by White Fly, *Bemisia tabaci* Gennadius (*B. gossypiperda* Misra and Lamba).—The malady has been reported from the Sudan, Africa, cotton areas on domestic and American cottons (Kirkpatrick, 1931). Leaf curl without mottling occurs on some types, and a typical mosaic with some shortening of internodes is common on American long-staple types. Initial transmission by the white fly *Bemisia tabaci* Gennadius from second growth sprouts is common. Resistant or tolerant varieties in domestic, Asiatic, and American cottons offer the best means of control.

3. Angular Leaf Spot, or Bacterial Blight,[1] *Xanthomonas malvacearum* (E. F. Sm.) Dows.—The bacterial blight is distributed widely in the cotton areas of the world. In the United States, the disease is more severe in the south central and south western sections of the cotton belt,

[1] Credit is given the Sub-committee on Cotton Diseases, Southern Experiment Station Committee or Research Council for the information contained in the report, Cotton Diseases, March, 1945 (mimeographed). Good literature lists are included.

where it occurs on the cultivated cottons and develops from inoculation on *Gossypium thurberi* Tod (*Thurberia thespesioides* A. Gray).

Symptoms and Effects.—The small round spots occur on the cotyledons during the early seedling stage of growth and angular lesions on the older plants. The disease on the leaves appears first as watersoaked spots that enlarge to angular brown to black lesions. The spots occur on the leaves from the seedling to the mature plant stage during periods of high temperatures. Black elongate lesions occur on the young stems, sometimes

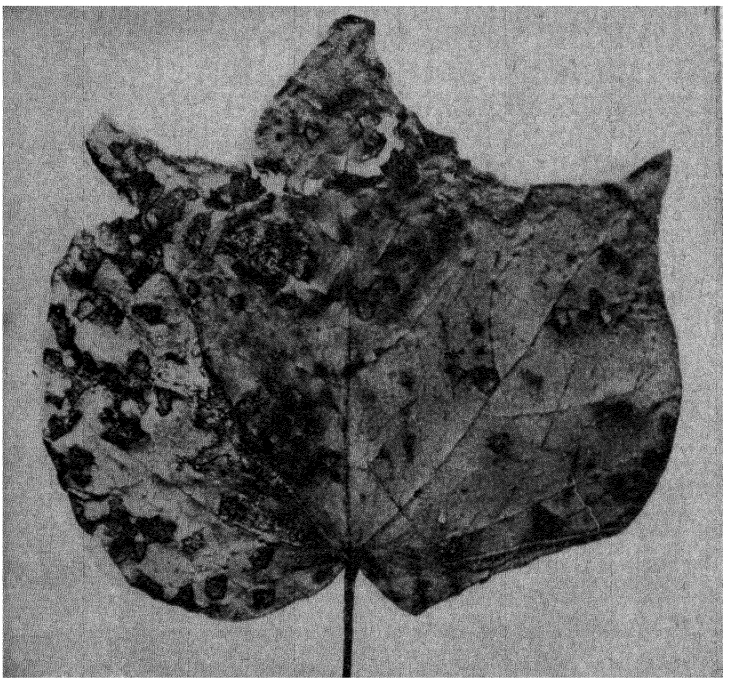

FIG. 88. Angular leaf spot of cotton caused by *Xanthomonas malvacearum*.

causing girdling and death of the stem. This type of symptom is referred to as "black arm." Angular to circular black sunken spots and rotting occurs on the bolls (Brown and Streets, 1937, Neal and Gilbert, 1935) (Fig. 88). Boll rot occurs during hot, humid weather, especially when insect punctures are numerous. Secondary organisms frequently enter through the bacterial lesions to discolor or rot the boll. Entrance of the bacteria is through stomata or wounds (Edgerton, 1912, Tennyson, 1936).

The Bacterium.—*Xanthomonas malvacearum* (E. F. Sm.) Dows.
[*Phytomonas malvacearum* (E. F. Sm.) Bergey *et al.*]
[*Bacterium malvacearum* (E. F. Sm.) E. F. Sm.]
(*Pseudomonas malvacearum* E. F. Sm.)

The rods are rounded at the ends, with one polar flagellum, no spores, and form pale-yellow colonies in culture.

Etiology.—The organism survives in the fuzz on the seeds, probably in the seed, and on crop residue. The external infestation of the seed and early seedling infection of the cotyledons is common. Under conditions of high moisture at harvest or overwintering in the fields, seed infection apparently occurs (Archibald, 1927). Volunteer seedlings from diseased fields frequently show infection of the cotyledons. Crop residue offers a means of infecting the new crop (Hare and King, 1940). The spread of the bacteria from infected cotyledons to leaves and other plant parts is associated with high temperatures, high humidity, wind, rain, and irrigation water, according to Brown (1942), Faulwetter (1917), Massey (1927), Stoughton (1928, 1930, 1931, 1932, 1933), and others. Water-soaking of the tissues prior to infection probably aids infection. Apparently environmental conditions of the western drier section are more favorable for the disease than those of the eastern section of the United States.

Control of the disease by seed treatment, crop rotation, and resistant varieties is practical. The use of sulphuric acid in delinting the seed gives good control in noninfested soil, according to Bain (1939) and Brown and Gibson (1925). The mercury dusts control seedling blight when they are used after the acid treatment as well as without delinting. Rotation of crops and deep plowing with irrigation and tillage well in advance of planting are useful in reducing the incidence of the disease from crop residue. Resistant varieties in some types of cotton are available. Knight and Clouston (1939, 1941, 1944) reported two factor pairs and modifiers in angular leaf spot resistance studies with old world types, sea island, and American upland varieties. Seedling reaction to the disease is similar to mature plant reaction, according to Weindling (1944).

4. Anthracnose, *Glomerella gossypii* Edg.—The disease is distributed widely on cotton. Severe damage to seedling stands and to bolls and seed occurs in the eastern humid section of the cotton belt of the United States. The disease is rare in the western drier area. The malady ranks first of those causing boll and seedling damage in the Southeastern United States, according to Edgerton (1912), Weindling *et al.* (1941), and others.

Symptoms and Effects.—The lesions and rotting occur on the cotyledons, leaves, stems, and bolls. Small reddish to light-colored spots or necrosis of the marginal tissues are common on the cotyledons. Similar oblong brown cankers occur on the hypocotyl and young stem (Harrold, 1943). Girdling of the stem occurs under conditions of high humidity. The lesions frequently are covered with the pink mass of spores in poorly

developed acervuli. Limited dead brown spots occur on the leaves and stems, especially associated with injuries or angular leaf spots (Fig. 89). The symptoms on the bolls vary, depending upon the time and manner of infection. Infection on the side of the partly developed boll results in a sunken brown spot with reddish margin. Slimy pink masses of spores are common in the center of the lesion during humid weather. Infection

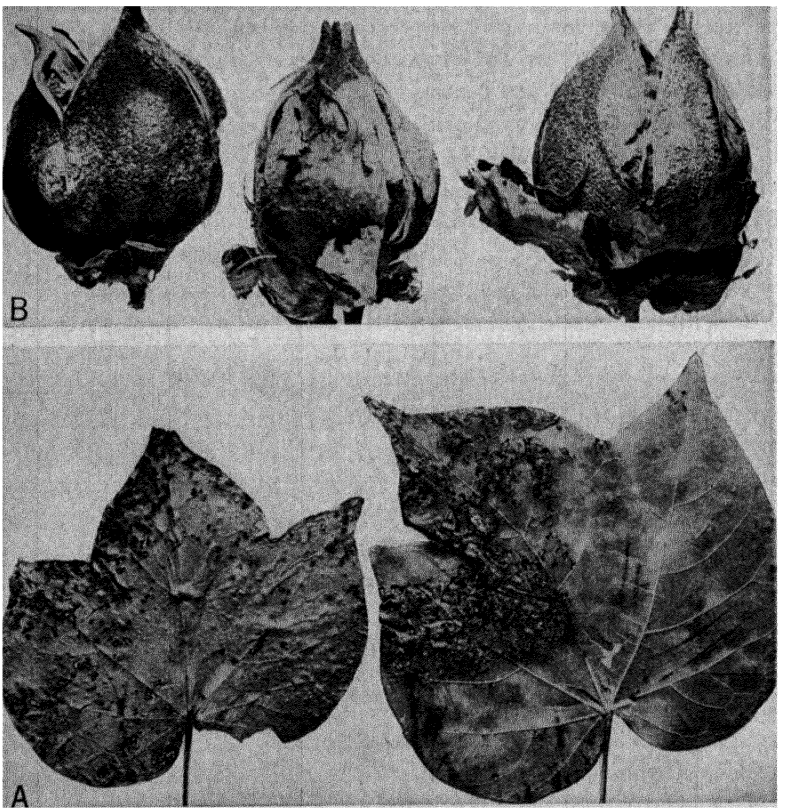

FIG. 89.—Anthracnose of cotton caused by *Glomerella gossypii*. (*A*) leaf lesions; (*B*) boll lesions.

through the dead pistil usually results in an internal rot and drying out of the boll. The general rot of the boll is associated with other organisms, especially *Xanthomonas malvacearum.* Acervuli with dark setae develop on the lesions after necrosis of the tissues.

The Fungus.—*Glomerella gossypii* Edg.
(*Colletotrichum gossypii* South.)

The perithecia, usually submerged in the dead tissues, are subglobose to pyriform, dark brown, with the beaks protruding, and 80–120 by 100–160 microns in size. The

asci are numerous, clavate in shape, and intermixed with slender paraphyses. The ascospores, arranged uniseriatly or irregularly biseriatly, are elliptical, sometimes slightly curved, hyaline, and measure 5–8 by 12–20 microns. The conidia develop on the mycelium or in acervuli, depending upon moisture. Acervuli vary in size and shape, and the setae are dark with hyaline tips. Conidiophores are short, closely packed, and hyaline. Conidia are cylindrical, generally straight, hyaline, one-celled, 3.5–7 by 12–25 microns in size, and are held in a mucilaginous mass on the acervulus. Ullstrup (1938) reported considerable variability in the fungus.

Etiology.—External seed infection is general, but the internal infection is limited; both result in poor stands and abundant conidial inoculum from the lesions on the cotyledons. Low temperature and low moisture increase the amount of seedling blight and damping-off of seedlings (Arndt, 1944). Spread of the infection to the plant tissues aboveground from secondary inoculum occurs during humid weather throughout the growing season. Crop residue is an additional source of inoculum.

Control.—Crop rotation and seed treatment are important control measures. Ceresan and Improved Ceresan control the seed infection borne externally and thus reduce the seedling infection and inoculum produced on infected seedlings. Varieties show some differences in susceptibility. Varieties resistant to *Xanthomonas malvacearum* show less boll rot from anthracnose.

5. Boll Rots and Seedling Blight.—A number of organisms are associated with boll rots and seedling blight in various sections of the world. The fungi associated with these diseases vary in different areas. The two diseases discussed previously are probably the most important of the group. Many of the secondary boll rots are due to infection following boll weevil injuries. *Aspergillus* and *Rhizopus* spp. cause boll rots following injuries and are common in the Southwestern United States. The former produces pink to brown lesions, the latter olive green to brown. Pink boll rot and root rot caused by *Gibberella fujikuroi* (Saw.) Wr. (*Fusarium moniliforme* Sheldon) is relatively common (Harrold, 1943, Woodroff, 1927). In addition *Fusarium, Diplodia, Sclerotium* spp. and other fungi are associated with either boll rots, seedling blights, or both (Gottlieb and Brown, 1941, Ray and McLaughlin, 1942).

Seed treatment is used extensively as a control of the seedling blights. The organic mercury compounds, especially Ceresan, are effective in increasing the stands of healthy plants. Rotation of crops using winter legumes in the rotation, soil preparation, and soil fertility are important preventative measures.

6. Fusarium Wilt, *Fusarium oxysporum* f. *vasinfectum* (Atk.) Synder and Hansen.—The wilt is distributed throughout the world in areas of sandy acid soil. The disease is among the oldest cotton maladies in the United States and causes heavy losses when susceptible varieties are grown on the sandy soils (Orton, 1900). Fusarium wilt is interrelated

closely with the root knot nematode [*Heterodera marioni* (Cornu) Goddey] and potash deficiency. The root lesion nematode [*Pratylenchus pratensis* (deMan) Filip.] probably also produces avenues of entrance for the wilt fungus (Smith, 1941). The Fusarium wilt is prevalent in the Southeastern United States into eastern Texas on cotton, okra, Burley tobacco and the coffee weed (*Cassia tora* L.). It is important again in the lighter soils of the Southwestern irrigated sections where Acala Upland and American-Egyptian varieties are grown.

Symtoms and Effects.—The plants show considerable variation in symptoms, depending upon the degree of resistance of the variety and environmental conditions. The diseased plants are small with smaller leaves and bolls. The leaves are yellow to brown and drop from the base upward. Wilting of the leaves and premature death of the plants occurs in susceptible varieties. Discoloration of the vascular elements and some plugging of the bundles is evident in sections of the root and stem. The symptoms apparently are caused by products of a toxic nature produced by the association of fungus and cotton plant rather than by the plugging of the xylem vessels, as reviewed by Gottlieb (1944) and Neal (1928). The variability in pathogenicity of the fungus is discussed by Armstrong *et al.* (1940).

The Fungus.—*Fusarium oxysporum* f. *vasinfectum* (Atk.) Snyder and Hansen

(*Fusarium vasinfectum* Atk.)

The species *Fusarium oxysporum* Schl., as amended by Snyder and Hansen (1940), gives sufficient latitude morphologically to include the *Fusarium* spp. of section Elegans (Wollenweber and Reinking, 1935) or the members of this genus producing the wilt diseases. The physiologic varieties producing wilt on the respective plants or groups of plants are designated by the use of trinomials. Smith (1899), in the early investigations on this group, suggested this when he considered watermelon, cotton, and cowpea wilts as caused by varieties of the same species, and certain others have followed this practice.

The conidiophores are verticillately branched, and they form in sporodochia, reduced pionnotes, or less frequently, on the mycelium direct. Ellipsoidal unicellular microconidia, averaging 2–3.5 by 5–12 microns in size, occur commonly. Sickle-shaped macroconidia, hyaline, mostly 3-septate, but sometimes 4- and 5-septate, typically 3–4.5 by 40–50 microns in size are common. Vegetative resting cells (chlamydospores) form either terminally or intercalary, and sclerotia are common. The ascigerous stage is not known.

Etiology.—Soil infestation is common in light acid or neutral soils. Infection occurs through the root system, with the fungus developing in the xylem. The wilting occurs during the growing season due to excretory toxic substances produced in the vascular elements.

Control is largely by the use of resistant varieties and crop practices. Varieties resistant to both wilt and root knot give better yields in infested

soils (Smith, 1941, Young and Humphrey, 1943). Wilt resistance is probably conditioned by a single factor pair (Fahmy, 1931, Kulkarni, 1937). In Egypt, the Asiatic cottons are susceptible as a group, whereas the American and Egyptian cottons contain resistant strains. The incorporation of organic material in the soil and a proper balance of potash fertilizer decrease the losses from wilt (Young and Tharp, 1941). Rotations to reduce nematode infestation decrease wilt damage.

7. Verticillium Wilt, *Verticillium albo-atrum* Reinke and Berth.—The disease is distributed generally, and apparently it is associated with alkaline soils. This wilt is prevalent in the Mississippi delta and the Southwestern United States. Verticillium wilt is severe in cool soils, especially when the plants are blossoming or later. Mottling of the leaves with pale-yellow irregular areas, later turning brown, are the most characteristic early symptoms. The infected plants ripen prematurely, and boll development is stopped. The interior xylem of the root and stem shows browning as in other plants infected with this wilt fungus.

The Fungi.—*Verticillium albo-atrum* Reinke and Berth. and *V. dahliae* Kleb.

Conidiophores are branched verticillately, and conidia are borne singly at the apex of the branches. Conidia are globose to ovoid, hyaline to lightly colored. The two species commonly associated with the wilts are differentiated by Berkeley *et al.* (1931) by the difference in type of resting mycelium. *Verticillium albo-atrum* produces dark torulose resting hyphae, whereas *V. dahliae* produces pseudo-sclerotia and sclerotia.

Both species are common in most of the areas where the disease is severe on crop plants. The rapid dissemination of the disease on cotton in the Southwestern United States is unexplained (Rudolph and Harrison, 1944). Tolerance to this wilt is associated with late maturity in most varieties. Wilt-tolerant varieties of the American types are being used in the Southwestern United States. Resistant varieties of the *Gossypium barbadense* L. type are more general than in the other species.

8. Phymatotrichum Root Rot, *Phymatotrichum omnivorum* (Shear) Dugg.—The Phymatotrichum or cotton root rot is found on more than 2000 different kinds of cultivated and wild plants. Losses in cotton are high in Texas, Arizona, and Mexico. Insofar as is known, the disease is limited to Southwestern North America.

Symptoms and Effects.—In local areas in the fields the plants wilt slightly and turn brown. The first symptom visible on the plant is the yellowing and bronzing of the leaves. Slight wilting of the leaves occurs as the next transitory symptom. Soon after wilting the leaves become brown and dry but remain attached to the plant (Fig. 90). Root rot is evident below the soil surface by the browning of the bark and cambium tissues and by the strands of the mycelium on the surface of the rotted

roots. Spore mats appear on the soil surface near the dead plants when the soil is moist after summer rains or irrigation. The mats are first cottony and white, later becoming tan in color and powdery in texture. The root rot appears in patches when the plants are partly grown, and the areas of dead plants spread as the season advances.

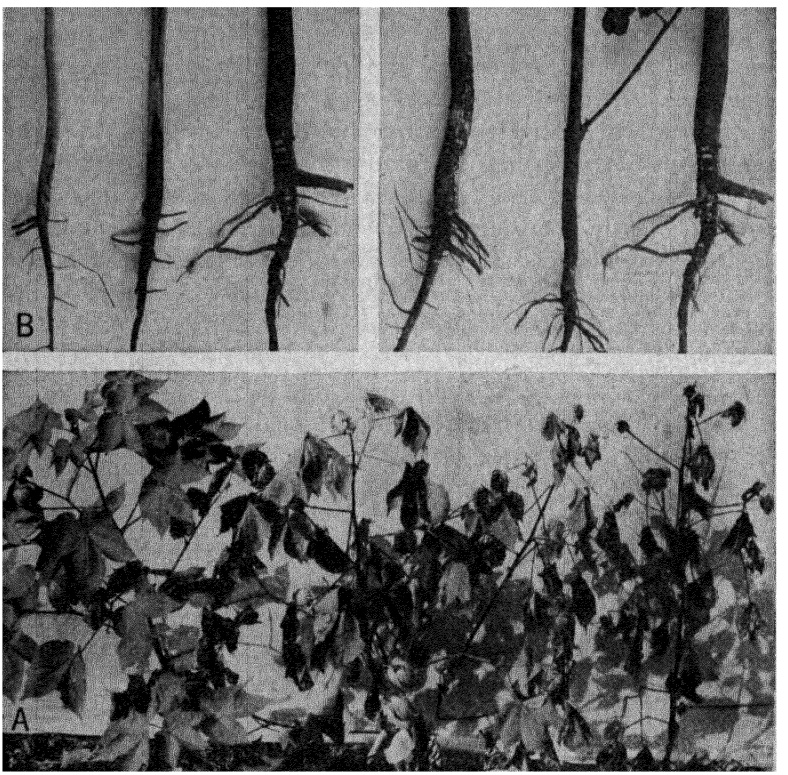

FIG. 90.—*Phymatotrichum* root rot of cotton, showing plants in various stages of wilting and browning (*A*) and the discoloration of the cortex of the root and stem base (*B*)

The Fungus.—*Phymatotrichum omnivorum* (Shear) Dugg. (*Ozonium omnivorum* Shear)

Streets (1937) gives a good description of the fungus and reviews the literature. The mycelium consists of two types: the less common large-celled mycelium, and the fine-celled. The mycelial strand consists of closely compressed hyphae with slender tapering side branches. These cruciately branched acicular hyphae are characteristic of the fungus when the strands are actively vegetative. Old strands are small, dark brown, and with little branching. The spore mat develops from the strands into a large-celled much-branched hyphal mass with swollen tips (conidiophores) bearing numerous globose to ovate smooth conidia 4.8 to 5.5 microns in diameter or 5–6 by 6–8 microns in the ovate-shaped conidia. Conidia apparently do not germinate or cause

infection. Sclerotia are round to irregular, small, light to dark brown, produced singly or in chains, and germinate to produce mycelium.

Etiology.—The mycelial strands and sclerotia persist in the soil for long periods. Soil infestation in the alkaline dark soils once established is rather permanent (Taubenhaus and Ezekiel, 1936). Infection and rotting of the roots of the numerous plants affected occurs during the growing season, especially from mid-summer into the autumn. The northern boundary of the general infestation apparently is determined by winter temperatures (Ezekiel, 1945). The monocotyledonous plants are immune to the root rot under field conditions, although corn is attacked under greenhouse conditions. Substances within the tissues apparently contribute to the immunity (Ezekiel and Fudge, 1938, Greathouse and Rigler, 1940). The fungus development in the soil is checked by the competition and antibiotic action of other soil organisms. Soil conditions favorable for the development of soil microflora apparently offers the best means of holding the root-rot parasite in check (Crawford, 1941, Mitchell, 1941).

Control.—Economical control measures are inadequate especially in the alkaline soils to which the fungus is adapted. Rotation with oats, corn, sorghum, and grasses reduces the damage. The use of winter cover crops, especially legumes, reduces the root rot in cotton in most of the areas where the disease is severe. The use of organic material high in nitrogen or with nitrogen fertilizer, especially when combined with deep tillage and aeration, decreases root-rot damage and increases cotton yields (Adams *et al.*, 1939, Blank, 1944, Jordan *et al.*, 1939). Early maturing varieties tend to escape the damage. Some difference in reaction of cotton varieties and selections is indicated by Goldsmith and Moore (1941), Streets (1937), Taubenhaus and Ezekial (1936), although the large number of *Gossypium* spp. and varieties tested to date do not indicate the presence of resistance to this disease.

9. Soreshin, *Rhizoctonia solani* Kuehn.—The seedling stem canker known as "soreshin" is distributed generally on cotton and is common in the United States. Losses in seedling stands due to the disease are large in some areas and seasons, making necessary the replanting of large acreages.

Symptoms.—The characteristic symptom is the stem canker or soreshin near the soil line, although seedling blight is very common earlier in the season. The seedlings blight before or after emergence during cold, wet weather. The seedling rot develops first in the hypocotyl with little discoloration of the cortical tissues. Necrosis of the hypocotyl tissues and blighting of the seedling occurs in cold, wet soils. Under conditions less favorable for disease development, the hypocotyl lesions later appear as reddish-brown sunken cankers extending into the base of the stem.

These cankers range from linear cortical lesions to completely girdling the stem near the soil line. Partial recovery of the plants with new root growth occurs, especially in older plants, as the soil becomes warm. Angular brown shot-hole type leaf spots occur less frequently than the stem lesions (Neal, 1942, 1944).

The fungus is soil-borne and attacks a wide range of plants. The disease develops on cotton during periods of cold, wet weather. Crop rotations, good soil preparation, seed treatment with the mercury dusts and Arasan, and planting after the soil is warm are important control measures (Fahmy, 1931). Seed treatment is not effective in controlling the later cankering of the stems, although early hypocotyl invasion and seedling blight is reduced by the residual protective action of the dust fungicide.

10. Rust, *Puccinia stakmanii* Presley.—The aecial stage of *Puccinia stakmanii* occurs on cotton in the Southwestern United States. Under favorable conditions, infection is heavy and causes reduction in yields. The uredial and telial stage of the rust occur on *Bouteloua* spp., desert grasses common through the area (Presley and King, 1943).

Symptoms.—The circular slightly elevated orange-yellow to citron-yellow aecia occur chiefly on the undersurface of the cotton leaves (Fig. 91).

The Fungus.—*Puccinia stakmanii* Presley
(*Aecidium gossypii* Ell. and Ev.)
(*Puccinia hibisciata* Kell.)
(*Puccinia schedonnardi* Kell.)

The aecia develop chiefly on the leaves as slightly elevated spots, 2 to 5 mm. in diameter, with the conspicuous peridia first orange and later fading to yellow, and the margin of the peridium lacerate or recurved. Aeciospores are globose or broadly oblong, pale yellow to colorless, and finely verrucose. Uredia are formed chiefly in the leaves of the grasses, and they are pale brown with the epidermis of the leaf turned back. Urediospores are globoid or broadly ellipsoid, echinulate, with equatorial germ pores which distinguish this species from the others common on the grasses. Telia are abundant in the uredia, and they are formed also independently as naked sori. Teliospores are oblong to broadly ellipsoid, only slightly or not constricted at the septum, and rounded at both ends, the pedicel is long and sometimes attached at an angle.

Etiology.—The uredia and telia develop on the grasses during the spring and early summer. Sporidial inoculum from the teliospores in the grasses is abundant during the summer rains and results in the aecial infection on the cotton. The presence of rusted grasses in the fields or in waste areas near the fields and summer rains determine the amount of rust damage on the cotton. Most American cotton varieties are susceptible, whereas the Asiatic cottons are resistant or moderately susceptible.

A second rust, *Cerotelium desmium* (Berk. and Br.) Arth., occurs sparingly on cotton in the Southeastern United States and more generally

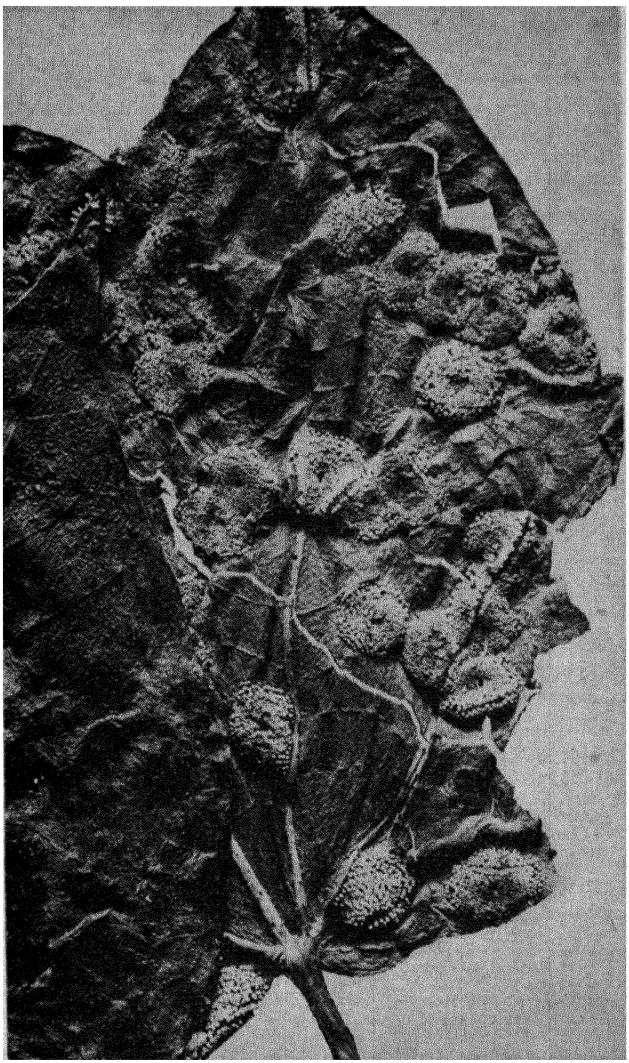

FIG. 91.—Part of a cotton leaf showing the densely massed aecia of *Puccinia stakmanii* on the leaf surface.

in South America, West Indies, India, and the South Pacific. Only the uredial stage is known.

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CHAPTER XVII

FLAX DISEASES

Varieties of common flax, *Linum usitatissimum* L., consist of two types: fiber and seed. These differ considerably in character of plant growth. The fiber types develop tall slender stems, produce a high content of good quality fiber, and bear seeds of low oil content. Seed flax varieties develop shorter stems with more tendency to branch and usually bear larger seeds of higher oil content. Flax is adapted to a wide range of environmental conditions but does best in cool climates. It is grown in the warmer regions of the temperate zones as a winter crop and in the cooler regions as a summer crop. Seed flax varieties are more generally grown in the drier areas, whereas fiber flax is grown in the humid sections.

A wild species, *Linum angustifolium* Huds., apparently is related closely to the cultivated flax. Both are of Asiatic or European origin, have 15 pairs of chromosomes (Kiluchi, 1929, Ray, 1944, Tammes, 1928), and hybridize readily. The other wild *Linum* spp. of the Old and New World that have been investigated have not been compatible in crossing with common flax, although several have the same number of chromosomes (Ray, 1944). Some of these wild *Linum* spp. are susceptible to important diseases of cultivated flax, notably pasmo and rust.

Flax improvement in North America has centered primarily about the successful battle against diseases (Dillman, 1936), although more recently attention has been directed toward combining disease resistance with improved oil content and quality. Flax wilt played an important role in the development of the crop in North America. As early as 1890, Lugger (1896) conducted experiments on the control of wilt by the use of fertilizers and seed treatments. Bolley (1901) described the disease, named the parasite, and reported on wilt-resistant varieties; although unknown to him, Broekema (1893) in the Netherlands, described a similar disease complex and reported resistance to the malady; and in 1896, in Japan, Hiratsuka described the disease as being caused by a species of *Fusarium*. While losses from flax wilt, rust, and pasmo are greatly reduced by the use of resistant varieties, these and other diseases cause heavy losses in this crop in some years.

1. Heat Canker, Nonparasitic.—Heat injury of the cortical tissues of hypocotyl and stem near the soil line is common on flax (Reddy and

Brentzel, 1922) and other succulent herbaceous plants (Hartley, 1918, Harvey, 1923, Tubeuf, 1914). Surface temperatures in the dark-colored soils frequently are high enough to kill the cells of young cortical tissues before the plants are large enough to shade the surface. Cankers resulting from such injury are common in flax in the semihumid plains and at high

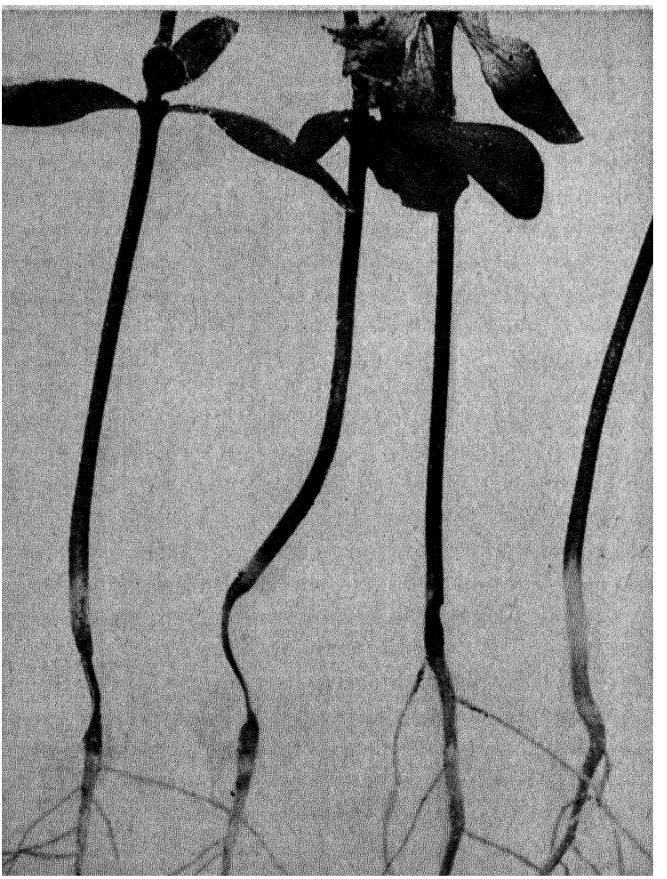

FIG. 92.—Heat canker of flax caused by high soil temperatures near the soil surface.

altitudes throughout the world. The cortical tissues collapse, resulting in the death of the young seedlings or in sunken brown lesions on the stems. The stems usually enlarge above the canker in the plants that survive the initial injury (Fig. 92). Cortical rotting organisms frequently invade the injured tissues to increase the damage (Reddy and Brentzel, 1922). Preventing excessive soil temperatures by early planting, by drilling the rows north and south so as to secure maximum shading, by

higher rates of seeding, by use of a nurse crop, by mulching the soil surface, and by irrigation are means of reducing this type of damage.

2. Seedling Blight and Root Rot, *Pythium* Spp. and Other Fungi.—The seedling disease complex known as seedling blight, scorch, fire, and root rot is common in the flax-producing areas of the world. The Pythium root rot is probably the most important single factor in this complex. The literature is reviewed by Berkeley (1944).

The symptoms of the disease complex vary considerably. The seedlings blight in the early stages of germination or after emergence in the more severe manifestations of the malady. When the disease develops following the seedling stage, the plants are dwarfed, the lower leaves turn brown, and the root system is reduced by a brown soft rot. The cortical tissues are invaded, followed by the rotting of the vascular tissues under conditions of high soil moisture and high temperature.

The fungus *Pythium debaryanum* Hesse reduces stands and causes root rot, especially in the prairie soils of North America. Its morphology is given in Chap. XIII.

According to Diddens (1932) and Middleton (1943), *Pythium aphanidermatum* (Edson) Fitzp., *P. splendens* Braun, *P. vexans* DBy., *P. megalacanthum* DBy., *P. mamillatum* Meurs, *P. irregulare* Buis., and *P. intermedium* DBy. are associated less commonly with root rot of flax. *Olpidium brassicae* (Wor.) Dang. (*Asterocystis radicis* DeWild.), *Thielaviopsis basicola* (Berk. and Br.) Ferr., and other fungi are associated with the root rot.

The soil-borne organisms invade the seedling tissues under favorable environmental conditions, and they cause severe damage to stands when mechanically injured seed is used. Crop rotation and seed treatment with the organic mercury compounds or Arasan help protect stands.

3. Powdery Mildew, *Erysiphe cichoracearum* DC.—The powdery mildew is probably of no economic importance in North America, Europe, and Asia, although it is reported abundant on flax in Siberia. The powdery superficial gray mycelium and conidia are formed on the leaves, stems, and floral structures.

The Fungus.—*Erysiphe cichoracearum* DC.
(*Oidium lini* Skoric)

Perithecia are scattered, reddish brown, and develop simple appendages. Asci are stalked, clavate, hyaline, and usually contain two oblong to ovate ascospores. The ovate hyaline conidia are borne in chains from the apex of the conidiophore. Allison (1934) reported that only a few varieties were infected in the Minnesota plots. Homma (1928) reported *Erysiphe polygoni* DC. on flax in Japan.

4. Pasmo, *Mycosphaerella linorum* (Wr.) Garcia-Rada.—Pasmo occurs in the seed- and fiber-producing areas of North and South America, New Zealand, and Europe. The disease causes defoliation, reduces yield of

seed, and damages the fiber (Brentzel, 1930). Newhook (1942) reported the disease as general on wild flax (*Linum marginale* A. Cunn.) in New Zealand.

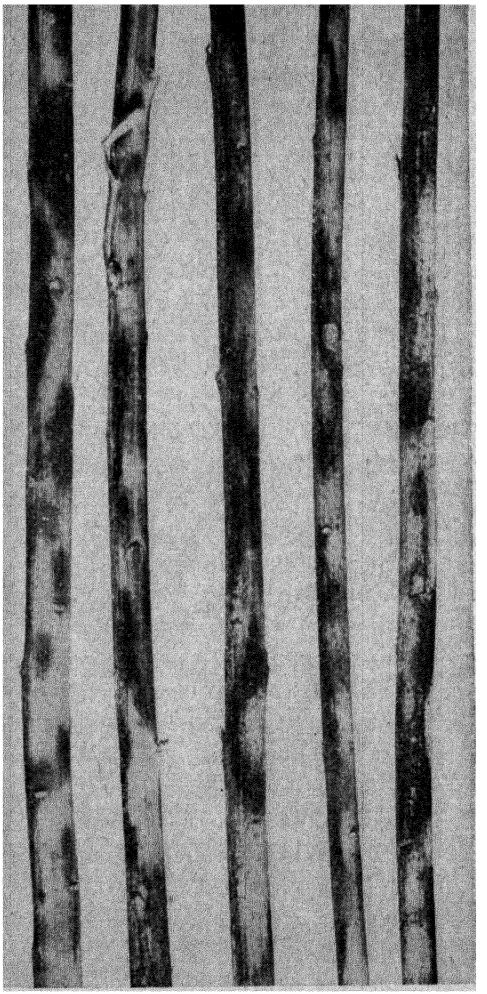

FIG. 93.—Pasmo disease caused by *Mycosphaerella linorum* on flax stems.

Symptoms.—The symptoms of the disease are striking and easily recognized, especially during the latter part of the growing season. Lesions develop first on the cotyledons and later on the lower leaves of the seedlings. The lesions are generally circular in outline and vary in color from greenish yellow to dark brown, depending upon age. Pycnidia

develop abundantly in the older lesions on the cotyledons and on the leaves. Later the stem lesions develop, first as small elongated lesions which then enlarge and coalesce, extending around the stem as well as longitudinally. The infected areas alternate with green tissue until infection becomes severe; then the stems brown as the plants are defoliated in the spots where the disease is severe (Fig. 93). Lesions also occur on the bolls. The pycnidia develop on the stem lesions as they turn brown. Perithecia occur sparingly on the old dead stems.

The Fungus.—*Mycosphaerella linorum* (Wr.) Garcia-Rada
(*Sphaerella linorum* Wr.)
(*Phlyctaena linicola* Speg.)
(*Septogloeum linicola* Speg.)
[*Septoria linicola* (Speg.) Gar.]

The perithecia formed on the dead stems are spherical to oval, 70–100 by 60–90 microns in size, and scattered. The asci are oblong to clavate or subcylindrical, sessile, straight or tortuous, hyaline, with eight spores irregularly biseriate or uniseriate. The ascopores are fusiform, mostly curved, aseptate, hyaline, and measure 2.5–4 by 11–17 microns. The submerged pycnidia are subglobose, 62 to 126 microns in diameter, with small ostioles when fully developed. The conidia are subcylindrical, tapering slightly to the ends, straight or curved, usually 3-septate, but some are 7-septate when mature, the 3-septate conidia measure 1.5–3.0 by 12–28 microns and are hyaline. Wollenweber (1938) described the ascigerous stage as *Sphaerella linicola* Wr. but in a note at the time of publication changed the binomial to *S. linorum* Wr., as Naoumoff (1926) had described *Mycosphaerella linicola* Naum. on flax stems in Russia. Wollenweber decided the two were not the same, as the perithecia described by Naoumoff were larger (200 microns). The descriptions are similar otherwise. Naoumoff (1926) described *Ascochyta linicola* Naum. and Wass. causing stem browning and *Phoma linicola* Naum. on the stems late in the season. The latter fungus is probably the same as described on *Linum mucronatum* in 1914 from Mesopotamia as *P. linicola* Bub., from Belgium in 1926 on *L. usitatissimum* as *P. linicola* March. and Verpl., and from Ireland as *Phoma* sp. Pethybridge *et al.*, 1921. Rost (1937) discussed the distribution of *M. linorum* and Garassini's transfer of the conidial stage to the genus *Septoria*. Specialization of the parasite was studied by Rodenhiser (1930).

Etiology.—The fungus persists on old straw, both as mycelium and conidia. The perithecial stage apparently is unimportant in the etiology of the fungus, and it is not common in North America. Primary and secondary inoculum is largely from conidia. Continued wet weather late in the growing season increases the spread and damage.

Control.—Rotation and removal of straw or covering the straw in plowing helps reduce the inoculum. Seed treatment reduces the seed-borne conidia. Resistant varieties, such as Buda (C.I. 336), Bison (C.I. 389), Koto (C.I. 842), and Crystal (C.I. 982), reduce the damage materially.

5. Browning and Stem Break, *Polyspora lini* Laff.—The disease is distributed generally on flax. It is of little economic importance in North

America, but causes damage to fiber flax in Europe, New Zealand, and Asia.

Symptoms.—The symptoms of browning are conspicuous on the seedlings and on plants approaching maturity. Browning is largely a seed-borne disease, and the primary light-gray to brown circular lesions with darkened margin develop on the cotyledons. From the primary lesions the infection spreads to the cotyledonary node where a canker is formed. At any subsequent stage the stem may break at the canker. Since most stems are not completely severed they become semierect, but ripen prematurely producing little seed. Circular gray to brown lesions develop on the leaves, capsules, and stems late in the season. The development of the fungus is largely in the cortical and parenchymatous tissues. Minute ascervuli-like structures bearing abundant conidia develop on the lesions during periods of high moisture.

The Fungus.—*Polyspora lini* Laff.

Small indefinite stromata are formed usually over the stomatal cavities. The hyaline conidiophores form as branches from this mycelial mat and bear masses of conidia from the swollen tips. No setae are formed. The conidia are oval to cylindrical, straight, with bluntly pointed ends, hyaline, and nonseptate. The width of the conidia is constant at about 4 microns; the length is 9 to 20 microns (Henry, 1925, 1938, Lafferty, 1921, Schilling, 1922).

Etiology.—The fungus is primarily seed-borne but may persist in the crop residue. Inoculum from the cotyledons and infected seedlings initiate the general infection. Secondary spread occurs during periods of high moisture (Flor, 1936, Henry, 1925, Lafferty, 1921).

Control.—The use of disease-free seed, seed treatments, and resistant varieties are important in control of browning. Seed treatment with the organic mercury compounds reduces seed infection (Henry, 1938, Muskett and Calhoun, 1941). Hot water-treatment, 10 minutes at 126°F., is recommended by Baylis (1941). The variety Rio (C.I. 280) is highly resistant to the disease (Flor, 1925, Baylis, 1941).

6. Anthracnose, *Colletotrichum linicolum* Pethyb. and Laff.—The anthracnose occurs on both the grain and fiber flax varieties in humid, cool areas throughout the world. Damage is severe in the California seed flax area on Punjab (C.I. 20) grown in the winter and in the fiber flax regions of Europe and Asia. Bolley (1903) first described the disease as "flax canker" and named the organism *Colletotrichum lini* Bolley, but he did not include a description of the fungus.

Symptoms.—The symptoms are typical of most of the anthracnose diseases. Cankers on the cotyledons are circular zonated sunken brown spots that spread under cool and moist conditions to involve the cotyledons and apex of the stem. Seedling blight occurs either before or after emergence, in the latter case usually as a stem canker at the soil line.

Leaf spots and stem cankers are common during the growing season, especially under conditions of high moisture. Brown spots form on the capsules, and less conspicuous lesions are found on the seed. Acervuli develop on the mature lesions.

The Fungus.—*Colletotrichum linicolum* Pethyb. and Laff.
(*Colletotrichum lini* Bolley)
[*Colletotrichum lini* (West.) Toch.]

The acervuli are formed subepidermally, and they rupture the epidermis. Setae are erect, usually 3-septate, and dark brown. Conidiophores are short, hyaline, and mostly simple. Conidia are cylindrical, tapering toward both ends, straight to slightly curved, hyaline, and aseptate to diseptate when mature. Westerdijk (1916) reported a somewhat similar disease, suggested the *Colletotrichum* nature of the fungus, and named the fungus *Gloeosporium lini* Westerdijk, however, the fungus was probably *Polyspora lini.*

Etiology.—Infected seed and crop residue are the important sources of infection. Conidia produced on the seedling cankers furnish the primary inoculum. Secondary infection occurs whenever weather conditions are favorable. Stands are reduced and fiber is damaged by the disease (Hiura, 1924, Pethybridge and Lafferty, 1918, 1920).

Control.—The disease is controlled largely by the use of sound seed, seed treatment, rotation of crops, and resistant varieties. Buda (C.I. 326) and Crystal (C.I. 982) are resistant, whereas Punjab (C.I. 20) is highly susceptible (Ray, 1945).

7. Fusarium Wilt, *Fusarium oxysporum* f. *lini* (Bolley) Snyder and Hansen.—The wilt disease is distributed generally with flax culture (Baylis, 1940, Bolley, 1906). Wilt has been associated with the culture of flax in North America (Bolley, 1901) to the extent that the flaxseed industry was threatened prior to the development of resistent varieties.

Symptoms.—Flax plants are attacked by wilt at any stage in their development, and symptoms vary with varieties and with environmental conditions. Although primarily a wilt, seedling blight occurs when susceptible seedlings are grown at high temperatures. In typical wilt, the leaves turn yellow or grayish yellow, the apical leaves thicken, growth stops, and the plants die and turn light brown (Fig. 94). Frequently the plant is only stunted, in which case the leaves turn yellow and fall prematurely or the primary stem dies and new apparently healthy lateral branches develop from the first node. A late infection or a weak attack may be evidenced by premature ripening.

The Fungus.—*Fusarium oxysporum* f. *lini* (Bolley) Snyder and Hansen
(*Fusarium lini* Bolley)

Conidiophores are short and branched and usually form in erumpent sporodochia. Conidia are fusiform to falcate, 3-septate, and hyaline to light pink in mass; micro-

conidia are not abundant (see Chap. XVI for morphology). Specialization of the parasite is reported by Borlaug (1945) and Broadfoot (1926).

Etiology.—The fungus is primarily soil-borne; it persists for several years in the soil to invade the young plant through the roots, and develops

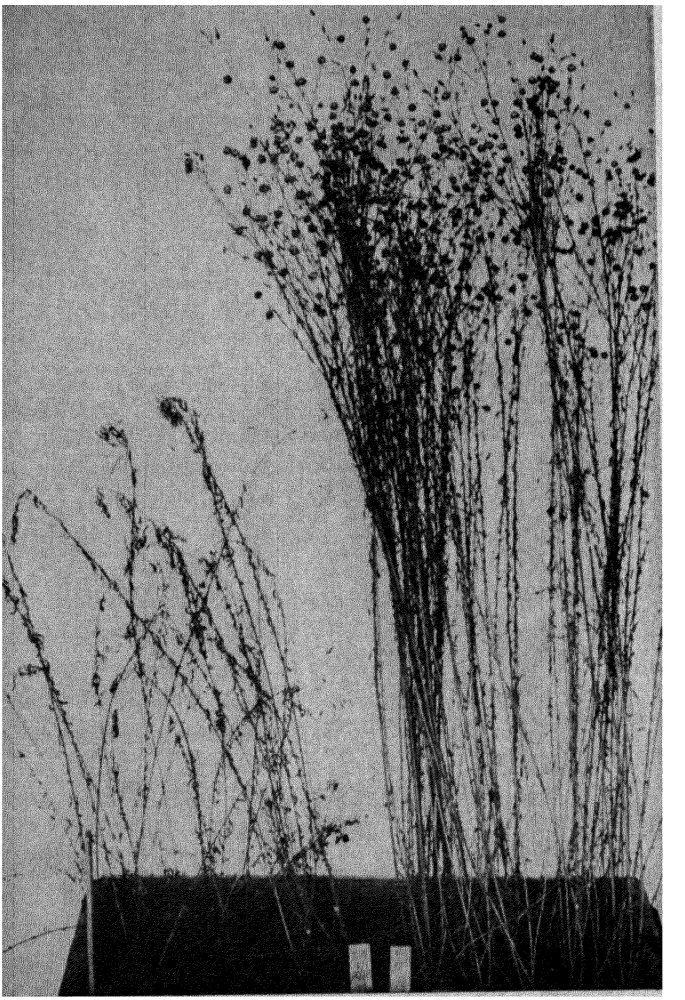

FIG. 94.—Flax wilt caused by *Fusarium oxysporum* f. *lini*. The disease is controlled by the use of wilt resistant varieties, right.

chiefly in the xylem vessels. High temperatures and low moisture are important factors in the development of the disease and the expression of resistance in most flax varieties (Tisdale, 1916, 1917). Fungus mycelium apparently must be present in the plant tissues to produce wilting (Schuster, 1944). Seed infection occurs and accounts for the spread of

the parasite to new areas (Bolley, 1924, 1926, Bolley and Manns, 1932).

Control.—Wilt-resistant varieties constitute the chief means of control (Bolley, 1901, 1932, Stakman *et al.*, 1919, Tisdale, 1917). Wilt resistance is conditioned by several factor pairs, and selected strains breed true for different degrees of resistance (Barker, 1923, Burnham, 1932). The nature of wilt resistance has been studied by Nelson and Dworak (1925), Reynolds (1931), Tisdale (1917), and others.

FIG. 95.—Flax rust caused by *Melampsora lini* showing the old aecia, the uredia, and the telia on the leaves and stems.

8. Rust, *Melampsora lini* (Pers.) Lév.—Flax rust occurs in the major flax-producing areas of the world. Specialized races of the rust parasite occur on both the cultivated and wild species of *Linum*. The rust causes damage to the fibers and reduces seed production.

Symptoms and Effect.—The flax rust parasite is an autoecious, long-cycle fungus producing pycnia (spermagonia), aecia, uredia, and telia on the flax plant. Pycnia and aecia usually occur during the early part of the growing season, and they appear as light-yellow to orange-yellow sori on the leaves and stems. The reddish-yellow uredia occur on the leaves, stems, and capsules during the growing season. The brown to black

telia, covered by the epidermis, occur chiefly on the stems, but also on the leaves and capsules late in the growing season (Fig. 95).

The Fungus.—*Melampsora lini* (Pers.) Lév.
(*Uredo miniata* f. *lini* Pers.)
(*Xyloma lini* Ehrenb.)
(*Melampsora liniperda* Palm)

The pycnia are subepidermal and are usually formed in the stomatal cavity. The round orange-yellow naked aecia occur on both surfaces of the leaf. Aeciospores are globoid, hyaline, and finely verrucose. The uredia are round to elongate, naked, reddish yellow changing to pale yellow as they mature, with paraphyses intermixed with the spores. Urediospores are elliptical to obovate, walls yellow, contents orange yellow, finely verrucose, and pores usually equatorial (Fig. 95). Telia may be round, but often are elongated and confluent, covered by the epidermis, slightly elevated, and brown to black. Teliospores are formed in a closely packed single layer, prismatic in shape, one-celled, smooth, brown, and germinate in place (Fig. 95).

Etiology.—The autoecious long-cycle rust produces all stages on the flax plant. The teliospores on the crop refuse germinate in the spring to produce the sporidia, which infect the young tissues of the flax plant. The pycnial stage develops, fusion of compatible haploid cells occurs to initiate the binucleate phase, and the aecial stage forms from the binucleate fusion hyphae. According to Allen (1934), the fungus is heterothalic. The primary uredial infection develops from the aeciospores throughout the early part of the growing season, and secondary infections from urediospores account for much of the later spread. Telia are formed around the uredia and from independent uredial infections as the flax plant matures. The telia persist in the flax straw to renew the cycle. The uredial stage continues development in regions where the flax plants are growing in both summer and winter; however, the telial material on the crop refuse is the common source of primary inoculum in most flax-producing areas.

Control.—Crop rotation and removal or plowing under of the flax refuse is important in controlling the epidemic development of the disease. The teliospores on small pieces of infected tissue frequently are carried with the seed; therefore, careful cleaning of flax seed is important, especially when seeding on new land.

Resistant varieties offer the best means of rust control, as reviewed by Flor (1941) and Vallega (1944). Ottawa 770B (C.I. 355), a seed flax, is resistant to the known physiologic races of North America and Europe and to most of the South American races. Bombay (C.I. 42), Punjab (C.I. 20), and Indian types 29 and 46 are resistant to all known races from Argentina (Flor, 1940, Straib, 1939, Vallega, 1944). J. W. S. (C.I. 388), a fiber flax, is resistant to the South American races except races 42 and 42A, but is susceptible to certain of the North American and European races. Saginaw Bombay (C.I. 671) is resistant to all the

known Argentina races (Flor, 1941, Vallega, 1944). Other varieties and hybrid selections are resistant to many of the 42 physiologic races differentiated. No flax variety is known to be immune from all races. Flor (1941) has shown that several factor pairs are involved in conditioning resistance to the various groups of physiologic races.

Physiologic specialization has been studied by Flor (1935, 1940), Straib (1939), Vallega (1933, 1944), Waterhouse and Watson (1941), and others. Some 42 physiologic races have been differentiated from North and South America, Europe, and Australia. The reaction of certain of the races on some of the differential varieties is influenced by environmental conditions.

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CHAPTER XVIII

TOBACCO DISEASES

The cultivated tobaccos comprise two species, *Nicotiana tabacum* L. and *N. rustica* L.; varieties of the former constitute the major commercial crop. A large number of additional species, however, are indigenous in the Western Hemisphere. The two cultivated species hybridize readily, as each contains 24 chromosome pairs apparently of similar origin. *N. tabacum* has been crossed with some 12 other species, with chromosome pairs ranging from 9 to 24. While in present experimental practice these wide crosses are not utilized extensively in breeding programs, there is, nevertheless, the probability of obtaining disease-resistance factors from a number of the wild species (East, 1928, Garner *et al.*, 1936).

The tobacco plant is adapted over a wide range of climatic and soil conditions of the world. The species are largely self-pollinated, and they are mostly annuals, varying greatly in type of growth and length of growing period.

Both the leaves and stems are used in commerce. The stems are used in the preparation of nicotine extracts. The quality of the leaf is influenced greatly by climatic conditions and soil composition, which tend to localize production of different types of commercial tobacco.

Diseases of the crop are important in both yield of leaf and quality of the product. Plant diseases frequently cause large losses in the crop, and disease investigations and control are important factors in the improvement of the crop. Wolf (1935) has discussed tobacco diseases in detail, and reference should be made to this volume for the more complete information.

1. Leaf Spotting and Yellowing, Nonparasitic.—The quality of the tobacco leaf is associated with soil fertility and climate as well as the tobacco variety. "Firing," "rusting," "spotting," and similar descriptive terms have been used to describe various soil nutrient deficiencies and other nonparasitic manifestations. Deficiencies in potash, phosphate, and magnesium influence the quality of the leaf and if acute are manifest by crinkling and yellowing of the leaf tip and margins, brown leaf blotches, and bleaching or chlorosis. The general use of commercial fertilizers in tobacco culture has reduced this type of malady in the more important tobacco areas. Other nonparasitic diseases such as frenching,

sunscald, lightning injury, and hail spots are not uncommon (Anderson, 1940, Johnson, 1924, Valleau *et al.*, 1942, Wolf, 1935).

2. Brown Root Rot, Cause Unknown.—The brown root rot of tobacco and other crops is present in most of the tobacco districts of North America. The malady was described first by Johnson (1915, 1939), and it has been studied extensively since, as reviewed by Berkeley (1944). The brown necrosis of the roots develops first as a brown discoloration

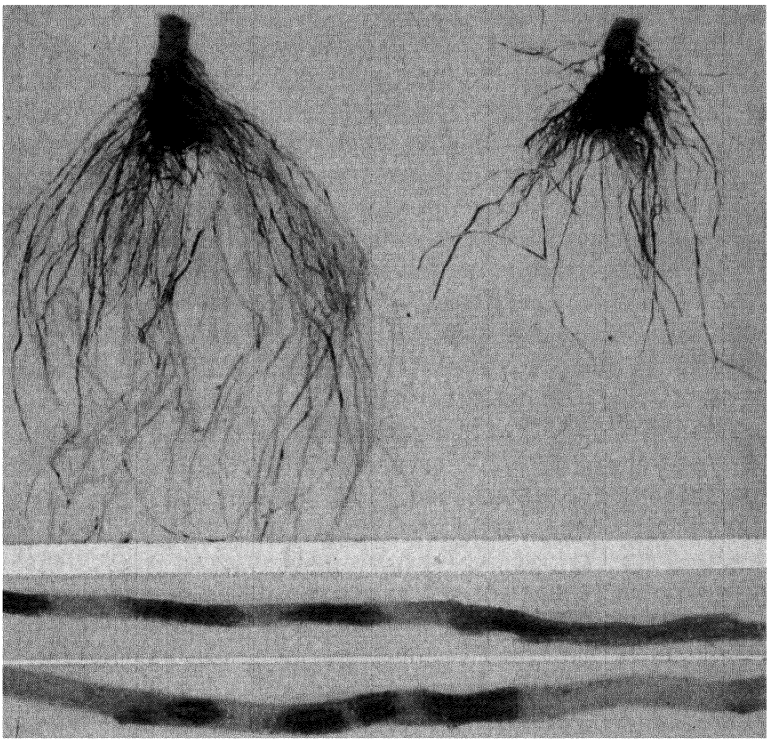

FIG. 96.—Tobacco roots showing the characteristic symptoms of the brown root rot, and enlarged pieces of root showing necrosis. (*Courtesy of J. Johnson.*)

of the cells of the cortical tissue, followed by cell necrosis and sloughing off of the cortex and the final collapse of the entire root system (Fig. 96). Numerous fungi are associated with the necrotic tissues as the rot develops, but none is capable of producing the disease under the conditions used in inoculation studies to date. The diseased plants are yellow and dwarfed, and they tend to wilt readily during bright days. The plants recover somewhat as the season advances.

The direct cause of the disease is not known, although it is associated with certain crops, especially corn, soybeans, orchard grass, and timothy,

grown on the land the year prior to tobacco. Treatment of soil with heat or formalin reduces the damage. The severity of the disease decreases with the continued production of tobacco. The affected plants frequently recover after a period of hot, dry weather, with little browning of the new roots that develop later.

Rotating tobacco with crops other than those tending to induce the disease or continuous tobacco culture reduces damage. Tobacco varieties, in most of the types, differ to some extent in their reaction to the malady (Koch and Haslam, 1938).

3. Mosaics, Infectious Viruses.—Tobacco mosaic has been associated closely with the historical investigations of virus diseases in plants. Mayer (1886) first described the mosaic on tobacco. Ivanowski (1892) demonstrated the transmissibility and the filterability of the virus principle using tobacco mosaic, and his work was confirmed and interpreted by Beijerinck (1898). The insect transmission of a tobacco mosaic disease was demonstrated by Allard (1914); however, Ball and Takami had demonstrated earlier the insect transmission of curly top of beet and the dwarf disease of rice, respectively (Chap. VII). The first chemical purification of a virus was accomplished (Stanley, 1935, 1936) using the tobacco mosaic. Numerous physical and chemical studies have been made on the tobacco viruses during the past two decades.

Many viruses and numerous strains are associated with the mosaic maladies of tobacco in various parts of the world. The ordinary tobacco mosaic is the more common and widely distributed of the group. In addition, the ring spot and streak viruses occur commonly on tobacco in North America (Berkeley and Koch, 1940, Valleau *et al.*, 1942). The tobacco plant is used extensively in the determination and characterization of the plant viruses. Where mosaic infection is heavy early in the season, losses in yield and quality occur (Johnson and Ogden, 1939, Valleau *et al.*, 1942). The viruses and strains occurring on tobacco have been listed and described by Holmes (1939), Johnson *et al.* (1939),[1] and Smith (1937).

Certain distinct diseases of the tobacco plant are caused by a specific virus or by two or more viruses in combination. This is true also in other crop plants, such as the potato and garden pea. Many viruses occur naturally on tobacco or can be transmitted to tobacco, where they induce reproducible symptoms. Each of the viruses is distinguished, in part, on the basis of symptoms induced upon the specific plants or varieties. Other differentiating or diagnostic characteristics include specific differences in the properties of the viruses, such as (1) longevity of infective principle in vitro, in specific plants, or in specific vectors; (2) tolerance to

[1] Manuscript of report of committee on description and nomenclature of plant viruses of the International Botanical Congress 1939.

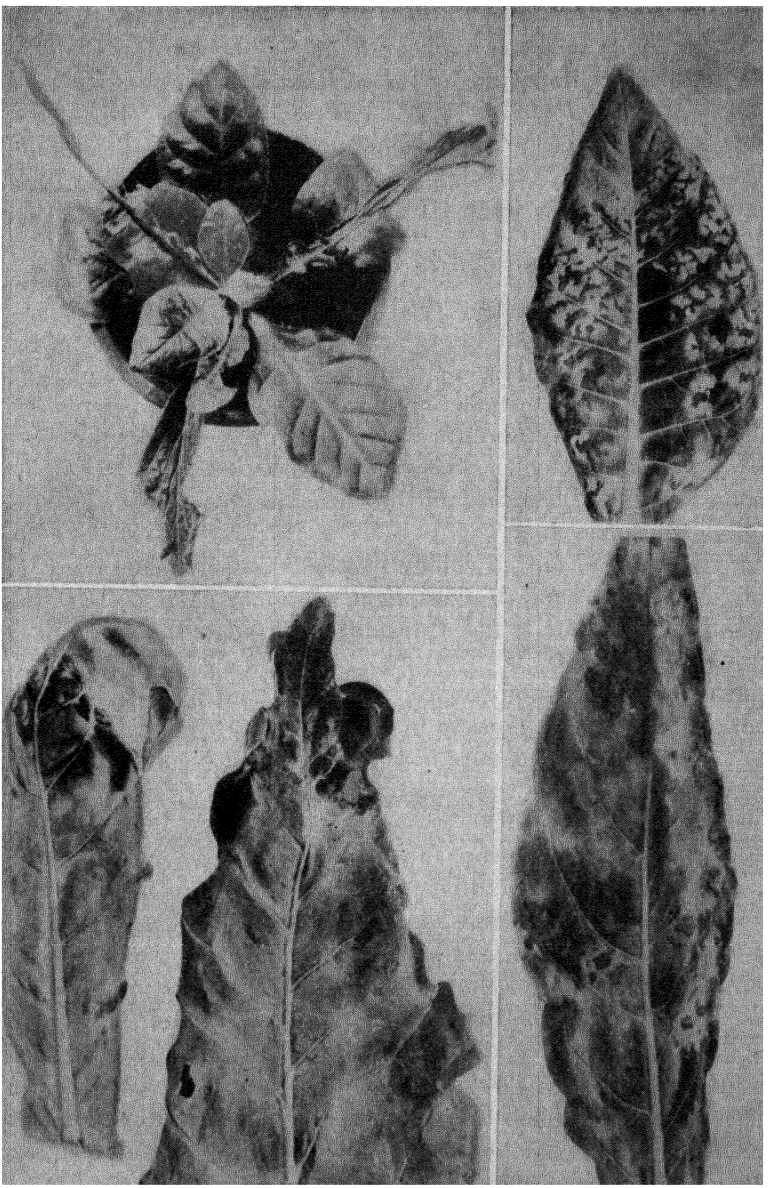

FIG. 97.—Plant and leaves showing the characteristic symptoms of the common tobacco mosaic. (*Courtesy of J. Johnson.*)

chemicals; (3) thermal inactivation; (4) filterability; (5) effect of dilution on infectivity; (6) agencies of transmission; and (7) serological and immunological relationships. No uniform system or standard of classifica-

tion of the viruses has been adopted as yet, and future progress is dependent largely upon a better understanding of the nature of the viruses. A few of the more important of the tobacco viruses are discussed briefly.

Common Tobacco Mosaic, Virus and Strains.—The ordinary tobacco mosaic is world wide in distribution, and it is the most common virus disease on the crop. The symptoms induced vary somewhat, depending upon age of the plant when infection occurs, the variety, and environmental conditions.

Symptoms.—In most varieties of tobacco the primary lesions are yellowish green, followed by clearing of the veins and greenish-yellow mottling. Dwarfing and distortion of the newly formed leaves with irregular crumpled swellings darker green in color frequently accompany the mottling (Fig. 97). Infections early in the development of the plant result in dwarfing of the entire plant, dwarfing and distortion of the flowers, mosaic-affected sucker growth, and frequently tissue necrosis on some varieties. Strains of the virus produce somewhat different symptoms on tobacco.

The virus is transmitted chiefly by slight abrasive contacts. Several aphids, *Myzus* spp. and *Macrosiphum solanifolii* (Ashm.), also transmit the virus. The virus is not transmitted through the seeds of mosaic tobacco.

Control.—The common tobacco mosaic is controlled by rotation and sanitation. The virus persists in undecomposed infected tobacco residues. Dried unsterilized mosaic leaves apparently are a source of reinfecting the plants by workers, either in the seed bed or in the field (Johnson and Ogden, 1939, Valleau and Johnson, 1937). Resistant varieties offer a possible means of control. In Ambalema, T. I. 488, and other collections from Columbia, South America, resistance to common tobacco mosaic apparently is dependent upon two recessive pairs of genes and probably modifying genes (Clayton *et al.*, 1938). Resistance as expressed by failure to show chlorosis after inoculation with tobacco mosaic was demonstrated in *Nicotiana glutinosa* L. and the first generation hybrid between this species and *N. tabacum* L. by Allard (1914). Holmes (1938), using an amphidiploid species (*N. digluta* Clausen and Goodspeed), found that the *N. glutinosa* response to infection with tobacco mosaic virus was transferable to *N. tabacum* through the medium of the amphidiploid species and that the reaction was inherited as a single dominant factor pair. Clayton and McKinney (1941) have shown that the genotypes with the glutinosa type of mosaic resistance developed systemic necrosis or that they were highly susceptible under field conditions; however, other investigators have reported satisfactory results with this type of resistance.

Tobacco Ring Spot, Virus and Strains.—The disease apparently is

distributed widely on tobacco and other crops and weeds, including soybean, sweetclover, and cowpea.

Symptoms.—Ring spot is essentially a foliage disease of tobacco, although on some plants, as the soybean, stems and buds are affected. The first symptom on tobacco leaves appears as small translucent rings of necrotic tissue. The necrotic tissue soon becomes bleached and dry as new rings form outside the primary ring and become separated from it by a zone of green tissue (Fig. 98) The ring pattern is circular to irregular, depending apparently upon the nearness to virus. Symptoms vary from the ring patterns to bleaching and yellowing of the leaves. Young leaves show the sequence of symptoms as they unfold, indicating a systemic infection. Stem necrosis occurs on some *Nicotiana* spp., although it is uncommon on commercial tobacco.

The virus and strains are transmitted by mechanical means and probably insect vectors. Perennial weeds and sweetclover apparently are concerned with the overwintering of the virus. Control of weeds near seed beds and rotation of crops are possible means of control.

Other Virus Diseases.—Tobacco streak and etch occur naturally in local or general areas of the United States and Canada. Streak is manifest by local or systemic irregular spots, lines, and ring-like necrotic lesions, usually followed by recovery. Systemic chlorotic mottling and necrotic etching are characteristic symptoms of the etch virus and strains (Fig. 98).

Permanent or transient symptoms on tobacco are produced by a large number of viruses. As stated earlier, tobacco is used as an assay suscept for many of the plant viruses.

4. Bacterial or Granville Wilt, *Pseudomonas solanacearum* E. F. Sm. — The bacterial wilt of tobacco and other Solanaceous plants is a common disease on tobacco in the Southern United States and the warmer areas of other countries. Losses from the disease are high in local sections in the Southeastern United States.

Symptoms.—The symptoms usually appear several weeks after transplanting. The leaves wilt, turn yellow, and finally brown as the plants die. The stems show yellow discolored areas in the xylem tissues and white bacterial masses in the vascular bundles of stems and leaf veins.

The Bacterium.—*Pseudomonas solanacearum* E. F. Sm.
[*Phytomonas solanaceara* (E. F. Sm.) Bergey *et al.*]
(*Bacillus solanacearum* E. F. Sm.)
[*Bacterium solanacearum* (E. F. Sm.) E. F. Sm.]

The rods are motile by means of one polar flagellum, have no spores, no capsules develop, and the colonies are opalescent white to brown.

Etiology and Control.—The bacteria persist in the soil in association with crop residue. The disease occurs on other Solanaceous crops, some

legumes, and several weeds. Infection occurs through the roots, especially where injured in transplanting, by nematodes, or by insects. Crop rotation, use of cover crops, balanced fertility, and seed-bed sterilization aid in reducing the disease (Garriss and Ellis, 1941).

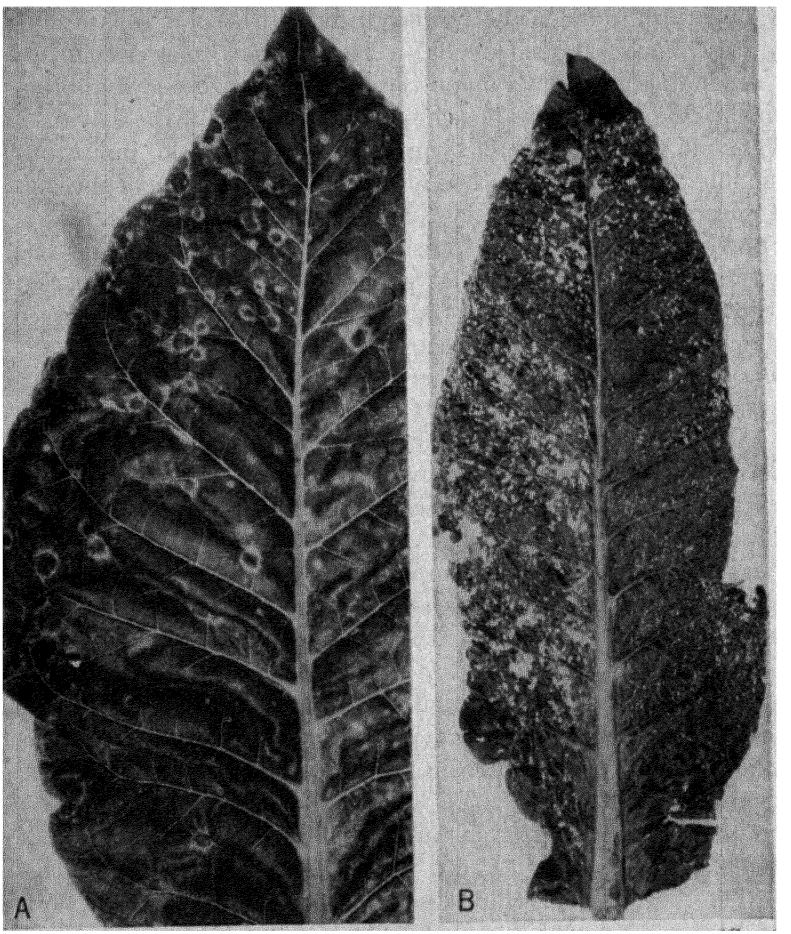

FIG. 98.—Tobacco leaves showing (*A*) ring spot and (*B*) streak symptoms. (*Courtesy of J. Johnson.*)

5. Bacterial Leaf Spot or Wildfire, ***Pseudomonas tabaci*** (Wolf and Foster) Stapp.—Wildfire occurs in the tobacco districts of North America. The disease is found on other plants, notably the soybean. The occurrence of the disease is sporadic, and damage is associated with weather conditions; although, losses on tobacco are probably greater than from any of the other bacterial leaf diseases.

Symptoms.—Spots on the leaves are circular to angular as they coalesce. The spot consists of a small light tan to brown dead infection center surrounded by a halo of chlorotic tissue similar to that of halo blight of oats (Chap. VI). No exudate is formed on the lesions. Infection of very young tissues results in a more general chlorosis of the infected area (Fig. 99). The disease generally appears on the lower leaves first and spreads rapidly during heavy rains or long periods of high moisture when the tissues are water-soaked or the stomatal cavities are filled with moisture, as reviewed by Valleau *et al.* (1943).

The Bacterium.—*Pseudomonas tabaci* (Wolf and Foster) Stapp
[*Phytomonas tabacae* (Wolf and Foster) Bergey *et al.*]
(*Bacterium tabacum* Wolf and Foster)

The rods are motile by means of polar flagella, have no spores, no capsules are formed, and the colonies are white.

Etiology and Control.—The bacteria apparently persist in crop refuse to furnish inoculum for the new crop. Conditions favorable for water-soaking of the leaf tissue or for filling the stomatal cavities with water are associated with rapid spread and development of the disease. Open stomata are important in the entrance of the inoculum in water into the stomatal cavity (Valleau *et al.*, 1943). Seed-bed sanitation is essential to prevent the damage and spread of the disease on the seedlings. Applications of Bordeaux mixture to the seed bed help control the disease. Rotation of crops and elimination of the infected crop residues are important in the field control of the disease. Some of the commercial varieties are moderately resistant to the disease.

6. Angular Leaf Spot or Black Fire, *Pseudomonas angulata* (Fromme and Murray) Stapp.—The disease occurs in all tobacco areas of the United States, where under favorable environmental conditions late in the season the quality of leaf tissue is damaged. The small spots resulting from the late infections are conspicuous in the cured tobacco.

The leaf spots vary considerably in size and shape. In the seed bed the spots are small, angular, and black or dark brown in color. In the field the spots are small and angular at first but frequently develop to larger lesions (Fig. 99). The larger spots are zonate, tan to dark brown, and small amounts of exudate occur on the surface (Fromme and Wingard, 1922). The disease develops rapidly under high moistures and temperatures (Valleau *et al.*, 1943).

The Bacterium.—*Pseudomonas angulata* (Fromme and Murray) Stapp
[*Phytomonas angulata* (Fromme and Murray) Bergey *et al.*]
(*Bacterium angulatum* Fromme and Murray)

The rods are motile by means of polar flagella, have no spores, no capsules are formed, and the colonies are dull white.

The etiology and control are similar to that for wildfire.

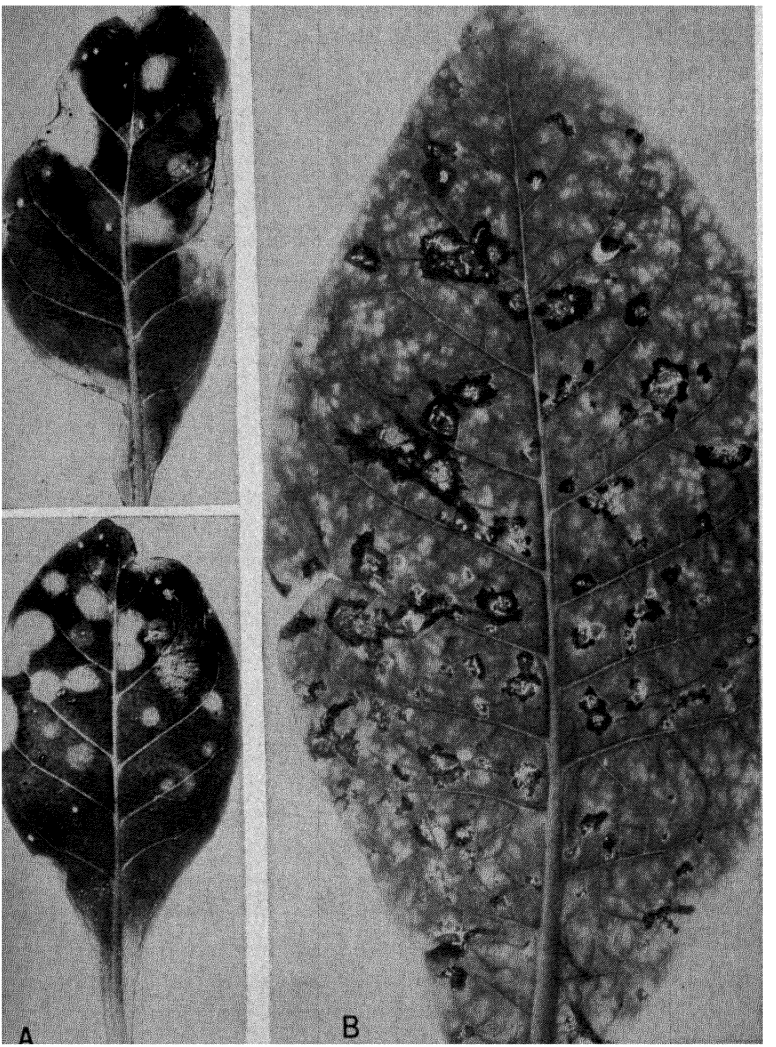

FIG. 99.—(*A*) Leaves of tobacco seedlings showing the lesions and fire produced by *Pseudomonas tabaci*. (*B*) A mature leaf showing yellow blotches of potash deficiency and the angular, black lesions with light centers characteristic of black fire or angular leaf spot caused by *P. angulata*. (*Courtesy of J. Johnson.*)

7. Pythium Damping-off and Stem Rot, *Pythium* Spp.—The disease occurs in the seed bed and soon after transplanting into the field. Dam-

age from the disease is general, but especially severe in the tropics. The disease has been studied extensively in India and Sumatra, as summarized by Middleton (1943).

Symptoms.—The damping-off occurs in the early to late seedling stage as a seed-bed rot. The characteristic brown soft rot and white surface mycelium occurring in local areas in the bed are indicative of the disease. In the field the dark-brown lesions develop at the base of the stem and frequently extend into the leaves (Anderson, 1940).

The Fungi.—Several species of *Pythium* cause the malady. The most common species is *Pythium debaryanum* Hesse. (The morphology is given in Chap. XIII). A second species occurs on tobacco in the Eastern United States (Anderson, 1940) and generally through Asia, Africa, and the Pacific area.

Pythium aphanidermatum (Edson) Fitzp.
(*Rheosporangium aphanidermatus* Edson)
(*Pythium butleri* Subr.)

Sporangia are lobulate, branched, inflated, and freely produced. Oögonia are spherical, usually terminal, and 22 to 27 microns in diameter. Antheridia are usually monoclinous, typically intercalary, and occur one or two per oögonium. Oöspores are aplerotic, single, smooth, moderately thick-walled, and 17 to 19 microns in diameter. *Pythium deliense* Meurs occurs on tobacco in Sumatra.

Etiology and Control.—These fungi are common on certain types of decomposing organic material in the soil. The invasion and rotting of the plant tissues occurs under conditions of high moisture and low light intensity. Sterilization of seed beds, drainage, ventilation, and the use of fungicidal sprays are all important as control measures.

8. Black Shank, *Phytophthora parasitica* var. *nicotianae* (Breda de Haan) Tucker.—The disease occurs in the Southern United States where it has spread from the Florida-Georgia tobacco sections. The black shank occurs generally in the tropical sections of South America and the Pacific tobacco areas. The disease causes considerable damage in certain types of tobacco, but resistant varieties are coming into general use (Gratz and Kincaid, 1938, Tisdale, 1931).

Symptoms.—Symptoms are evident by the blackened stalk and the subsequent rapid wilting of the tops. The disease occurs infrequently in the seed bed as a damping-off of the seedlings and the blackened rotted basal portion of the stems. In the field the black rot of the stem starts near the soil line and extends up the stem and down into the roots. Necrosis of the stem tissues is rapid, and a spongy rot develops. The plants wilt rapidly and collapse. Large brown leaf lesions occur in wet weather. The disease spreads from local areas as the season advances (Tisdale, 1931).

The Fungus.—*Phytophthora parasitica* var. *nicotianae* (Breda de Haan) Tucker
(*Phytophthora nicotianae* Breda de Haan)
(*Phytophthora tabaci* Saw.)

Tucker (1931) placed the fungus as a variety of *Phytophthora parasitica* Dast., as it was similar in morphology and varied chiefly in being parasitic on tobacco stems. Mycelial growth is abundant on media at high temperatures. Sporangia are broadly ovate, papillate, and average 25 by 30 microns in size. Vegetative resting spores (chlamydospores) are abundant in culture. Oöspores are globose, about 15 to 20 microns in diameter although the size is variable, the membrane is thick and smooth.

Etiology.—The fungus persists in the soil in association with crop refuse and reinfects the plants near the soil line. The fungus is spread through infected tobacco refuse, surface water, implements, and other agencies.

Relatively long rotations and sanitation help prevent the rapid spread of the disease. Resistant varieties are used in the areas where the disease is severe. Tisdale's (1931) results indicated that several factors are probably involved in resistance to the disease.

9. Downy Mildew or Blue Mold, *Peronospora tabacina* Adam.—The downy mildew was first reported from Australia, where it occurs on the cultivated and some wild species. The first outbreak of the disease in the United States occurred in Florida and Georgia in 1921. Since then downy mildew or blue mold has spread through the central, eastern, and southern tobacco areas (Anderson, 1937, 1940, Koch, 1941). Similar diseases earlier were reported occasionally on wild species of *Nicotiana* in North and South America. More recently, the disease has become prevalent on cultivated tobacco in Argentina and Brazil (Godoy and Caste, 1940, Wolf, 1939). The disease causes loss of plants in the seed bed and delayed planting. The damage from the disease is sporadic, as the development is influenced greatly by temperature and moisture.

Symptoms.—The appearance of the infected plants is variable, depending upon weather conditions, age of the plants, and stage of disease development. The disease in the seed bed first appears as pale-green to yellow indefinite lesions on the upper leaf surface and a gray to brown downy mass of conidiophores on the undersurface of the leaf. In the young plants, and especially the leaf, tissues brown and collapse, giving the bed a scalded appearance (Fig. 100). The lesions with conidiophores and conidia are more characteristic on the older plants in the beds and in the field. The older plants generally recover and produce new leaves.

The Fungi.—*Peronospora tabacina* Adam
Peronospora hyoscyami DBy.
Peronospora nicotianae Speg.

Fig. 100.—Downy mildew or blue mold of tobacco caused by *Peronospora tabacina*. (*Courtesy of J. Johnson.*)

According to Adams (1933), Angell and Hill (1932), Clayton and Stevenson (1935), and Wolf (1939), three species occur on different suscepts: *Peronospora tabacina* Adam on tobacco and certain wild species of the genus, *P. nicotianae* Speg. on some wild tobacco species in South America and possibly North America, and *P. hyoscyami* DBy. on the black nightshade (*Hyoscyamus niger* L.); the latter binomial formerly was used for the fungus on tobacco. The morphology of *P. tabacina* only is given.

One or more conidiophores emerge from the stomata, usually on the lower surface of the leaf. The conidiophores are five to eight times dichotomously branched, the curvature of the branches increasing to the ultimate branches which diverge obtusely, slightly curved, or recurved and end bluntly. The conidia are ovoid to ellipsoid, hyaline to dilute violet, measure 13–19 by 16–29 microns, and germinate to produce germ tubes from the side of the conidium. Oögonia and oöspores are produced in the necrotic tissues. The oögonia are terminal, with antheridia forming as a branch from the oögonial stalk. The oöspores are globose, dark brown, epispore smooth or slightly roughened, and 35 to 60 microns in diameter. The intercellular mycelium and branched haustoria are described by Henderson (1937).

Etiology.—Both conidia and oöspores function as primary inoculum under certain conditions. The conidia produced on tobacco plants, second growth, or perennial wild species in the areas of mild winter reinfect the seed beds. The oöspores are the more important source of primary infection for new seed beds in most of the tobacco sections. While the conidia are relatively short-lived, secondary spread and wide distribution of the parasite occurs from this source of inoculum (Anderson, 1937, Hill and Angell, 1933). Temperature, sunlight, and moisture are limiting factors in the development of disease (Dixon *et al.*, 1936).

Control.—Location of seed beds away from oöspore inoculum and use of fungicides offer the best means of control. Steam sterilization as generally practiced, good drainage, and exposure to sunlight are advisable. The use of copper sprays and gasses such as benzol and paradichlorbenzene give control in the seed bed (Clayton, 1938). Clayton (1945) reported the commercial varieties susceptible in the early seedling stage and varying somewhat in susceptibility in the mature plant stage. He also reported certain of the wild species of tobacco as resistant even in the seedling stage. Angell and Hill (1932) listed all the Australian species as susceptible. Fertile hybrids between the resistant wild species and tobacco are uncommon, and they are difficult to use.

10. Brown Leaf Spot, *Alternaria tenuis* Nees.—During periods of high temperature and moisture, large circular brown spots develop on the mature leaves and on leaves after harvest. Spots on the leaves from contact infection through dead corolla tissue also occur in shade tobacco. The fungus sporulates on the surface of the lesions.

11. Fusarium Wilt, *Fusarium oxysporum* var. *nicotianae* (J. Johnson) Snyder and Hansen.—The wilt occurs rather generally in the sandy soils of the warmer tobacco sections (Fig. 101). This variety of the fungus as well as certain of those parasitizing other crops, as cotton and tomato,

apparently are capable of producing wilt in tobacco. Root knot and potassium deficiency apparently increase the damage from wilt. Wilt-

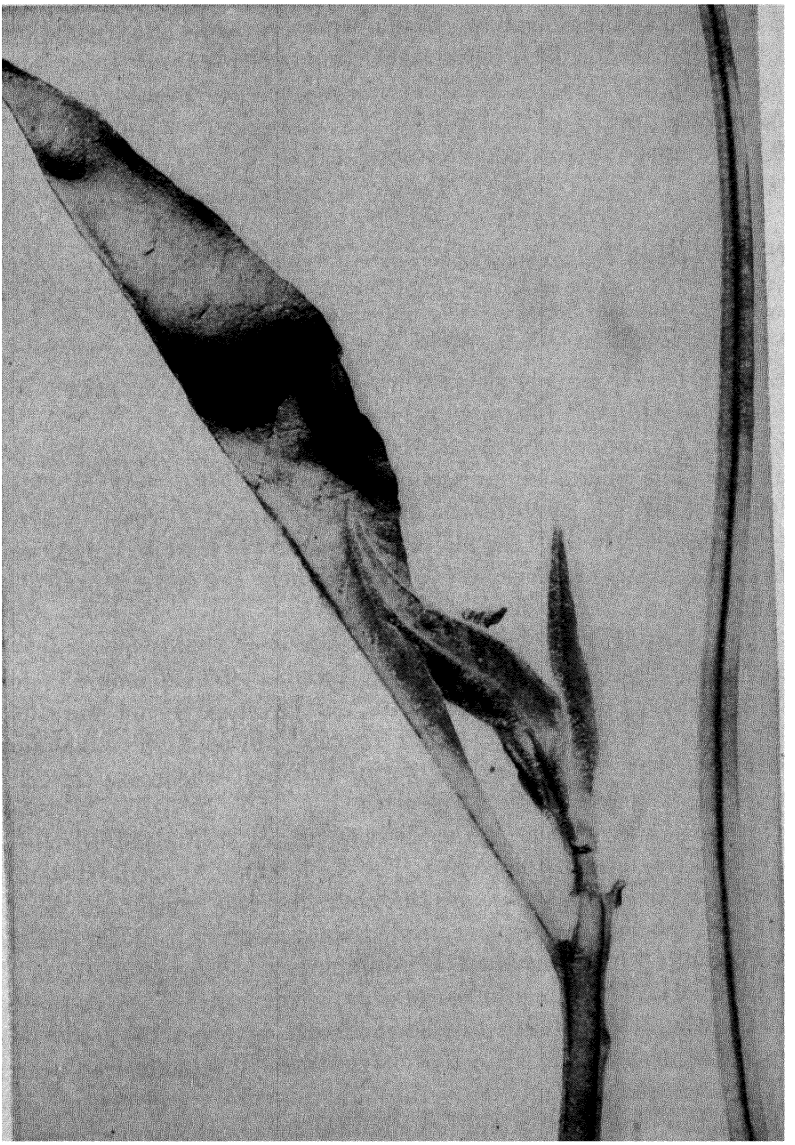

FIG. 101.—Fusarium wilt of tobacco showing the narrow, thickened leaves and brown discolored stem interior. (*Courtesy of J. Johnson.*)

resistant varieties offer the best means of control. The morphology of the fungus is given in Chap. XVI.

12. Frogeye Leaf Spot, *Cercospora nicotianae* Ell. and Ev.—This leaf spot is distributed widely throughout the world, and it is of economic importance chiefly in the more tropical, humid areas. The symptoms are zonate white and yellow circular spots with the gray conidiophores present later on the dead bleached portion of the lesion. Infections, just prior to harvest, produce small green spots on the cured leaves. Smaller zonate spots appear on the bracts, calyx, and capsules (Hill, 1936).

The Fungus.—*Cercospora nicotianae* Ell. and Ev.
(*Cercospora raciborskii* Sacc. and Syd.)
(*Cercospora solanicola* Atk.)

The dark-brown septate conidiophores arise in groups from masses of mycelium in the tissue. The conidia are borne from the terminal cell, and they are displaced by elongation of the conidiophore. The conidia taper gradually toward the apex, are hyaline, have 0 to 16 septations, and are 90 to 300 microns long.

Etiology.—The fungus persists in crop refuse, and it is carried over on the seed. Seed-bed infections occur from tobacco trash or conidia on the seed. Secondary spread from conidia is general during hot, humid weather. The conidia and mycelium in dry crop refuse remain viable for one or more years. Seed treatment and sanitation apparently control the disease in areas where living plants do not survive from season to season. Fungicidal sprays are necessary in areas where the conidial inoculum is abundant.

13. Black Root Rot, *Thielaviopsis basicola* (Berk. and Br.) Ferr.—This fungus produces a root rot on a large number of field crops, including cotton, cowpeas, flax, lupines, peanuts, red clover, soybean, and tobacco, and it is world wide in distribution especially in cold, wet alkaline soils (Johnson, 1916). Prior to the use of resistant varieties, the disease caused large losses in yield of tobacco.

Symptoms and Effects.—The disease proper is limited to the root system and the base of the stem. The depletion of the root system and the black rotted root stubs are the characteristic symptom. The depleted root system results in retarded uneven growth of the plants, a yellow or chlorotic appearance, and temporary wilting (Fig. 102). The rapid recovery of the plants as soon as warm weather prevails is also characteristic of the disease. The fungus invades the root cortex by entrance around the branch root ruptures, mechanical injuries, or direct penetration and advances through the cortex and central steele, producing the blackened condition simultaneous with the necrosis of the tissues.

The Fungus.—*Thielaviopsis basicola* (Berk. and Br.) Ferr.
[*Thielavia basicola* (Berk. and Br.) Zopf.]
(*Thielavia basicola* Zopf.)

Two types of spores are produced under different environments and ages of the mycelium. The endoconidia are borne on the young mycelium, especially in culture. The endoconidiophores are phialides comprising a bulbous base and an elongated tube 50 to 90 microns long, gradually tapering to 3 to 7 microns in diameter at the end. They arise as a branch from near the center of a hyphal cell. The endoconidia are formed in chains within the conidiophore and are extruded singly or in chains. The conidia vary

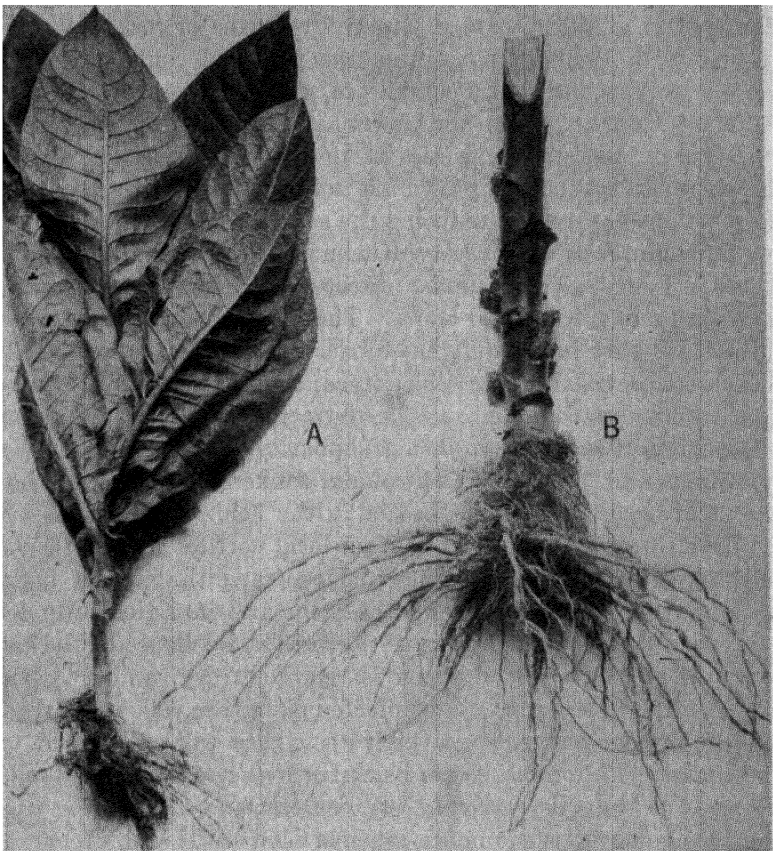

FIG. 102.—Black root rot of tobacco caused by *Thielaviopsis basicola*, showing the rotted roots and small plant in a susceptible variety (*A*) compared with the root system of a resistant plant (*B*). (*Courtesy of J. Johnson.*)

in size, measuring 8–30 by 3–6 microns, are cylindrical with rounded ends, hyaline, and germinate to produce mycelium (Brierley, 1915). The thick-walled resting spores originate in chains or clusters laterally or terminally from any part of the mycelium; they are hyaline at first, but soon become thick-walled and brown, short cylindric with angular ends, about 12 by 5–8 microns in size, and separate at maturity. The ascigerous stage (Thielavia) was formerly associated with this species, but according to McCormick (1925) the relationship is questionable. Rawlings (1940) has shown specialization of the parasite.

Etiology.—The fungus infestation persists in alkaline soils for an indefinite period and invades the roots of plants during cool, wet weather. The parasitic activity of the fungus is greatly restricted as the soil temperature rises, probably associated with both the activity of the fungus and the resistance of the plant suscepts (Conant, 1927, Doran, 1929, Jewett, 1938, Johnson and Hartman, 1919).

The control of the disease is largely by means of resistant varieties. Johnson (1916, 1930) demonstrated the practical control by resistant tobacco selections and that a complex of several factors were involved in resistance. Conant (1927) and Jewett (1938) have studied the nature of resistance. Resistant varieties of the various types of tobacco are used wherever tobacco is grown on soils infested with the parasite. Crop rotations are used in reducing the severity of the infestation.

14. Soreshin, *Rhizoctonia solani* Kuehn and *Sclerotium bataticola* Taub., and Other Fungi.—These similar diseases are distributed generally on tobacco and many other field crops. The fungi causing the stem cankering (soreshin) vary in the different tobacco-producing sections. Rhizoctonia is more prevalent in the cooler areas; *Sclerotium bataticola* and other species occur more commonly in the warmer areas. These fungi attack the basal stem tissues and produce a similar type of disease.

Symptoms and Effects.—Dark-colored cankers or rotting at the base of the stem occur in the seed bed or in the field. The lesions usually occur near the soil line and vary from local cortical lesions to cankers developing into the woody tissues and central pith. Under favorable conditions, the lesions extend up the stem and into the lower leaves. The invaded tissues show extensive necrosis and collapse of the tissues. The brown to black rotted lesion is relatively dry, and sclerotia are common on the rotted area. The infected plants are yellow and dwarfed, frequently break over, and in severe rotting they wilt and brown.

The Fungi.—*Rhizoctonia solani* Kuehn or *Pellicularia filamentosa* (Pat.) Rogers (*Corticium vagum* Berk. and Curt.)

The morphology of the fungus is given in Chaps. VII and XI.

Sclerotium bataticola Taub.

The morphology of the fungus and its several stages are discussed in Chap. IX.

Sclerotium rolfsii Sacc. also occurs on tobacco in the Southern United States.

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APPENDIX A

DISEASES OF FIELD CROPS ARRANGED BY CAUSAL FACTOR

(Suggested for a three-hour course)

1. Nonparasitic Diseases
   - Gray speck disease of cereals—manganese deficiency
   - Alfalfa yellows—boron deficiency
   - Leaf spotting "rust" of cotton—potash deficiency
   - Frost injury and winterkilling in wheat
   - Cold and winter injury in alfalfa
   - Heat canker in flax
   - Brown root rot of tobacco
2. Virus Diseases
   - Vectors largely aphids
     - Common tobacco mosaic
     - Tobacco ring spot
     - Alfalfa and clover mosaics
     - Corn and sugarcane mosaics
   - Vectors largely leaf hoppers
     - Alfalfa dwarf
     - Corn and sugarcane streak diseases
     - Chlorotic streak of sugarcane
     - Dwarf disease of rice
   - Vectors unknown but symptoms similar to streaks
     - Wheat mosaics
3. Bacterial Diseases
   - Bacterial blights
     - Bacterial blight of barley, wheat, rye, grasses—*Xanthomonas translucens*
     - Bacterial streak of sorghums—*Xanthomonas holcicola*
     - Bacterial stripe of sorghums—*Pseudomonas andropogoni*
     - Angular leaf spot of tobacco—*Pseudomonas angulata*
     - Angular leaf spot of cotton—*Xanthomonas malvacearum*
     - Bacterial pustule of soybean—*Xanthomonas phaseoli* var. *sojense*
   - Halo blights
     - Halo blight of oats and grasses—*Pseudomonas coronafaciens*
     - Wild fire of tobacco and soybean—*Pseudomonas tabaci*
   - Bacterial wilts
     - Alfalfa wilt—*Corynebacterium insidiosum*
     - Stewart's wilt of corn—*Bacterium stewartii*
     - Granville wilt of tobacco—*Pseudomonas solanacearum*

4. Chytridiales
   - Crown wart of alfalfa—*Urophlyctis alfalfae*
   - Physoderma disease of corn—*Physoderma zea-maydis*
5. Peronosporales
   - Pythium diseases
     - Root rot of corn and sugarcane—*Pythium arrhenomanes*
     - Root rot of cereals and grasses—*Pythium graminicolum*, etc.
     - Root rot and damping-off of legumes, etc.—*Pythium debaryanum*
   - Downy mildews
     - Downy mildew of alfalfa—*Peronospora trifoliorum*
     - Blue mold or downy mildew of tobacco—*Peronospora tabacina*
     - Downy mildew of the cereals and grasses—*Sclerospora macrospora*
     - Downy mildew of millets—*Sclerospora graminicola*
6. Perisporiales (Powdery Mildews)
   - Powdery mildew of cereals and grasses—*Erysiphe graminis*
   - Powdery mildew of legumes—*Erysiphe polygoni*
7. Hypocreales
   - Ergot of cereals and grasses—*Claviceps purpurea* and *C. paspali*
   - Gibberella head blight, root rot, and seedling blight of cereals—*Gibberella zeae*, *Fusarium culmorum*, etc.
   - Corn ear and stalk rot and seedling blight—*Gibberella zeae* and *G. fujikuroi*, etc.
   - Sooty blotch of clovers—*Cymadothea trifolii*
8. Sphaeriales
   - Pasmo disease of flax—*Mycosphaerella linorum*
   - Spring black stem of legumes—*Mycosphaerella lethalis*, *Ascochyta imperfecta*, *Phoma trifolii*
   - Summer black stem and leaf spot of legumes—*Mycosphaerella davisii* and *Cercospora zebrina*
   - Culm rot of rice—*Leptosphaeria salvinii*
   - Take-all of wheat and grasses—*Ophiobolus graminis*
   - Corn leaf blights—*Cochliobolus heterostropus*, *Helminthosporium turcicum*, and *H. carbonum*
   - Spot blotch of barley and wheat—*Helminthosporium sativum* (See also *Cochliobolus miyabeanus* (*H. oryzae*)
   - Net blotch of barley—*Pyrenophora teres* (See also *P. bromi* and *P. avenae*)
   - Stripe disease of barley—*Helminthosporium gramineum*
9. Pezizales
   - Sclerotinia root rot and crown rot on red clover—*Sclerotinia trifoliorum*
   - Pseudopeziza leaf spot of alfalfa—*Pseudopeziza medicaginis*, *P. trifolii*, *P. meliloti*
   - Yellow leaf spot of alfalfa—*Pseudopeziza jonesii* or *Pyrenopeziza medicaginis*
10. Sphaeropsidales
    - Diplodia ear rot and stalk rot of corn—*Diplodia zeae*, *D. macrospora* (See also *Physalospora zeae* and *P. zeicola*)
    - Septoria leaf blotches of cereals and grasses—*Septoria* spp.
11. Melanconiales (Anthracnoses)
    - Northern anthracnose of clovers—*Kabatiella caulivora*
    - Southern anthracnose of clovers—*Colletotrichum trifolii*
    - Anthracnose of cereals and grasses—*Colletotrichum graminicolum*
    - Anthracnose of cotton—*Glomerella gossypii*
    - Red rot of sugarcane—*Physalospora tucumanensis*

12. Moniliales
    - Phymatotrichum root rot—*Phymatotrichum omnivorum*
    - Scald of cereals and grasses—*Rhynchosporium secalis* and *R. orthosporium*
    - Cerospora leaf spot of rice—*Cercospora oryzae*
    - Fusarium wilt of crops—*Fusarium oxysporum*
      (See also Verticillium wilt of cotton—*Verticillium albo-atrum*
13. Autobasidiomycetes
    - Rhizoctonia root rot and stem blight of crops—*Pellicularia filamentosa*
    - Typhula snow mold or scald of cereals and grasses—*Typhula itoana, T. idahoensis*, etc.
14. Ustilaginales (Smuts)
    Ustilago spp.
    - Covered smuts of cereals and grasses—*Ustilago hordei, U. kolleri, U. bullata*, etc.
    - Intermediate or black loose smuts of cereals and grasses—*Ustilago avenae, U. nigra*
    - Loose smuts of cereals and grasses—*Ustilago nuda, U. tritici*, etc.
    - Stalk smuts of grasses—*Ustilago spegazzinii*, etc.
    - Stripe leaf smut of grasses—*Ustilago striiformis*
    - Corn smut—*Ustilago maydis*

    Sphacelotheca spp.
    - Kernel smuts of sorghum—*Sphacelotheca cruenta, S. sorghi*

    Tilletia spp.
    - Kernel smuts (bunt) of wheat and grasses—*Tilletia caries, T. foetida*, etc.

    Neovossia spp.
    - Kernel smut of rice—*Neovossia horrida*

    Entyloma spp.
    - Leaf spot smut of rice and grasses—*Entyloma lineatum, E. irregulare*, etc.

    Urocystis spp.
    - Flag smut of wheat and grasses—*Urocystis tritici, U. agropyri*, etc.
15. Uredinales (Rusts)
    Melampsora spp.
    - Flax rust—*Melampsora lini*

    Uromyces spp.
    - Rust of legumes—*Uromyces striatus, U. trifolii*

    Puccinia spp.
    - Stem rust of cereals and grasses—*Puccinia graminis*
    - Stripe rust of cereals and grasses—*Puccinia glumarum*
    - Leaf rust of cereals and grasses—*Puccinia coronata, P. rubigo-vera, P poae-sudeticae*, etc.
    - Cotton rust—*Puccinia stakmanii*

APPENDIX B

BACTERIA AND FUNGI PARASITIC ON FIELD CROPS

SCHIZOMYCETES	Suscepts	Page
Eubacteriales		
Erwinia flavida (G. Fawc.) Magrou	Sugarcane	190
Bacterium agropyri (O'Gara) Stapp	Grasses	258
B. albilineans Ashby	Sugarcane	187, **188**
B. panici Elliott	Millets	99
B. rubrisubalbicans (Christopher and Edg.) Burgw	Sugarcane	189
B. stewartii E. F. Sm.	Corn	67, 68, **69**
Xanthomonas holcicola (Elliott) Starr and Burk	Sorghums	**168**, 169
X. malvacearum (E. F. Sm.) Dows	Cotton	341, **342**, 343, 344, 345
X. phaseoli var. *sojense* (Hedges) Starr and Burk	Soybeans	331, **332**
X. rubrilineans (Lee *et al.*) Starr and Burk	Sugarcane	**188**, 189
X. translucens (L. R. Jones, A. G. Johnson, and Reddy) Dows	Cereals	21, **22**, 23, **110**, 256
X. translucens f. sp. *cerealis* Hagb.	Grasses, cereals	257
X. translucens f. sp. *phleipratensis* Wallin and Reddy	Grasses	258
X. translucens f. sp. *secalis* (Reddy, Godkin, and A. G. Johnson) Hagb.	Rye, grasses	151
X. translucens f. sp. *undulosa* (E. F. Sm., L. R. Jones, and Reddy) Hagb.	Wheat	201, **202**
X. vasculorum (Cobb) Dows	Sugarcane	69, 186, **187**, 188
Pseudomonas alboprecipitans Rosen	Grasses, corn	99
P. andropogoni (E. F. Sm.) Stapp	Sorghums	**168**, 169
P. angulata (Fromme and Murray) Stapp	Tobacco	**375**, 376
P. atrofaciens (McCull.) Stapp	Wheat	202
P. coronafaciens (Elliott) Stapp	Oats	108, 109, **110**
P. coronafaciens var. *atropurpurea* (Reddy and Godkin) Stapp	Grasses	**256**, 257
P. glycinea (Coerper) Stapp	Soybeans	331
P. itoana Toch	Rice	134, **135**
P. medicaginis Sackett	Alfalfa	**297**, 325
P. sojae Wolf = *P. glycinea* (Coerper) Stapp	Soybeans	331
P. solanacearum E. F. Sm	Tobacco	373
P. striafaciens (Elliott) Starr and Burk	Oats	110
P. syringae v. Hall	Sorghums	**169**, 317, 325
P. tabaci (Wolf and Foster) Stapp	Tobacco	331, 374, **375**, 376
P. trifoliora (L. R. Jones *et al.*) Burk. = *P. syringae* v. Hall	Clover	**317**

SCHIZOMYCETES	Suscepts	Page
Actinomycetales		
Corynebacterium insidiosum (McCull.) H. L. Jens	Alfalfa	295, **296**, **325**
C. rathayi (E. F. Sm.) Dows	Grasses	258
PHYCOMYCETES		
Chytridiales		
Olpidiaceae		
Olpidium trifolii (Pass.) Schroet	Clover	**298**, 299, 299, **325**
(*Asterocystis radicis* DeWild.) = *Olpidium brassicae* (Wor.) Dang	Flax	357
Cladochytriaeaceae		
Urophlyctis alfalfae (Lageh.) Magn	Alfalfa	297, **298**, 299, **325**
(*U. trifolii* (Pass.) Magn.) = *Olpidium trifolii* (Pass.) Schroet	Clover	**298**, 299, **325**
Physoderma zea-maydis Shaw	Corn	70
OOMYCETES		
Peronosporales		
Pythiaceae		
Pythium aphanidermatum (Edson) Fitzp	Tobacco, flax	71, 357, **377**
P. aristosporum Vanterpool	Wheat	203
P. arrhenomanes Drechs	Cereals, grasses	70, 71, 72, 170, 171, 189, 203, 258
P. debaryanum Hesse	General	258, **299**, 325, 332, 357, **377**
P. deliense Meurs	Tobacco	**377**
P. graminicolum Subr	Cereals	70, 71, 189, 203, 258
P. intermedium DBy	Flax	357
P. irregulare Buis	Flax	357
P. mamillatum Meurs	Flax	357
P. megalacanthum DBy	Flax	357
P. splendens Braun	Alfalfa, flax	299, 357
P. tardicrescens Vanterpool	Wheat	203
P. ultimum Trow	Alfalfa	299
P. vexans DBy	Alfalfa, flax	299, 357
P. volutum Vanterpool and Truscott	Wheat	203
Peronosporaceae		
Phytophthora cactorum (Leb. and Cohn) Schroet	Sweetclover	299, 325
P. megasperma Drechs	Sweetclover	299
P. parasitica Dast	General	378
P. parasitica var. *nicotianae* (Breda de Haan) Tucker	Tobacco	**377**, **378**
Peronospora hyoscyami DBy	Nightshade	**378**, 390
P. manshurica (Naum.) Syd	Soybeans	**332**, **333**
P. nicotianae Speg	Tobacco	**378**, 390
P. tabacina Adam	Tobacco	**378**, 379, 380
P. trifoliorum DBy	Alfalfa, clover	299, 300, **301**, 325

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Peronosporales, *Peronosporaceae*, *Sclerospora butleri* Weston	Grasses	75
S. farlowii Griff.	*Chloris* sp.	75
S. graminicola (Sacc.) Schroet.	Grasses, millets	73, 75, 99, 100, **101**, 171, 204, 259
S. indica Butl.	Corn	73
(*S. javanica* Palm) = *S. maydis* (Rac.) Butl.	Corn	73
S. macrospora Sacc.	Cereals, grasses	73, 75, 110, **204**, 259
S. magnusiana Sorok.	Equisetum	75
S. maydis (Rac.) Butl.	Corn	**73**, 75
S. miscanthi Miy.	*Miscanthus* sp.	75
S. noblei Weston	Sorghum, grasses	75
S. northi Weston	*Erianthus* sp.	75
S. oryzae Brizi	Rice	75
S. philippinensis Weston	Corn, sorghum, sugarcane	**73**, 75
S. sacchari Miy.	Sugarcane, corn	**73**, 75, 190
S. sorghi (Kulkarni) Weston and Uppal	Sorghum	73, 75, **171**
S. spontanea Weston	Sugarcane, corn	**73**, 75
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Mitrula sclerotiorum Rostr.	Clover	321
Perisporiales		
Erysiphaceae		
Erysiphe cichoracearum DC.	Flax	357
E. graminis DC.	Cereals, grasses	25, 26, **27**, 46, 259
E. graminis avenae El. Marchal	Oats, grasses	111
E. graminis hordei El. Marchal	Barley, grasses	26, **27**, 28
E. graminis secalis El. Marchal	Rye, grasses	151
E. graminis tritici El. Marchal	Wheat, grasses	205
E. polygoni DC.	Legumes	**317**, 325, 334, 357
Microsphaera alni (DC.) Wint.	Clover	317
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Hypocreaceae		
Claviceps microcephala (Wallr.) Tul.	Grasses	260
C. paspali F. L. Stevens and Hall	Grasses	259
C. purpurea (Fr.) Tul.	Cereals, grasses	32, 135, 152, 153, **154**, 155, 157, 205, 259, 260, 261
C. pusilla Ces.	Grasses	259
(*Ustilaginoidea oryzae* (Pat.) Bref.) = *U. virens* (Cke.) Tak.	Rice	135
U. setariae Bref.	Grasses	135
U. virens (Cke.) Tak.	Grasses	135
Epichloe typhina (Fr.) Tul.	Grasses	262

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graminicola (Berk. and Br.) Wr.	Cereals, grasses	152, 210, **211**, 262
C. graminicola var. *neglecta* Krampe	Cereals	211
C. nivale Fr. and *C. nivale* f. *graminicola* (Berk. and Br.) Snyder and Hansen according to Snyder and Hansen (Wheat, 1945)		212
Gibberella fujikuroi (Saw.) Wr.	Cereals, grasses, cotton	74, 76, 77, 78, **80**, 135, 171, 190, 345,
G. fujikuroi var. *subglutinans* Edwards	Cereals, grasses	77, **80**, 190
(*G. saubinetti* (Mont.) Sacc.) = *G. zeae* (Schw.) Petch	Cereals, grasses	28, 74
G. zeae (Schw.) Petch	Cereals, grasses	28, 29, 30, 74, 76, 77, 79, 135, 171, 207, 210, 263
Dothidiaceae		
Phyllachora graminis (Fr.) Fckl.	Grasses	262
Cymadothea trifolii (Fr.) Wolf	Clover	**318**, 325
[*Dothidella trifolii* (Fr.) Bayl.-Elliott] = *Cymadothea trifolii* (Fr.) Wolf	Clover	318
Pseudoplea trifolii (Rostr.) Petr.	Clover	**319**, **320**, **325**
[*Saccothecium trifolii* (Rostr.) Kirsch.] = *Pseudoplea trifolii* (Rostr.) Petr.	Clover	319
Sphaeriales		
Mycosphaerellaceae		
Mycosphaerella davisii F. R. Jones	Alfalfa, clover	303, **304**, 325
M. lethalis Stone	Sweetclover	301, 302, **303**, 325
M. linicola Naum.	Flax	359
M. linorum (Wr.) Garcia-Rada	Flax	357, 358, **359**
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Physalospora tucumanensis Speg.	Sugarcane	190, 191, **192**
P. zeae Stout	Corn	83, **84**
P. zeicola Ell. and Ev.	Corn	83
Leptosphaeria avenaria G. F. Weber.	Oats, grasses	113
L. herpotrichoides DeN.	Cereals	215
L. pratensis Sacc. and Briard	Alfalfa, clover, etc.	305, **306**, 325
L. salvinii Catt.	Rice, grasses	135, 136, **137**
Ophiobolus graminis Sacc.	Cereals, grasses	212, 213, **214**, 263
O. graminis var. *avenae* Turner	Oats, grasses	214
O. miyabeanus Ito and Kuribay. See also *Cochliobolus miyabeanus* (Ito and Kuribay.) Drechs.	Rice	139
O. oryzae Miyake	Rice	139
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O. setariae Ito and Kuribay. See also *Cochliobolus setariae* (Ito and Kuribay.) Drechs.	Millets	101
Cochliobolus heterostrophus Drechs.	Corn	85, 86, 87, **88**, 267
C. miyabeanus (Ito and Kuribay.) Drechs.	Rice	138, **139**, 140
C. sativus (Ito and Kuribay.) Drechs. See also *Ophiobolus sativus* Ito and Kuribay	Cereals, grasses, rice	**40**, 41, 216
C. setariae (Ito and Kuribay.) Drechs. See also *Ophiobolus setariae* Ito and Kuribay	Millets	101
C. stenospilus (Carpenter) Matsu. and Yamamoto	Sugarcane	192, **193**
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Pyrenophora avenae Ito and Kuribay	Oats	**111**, 112
P. bromi Died.	*Bromus* spp.	**264**, 265
P. graminea Ito and Kuribay. See also *Helminthosporium gramineum* Rab.	Barley	36
P. teres (Died.) Drechs.	Barley	**32**, 33, 34
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Pleospora herbarum (Fr.) Rab.	Clover, alfalfa	321, **322**, 325
P. rehmiana Staritz	Legumes	302
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Glomerella glycines Lehman and Wolf	Soybeans	334
G. gossypii Edg.	Cotton	343, **344**
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Diaporthe sojae Lehman	Soybeans	334, **335**
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Sclerotinia trifoliorum Eriks.	Clovers, etc.	**320**, 321, 325
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Pseudopeziza medicaginis (Lib.) Sacc.	Alfalfa	**307**, **308**, 325
P. jonesii Mannf. See also *Pyrenopeziza medicaginis* Fckl.	Alfalfa	**307**, **308**, 325
P. meliloti Syd.	Sweetclover	**307**, **308**, 325
P. trifolii (Biv.-Bern.) Fckl.	Clover	**308**, 325
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Ustilaginaceae		
Ustilago aculeata (Ule) Liro	*Agropyron* and *Elymus* spp.	276
U. avenae (Pers.) Rostr.	Oats	46, 51, **114**, 115, 116, 117, 118, 119
[*U. bromivora* (Tul.) Fisch de Waldh.] = *U. bullata* Berk.	Grasses	280
U. bullata Berk.	Grasses	279, 280
U. crameri Koern.	Millet, grasses	46, 102, **103**
U. echinata Schroet.	Reed canary grass	276
U. halophila Speg.	*Distichlis* spp.	278
U. hordei (Pers.) Lagerh.	Barley	46, 52, 53, 54, **55**
[*U. hypodytes* (Schlecht.) Fr.] = *U. spegazzinii* Hirsch., *U. williamsii* (Griff.) Lavrov, *U. halophila* Speg., and *U. nummularia* Speg.	Grasses	277, 278
U. kolleri Wille	Oats	46, 54, 115, 116, 117, 118, **119**, 121
U. longissima (Schlecht.) Meyen	*Glyceria* spp.	276
(*U. lorentziana* Thuem) = *U. bullata* Berk.	Grasses	280
U. maydis (DC.) Oda	Corn	46, **89**, 90
(*U. medians* Bied.) = *U. nigra* Tapke and *U. nuda* (Jens.) Rostr.	Barley	50, 51, 114
U. mulfordiana Ell. and Ev.	*Fescue* spp.	280
U. neglecta Niessl	*Setaria* spp.	103
U. nigra Tapke	Barley	46, 50, **51**, 52, 53, 54, 114
U. nuda (Jens.) Rostr.	Barley	45, 46, **47**, 48, 49, 222
U. nummularia Speg.	Grasses	278
U. perennans Rostr.	*Arrhenatherum* sp.	114
U. scitaminea Syd.	Sugarcane	193
U. sitanii G. W. Fisch.	*Sitanion* and *Hordeum* spp.	280
U. spegazzinii Hirsch.	Grasses	277
U. spegazzinii var. *agrestis* (Syd.) G. W. Fisch. and Hirsch.	Grasses	277
U. striiformis (West.) Niessl	Grasses	274, 275, **276**
U. tritici (Pers.) Rostr.	Wheat	46, 48, 161, **222**, 223
U. williamsii (Griff.) Lavrov	*Oryzopsis* and *Stipa* spp.	278
[*U. zeae* (Beck.) Ung.] = *U. maydis* (DC.) Cda.	Corn	46, **89**
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Ustilaginales, *Ustilaginaceae*, *Sphacelotheca destruens* (Schlecht.) Stevenson and A. G. Johnson	*Panicum* spp.	46, 102, **103**
S. reiliana (Kuehn) Clint.	Sorghum, corn	46, 91, **92**, 178
S. sorghi (Lk.) Clint	Sorghum	46, 176, **177**, 178, 280
Sorosporium syntherismae (Pk.) Farl.	*Cenchrus* and *Panicum* spp.	280
Tolyposporium penicillariae Bref	Millet	102
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Tilletia asperifolia Ell. and Ev	*Muhlenbergia* sp.	281
T. caries (DC.) Tul	Wheat	46, 223, 225, **226**, 228, 229, 281
T. cerebrina Ell. and Ev	*Deschampsia* spp.	281
T. corona Scribn	Southern grasses	147
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T. guyotiana Hariot	*Bromus* spp.	280
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(*T. horrida* Tak.) = *Neovossia horrida* (Tak.) Padwick and Azmatullah Kahn	Rice	46, **147**
(*T. levis* Kuehn) = *T. foetida* (Wallr.) Liro.	Wheat spp.	46, 226
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E. lineatum (Cke.) J. J. Davis	Wild rice, rice	46, **148**, 276
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Melampsora lini (Pers.) Lév	Flax	**363**, **364**
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(*Puccinia anomala* Rostr.) = *P. hordei* Otth.	Barley	**57, 58**
P. coronata Cda.	Oats, grasses	**123**, 124, 125, 126, 127,128, 281,282, 283, 284
(*P. dispersa* Eriks. and E. Henn.) = *P. rubigo-vera* (DC.) Wint.	Rye, grasses	164
P. glumarum (Schm.) Eriks. and E. Henn.	Cereals, grasses	57, **163**, 238, 239, **240**, 281, 282,283, 285
P. graminis Pers.	Cereals, grasses	46, 56, **232**, 233, 235, 243, 281, 282, 283, 284
P. graminis agrostidis Eriks.	Grasses	**281**, 282, 283
P. graminis avenae Eriks. and E. Henn.	Oats, grasses	119,**120**,121,282,283
P. graminis phlei-pratensis (Eriks. and E. Henn.) Stakman and Piemeisel	Timothy, grasses	**281**, 282, 283
P. graminis poae Eriks. and E. Henn.	*Poa* spp., grasses	**281**, 282, 283
P. graminis secalis Eriks. and E. Henn.	Rye, grasses	56, **163**, 282, 283
P. graminis tritici Eriks. and E. Henn.	Wheat, grasses	56, 231, **232**, 237, 238, 240, 243, 282, 283
P. hordei Otth. See also *P. anomala* Rostr.	Barley	57, **58**
P. montanensis Ell.	Grasses	284
P. poae-sudeticae (West.) Jørst.	*Poa* spp.	284
P. polysora Underw.	Corn	93
P. purpurea Cke.	Sorghum	179
P. rubigo-vera (DC.) Wint.	Cereals, grasses	46,164,240,**242**,281, 282, 283, 284
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P. rubigo-vera tritici (Eriks. and E. Henn.) Carleton	Wheat, grasses	57, 241, **242**, 243
P. sorghi Schw.	Corn	92, 93
P. stakmanii Presley	Cotton, grasses	350
(*P. triticina* Eriks.) = *P. rubigo-vera tritici* (Eriks. and E. Henn.) Carleton	Wheat, grasses	242
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(*C. vagum* Berk. and Curt.) = *C. solani* (Prill. and Del.) Bourd. and Galz.	General	**145**, 146, 384
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T. idahoensis Remsberg	Cereals, grasses	221
T. itoana Imai	Cereals, grasses	**221**, 273
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(*P. linicola* Naum.) = *P. linicola* Bub.	Flax	359
(*P. linicola* March. and Verpl.) = *P. linicola* Bub.	Flax	359
P. medicaginis Malbr. and Roum. = *Ascochyta imperfecta* Pk.	Alfalfa	302
P. trifolii E. M. Johnson and Valleau	Clover	301, 302, **303**, 318, 325
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Macrophomina phaseoli (Maubl.) Ashby. See also *Sclerotium bataticola* Taub.	General	**174**, 337
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A. caulicola Laub.	Sweetclover	**303**, 325
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A. linicola Naum. and Wass.	Flax	359
[*A. meliloti* (Trel.) J. J. Davis] = *Mycosphaerella lethalis* Stone	Sweetclover	303
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(*Diplodia frumenti* Ell. and Ev.) = *Physalospora zeicola* Ell. and Ev.	Corn, general	83
D. macrospora Earle	Corn	80, **83**
[*D. maydis* (Berk.) Sacc.] = *D. zeae* (Schw.) Lév.	Corn	80, **81**
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[*S. meliloti* (Lasch.) Petr.] = *Leptosphaeria pratensis* Sacc. and Briard.	Sweetclover	306
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Selenophoma bromigena (Sacc.) Sprague and A. G. Johnson	*Bromus* spp.	**271**, **272**, **273**

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(*Septoria avenae* Frank) = *Leptosphaeria avenaria* G. F. Weber	Oats, grasses	113
S. bromi Sacc.	*Bromus* spp.	271, 272
S. elymi Ell. and Ev.	*Elymus* and *Agropyron* spp.	272
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S. infuscans (Ell. and Ev.) Sprague	*Elymus* spp.	273
S. jaculella Sprague	*Bromus* spp.	271
S. macropoda Pass.	*Poa* spp.	272
S. nodorum Berk.	Wheat, grasses	45, 217, 218, **219**, 220
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S. secalis Prill. and Del.	Rye, grasses	159, **160**
S. secalis var. *stipae* Sprague	*Agrostis* and *Stipa* spp.	271
S. sojae Syd. and Butl.	Soybeans	336
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S. tritici Rob.	Wheat, oats, grasses	217, 218, **219**, 220, 271
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Polyspora lini Laff.	Flax	359, **360**, 361
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(*C. falcatum* Went.) = *Physalospora tucumanensis* Speg.	Sugarcane	190, 191, **192**
C. graminicolum (Ces.) G. W. Wils.	Cereals, grasses	44, 113, 158, **159**, 172, 173, 191, 192, 324
(*C. lineola* Cda.) = *C. graminicolum* (Ces.) G. W. Wils.	Sorghum	172, 191, 192
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R. secalis (Oud.) J. J. Davis	Barley, rye, grasses	42, **43**, 44, 159, **268**, 269
Mastigosporium album Riess	Grasses	268
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M. cylindricum Sprague	Grasses	268
M. rubricosum (Dearn. and Barth.) Sprague.	Grasses	268
Piricularia oryzae Cav	Rice	141, **142**
Cercosporella herpotrichoides Fron	Cereals	215
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Thielaviopsis basicola (Berk. and Br.) Ferr..	Tobacco, flax, general	357, **382**, 383
Nigrospora oryzae (Berk. and Br.) Petch..	Corn, rice	84
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N. sphaerica (Sacc.) Mason	Corn, barley	**84**, 85
Scolecotrichum graminis Fckl	Grasses	**269**, 270
S. graminis avenae Eriks	Oats, grasses	113
(*Helminthosporium avenae* Eidam) = *Pyrenophora avenae* Ito and Kuribay....	Oats	**111**, 112
H. bicolor Mitra	Wheat	216
(*H. bromi* Died.) = *Pyrenophora bromi* Died.	*Bromus* spp.	264
H. californicum Mackie and Paxton	Barley	42
H. carbonum Ullstrup	Corn	85, 86, 87, **88**
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H. dictyoides Drechs	*Festuca* sp.	265, **267**
H. erythrospilum Drechs	*Agrostis* spp.	266
H. giganteum Heald and Wolf	Grasses	266
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H. halodes var. *tritici* Mitra	Wheat	216
H. leucostylum Drechs	Millet, grasses	101
(*H. maydis* Nishikado and Miyake) = *Cochliobolus heterostropus* Drechs	Corn	85, 86, 87, **88**
H. miyakei Nishikado	*Eragrostis* sp.	267
H. monoceras Drechs	*Echinochloa* sp.	101
H. nodulosum (Berk. and Curt.) Sacc	Millet, grasses	101
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H. sacchari (Breda de Haan) Butl	Sugarcane	192, **193**
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