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CORN DISEASES

**IN THE UNITED STATES
AND THEIR CONTROL**

AGRICULTURAL RESEARCH
SERVICE • UNITED STATES
DEPARTMENT OF AGRICULTURE
IN COOPERATION WITH
PURDUE UNIVERSITY
AGRICULTURAL
EXPERIMENT STATION

AGRICULTURE HANDBOOK NO. 199

CONTENTS

	Page
Factors affecting the development of corn diseases.....	1
Environment.....	2
Resistance of host.....	2
Causal organisms.....	2
Control of corn diseases.....	2
Seed rot and seedling blight.....	3
Stalk rots and root rots.....	5
Diplodia stalk rot.....	6
Gibberella stalk rot.....	7
Charcoal rot.....	7
Pythium stalk rot.....	8
Other stalk rots.....	8
Pythium root rot.....	9
Ear rots.....	9
Diplodia ear rot.....	9
Fusarium kernel rot.....	10
Cob rot.....	10
Gibberella ear rot.....	11
Gray ear rot.....	11
Other ear rots.....	12
Storage rots and kernel disorders.....	12
Leaf diseases.....	14
Northern corn leaf blight.....	15
Southern corn leaf blight.....	16
Helminthosporium leaf spot.....	17
Bacterial wilt.....	18
Minor leaf diseases.....	19
Bacterial leaf blight and stalk rot.....	20
Bacterial stripe.....	21
Zonate leaf spot.....	21
Purple sheath spot.....	22
Holcus spot.....	22
Cercospora leaf spot.....	22
Anthracnose.....	22
Brown spot.....	22
Corn rusts.....	23
Common corn rust.....	23
Southern corn rust.....	24
Tropical corn rust.....	25
Downy mildews.....	25
Crazy top.....	25
Downy mildew.....	26
Virus diseases.....	27
Corn stunt.....	27
Sugarcane mosaic.....	27
Celery stripe.....	27
Leaf fleck.....	27
Corn smuts.....	28
Common smut.....	28
Head smut.....	29

CORN DISEASES

IN THE UNITED STATES AND THEIR CONTROL

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The corn (*Zea mays* L.) crop of the United States is subject to a number of diseases that reduce both yield and quality. Yearly losses range from about 2 to 7 per cent, but in some localized areas one or more diseases may become acute and destroy a considerably higher proportion of the crop. Ear and kernel rots decrease yield, quality, and feeding value of the grain. Stalk diseases may not only lower yield and quality but also make harvesting difficult. When leaves are damaged by diseases, the production of carbohydrates to be stored in the grain is lessened, and immature, chaffy ears result.

The diseases of corn may be classified as parasitic and non-parasitic. Most of the parasitic or infectious diseases of corn are caused by fungi, a few by bacteria, and a few by viruses. Fortunately no known virus diseases of corn have been found in the Corn Belt of the United States. Some parasitic diseases are caused by nematodes. These very small, slender, wormlike animals live in

the soil and may feed on the roots of corn. They have not become serious in the main corn-growing areas of this country, but they are prevalent in the Southern States.

Nonparasitic diseases result from unfavorable climatic and soil conditions. Deficiencies in the soil of nitrogen, phosphorus, or potassium cause some of the most frequently observed nonparasitic diseases of corn. Occasionally corn may suffer from the lack of essential minor elements in the soil.

In contrast to some of the diseases in certain other crops, the diseases of corn seldom become severe over very wide areas. The production of corn in any given locality of the United States has not been limited by disease where soil and weather conditions have been favorable for the crop, nor has it been necessary to stop growing the crop over a wide area because of disease.

In this handbook only the parasitic diseases caused by fungi, bacteria, and viruses will be treated in detail.

FACTORS AFFECTING THE DEVELOPMENT OF CORN DISEASES

Diseases of corn, like those of other crops, vary in severity from year to year and from one locality or field to another depending on the environment, the resistance of the host, and the causal organism. All three factors must be present for the development of a disease.

For example, if the environment is favorable for a disease and the causal organism is present, but the host is highly resistant, little or no disease will develop. Similarly, if the causal organism is present and the host is susceptible, but the environment is unfavorable, the disease may not appear.

Environment

Many corn diseases develop best when there is plenty of moisture during the growing season. Either rain or heavy dew is necessary for spores of disease-producing fungi to germinate and to penetrate the plant. Some diseases of corn such as seed rot and seedling blight thrive when the soil temperature is low for an extended period following planting. Bacterial wilt, or Stewart's disease, is favored by mild winter temperatures. Under such conditions the corn flea beetles (*Chaetocnema pulicaria* Melsh.), which carry the disease-producing bacteria through the winter and spread the disease from plant to plant, survive in large numbers. Crazy top of corn becomes prevalent only when the soil is waterlogged for several days during the very young seedling stage. The temperature and moisture of both soil and air may thus determine the development of corn diseases.

Soil fertility may affect the severity of some infectious diseases of corn, particularly certain stalk rots. Maintaining soils at highly productive levels by judicious use of proper fertilizers tends to produce vigorous plants that do not die prematurely. However, some diseases seem not to be affected to any great extent by soil fertility, and a general statement cannot be made that a fertile soil means healthy corn plants.

Resistance of Host

Great differences exist among inbred lines and hybrids as to their relative resistance to various diseases. Their inherent resistance or susceptibility may often determine whether an epidemic of a given disease will occur. Resistance to most corn diseases is determined by several genes. In only three diseases of corn is resistance inherited as a monogenic dominant. It is the genetic resistance of the host that can be manipulated by the corn breeder and through which he can modify inbred lines and their hybrid combinations so as to produce high levels of disease resistance.

Causal Organisms

No disease epidemic will occur if the specific causal organism—a fungus, bacterium, or virus—is absent, even though the environment is favorable and the host susceptible. The causal organisms are as variable in their virulence as the hosts they attack are in their relative resistance. There are, for example, several physiologic races of *Puccinia sorghi* Schw., the fungus causing common corn rust. These vary only in their virulence on different inbred lines of corn. However, in the United States the problem of physiologic races within the species of fungi and of bacteria causing diseases in corn has not been a deterrent to breeding for disease resistance.

CONTROL OF CORN DISEASES

Two ways to control corn diseases are the use of disease-resistant hybrids and the application of fungicides to the seed. Other practices such as spraying plants with fungicides, adjustment of soil fertility by intelligent use of fertilizers, and maintenance of

good drainage tend to reduce losses from certain diseases, but these have limited application.

The incorporation of genetic resistance into agronomically desirable hybrids by breeding methods seems to be the most efficient and permanent means of

controlling corn diseases. None of the hybrids in use are highly resistant to all diseases and some are extremely susceptible. Much remains to be done in developing disease-resistant hybrids by the plant breeder and plant pathologist. It may be difficult to obtain high resistance to all diseases, but on the basis of past accomplishments it appears to be entirely possible to develop adapted, high-yielding corn hybrids that are resistant to the major diseases in a given area. The resistance of a hybrid to a disease is generally proportional to the number of resistant inbred lines in its pedigree. Thus a hybrid comprised of four resistant inbred lines is likely to be more resistant than one possessing only a single resistant inbred line in its pedigree.

Where a disease is of sufficient economic importance to warrant control, local State agricultural experiment stations should be consulted for recommendations on the use of resistant hybrids for the particular area.

The treatment of seed corn with a fungicide, either in a dust or a slurry, may control seed rots and seedling diseases but no other diseases. Since 1935 considerable advancement has been made in improving seed corn treatments, and good fungicides are on the market that are specifically prepared for this purpose. These should be used according to the recommendations of the manufacturer.

Spraying corn plants with fungicides to control leaf diseases is successful in southern Florida. This practice is economically feasible there because it is confined to market-garden sweet corn,

which is a high value crop. In many seasons leaf blights are so severe in southern Florida that unsprayed sweet corn fails to yield marketable ears.

Crop rotation and destruction of diseased plant parts have been suggested as control measures for certain plant diseases. Such practices are most effective where the crop is grown in a limited area or if the specific disease-producing agent is strictly soilborne. In the Corn Belt of the United States or other areas where corn is extensively grown, it is unlikely that destroying diseased plant parts can be sufficiently thorough to be effective in eliminating diseases. With few minor exceptions rotation of crops has little effect on reducing diseases of corn. In river-bottom fields where, by necessity, corn is often grown successively over several years, some diseases appear to be no more prevalent than where rotation is practiced. Rotation is probably more beneficial to corn in improving tilth and conserving fertility than in reducing diseases.

Maintaining well-balanced fertility of the soil can help to lessen the effects of some corn diseases. Certain stalk rots are more severe when corn is planted where potassium is deficient and nitrogen is in excess. The effect of soil fertility on corn diseases depends not only on the specific disease but also on the particular mineral deficiencies in the soil. Much is to be learned about the relationship of soil nutrients to the diseases of corn. Since yield is a primary factor in corn production, every effort should be made to build up and maintain soils at maximum fertility levels.

SEED ROT AND SEEDLING BLIGHT

The period during which seed germinates and the seedling be-

comes established is very critical in the life cycle of the corn plant.

Severe infection may kill the embryo before germination. Where infection takes place after germination, seedlings may be destroyed before or after emergence (pl. 1, A). Seedlings that survive attack are usually retarded in vigor and develop into less productive plants than plants from healthy seedlings.

The prevalence of seedling blight varies considerably depending, in part, on the weather conditions following planting. Seedling diseases are more prevalent in cold, wet soil than in warm soil. Under the latter condition germination is rapid, and the seedling soon becomes established and escapes infection by soil-inhabiting, pathogenic fungi. In cold, wet soils germination is retarded and the disease-producing fungi can invade the embryo or young seedling. A soil temperature of about 50° F. is favorable for seedling blight, because at this temperature germination is very slow and the soilborne pathogens can grow and invade seed and seedlings.

In addition to the effect of cold, wet soil, other factors such as age of the seed, degree of finish or maturity, mechanical damage, and genetic resistance to invasion by disease-producing fungi affect the severity of seed rot and seedling blight. Seed stored for 2 or more years often becomes increasingly susceptible to seed and seedling diseases when planted under unfavorable conditions. Dry seed of 12-percent moisture or less stored at about 35° F. at a low humidity will generally germinate and grow in cold soil. Immature or poorly finished seed is almost invariably susceptible to seedling blight. Mechanical damage to the seedcoat, such as breaks or cracks that occur during harvesting or processing, affords ave-

nues by which soilborne fungi can invade the seed. Inbred lines and their hybrid combinations differ in their resistance to seed rot and seedling blight. In general, sweet corn is much more susceptible to seedling diseases than is dent corn, and popcorn is most resistant. This susceptibility may be due to genetic factors, thinner seedcoat, and ready availability of the sugary endosperm as nutrition for the pathogenic fungi.

With the introduction of hybrid corn there has been less seed rot and seedling disease than when open-pollinated corn was used because of better culling of seed ears, rapid artificial drying, selection for resistance, and use of effective seed-treating materials.

Symptoms.—The symptoms of seed rot and seedling blight range from complete killing of the embryo before seed germination in severe infection to small discolored lesions on the roots and lower part of the sprout in mild infection (pl. 1, B). Although several fungi may parasitize the seed and seedling, the symptoms are generally similar. Lesions on the mesocotyl are often brown and sunken. Invasion of roots by pathogenic fungi is indicated by discolored, water-soaked, flaccid lesions. Aboveground symptoms appear as wilting and dying of the seedling leaves.

Causal Organisms.—A number of fungi can cause seed rot and seedling blight of corn. The most important of these are *Pythium irregulare* Buis., *P. debaryanum* Hesse, *P. paroeandrum* Drechs., *P. ultimum* Trow, *P. splendens* Braun, *P. vexans* DBy., and *P. rostratum* Butl. The members of this genus are strictly soilborne. Their distribution is determined by the soil type and cropping history. Muck soils frequently have a large population of one or more

species of *Pythium* that are pathogenic on corn. In general, the species attacking seed and seedlings are able to thrive at temperatures somewhat lower than those favoring rapid germination of corn.

In addition to the several species of *Pythium*, any one of the ear rot fungi may also infect seed and seedlings. These fungi are most often seedborne and already established in the seed at harvest. One of the most important economically is *Diplodia maydis* (Berk.) Sacc., which when in the seed often destroys the embryo before germination. *Gibberella zeae* (Schw.) Petch is found in the cooler areas where corn is grown and can cause severe seed rot and seedling blight in cold soil. *Penicillium oxalicum* Currie & Thom can cause seedling blight even in warm soil. This fungus attacks the mesocotyl near the seed and secretes oxalic acid, which kills cells in advance of the fungus. Seedlings infected with *P. oxalicum* often show wilted and necrotic streaks on their leaves, presumably due to toxic effects of oxalic acid translocated from the point of infection.

A few less virulent fungi sometimes associated with diseased seedlings are *Rhizoctonia zeae* Voorhees in the Gulf States, *R. bataticola* (Taub.) Butl., *Nigrospora oryzae* (Berk. & Br.) Petch, and *Aspergillus* spp. *Fusarium*

moniliforme Sheld. is often isolated from blighted seedlings, but most of the work done with it suggests that it is largely a secondary invader following a more vigorous parasite. Several other fungi have been found in diseased corn seedlings, but their virulence has never been proved.

Control.—The use of fungicides for seed treatment is necessary to control seedling diseases. Good seed-treating materials are available and protect the seed from invasion by soilborne pathogens during the critical early stages of germination. However, these materials have little if any effect on seedborne pathogens already established within the seed prior to planting. With the advent of hybrid corn, the application of a seed-treating material by the seed producer has become almost universal.

Another way to reduce the predisposition of seed corn to attack by pathogens after planting is to adjust harvesting and processing machinery so that there is minimum physical injury to the seed-coat. The use of resistant hybrids is still another means of avoiding seedling diseases and insuring good stands of corn. Delay in planting until the soil has become warm and until the danger of extended periods of cold weather has passed minimizes the hazard of poor stands due to seedling blight.

STALK ROTS AND ROOT ROTS

Stalk rots and root rots of corn are widespread in most areas where the crop is grown. In seasons when these diseases are severe, plants may die 2 to 3 weeks before becoming fully mature. As a result, the ears are light weight and poorly finished. However, more frequently the greatest damage is caused by stalk

breakage and root lodging, which make harvesting difficult, and many ears are lost on the ground.

Three stalk rots—diplodia, gibberella, and charcoal rot—are similar in that their respective causal organisms show the same general type of parasitism. The fungi responsible for these dis-

eases do not ordinarily attack young physiologically active plants but those approaching maturity. However, pythium stalk rot is a different type of disease, because young plants may be attacked well before the silking stage.

Diplodia Stalk Rot

Probably the most common stalk rot in the Corn Belt is diplodia stalk rot. The factors affecting the prevalence of the disease are not fully known, but dry weather in the early-growing season followed by ample rainfall for 2 to 3 weeks after silking appears to favor the development of this disease. Severe infection by any of the leaf diseases or destruction of leaves by hail or insects predisposes stalks to infection. Soil fertility may affect the severity of diplodia stalk rot. Observations and a few experiments have shown that more stalk rot occurs where soils are excessively high in nitrogen and low in potassium than where fertility is ample and balanced.

Symptoms.—When infection takes place before plants reach maturity, the leaves suddenly become grayish green similar to the effects of frost injury (pl. 2, A), the lower parts of the stalk turn from green to tan or brownish, and the pith becomes soft (pl. 2, B). When diseased stalks are split open, the pith is disintegrated and discolored, and only the water-conducting vascular bundles are left intact (pl. 2, C). Diseased stalks are weakened and break readily in the wind and rain. Occasionally minute black pycnidia, the fruiting bodies of the fungus, form in the fall just beneath the surface of the lower internodes of the stalk (pl. 2, E).

Causal Organism.—Diplodia stalk rot is caused by *Diplodia*

maydis (*D. zae* (Schw.) Lév.). The fungus produces two types of asexual spores. The most common is long, elliptical, straight to slightly curved, two celled, and olive colored to brown. The second spore type, less frequently seen, is colorless, long, narrow, and threadlike. Both are produced in small, black, flask-shaped pycnidia, which develop on infected parts of the host in the field in either the current year or the following spring (pl. 2, E). When spores are mature, they are exuded from the pycnidia during warm, moist weather and carried by the wind to healthy plants where they infect the stalk or ear. No sexual stage of the fungus is known.

Infection takes place most frequently at the crown of the plant and spreads into the stalk and the roots. Infection sometimes occurs at the nodes between the crown and the ear. Although the fungus spreads some distance in the stalk, it does not invade the entire plant. Generally the fungus does not grow from the crown through the stalk and into the ear to cause a rot there.

Control.—One way to reduce stalk rot is to use resistant hybrids. Local State agricultural experiment stations should be consulted for adapted resistant hybrids. Full-season hybrids are generally more resistant than those that mature early in a given area. Properly balanced soil fertility will tend to reduce the abundance and severity of diplodia stalk rot.

Where soil is infertile or potassium is deficient and nitrogen proportionately excessive, this disease is often more severe. The application of the proper kinds and amounts of fertilizer based on soil and tissue tests may help to minimize stalk rotting and breakage.

Gibberella Stalk Rot

Gibberella stalk rot is widely distributed in the United States and more prevalent in the northern and eastern parts of the country than diplodia stalk rot. So far as is known the environmental factors affecting both diseases are similar. The diseases are somewhat alike in that both usually attack plants several weeks after silking.

Symptoms.—The sudden onset of grayish-green color of leaves of early-infected plants resembles the appearance of plants affected with diplodia stalk rot at the same stage of development. The softening and discoloration of the exterior of lower internodes are also very similar in both diseases (pl. 3, A and B). When stalks affected with gibberella stalk rot are split, they will generally show a reddish discoloration of the diseased area. The shredded appearance of the pith is otherwise much the same as in diplodia stalk rot.

Causal Organism. — Gibberella stalk rot is a fungus disease caused by *Gibberella zeae*. The fungus has both asexual and sexual spores. The former are long, curved, and spindle shaped, with several cross walls. These spores are produced on the mycelium of the fungus, which grows from diseased plant parts during warm, moist weather. The asexual stage is known as *Fusarium graminearum* Schwabe. The sexual spores, or ascospores, are produced in black flask-shaped perithecia, which develop on the surface of diseased cornstalks in the fall or during the spring of the following year (pl. 3, C). Ascospores are exuded from the perithecia during warm, moist weather and carried by the wind to infect ears or stalks. This fungus also attacks wheat and barley,

where it causes the disease known as scab.

Control.—This disease may be controlled by using full-season resistant hybrids and by adjusting soil fertility to proper balance where necessary.

Charcoal Rot

Charcoal rot is found mostly in the drier areas where corn is grown, principally in the eastern Great Plains and in California. Occasionally it becomes common in the eastern Corn Belt.

Symptoms.—The disease first attacks the roots of seedlings and young plants. Lesions are brown and water soaked and later become black. When the plant approaches maturity, the disease spreads into the crown and lower internodes of the stalk. Infected stalks may often be recognized by grayish streaks on the surface of lower internodes. Internal



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FIGURE 1.—Typical shredding and grayish-black lower internodes of a cornstalk affected with charcoal rot.

parts of the stalk are shredded and grayish black (fig. 1). Minute black specks, or sclerotia, of the fungus are scattered over the surface of the fibrovascular bundles.

Causal Organism.—*Macrophoma phaseoli* (Maubl.) Ashby (*Rhizoctonia bataticola*) (*Sclerotium bataticola* Taub.) is the causal organism of charcoal rot. The fungus appears to be composed of several strains, which are differentiated on the basis of the size of sclerotia and the presence or absence of pycnidia. The spores are colorless, oval, and single celled and are borne in black flask-shaped pycnidia. Those strains of the fungus attacking corn evidently do not form spores. The sclerotia are black and globular to irregular in shape. They are a means by which the fungus is disseminated and by which it overwinters. The fungus has a wide host range, attacking sorghum, beans, and several other crops.

Control.—The disease may be controlled by irrigation, where practical, and by long rotations with crops that are not natural hosts of the fungus. Little is known about the relationship of soil fertility to the disease or the relative resistance of inbred lines and hybrids. Since the disease invades the crown and stalk as maturity is approached, balanced soil fertility and the use of full-season adapted hybrids would probably tend to reduce the severity of charcoal rot.

Pythium Stalk Rot

Pythium stalk rot is a minor disease that becomes acute only in localized areas. It seems to be confined to the Eastern and Southeastern United States, but it has been reported in some parts of Southern Europe and South America. The disease is favored by extended periods of hot, humid

weather. It is often encountered in river-bottom fields when air drainage is poor and humidity high.

Symptoms.—The disease is generally first recognized when plants fall over. The rotted part of the stalk is usually confined to a single internode just above the soil line. The diseased area is brown, water soaked, soft, and collapsed (pl. 3, D). The stalks are not broken off completely by the disease, and the plants live for several weeks because the vascular bundles remain intact.

Causal Organism.—Pythium stalk rot is caused by *Pythium aphanidermatum* (Edson) Fitz. (*P. butleri* Subr.). The fungus thrives at high temperatures, and unlike other stalk-rotting fungi it can attack young vigorously growing plants before silking time and the onset of maturity. The fungus is strictly a soil inhabitant.

Control.—No specific control for the disease is known, although differences in resistance are found among inbred lines.

Other Stalk Rots

A bacterial disease attacking both stalks and leaves of corn has been observed in North Carolina where overhead irrigation is used. A similar disease has been found in Wisconsin under comparable conditions. Stalks become soft, water soaked, and tan or brown. They topple over at the point of infection. Leaves are invaded at their base near the sheaths and ultimately become badly shredded. The causal organism, which is extremely virulent, appears to be a species of the genus *Erwinia* and distinct in certain characteristics from any other bacteria that cause stalk rots of corn.

Another bacterial stalk rot with symptoms very similar to those caused by *Pythium aphaniderma-*

tum has been reported on corn. The causal organism is *Erwinia dissolvens* (Rosen) Burk. A third stalk rot caused by the bacterium *Pseudomonas lapsa* (Ark) Burk. has been reported from California. Like the stalk rot caused by *E. dissolvens*, it closely resembles pythium stalk rot. Both of these bacterial stalk rots seem to be rare and limited in extent.

Pythium Root Rot

Pythium root rot is widely distributed, but evidently it is not so destructive to corn as to sugar-

cane and sorghum. Small feeding roots are first attacked, and lesions become light to dark brown and flaccid. In later stages large areas of the roots may become infected and severe lodging results. *Pythium debaryanum* and *P. graminicola* Subr., both of which have a wide host range, have been isolated from infected roots. The disease usually becomes severe where soil is poorly drained, cold, and wet. Inbred lines differ in resistance to the disease.

EAR ROTS

Corn is susceptible to a number of ear rots, five of which are widespread throughout the main corn-growing areas of the United States. They include diplodia ear rot, fusarium kernel rot, cob rot, gibberella ear rot, and gray ear rot. The diseases can, at times, reduce yield, quality, and feeding value of the grain. However, severe epidemics are usually limited to localized areas and are of less general economic importance than stalk rots.

Diplodia Ear Rot

Of the ear rots in the Corn Belt, diplodia ear rot, or dry rot, is the most common. In some years and in restricted areas this ear rot has caused appreciable damage. Wet weather extending from silking until 2 to 3 weeks thereafter appears to be ideal for infection of the ears, particularly if the early summer has been relatively dry so that spores of the fungus have not been dispersed before the ears were formed.

Symptoms.—Husks of ears infected early appear bleached (pl. 4, A) in contrast to the green healthy ears. When infection takes place within 2 weeks after silking,

the entire ear becomes grayish brown, shrunken, and very light weight by harvest (see cover). Such ears usually remain upright, with husks stuck tightly together owing to the growth of the fungus between them. Black pycnidia of the fungus are often found at the base of the husks and on the sides of the kernels of such badly infected ears. Ears infected later in the growing season may show no external signs of the disease until the ear is broken or kernels are removed. A white mold will then be found growing between the kernels, and their tips will be discolored. Infection usually begins at the base of the ear and progresses toward the tip. Ears appear to be most susceptible to infection from silking until about 3 weeks later.

Causal Organism.—This ear rot is caused by *Diplodia maydis*, the same fungus associated with diplodia stalk rot. It is described under that disease.

Control.—Inbred lines vary in their resistance to this ear rot and tend to transmit their reaction to this disease to their double-cross combinations. No inbred line or hybrid is completely resistant, but the most susceptible inbreds are

usually discarded before they are in hybrid combinations.

Fusarium Kernel Rot

Fusarium kernel rot is probably the most widespread disease attacking corn ears. It is present to some extent every year in the Corn Belt, but it seems to be more prevalent in the western part of this area. In central California the disease is sometimes particularly prevalent. Dry weather seems to favor its spread and development. The total loss caused by the disease is probably less than that caused by diplodia ear rot because of the scattered infections rather than complete involvement of the ears.

Symptoms.—The first symptom of fusarium kernel rot is a pink discoloration of caps of individual kernels or groups of kernels scattered over the ear (pl. 4, B). The color of infected kernels may vary somewhat from faint pink to reddish brown depending somewhat on the moisture content of the grain. As the disease progresses, infected kernels show a powdery or cottony-pink mold growth. Infection often becomes established around points where corn ear-worms (*Heliothis zea* (Boddie)) have entered the ear or channeled between the rows of kernels.

Causal Organisms.—Fusarium kernel rot is caused by *Fusarium moniliforme* and a closely related fungus, *F. moniliforme* var. *subglutinans* Wr. & Reinking, both of which produce an abundance of small, colorless, one-celled spores borne in chains or in false heads. These are microconidia. A few curved to spindle-shaped multicellular macroconidia are also formed on the mycelium. Occasionally the sexual stages *Gibberella fujikuroi* (Saw.) Wr. and *G. fujikuroi* var. *subglutinans* Edwards can be found on old corn-

stalks. They are represented by small black perithecia, which bear ascospores. The latter ooze from the perithecia during warm, moist weather. The sexual stage resembles that of *G. zea* in some respects.

Control.—No specific control for the disease can be recommended other than to avoid hybrids that tend to be susceptible. Hybrids with poor husk cover or with weak seedcoats in which kernels tend to "pop" or "silk cut" are susceptible to infection.

Cob Rot

Cob rot is widely distributed and present to some extent every year. Corn that has been killed or in which normal development has been arrested because of stalk rot, leaf blights, cold, or root injury is susceptible to cob rot. Corn grown on poor soil appears to be more susceptible than that raised on fertile soil, possibly because of premature dying from lack of proper nutrition.

Symptoms.—Affected ears are light weight, and the kernels are slightly bleached, poorly finished, and easily pressed into the cob. Shanks and bases of badly infected ears are often shredded, particularly when ears are picked mechanically or later when they are shelled. Close examination of infected ears shows very small black spore masses scattered in the shredded pith of the cob and on the tips of the kernels (pl. 4, C). The chaff on infected ears is often brown or chocolate colored instead of the normal bright red. In affected white hybrids the chaff will be pale yellowish or gray.

Causal Organism.—Cob rot is caused by *Nigrospora oryzae* (Berk. & Br.) Petch (*Basisporium gallarum* Möll.), a fungus that overwinters on old plant refuse in

the field. Only an asexual stage is known. Spores are globular to ovoid, black, and borne singly on the mycelium.

Control.—Since the fungus attacks ears of weakened plants, full-season adapted hybrids that are resistant to stalk rot and leaf blight are recommended.

Gibberella Ear Rot

Gibberella ear rot, sometimes called red ear rot, is found more frequently in the cooler, more humid areas where corn is grown. Corn infected with this disease is particularly toxic to hogs. When such corn is offered on the ear, the animals will refuse it, but when it is ground into meal they have no choice. Vomiting and dizziness are symptoms after hogs consume the infected corn. In severe cases death may follow.

Symptoms.—A reddish mold that starts at the tip of the ear is the primary and distinguishing symptom of gibberella ear rot (pl. 4, *D*). Ears infected early may become completely rotted; the husks adhere tightly and a pinkish mold grows between them.

Causal Organism.—This disease is caused by *Gibberella zeae*, the same fungus responsible for gibberella stalk rot. It is described under that disease.

Control.—Since gibberella ear rot has generally been of minor economic importance, no efforts have been made to control it through breeding for resistance.

Gray Ear Rot

Gray ear rot seems to be widely distributed over the Eastern United States, but rarely has it been severe and then only in very restricted areas. Extended periods of wet weather for several weeks after silking seem to favor the disease.

Symptoms.—In its early stages

this disease resembles diplodia ear rot because of the grayish-white mold that develops on and between the kernels near the base of the ear. In ears that have been infected early, the husks are tightly adherent and bleached (pl. 5, *A* and *B*). At harvest such ears are slate gray, and because of their light weight they are held upright. When an infected ear is broken, the cob will often show very small black specks, or sclerotia, scattered through it. Badly infected kernels show black streaks or specks beneath the seedcoat (pl. 5, *C*). In advanced stages the disease is easily distinguished from diplodia ear rot by the slate-gray color of the ear and black sclerotia in the pith of the cob and under the seedcoat.

Causal Organism.—The disease is caused by *Physalospora zeae* Stout, which produces the perithecia of the sexual stage and pycnidia of the asexual stage in large lesions on corn leaves (pl. 5, *D*). Occasionally infection takes place on the tassel neck or under the sheath of the uppermost leaf of the plant. The perithecia and the pycnidia that may develop in the same lesion are black and imbedded in the leaf tissue. Ascospores and pycnidiospores are oval or ellipsoid, colorless to pale green, and one celled. The fungus overwinters on infected leaves, and spores mature the following growing season to infect leaves and ears. Perithecia and pycnidia are not found on the ears. Only the sclerotia, which are small, hard, black knots of the mycelium, are found in the rotted ears and kernels. These structures are resistant to extremes of the environment and serve as a means of survival and propagation of the fungus. The name of the asexual stage of the fungus is *Macrophoma zeae* Tehon & Daniels.

Control.—No control for the disease is known other than the use of adapted hybrids. Since the disease is so similar to diplodia ear rot as to time and place of infection on the corn plant, resistance to the two diseases might be rather closely correlated.

Other Ear Rots

Several other ear rots and kernel rots of corn have been described. However, they are confined to limited geographical areas, occur very rarely, or are otherwise of negligible consequence in the pathology of the corn crop.

Physalospora ear rot is confined largely to the Gulf States and is similar in some respects to gray ear rot. Severe infection is characterized by dark-brown to black felty mold growth on all parts of the ear. Mildly infected ears may show a few blackened kernels confined near the base where most infections begin. The causal fungus *Physalospora zeicola* Ell. & Ev. has both a sexual and an asexual stage; the latter is called *Diplodia frumenti* Ell. & Ev. Pycnidiospores are oval, two celled, and striate. They develop on cornstalks infected by the fungus. *D. frumenti* is distinguished from *Macrophoma zeae* on the basis of size, shape, color, and septation of the pycnidiospores.

Rhizoctonia ear rot, found also in the Gulf States, is recognized in its early stages by a salmon-pink mold growth on the ear. With age the infected ears become dull gray, and numerous small sclerotia ranging from white through salmon color to brown are formed on the outer husks. The disease is caused by *Rhizoc-*

tonia zeae, a fungus not known to produce spores, but it is propagated and spread only by the sclerotia. Warm, humid weather seems favorable for infection.

Southern diplodia ear rot is found in the Southeastern United States. The symptoms of the disease are similar to those of diplodia ear rot (see cover). Southern diplodia ear rot is caused by *Diplodia macrospora* Earle, a fungus similar to *D. maydis*, but differing mainly in having larger spores. The fungus also attacks leaves of corn and produces small, round to oval lesions.

Penicillium ear rot is occasionally observed, particularly on inbred lines. The characteristic appearance is a powdery green mold on and between the kernels, which are often bleached. Infections are frequently associated with corn earworm channels. The causal organism is most frequently *Penicillium oxalicum*, but occasionally other species of the genus have been isolated from diseased ears.

Aspergillus ear rot is another disease that is comparatively rare. Black powdery mold grows on and between the kernels. *Aspergillus niger* v. Tiegh. is the fungus most frequently associated with this ear rot, although other species, some of which are greenish yellow, have been isolated from diseased ears.

Cladosporium kernel rot occurs occasionally in some years. Symptoms are characterized by dark greenish-black kernels scattered over the ear. *Cladosporium herbarum* S. F. Gray (*Hormodendrum cladosporioides* (Fres.) Sacc.) is the causal organism of the disease.

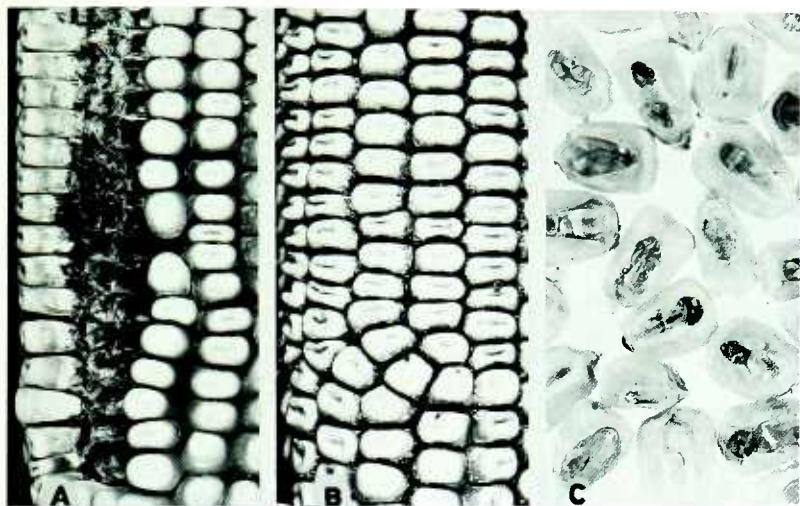
STORAGE ROTS AND KERNEL DISORDERS

Storage rots may develop on either cribbed ear corn or shelled

corn in a bin if the moisture content of the kernels and the air

temperature are high enough to permit fungi to grow. Storage rots reduce both feeding value and market grade of the corn.

Some of these typical storage molds are able to grow on corn with moisture contents as low as 14 to 18 percent.



BN-11245X, BN-11253X, BN-11249X

FIGURE 2.—Storage rots of corn: *A* and *B*, Mold growth between and at base of kernels; *C*, “blue-eye” of stored shelled corn. These rots develop when corn is stored before moisture content is sufficiently reduced.

Typical mold development on and between kernels and at their base are the first symptoms of these rots (fig. 2, *A* and *B*). When storage rots are allowed to develop, the kernels will cake together because of extensive mold growth. One storage rot called “blue-eye” is characterized by the bluish-green germ due to growth of the mold (fig. 2, *C*).

Storage rots can be due to any one of the several fungi causing ear and kernel rots in the field. Several species of *Aspergillus* and of *Penicillium* are the most common storage-rot fungi and frequently are referred to as typical storage molds. These are *A. glaucus* Lk. ex Fr. (group), *A. flavus* Lk. ex Fr., *A. candidus* Lk., *A. niger*, *A. ochraceus* Wilhelm, *P. rugulosum* Thom, *P. palitans* Westl., and *P. chrysogenum* Thom.

Storage molds can be controlled by drying shelled corn to 12-percent moisture or slightly less. Ear corn stored in well-ventilated cribs is ordinarily in no danger from storage rots if the moisture content is 18 percent or less. In some seasons when weather conditions do not permit proper drying, considerable mold may develop on ears in the field. Such corn should be artificially dried with crib driers to a moisture content low enough to stop mold growth.

Kernel disorders actually are not diseases, but because they provide opportunities for fungi to invade kernels they are closely related to corn pathology.

“Popped kernel” is a disorder appearing as irregular breaks in the seedcoat over the crown of the kernel (fig. 3, *A*). The kernels appear as partially expanded pop-

corn kernels. This disorder is more common on a few inbred lines than on hybrids.

"Silk-cut," another disorder, is characterized by a horizontal cut or split in the seedcoat over the sides of the kernel (fig. 3, B). It has nothing to do with the silk of the ear, but it seems to be associated with certain inbred lines and their combinations.

Both "popped kernel" and "silk-cut" are caused by weak seedcoats, which appear to be unable to expand as the kernels develop and consequently break at the weakest point. The rupturing of the seedcoat exposes the starch of the endosperm, which often becomes overrun with fungi, particularly *Fusarium moniliforme*.

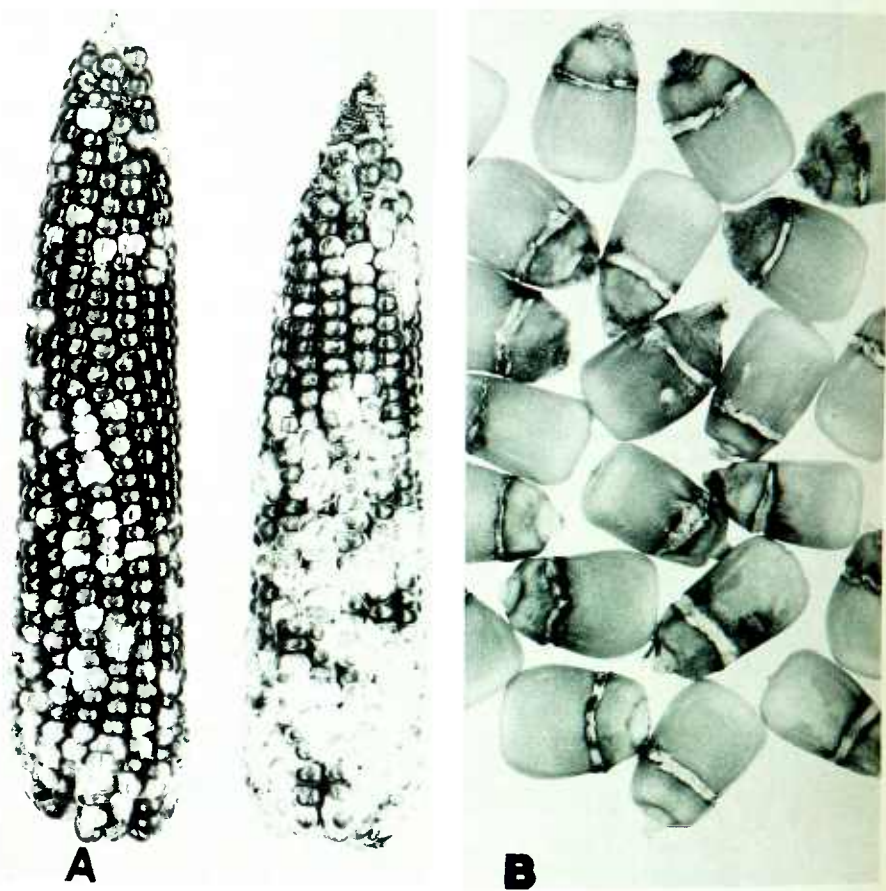


FIGURE 3.—Kernel disorders: A, "Popped-kernel"; B, "silk-cut." These are due to weakness of the seedcoat. Such breaks expose the starch and predispose kernels to infection by molds.

LEAF DISEASES

Certain leaf diseases of corn have increased in economic importance in the United States since 1940. During this period there were several seasons when one or

more of these diseases were severe. The economic importance of some of these diseases was almost coincident with the use of hybrid corn. However, this fact

does not mean that hybrid corn is necessarily inferior to open-pollinated corn in resistance to certain leaf blights.

Several reasons may account for this apparent association of the prevalence of some leaf blights and the use of hybrid corn. (1) During the late 1930's and early 1940's there were several years when weather conditions favored the development of both northern corn leaf blight and southern corn leaf blight in the eastern Corn Belt. This was at a time when hybrid corn was just being introduced into this area and there was much interest in the crop. (2) Hybrid corn is genetically much more uniform than open-pollinated corn. Consequently, when a disease attacks, the plants all tend to react similarly. Whereas open-pollinated corn, although not highly resistant, is much more heterogeneous, and the degree of resistance to diseases may vary from one plant to another.

Leaf blights vary in prevalence and severity from year to year and from one locality to another, depending largely on environmental conditions. Humid weather along with heavy dew favors the spread and development of leaf blights caused by fungi. Leaf blights can be found on corn in both poor and rich soil. Soil fertility does not seem to affect these diseases as much as weather conditions and genetic constitution of the plants.

Northern Corn Leaf Blight

Northern corn leaf blight is found in most humid areas where corn is grown. In the United States it frequently occurs in the eastern Corn Belt and extends to the Atlantic coast and southward. Under favorable conditions it may occasionally become locally severe in the northern and central Corn

Belt. The time when the disease first appears is determined largely by weather conditions. In some years it may be found before silking, whereas under less favorable conditions, such as hot, dry weather, there may be no trace of it. In southernmost Florida where temperature and humidity favor growth and reproduction of the fungus during most of the year, infection may become established on seedlings.

Reductions in grain yield may exceed 30 percent or more if the disease becomes well established 2 to 3 weeks after silking. The later the disease appears, the less yield is reduced. In addition to losses in yield during a severe epidemic, the feed value of fodder is lowered and plants become predisposed to stalk rot.

Symptoms.—Northern corn leaf blight is recognized by long, elliptical, grayish-green or tan spots on the leaves. When fully expanded the spots may be 1½ by 6 inches (pl. 6, A). These lesions appear first on the lower leaves, and as the season progresses the number increases until, under severe conditions, nearly all the leaves are covered with them and little green tissue remains. Under such circumstances the plants appear dead and gray, as though injured by frost. Many spores of the fungus are produced on the lower surface of the lesions under humid conditions. Often the spores are arranged in concentric zones, and a faint targetlike pattern in the lesions is evident. Lesions may form on the husks. Since the kernels are not attacked, the possibility of distributing the disease by seed is remote.

Causal Organism.—*Helminthosporium turcicum* Pass. is the fungus causing northern corn leaf blight. The fungus and the disease were first described in Italy in

1876. The fungus overwinters on infected corn leaves, and during the following summer spores are formed on the mycelium located in and on old lesions. The newly formed spores are carried by the wind to growing corn leaves where, if moisture is present, they germinate and penetrate and thus establish the disease. The fact that spores are carried for some distance during the summer may account for isolated epidemics in the northern Corn Belt. Germination and penetration take place within 6 to 18 hours when water is on the leaves and the temperature is within 65° to 80° F. Lesions are recognizable 7 to 12 days after infection, and new spores are formed on them to continue the disease cycle. Asexual spores are long and spindle shaped and have one to nine cross walls. A sexual stage, *Trichometasphaeria turcica* Luttrell, has been described, in which small black perithecia bearing ascospores were produced. This stage was obtained in pure culture in the laboratory by mating appropriate isolates of the fungus.

Besides corn the fungus also attacks sorghum (*Sorghum vulgare* Pers.), sudangrass (*S. sudanense* (Piper) Stapf.), johnsongrass (*S. halepense* (L.) Pers.), and teosinte (*Euchlaena mexicana* Schrad.). Cross inoculations with isolates from these hosts suggest the presence of physiologic races within the fungus. No differential pathogenicity has been found among isolates from corn. This fact makes the work of the corn breeder less complicated, because he does not need to breed for resistance to different races of the fungus.

Control.—The most effective and efficient means of controlling northern corn leaf blight is to use resistant hybrids. Resistant hy-

brids adapted to southern Indiana, Illinois, and Ohio are available. Other resistant hybrids of different adaptation are being developed.

Resistance to this disease is determined by a large number of genetic factors, or genes, but it is possible through breeding to transfer such resistance to susceptible but otherwise desirable inbred lines for use in hybrid combinations. The amount of resistance in a double-cross hybrid is generally proportional to the number of blight-resistant inbred lines in the pedigree.

To control the disease on sweet corn in southern Florida, the plants are sprayed with a fungicide. This practice is economically feasible because of the premium paid for the crop. Seed treatment or crop rotation has no effect on the disease.

Southern Corn Leaf Blight

Southern corn leaf blight, like northern corn leaf blight, is found wherever corn is grown under humid conditions. It is confined chiefly to the Southeastern United States, but it does occur in Missouri, Illinois, Indiana, Ohio, and eastward to the Atlantic coast. The disease thrives at slightly higher temperatures than northern corn leaf blight, and consequently it is not found so far north as the latter.

Symptoms.—Typical lesions of this leaf blight are parallel sided and grayish tan, ranging from minute spots up to ½ by 1½ inches (pl. 6, B). The tips of ears and the silks of sweet corn may be attacked during an extremely heavy infestation. Similar infections have not been observed in dent corn.

Causal Organism.—The disease is caused by the fungus *Helminthosporium maydis* Nisik. & Miy-

ake. Spores are long, narrow, and slightly curved; they have from 3 to 13 cross walls. A sexual stage, *Cochliobolus heterostrophus* Drechs., bears long threadlike ascospores in black flask-shaped perithecia. Since the sexual stage is found rarely in nature, it is doubtful whether it functions in propagating the fungus. No specialized pathogenic races of the fungus are known, although the disease has been reported to attack teosinte.

Control.—Like northern corn leaf blight, the control of this disease is best accomplished by using resistant hybrids. The few released hybrids previously referred to as resistant to northern corn leaf blight also are highly resistant to southern corn leaf blight. The disease is controlled

in southern Florida by spraying with a fungicide. Resistance is determined by several genes, which appear to be independent of those governing resistance to northern corn leaf blight. The resistance of a double-cross hybrid is proportional to the number of resistant inbred lines in the pedigree.

Helminthosporium Leaf Spot

Helminthosporium leaf spot is of minor economic importance. It is encountered on a few susceptible inbred lines. Farmers are not troubled with the disease, because it does not occur on commercial single- or double-cross hybrids. There are two physiologic races of the fungus: Race I is highly virulent and specialized in its parasitism to a few inbred

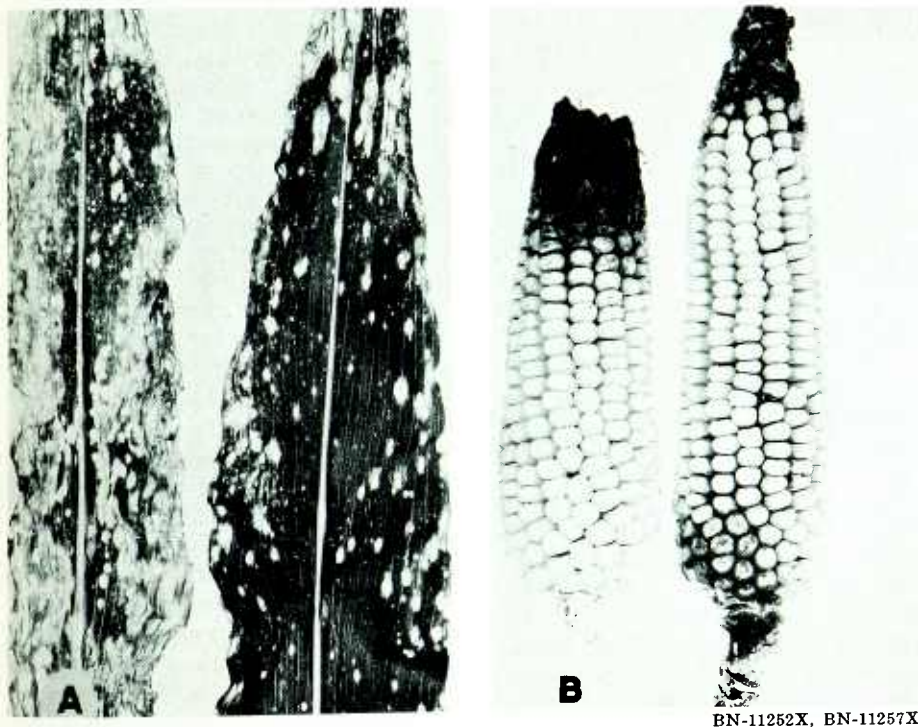


FIGURE 4.—A, Leaf lesions typical of those caused by race I of *Helminthosporium carbonum*; B, corn ear infection typical of that caused by race I or race II of this fungus.

lines of corn; race II is much less virulent and shows no distinct host specialization. Humid weather favors rapid spread of the disease.

Symptoms.—The disease caused by race I of the pathogen is recognized by tan, oval to circular lesions, ranging from very small spots up to $\frac{1}{2}$ by 1 inch (fig. 4, A). A pattern of concentric zones is often evident in the lesions. Under ideal conditions lesions become abundant and the fungus produces spores profusely on leaf sheaths. The ears are also readily attacked and have a black felty mold over the kernels and the ear appears charred. Symptoms produced by race II on field-grown plants are oblong chocolate-colored spots ranging from very small up to $\frac{1}{4}$ by 1 inch. The ear rot caused by race II is indistinguishable from that caused by race I (fig. 4, B). The leaf disease caused by race II is very rarely found.

Causal Organism.—*Helminthosporium carbonum* Ullstrup is the fungus responsible for the disease. Asexual spores are olive brown, long-elliptical to spindle shaped, with rounded ends bearing from 2 to 12 cross walls. The two races of the fungus are alike in cultural behavior, size, shape, and color of spores, but they are distinguished only on the basis of symptoms produced on corn leaves and the specialization in parasitism of race I to a few inbred lines. A sexual stage of the fungus, *Cochliobolus carbonus* Nelson, has been produced in pure culture by growing in association appropriate mating types of the fungus. The perithecia and ascospores resemble those of *C. heterostrophus* in size, shape, and color.

Control.—This disease can be easily controlled by using resist-

ant inbred lines. Since a single gene determines resistance, it is rather simple to incorporate resistance into a susceptible line by breeding methods.

Bacterial Wilt

Bacterial wilt, sometimes referred to as Stewart's disease, is widespread over the Eastern United States. It is much more severe on sweet corn than on dent corn.

This disease, unlike the three previous fungus diseases, does not require damp weather and heavy dew for spread and development. Bacterial wilt is carried through the winter within the bodies of corn flea beetles. When these insects come out of hibernation late in the spring, they feed on young corn plants and thus start infections on the leaves. During the growing season the beetles continue to spread the disease from infected to healthy plants. The prevalence of bacterial wilt varies from year to year depending on the number of corn flea beetles that survive the winter. When winters are mild, usually large numbers of beetles survive and thus start infections and spread the disease during the growing season. Cold winters reduce the number of beetles, and consequently there is little primary infection and spread of the disease is retarded.

Symptoms.—In sweet corn, susceptible hybrids wilt rapidly and resemble plants with an inadequate water supply (pl. 6, C). Infected plants that do not die are stunted and may produce no ears. Diseased plants frequently show long, irregular, pale-green to yellowish streaks on the leaves. These streaks become dry and brown. The vascular bundles in the plants become filled with bac-

teria. When the stalks are cut, yellowish masses of bacteria ooze out on the cut surface. If small pieces of infected leaf tissue are placed in a drop of water and observed under a microscope, masses of bacteria will be seen flowing out from the cut ends of the vascular bundles. In severely infected plants cavities may form in the pith of the stalk. In such plants the bacteria spread throughout the vascular system, passing through the cob into the kernels. Infected kernels may thus be a means of spreading the disease to new localities.

Dent corn is generally much more resistant than sweet corn and seldom does the disease spread through the entire plant, except in a few very susceptible inbred lines, and then the symptoms are as described for sweet corn. In dent corn the characteristic symptoms on the leaves are the long, irregular, pale-greenish streaks that turn yellow, then die, and become straw colored (pl. 6, *D*). This is known as leaf blight or late-infection phase, since it generally becomes prevalent after tasseling. These dead, dry lesions sometimes become covered with saprophytic fungi, which mistakenly may be assumed to cause the disease. The long streaks always originate at the feeding wounds of corn flea beetles. Where infection is severe, much of the leaf area may be destroyed, the yield is reduced, and the plants become more susceptible to stalk rot. The infection of dent corn kernels is exceedingly rare and occurs only where the disease is most severe on susceptible inbred lines.

Causal Organism. — Bacterial wilt is caused by a species of bacterium, *Xanthomonas stewartii* (E. F. Sm.) Dows. The cells are short, nonmotile rods. Colonies on nutrient agar are small, slow growing, and pale yellow.

Control. — The most practical way to control this disease is to use resistant hybrids. The hybrid Golden Cross Bantam was the first sweet corn developed for resistance to this disease. It is widely adapted over most of the eastern Corn Belt. Several other sweet corn hybrids have been developed that combine resistance to bacterial wilt with high yield and quality. In both sweet and dent corn the earlier and shorter inbred lines appear to be more susceptible than the later and taller inbred lines. In sweet corn, Country Gentleman and Evergreen are generally more resistant than the Bantam types.

Early spraying or dusting plants with an insecticide to kill corn flea beetles is partly effective in reducing spread of the disease. There is some indication that adequate levels of potassium in the soil tend to minimize the disease, whereas high levels of nitrogen predispose plants to susceptibility. Seed treatments or spraying plants with fungicides has no effect on controlling the disease.

In dent corn there appears to be a positive correlation between resistance to the late-infection phase of this disease and resistance to northern corn leaf blight.

Studies conducted several years ago on bacterial wilt indicate that two major genes and one minor gene operate in determining resistance to the disease.

MINOR LEAF DISEASES

Several fungi and bacteria attack corn leaves but are of little economic importance, either be-

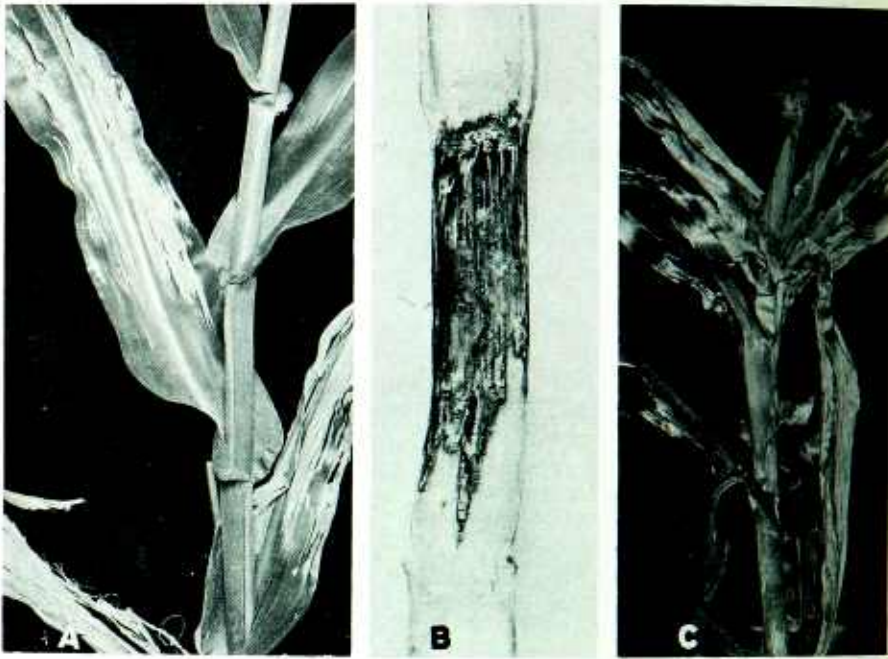
cause they rarely occur or because they do little damage. They will be treated briefly.

Bacterial Leaf Blight and Stalk Rot

Bacterial leaf blight was first described about 1930 as occurring in some of the southern and central areas of the United States. The disease seems to be favored by rather high temperature. It

ding, especially after wind and rain.

Stalk rot usually occurs just above the point where the ear is attached and causes a dark-brown rot of the pith (fig. 5, *B*). As the rot progresses, the tops of the plants die. Affected plants are dwarfed and often develop mul-



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FIGURE 5.—Bacterial leaf blight and stalk rot: *A*, Leaf lesions; *B*, stalk rot, showing discoloration of internode; *C*, multiple ear formation on a plant affected with bacterial leaf blight and stalk rot.

has been of minor economic importance.

Symptoms.—The disease may appear in localized areas in the field. Leaf lesions range from small elliptical spots to long narrow stripes, extending nearly the full length of the leaf (fig. 5, *A*). These lesions often coalesce and affect most of the leaf width. At first the lesions are olive green and water soaked and later become tan and dry. Badly diseased leaves show characteristic shred-

ded leaves, which are usually sterile (fig. 5, *C*).

Causal Organism.—The bacterium causing the disease is *Pseudomonas alboprecipitans* Rosen. The cells are short, rod shaped, and motile. The organism was first described on foxtail grass (*Setaria* spp.), where it causes a leaf disease.

Control.—Because of its minor economic importance, no control measures have been developed for this disease.

Bacterial Stripe

Bacterial stripe seems to thrive during and after extended periods of warm, wet weather. Lesions

and is the same organism responsible for bacterial stripe of *Sorghum* species. This disease is not of economic importance and has



BN-11256X, BN-11250X

FIGURE 6.—Bacterial stripe: A, Typical narrow linear lesions on lower leaves; B, striped bleached leaves, which are not infected, develop after severe infection on lower leaves.

are long, narrow, generally parallel sided, olive to amber colored, and water soaked (fig. 6, A). They first appear on the lowest leaves. On very susceptible inbred lines most of the leaves below the ear may be killed by the disease. Under such conditions where infection is severe, the upper leaves at first have pale-yellow or greenish-white stripes. The stripes enlarge, coalesce, and continue to become lighter until the upper leaves are almost completely white (fig. 6, B). These bleached leaves are not infected but show a secondary effect of infection on the lower leaves. *Pseudomonas andropogonis* (E. F. Sm.) Stapp is the bacterium causing this disease

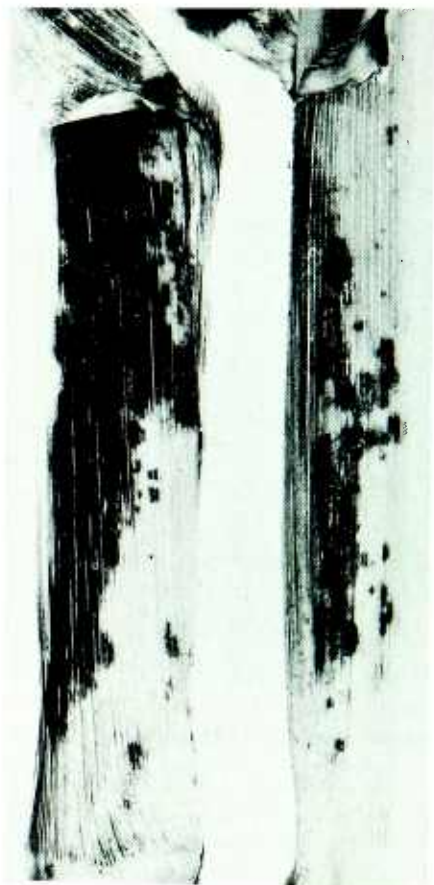
been acute only on a few very susceptible inbred lines of corn.

Zonate Leaf Spot

Zonate leaf spot is found in the Gulf States. Lesions are reddish brown and water soaked. As they enlarge up to 1 to 2 inches in diameter, a very definite target-like or zonate pattern appears. The disease occurs on sorghum, sudangrass, johnsongrass, and sugarcane (*Saccharum officinarum* L.), as well as on corn. The causal organism is the fungus *Gloeocercospora sorghi* D. Bain & Edg., which produces numerous long, narrow, needlelike spores in a slimy matrix on the surface of the lesions.

Purple Sheath Spot

Purple sheath spot is rather widespread, but as far as can be determined it causes no measurable damage to plants. Purplish-brown irregular spots of varying sizes become conspicuous on the leaf sheaths, usually after silking



BN-11280X

FIGURE 7.—Purple sheath spot.

(fig. 7). Beneath these spots the inner surface of the leaf sheath shows a discoloration and breakdown of the tissue. Inbred lines

differ considerably in their reaction to this disease. Investigations have shown that several fungi and bacteria live saprophytically on debris that collects behind the leaf sheaths and become mildly parasitic as the host ages.

Holcus Spot

Holcus spot attacks the leaves of several grasses, including fox-tail millet (*Setaria italica* (L.) Beauv.), sudangrass, johnsongrass, and some varieties of sorghum, in addition to corn. The lesions are first dark green and water soaked; later they become dry and brown with a reddish margin. They are round to elliptical and range from small spots up to about one-half inch in diameter. The causal organism is the bacterium *Pseudomonas syringae* v. Hall, which invades its hosts through stomata.

Cercospora Leaf Spot

Occasionally cercospora leaf spot is found on corn leaves as they approach maturity. The spots are gray to tan and narrow, ranging from small spots up to $\frac{1}{4}$ by 2 inches. The causal organisms *Cercospora zea-maydis* Tehon & Daniels and *C. sorghi* Ell. & Ev. differ slightly in the size of spores and in the symptoms they produce.

Anthracnose

Anthracnose is rarely found in the United States. Localized outbreaks have occurred under extremely humid conditions. Small, oval, tan spots on the leaves are the characteristic symptoms caused by *Colletotricum graminicola* (Ces.) G. W. Wils.

BROWN SPOT

Brown spot, or physoderma disease, of corn is often prevalent in the Southeastern United States, although it has been reported as

far west as Kansas and north to South Dakota. Warm, humid weather favors its development and spread.

Symptoms.—Initial symptoms occur mainly below the ear on leaf blades, leaf sheaths, and stalks. Lesions first appear near the base of the leaf blade as yellowish spots that later turn brown (pl. 7, A). Spots coalesce to form large blotches. Infections on the leaf sheath resemble purple sheath spot (pl. 7, B). Stalks become infected at the nodes beneath the sheaths. Cells of infected tissues disintegrate and expose pustules containing the reddish-brown sporangia of the fungus. In severe infection stalks break at the nodes and leaf sheaths below the ear (pl. 7, C) and most of the leaves die prematurely (pl. 7, D).

Causal Organism.—The fungus causing brown spot, *Physoderma maydis* Miyabe (*P. zae-maydis* Shaw), is relatively simple in structure. Sporangia, which are produced abundantly in the pustules, are golden brown, smooth,

thick walled, and generally globe shaped except for a flat area on one side. When sporangia germinate, a cap or lid opens to release a vesicle, which ruptures and liberates several uniciliate zoospores. These motile cells require water on the leaf surface to move about. They come to rest, round up, and penetrate the host. Germination of sporangia requires water and a temperature range from 73° to 85° F. The fungus overwinters in the sporangial stage on infected tissue.

Control.—Marked differences exist between inbred lines in resistance to this disease. Where brown spot is troublesome, State agricultural experiment stations should be consulted for recommended resistant hybrids. Crop rotation and sanitation are ineffective in practical control of the disease.

CORN RUSTS

In the United States two rusts are found on corn—common corn rust, which is almost universal where corn is grown, and southern corn rust, which is confined generally to the southeastern areas. Tropical corn rust is found in some of the tropical areas of the Western Hemisphere. Common corn rust generally does not cause much damage to the plant, although exceptions have been noted, especially in sweet corn, where severe infection reduced yield. Southern corn rust has never been reported as severe in the United States, but in parts of Africa the disease has caused heavy yield losses.

Common Corn Rust

In the Corn Belt common corn rust often appears soon after silking, although in some years it may appear much earlier. Cool,

humid weather favors development of this disease.

Symptoms.—This rust is recognized by oval to elongate cinnamon-brown pustules scattered over both surfaces of the leaves (pl. 8, A). As corn matures, the pustules become brownish black owing to the replacement of the reddish summer spores, or urediniospores, by black winter spores, or teliospores. The pustules may appear on any of the above-ground parts of the plant, but they are most abundant on the leaves. The pustules of this rust, or uredinia and telia, break through the epidermis early in their development. This one characteristic differentiates this rust from southern corn rust.

Causal Organism.—*Puccinia sorghi* is the fungus causing common corn rust. The urediniospores are globose, finely echinu-

late, and cinnamon brown. They are carried by the wind and under favorable temperature and moisture conditions germinate and penetrate corn. Teliospores are two celled, brownish black, smooth, thick walled, and ellipsoid. Each spore is attached to a pedicel twice the length of the spore. Teliospores germinate after a ripening period—usually over winter—to form small basidiospores, which are delicate, thin walled, and single celled. These spores cannot infect corn, but they parasitize species of *Oxalis*, where the pycnial and aecial, or cluster-cup, stages arise after infection by the basidiospores. On the upper side of *Oxalis* leaves pycnia are formed. They contain minute spores, or spermatia. Fusion of these with receptive structures of the opposite mating type initiates the development of aeciospores in "cluster cups" on the lower surface of *Oxalis* leaves. The aeciospores are globoid to ellipsoid, finely verrucose, and pale yellow. They are carried by the wind to corn leaves, where they start infections that again give rise to the urediniospores. In the Central and Southern United States the urediniospores may overwinter and serve to initiate infection in the spring, thus bypassing the necessity of the alternate host *Oxalis*.

Control.—In the United States there has been no urgent need to control common corn rust. Most inbred lines are susceptible, but a few have shown resistance to one or more physiologic races of this rust. One collection of the Cuzco variety from Peru appears to be highly resistant to all cultures of *P. sorghi*. Since resistance is determined by a single dominant gene, transfer of such resistance to agronomically de-

sirable inbred lines should be relatively simple.

Southern Corn Rust

Southern corn rust was first reported in the United States from Massachusetts in 1879. Since then it has been found in several Southern States and as far north as Wisconsin. It is frequently encountered in the warm, humid areas of Central and South America. It has become economically important in parts of Africa and has been found in several countries in the Eastern Hemisphere. Southern corn rust apparently requires somewhat higher temperatures than common corn rust.

Symptoms.—The uredinia of this rust, which occur on both surfaces of the leaves, are definitely lighter in color, somewhat smaller, and more circular than those of common corn rust. The epidermis remains intact over the pustules for a longer time than in common corn rust. Telia are chocolate brown to black and often in circles around the initial uredinia (pl. 8, B). They are easily distinguished from those of common corn rust, because they are smaller and the leaf epidermis over their surface is retained for a longer time.

Causal Organism.—*Puccinia polysora* Underw. is the causal organism of southern corn rust. Urediniospores are moderately echinulate, yellowish to golden, globose to ovoid, and slightly larger than those of *P. sorghi*. Teliospores are chestnut brown to black, angular, ellipsoid, and slightly shorter and thicker than those of *P. sorghi*. The spores are two celled, the apical wall is only slightly thickened, and they are borne on a short pedicel one-fourth or less the length of the spore. No alternate host for *P. polysora* has been reported; con-

sequently, only the uredinial and telial stages are known.

Control.—This disease has not become sufficiently important economically in the United States to develop resistant hybrids. However, in Africa, where it has become a real problem, resistant hybrids have been produced. Two physiologic races of the rust have been reported there. A single dominant gene determines resistance to one of these races.

Tropical Corn Rust

Tropical corn rust is found only in the warm, humid parts of the Western Hemisphere and has not been reported in the United States. The uredinia that occur

mainly on the upper leaf surface are oblong, pale yellow, and covered by the epidermis, except for a small pore or slit. The telia are chocolate brown, encircle the uredinia, and remain covered by the leaf epidermis for a long time. The causal organism is *Physopella zae* (Mains) Cummins & Ramachar (*Angiopsora zae* Mains). Urediniospores are ovoid, colorless to yellow, and moderately echinulate. Teliospores are golden brown, one celled, oblong, angular, and borne in sessile chains of two spores. No alternate host has been found. Nothing is known about this rust as to inheritance of resistance.

DOWNY MILDEWS

Seven downy mildew diseases are known to attack corn. Only crazy top and downy mildew occur in the United States. The others are found in the Eastern Hemisphere, particularly in the Philippines, Indonesia, Southeast Asia, India, and Africa. All these diseases are caused by closely related species of *Sclerospora* and *Sclerophthora*. The symptoms are somewhat similar. They are characterized by chlorotic streaking, mottling, stunting, malformation of tassels and ears, and excessive tillering. Warm, very humid weather favors those downy mildew diseases found outside the United States.

Crazy Top

Crazy top is widespread over the United States. It has been reported from New York to California and from Wisconsin to Texas. However, it is seldom so prevalent as to cause any appreciable damage. The primary environmental factor determining the

presence of the disease is flooding or waterlogging of the soil during the period from germination of the kernels until seedlings are 4 to 6 inches high.

Symptoms.—The most conspicuous symptom characterizing this disease, and from which its name is derived, is the partial to complete proliferation of the tassel (pl. 8, C). Instead of the normal floral structures, these parts continue to grow and take on the shape of small leaves so that the tassel looks like a large plummy mass (fig. 8, A). Excessive ear shoots and an increased number of internodes above the ear and in the shank are characteristics of this disease. Pronounced stunting, narrow straplike leathery leaves, excessive tillering, and complete suppression of tassel and ear formation are typical symptoms of severely infected plants (fig. 8, B).

Causal Organism. — *Sclerophthora macrospora* (Sacc.) Thirum. (*Sclerospora macrospora* Sacc.) is the fungus causing crazy top



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FIGURE 8.—A, Typical crazy top symptoms; B, severe infection by *Sclerophthora macrospora*, showing complete suppression of tassels and ears and the narrow straplike leaves.

of corn. Sexual spores, or oöspores, are relatively large, thick walled, and yellowish. These presumably germinate to form sporangia, the asexual phase of the fungus. Sporangia, which are exceedingly rare on corn in the United States, are lemon shaped and germinate by releasing kidney-shaped biciliate zoospores. Little is known regarding the mode of infection, but it is likely that penetration takes place by zoospores, which are released when oöspores germinate in saturated soil. The parasitic mycelium is completely systemic. Oöspores are formed in great abundance in leaf sheaths and in the straplike narrow leaves of badly infected plants.

Sclerophthora macrospora, like all fungi causing downy mildews, is an obligate parasite. A large number of wild grasses are at-

tacked by this fungus. These hosts may be a means by which the parasite maintains itself in the absence of corn.

Control.—The most direct means of control is to provide proper soil drainage. Nothing is known concerning the relative resistance of hybrids. Seed treatment has no effect in controlling this disease.

Downy Mildew

Downy mildew is exceedingly rare. It has been reported on corn only twice in the United States. Grayish blotching and mottling of the leaves, together with chlorotic streaking and stunting, are symptoms on corn. The fungus *Sclerospora graminicola* (Sacc.) Schroet. attacks foxtail grass (*Setaria* spp.) occasionally. The size and shape of spore forms, although

somewhat similar to those of *Sclerophthora macrospora*, are sufficiently distinct to separate

the two species. The disease is of no economic importance in the United States.

VIRUS DISEASES

The virus diseases of corn that occur in the United States are corn stunt, sugarcane mosaic, celery stripe, and leaf fleck. They have been of little economic importance, and none occur in the Corn Belt. In certain tropical areas of the world some virus diseases of corn have become economically important.

Corn Stunt

Corn stunt was first found in California and later in the Rio Grande Valley of Texas. A distinctive symptom of this virus disease is the abnormal growth from the nodal buds below the ear. This growth gives a bushy appearance to the plants (pl. 8, D). These lateral branches may become 2 to 3 feet long. Where infection takes place early, all the internodes of the stalk are shortened so that the plant appears stunted. In later infections only the internodes above the ear are shortened. Leaves are light green and streaked with pale blotches. Often the tips and margins are reddish bronze. Several ears are frequently produced, but these are small and often only partially filled. The disease is transmitted by at least two leafhoppers — *Dalbulus maidis* (DeL. & W.) and *D. elimatus* (Ball). It does not seem to be transmitted through the seed. There are marked differences among inbred lines in resistance to the disease.

Sugarcane Mosaic

Sugarcane mosaic is sometimes found in the Gulf States on corn

when grown near sugarcane. Streaking and mottling of the leaves, together with partial or complete sterility of the ears, are the characteristic symptoms. The virus has a wide host range within the grass family. It is transmitted by the corn leaf aphid (*Rhopalosiphum maidis* (Fitch)) and two additional species of plant lice. The disease is not seedborne. With the introduction of resistant varieties of sugarcane, the incidence of the disease on corn has been reduced to a level where it is no longer of economic importance.

Celery Stripe

Celery stripe is a virus disease on corn in Florida. Longitudinal light-green streaks on the leaves and marked stunting constitute the main symptoms of the disease. Darkening of the vascular bundles in the stalk has been observed in old infected plants. The virus has a rather wide host range, among which are a number of wild plant species. The cotton or melon aphid (*Aphis gossypii* Glov.) is a natural vector of the virus. There is no evidence that the disease is of appreciable economic consequence.

Leaf Fleck

Leaf fleck is a virus disease of corn, which has been reported from California. Small pale spots that later become yellowish and ultimately affect the entire plant are the main symptoms. A single species of a wild grass, hardinggrass (*Phalaris tuberosa* var. *stenoptera* (Hack.) Hitchc.), is

the only other host. The disease is not seedborne and of no eco-

nomie importance. Three species of aphids can spread the disease.

CORN SMUTS

Corn is attacked by two smuts. Common smut, or boil smut, is widely distributed over the world and is present in nearly every area where corn is grown. Head smut of corn is of minor economic importance in the United States. It is found mainly in some of the Western States and rarely east of the Great Plains. Head smut is more prevalent in the Eastern Hemisphere than in the Americas.

Common Smut

Losses from common smut in the United States are variable, ranging from a trace up to about 6 percent in localized areas. It is doubtful whether losses in grain yield exceed 2 percent over very wide areas. However, in sweet corn the losses may be considerably higher. The number, size, and location of galls on the plant affect the amount of yield reduction. Large galls on and above the ear are more destructive than galls below the ear.

The relationship between weather conditions and the prevalence and severity of common smut is not clear. Dry weather generally favors smut. In the western Corn Belt common smut appears to be more prevalent. It is not known if the dry weather generally prevailing in that area predisposes the plants to infection or simply provides better means for the spread of the fungus. The incidence of smut seems to be higher among plants grown in soil especially high in nitrogen and particularly where heavy applications of barnyard manure have been made. Injuries due to hail or incurred during cultivation in-

crease the amount of smut by exposing tissue to invasion by the pathogen. Injuries resulting from detasseling in seed fields increase smut infection at that point of the stalk where the tassel is pulled out.

Symptoms.—The symptoms of common corn smut are usually conspicuous and easily recognized. All aboveground parts of the corn plant are susceptible (pl. 9, A-D). Galls are at first covered with a glistening white membrane. The interior of these galls soon becomes black and powdery, with the membrane rupturing to expose the sooty mass of spores. Galls on leaves seldom develop beyond the size of a pea. They become hard and dry and contain few spores (pl. 9, D). Embryonic tissue is particularly susceptible to attack. It is in areas where such tissue is abundant that galls are most frequently observed (pl. 9, A and B). Early infection may cause death of young plants, but this is very uncommon.

Causal Organism. — *Ustilago maydis* (DC.) Cda. is the fungus causing common corn smut. The chlamydospores, which constitute much of the black sooty galls, are brownish black, thick walled, heavily echinulate, and spherical to oval. They germinate to form a promycelium, on which one-celled, colorless, and very small elliptical basidiospores are borne. These basidiospores, or sporidia, are usually haploid, whereas the chlamydospores are diploid. Germination of the black chlamydospores takes place in water at about 50° to 95° F. Infection may take place by means of infection

hyphae either arising directly from the germinating chlamydo-spore or developing after the fusion of sporidia of the opposite mating types. Infection hyphae are binucleate; haploid hyphae are ordinarily incapable of penetrating the host. Penetration takes place through stomata, wounds, or directly through cell walls. The parasitic mycelium stimulates host cells to increase in size and number. Eventually chlamydospores develop in the mycelium within the galls and soon they are entirely converted to a black, powdery, spore mass.

Control.—The most effective means of control is to avoid very susceptible hybrids. Control through crop rotation and destruction of galls has been recommended, but it is unlikely that such methods would be effective or even practical where corn is extensively grown. In small garden plots the removal of galls may help to reduce the abundance of spores.

Head Smut

Head smut is unimportant economically in the United States and only rarely have local outbreaks in the Great Plains and some of the Western States been of consequence. The disease in sorghum caused by a different physiologic race of the same fungus appears to be more prevalent. Infection may take place over a wide range of soil temperatures. Dry soil is more favorable for infection than wet soil.

Symptoms.—The first symptoms of head smut are seen when tassels and ears appear. These parts may be partly or completely converted to a mass of brownish-black spores. The galls are first covered with a membrane that

soon breaks open to expose the powdery spore mass and vascular bundles of the host. These vascular bundles in the galls are one of the several features distinguishing this disease from common smut. If only parts of tassels and ears are converted to galls, it is not uncommon to find leafy proliferations on these parts. This stimulation of the formation of leaves in place of floral parts does not occur in common smut.

Causal Organism.—*Sphacelotheca reiliana* (Kühn) Clint. is the fungus causing head smut of corn. The chlamydospores are brown to black, thick walled, and generally spherical. They germinate to form a promycelium, on which sporidia are borne. The sporidia are small, colorless, thin walled, and single celled. Penetration of the host follows fusion of sporidia of the opposite mating types. The binucleate infection hyphae arising from such fusions penetrate the young seedling. The parasitic mycelium develops systemically in the host, and galls form after ears and tassels appear. The morphology and life cycle of the fungus are somewhat similar to those of *Ustilago maydis*. Seedling infection and the systemic nature of the parasitic mycelium differentiate head smut from common smut.

Control.—Because of the minor economic importance of the disease and its occurrence outside the main corn-growing areas of the United States, little is known regarding the relative resistance of inbred lines and hybrids. Seed treatment is somewhat effective in destroying externally borne spores on the seed, but it will not protect against infection in smut-contaminated soil. Crop rotation and seed treatment are recommended as partial control practices.



A

BN-11281X



BN-11274X

A, Poor stand of corn resulting from inadequate seed treatment and cold, wet weather after planting. B, Typical symptoms of seed rot and seedling blight; plant on left is healthy.



A BN-11272X



B BN-11267X



C BN-11259X



D BN-11260X

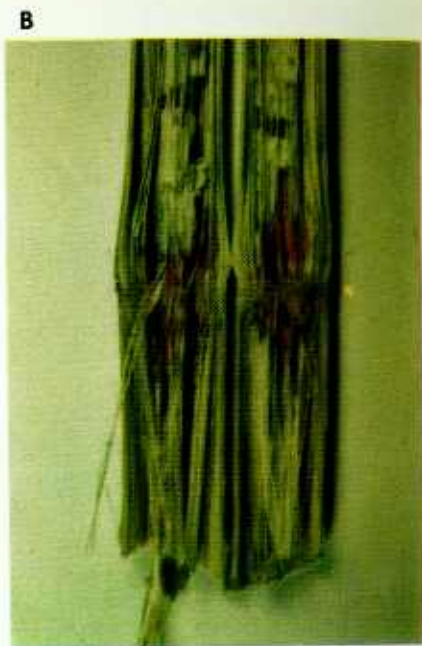


E BN-11269X

A, Premature death due to diplodia stalk rot; compare with healthy green stalks in background. B, Lower internodes of infected plants. C, Same stalk as in B but split open to show disintegration of pith. D, Lower internodes of healthy plant. E, Pycnidia of *Diplodia maydis* on infected stalk.



BN-11277X



BN-11291X



BN-11276X



BN-11270X

A, Crown of plant affected with gibberella stalk rot; B, breakdown and pink discoloration of pith of stalk affected with gibberella stalk rot; C, perithecia of *Gibberella zeae* on diseased stalk; D, pythium stalk rot, showing discoloration and collapse of lower internodes.

A

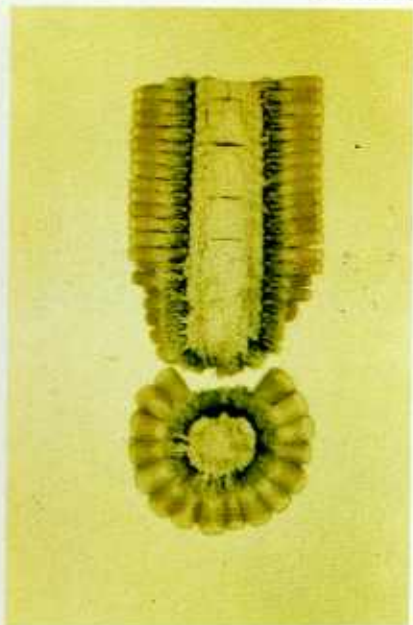


BN-11258X

B



BN-11271X



C

BN-11274X



D

BN-11275X

A, *Diplodia* ear rot, showing bleached husks; B, fusarium kernel rot; C, cob rot with typical shredding and black spore masses at tips of kernels; D, gibberella ear rot.



BN-11268X



BN-11279X



BN-11278X

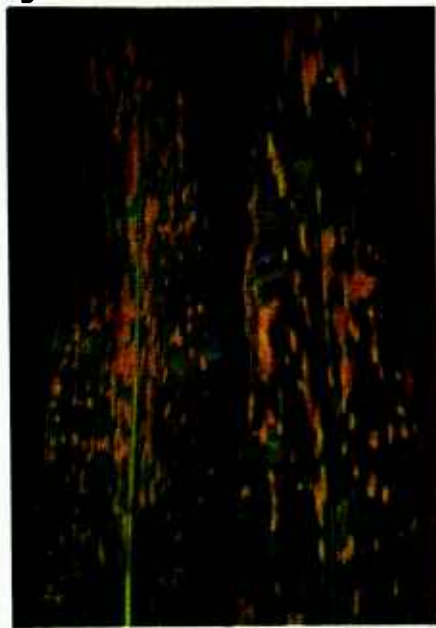


BN-11287X

- A, Discolored husks of ears affected with gray ear rot; B, husked ears, showing typical symptoms of gray ear rot; C, sclerotia, or minute black specks, of causal fungus beneath seedcoat; D, perithecia and pycnidia of *Physalospora zeae* in a leaf lesion.

A

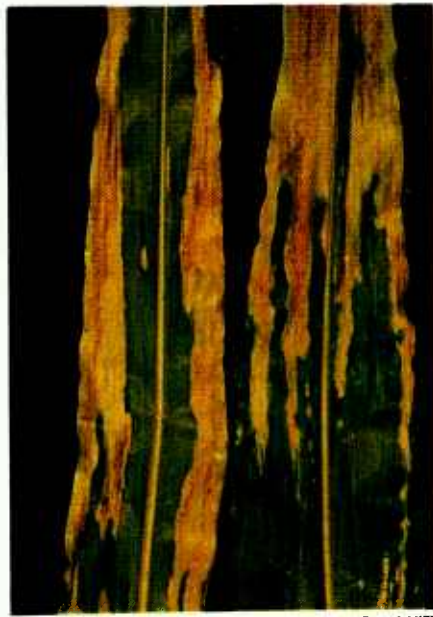
BN-11264X

B

BN-11294X



BN-11263X



BN-11265X

A, Typical lesions of northern corn leaf blight; **B**, narrow parallel-sided lesions characteristic of southern corn leaf blight; **C**, bacterial wilt of sweet corn, showing severe systemic infection; **D**, late-infection phase of bacterial wilt on dent corn.



A

BN-11261X



BN-11266X



C

BN-11293X

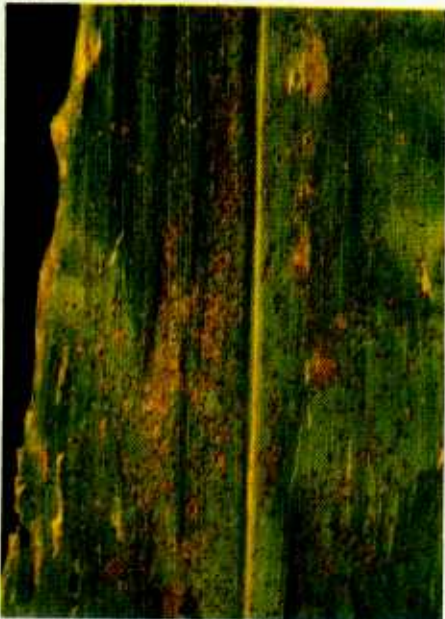


D

BN-11262X

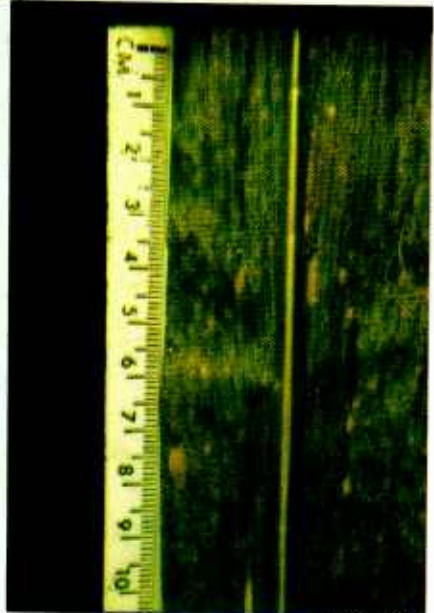
A, Brown spot disease, showing symptoms at base of leaf blades; *B*, symptoms of brown spot on leaf sheaths; *C*, stalk breaking resulting from severe brown spot infection; *D*, dead lower leaves and stalk infection typical of brown spot disease.

A



BN-11289X

B



BN-11290X



C

BN-11289X



D

BN-11282X

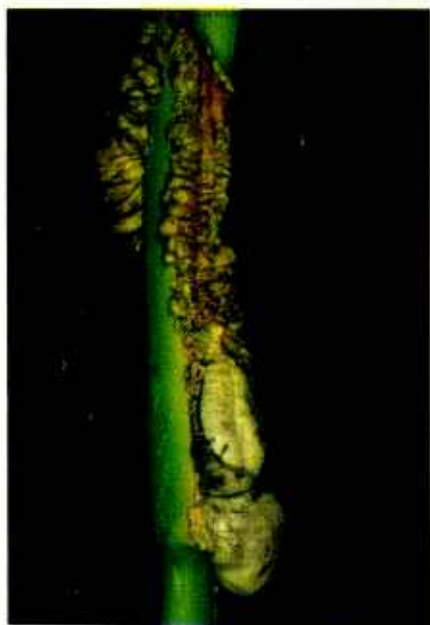
A, Common corn rust, showing pustules rupturing epidermis of leaf. B, Telia of southern corn rust, showing their circular pattern around uredinia; epidermis of leaf remains intact over telia for a long period. C, Leaf proliferation of tassel affected with crazy top. D, Corn stunt, showing stunting, bushiness, and yellowish-green streaking of leaves that typify the disease.



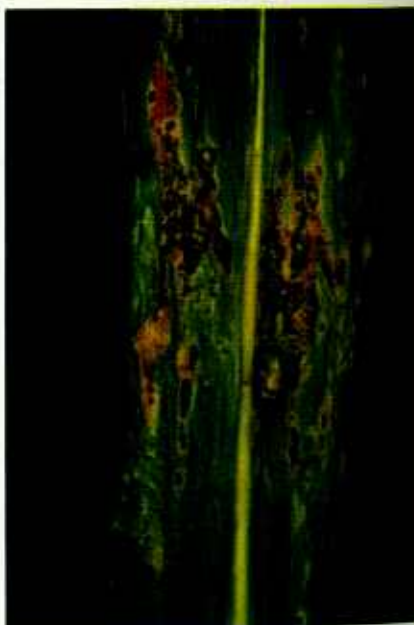
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BN-11286X



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A, Common corn smut on tassel; B, common corn smut affecting ear; C, infection of nodal buds; D, small dried smut galls on corn leaf.