DISEASES OF CORN IN THE MIDWEST
Diplodia seedling blight.

Diplodia ear rot.

Fusarium kernel rot.

Gibberella ear rot.

Aspergillus storage rot.

Fusarium (left) and Penicillium (right) storage rots.
DISEASES OF CORN IN THE MIDWEST

Agricultural Extension Services of Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota, Wisconsin, and U.S. Department of Agriculture cooperating

UNIVERSITY OF ILLINOIS COLLEGE OF AGRICULTURE COOPERATIVE EXTENSION SERVICE

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This circular was prepared by the following Extension plant pathologists, representing the North Central Region: M. C. Shurtleff, University of Illinois; R. C. Lambe, formerly at Iowa State University; D. S. Wysong, University of Nebraska; L. S. Wood, South Dakota State University; and J. L. Weihing, University of Nebraska.

Other Extension plant pathologists who gave leadership to the development of this publication are Dwight Powell, University of Illinois; E. G. Sharvelle, Purdue University; C. L. King and William Willis, Kansas State University; H. S. Potter and Nicky Smith, Michigan State University; H. G. Johnson, University of Minnesota; O. H. Calvert and Einar Palm, University of Missouri; H. L. Bissonnette, North Dakota State University; B. F. Janson and R. E. Partyka, Ohio State University; Gayle Worf and E. K. Wade, University of Wisconsin; and H. E. Smith, Washington, D.C.

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### GENERAL DISEASE SYMPTOMS

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<th>Disease</th>
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</thead>
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<tr>
<td>Stand poor and uneven. Seedlings wilt; may die.</td>
<td>Seed rots and seedling blights</td>
</tr>
<tr>
<td>Stalks break over readily; discolored or hollow inside. Roots decayed.</td>
<td>Stalk rots and root rots</td>
</tr>
<tr>
<td>Kernels, ears, and cobs moldy and rotted.</td>
<td>Ear and kernel rots</td>
</tr>
<tr>
<td>Moldy corn in crib or bin.</td>
<td>Storage rots</td>
</tr>
<tr>
<td>Kernels develop cracks.</td>
<td>Kernel disorders</td>
</tr>
<tr>
<td>Elongate dead areas in leaves.</td>
<td>Leaf diseases</td>
</tr>
<tr>
<td>Small, reddish-brown pustules on leaves.</td>
<td>Common rust</td>
</tr>
<tr>
<td>Silvery galls, filled with black dust, anywhere on plant aboveground.</td>
<td>Common smut</td>
</tr>
<tr>
<td>Plants stunted or dwarfed and bushy.</td>
<td>Maize dwarf mosaic</td>
</tr>
<tr>
<td>Leaves mottled light and dark green, later turning yellowish with red blotches and streaks.</td>
<td>Downy mildew (crazy top)</td>
</tr>
<tr>
<td>Tassel leafy and functionless; plants stunted and bushy. Ears sterile; often numerous and long.</td>
<td></td>
</tr>
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## A Variety of Corn Diseases in the Midwest

Corn in the Midwest may be attacked by a number of diseases that reduce its yield and quality. Yearly losses range from about 7 to 17 percent on the average, but in some localized areas may be much higher. Ear and kernel rots decrease yield, quality, and feeding value of the grain. Stalk diseases not only lower yield and quality but also make harvesting difficult. Leaf damage reduces the production of carbohydrates to be stored in the grain, resulting in immature, chaffy ears.

Corn diseases may be either infectious or noninfectious. Infectious diseases are caused by pathogens—fungi, bacteria, and viruses. Nematodes that feed on the roots of corn make wounds that are later invaded by root-rotting fungi.

Noninfectious diseases result from chemical or mechanical injury, genetic abnormalities, and unfavorable climatic and soil conditions. Nutritional deficiencies or imbalances, too much water, or high or low temperatures may cause symptoms much like those due to infectious organisms.

Unlike some diseases of other crops, corn diseases seldom become severe over wide areas. Up to now, diseases have not limited corn production in any area of the North Central states where soil and weather conditions have been favorable for the crop. Nor has it been necessary to stop growing corn over a wide area because of disease.

The potential now exists, however, for increased severity of corn diseases. One cause is genetic uniformity, typified by single cross hybrids. Another is the intensive cultivation of corn resulting from the recent adoption of continuous cropping, high plant populations, and heavy fertilization to achieve high yields. The high incidence of root and stalk rots in the past few years is largely due to the changes in corn culture.

This circular describes only the infectious diseases that have been recognized in the North Central states. One should not overlook the possibility of inroads by a new disease not yet recognized. The recent flare-up of maize dwarf mosaic illustrates the speed with which a new disease can sweep through an area.

### Estimated Average Corn Production and Disease Losses for the 12 North Central States, 1960-1966

<table>
<thead>
<tr>
<th>State</th>
<th>Acres corn harvested for grain</th>
<th>Acre yield, bu.</th>
<th>Production, bu.</th>
<th>Annual dollar loss from disease $</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ill.</td>
<td>9,252,142</td>
<td>81.9</td>
<td>757,750,430</td>
<td>100,023,057</td>
</tr>
<tr>
<td>Ind.</td>
<td>4,546,857</td>
<td>79.3</td>
<td>360,565,760</td>
<td>47,594,680</td>
</tr>
<tr>
<td>Iowa</td>
<td>10,323,571</td>
<td>77.8</td>
<td>803,173,824</td>
<td>106,018,945</td>
</tr>
<tr>
<td>Kans.</td>
<td>1,245,571</td>
<td>50.4</td>
<td>62,776,778</td>
<td>8,286,535</td>
</tr>
<tr>
<td>Mich.</td>
<td>1,490,428</td>
<td>62.1</td>
<td>95,555,759</td>
<td>12,217,336</td>
</tr>
<tr>
<td>Minn.</td>
<td>4,829,142</td>
<td>63.1</td>
<td>304,718,860</td>
<td>40,222,890</td>
</tr>
<tr>
<td>Mo.</td>
<td>3,027,857</td>
<td>60.0</td>
<td>181,671,420</td>
<td>23,980,627</td>
</tr>
<tr>
<td>Neb.</td>
<td>4,744,571</td>
<td>60.5</td>
<td>287,046,546</td>
<td>37,890,144</td>
</tr>
<tr>
<td>No. Dak.</td>
<td>216,142</td>
<td>34.7</td>
<td>7,500,127</td>
<td>990,017</td>
</tr>
<tr>
<td>Ohio</td>
<td>2,980,857</td>
<td>74.1</td>
<td>220,850,311</td>
<td>29,153,561</td>
</tr>
<tr>
<td>So. Dak.</td>
<td>2,785,142</td>
<td>39.5</td>
<td>110,013,109</td>
<td>14,521,730</td>
</tr>
<tr>
<td>Wisc.</td>
<td>1,592,285</td>
<td>72.5</td>
<td>115,440,663</td>
<td>15,238,167</td>
</tr>
<tr>
<td>All states</td>
<td>47,034,279</td>
<td>63.0</td>
<td>3,304,073,407</td>
<td>436,137,689</td>
</tr>
</tbody>
</table>

* Figures from U.S. Department of Agriculture Statistical Reporting Service, Crop Reporting Board.

** Based on an estimated average annual loss of 12 percent (see U.S. Department of Agriculture Agricultural Handbook No. 291, “Losses in Agriculture,” August 1962); and a price of $1.10 per bushel, the mean paid to farmers for corn, 1960-1966.

### Factors That Affect 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 presence of the pathogen, weather and soil conditions, and relative resistance or susceptibility of the corn. All three factors must be present and “in balance” for disease to develop. This can be put in the form of a simple diagram:

```
Susceptible plant
  /         \
Virulent pathogen  \
  |         |
  |         | Proper environment = DISEASE
```

Even when a disease-causing organism (pathogen) is present and the environment is favorable, little or no disease will develop if the corn hybrid is highly resistant. Similarly, disease probably will not develop if the organism is present and the corn is susceptible but the environment is unfavorable.

### Pathogens

Fungi and bacteria are minute forms of plant life. Those that cause diseases are called pathogens; those that obtain food only from dead plant material are called saprophytes. Most fungi reproduce and spread by spores, which correspond in function to the seeds of higher plants. The right combination of moisture and temperature is necessary for spores to germinate. The germinating spores grow into the living plant through natural openings or wounds. Fungi (though not bacteria) may also enter the plant directly.

Virus particles are complex molecules with physical and biological properties. They enter plants through wounds, which are often made by their insect carriers.

Some pathogens have several strains which differ in their virulence on the same lines of corn. For example, a number of strains or physiologic races of the fungus causing common corn rust are known to exist.
Environment

Many corn diseases develop best when moisture is abundant during the growing season. Rain, irrigation water, or heavy dew is necessary for spores of disease-producing fungi to germinate and penetrate the plant. Certain seed rots and seedling blights are favored by low soil temperatures before emergence. Bacterial wilt is most serious after mild winters. Downy mildew (crazy top) of corn occurs only when the soil is flooded or waterlogged while the seedlings are young. Temperature and moisture of both soil and air thus influence the development of corn diseases.

Soil fertility is another environmental factor that may affect the severity of some corn diseases, particularly certain stalk rots. If the soil is highly productive, it tends to produce vigorous plants. Fertile soil does not necessarily mean healthy corn, however, since some diseases are not affected by soil fertility.

Host resistance

Inbred lines and hybrids differ greatly in their ability to resist various diseases. Disease resistance or susceptibility often determines whether an outbreak of a given disease will occur. Resistance to most corn diseases is determined by one or more genes. These genes can be manipulated by the corn breeder to produce inbred lines and hybrids that combine high levels of disease resistance with other desirable characters.

CONTROL OF CORN DISEASES

At present, the most efficient and permanent way of controlling most corn diseases is to use adapted disease-resistant hybrids. Unfortunately no single hybrid is highly resistant to all diseases. In fact, some common hybrids are extremely susceptible to one or more diseases. Much therefore remains for the corn breeder and plant pathologist to do in developing disease-resistant hybrids. It may be impossible to obtain high resistance to all diseases, but on the basis of past accomplishments it appears possible to develop adapted, high-yielding corn hybrids that are resistant to the major diseases in a given area.

If a disease becomes important enough in your area to warrant control, consult your state agricultural experiment station or cooperative extension service for recommendations as to adapted, resistant hybrids.

Treatment of seed corn with a fungicide, such as thiram or captan, may control seed rots but not other diseases. Considerable progress has been made in improving seed corn treatments, and good fungicides or fungicide-insecticide combinations prepared for this purpose are available. All hybrid seed is now treated by the seedsmen.

Spraying corn with fungicides to control fungal leaf blights has a limited use for breeding nurseries and certain high-value seed-producing fields.

Crop rotation and destruction of diseased crop residues have been suggested as control measures for certain corn diseases. Such practices are most effective if the crop is grown in a limited area or if the specific pathogen is strictly soil-borne. In the North Central states where corn is intensively grown, it is unlikely that diseased plant parts can be destroyed completely enough to eliminate a disease. With a few exceptions crop rotation has little effect in reducing corn diseases. In river-bottom fields where corn has been grown continuously over a period of years, some diseases appear to be no more prevalent than where rotation is practiced. Rotation probably benefits corn more by improving soil tilth and conserving fertility than by reducing diseases.

Maintaining balanced soil fertility can help to lessen effects of some corn diseases. Certain stalk rots and northern leaf blight are often most severe where there is too little potassium and too much nitrogen. 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 needs to be learned about the relationships 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.

Proper seedbed preparation, weed and insect control, maintenance of a favorable pH, and maintenance of good soil drainage may help to control some diseases, although the effectiveness of these measures is limited.

SEED ROTs AND SEEDLING BLIGHTs

The period during which seed germinates and the seedling becomes established is very critical in the life of a corn plant. Severe infection by fungi may kill the embryo before germination. When infection takes place after germination, seedlings may be destroyed before or after emergence. Those that do survive attack are usually lower in vigor and develop into less productive plants than seedlings that have not been infected.

The prevalence of seedling blights varies considerably, partly depending on weather conditions after planting. If the soil is warm and moist, germination will be rapid and the seedling will soon become established, minimizing the danger of infection from soil-borne, disease-producing fungi. Very dry soil or cold, wet soil retards germination and emergence, making the seedling more susceptible to attack. Moreover, a soil temperature below 50° to 55° F. is favorable for most of the organisms causing seedling blights. Plant-
ing too deep increases the opportunity for fungi to attack the young sprouts.

The severity of seed rots and seedling blights is further affected by such factors as the age and condition of the seed and its genetic resistance to invasion by fungi. Even in cold soil, seed will usually germinate and grow if it has a moisture content of 12 percent or less, if it has been stored at a low humidity and at a temperature of about 35°F., and if it is less than 2 years old. Seed stored for 2 or more years often becomes increasingly susceptible to seed decays and seedling diseases, especially if it is planted under unfavorable conditions. Immature or poorly finished seed is almost always susceptible to seedling blight. Mechanical injuries to the seedcoat, such as breaks or cracks that occur during harvesting or processing, afford avenues of entry for soil-borne fungi.

Inbred lines and their hybrids differ in their resistance to seed rots and seedling blights. In general, dent or field corn is more resistant to seedling diseases than sweet corn, and popcorn is most resistant of all. The susceptibility of sweet corn may be due to a thinner pericarp and to the sugary endosperm that is readily available as food for fungi.

Because of better culling of seed ears, widespread use of grain dryers, selection for resistance, and use of effective seed-treatment fungicides, losses from seed rots and seedling diseases have been quite low in recent years.

Symptoms. Although a number of fungi may invade the seed and seedling, the symptoms of seed rot and seedling blight are generally similar. They may range from complete killing of the embryo before germinating to small discolored or rotted spots (lesions) on the roots and lower parts of the sprout (see inside front cover). Lesions on the sprout near the kernel are often brown and sunken; those on the root are discolored and water-soaked. Small feeding roots are often attacked first. Above-ground symptoms are yellowing, wilting, and death of the seedling leaves.

Seedling blight is easily confused with damage from wind and blowing sand, insect feeding, and chemical injury from misapplication of fertilizer or herbicide. Sometimes underground parts must be closely examined to determine the true cause of damage.

Cause. The most important pathogens causing seed rots and seedling blights are species of *Pythium*, especially *P. debaryanum* and *P. graminicola*. The distribution of these strictly soil-borne fungi is largely determined by soil type and cropping history. One or more species may be found in most soils, but populations are likely to be especially large in muck or heavy soils. In general, the species attacking seed and seedlings thrive at temperatures somewhat lower than those favoring rapid germination of corn. The disease is usually severe where soil is poorly drained, cold, and wet.

Some *Pythium* species may also infect large areas of older roots and cause severe lodging.

In addition to *Pythium*, any one of the ear rot fungi (page 11) may also infect seed and seedlings. These fungi are usually seed-borne and are already established in the seed at harvest. One of the most important is *Diplodia maydis*, which often destroys the embryo before germination. *Gibberella zeae*, found in the cooler corn-growing areas, can cause severe seed rot and seedling blight in cold soil. *Penicillium oxalicum* causes seedling blight even in warm soil. This fungus attacks the sprout near the seed. The leaves of seedlings infected with *P. oxalicum* often have wilted, brownish streaks (Fig. 1).

Other fungi sometimes associated with blighted seedlings are *Fusarium moniliforme*, *Rhizoctonia bataticola*, *Nigrospora oryzae*, and *Aspergillus* species. Several other fungi have been found in diseased corn seedlings, but their virulence has not been proved.

Control. Control of seedling diseases starts with adapted, disease-resistant hybrids. The seed should be of high quality without cracks. It is very important that harvesting and processing machinery be adjusted so that the seedcoat receives a minimum of physical injury.

A seed-protectant fungicide should also be used. Fungicides such as captan and thiram protect seed from infection by soil-borne fungi during the critical early stages of germination. No fungicide, however, has much effect on fungi already established in the seed before planting.

The hazard of seed rot and seedling blights can be further reduced by proper seedbed preparation and by correct placement of fertilizer, herbicide, and insecticide in the planting row. Another help is to delay planting until the soil is warm (above 55°F.) and the danger of long cold spells has passed.
STALK ROTS AND ROOT ROTS

Stalk rots and root rots are the most serious and widespread diseases of dent corn in the North Central states. Annual losses in a state often run as high as 8 to 10 percent or more, and losses greater than 25 percent have been reported in some areas. Some stalk rot is present in every field at harvest time.

When conditions favor rapid disease development, infected plants may die several weeks before ears are fully mature, resulting in ears that are poorly filled at the tips and in chaffy kernels. The greatest losses are often due to stalk breakage and root lodging, which make harvesting difficult and cause the loss of many ears on the ground. When rainfall is above normal, fungi soon destroy ears in contact with the soil.

Several different species of fungi cause stalk and root rots, the most common being the fungi causing Diplodia, Gibberella, Fusarium, and charcoal rots. The symptoms of these rots are all similar. They usually appear in plants that are approaching maturity rather than in young, actively growing plants.

Stalk rots may sometimes be caused by Pythium and bacteria. They differ from the more common rots both in symptoms and in time of infection, often attacking plants before silking.

Prevalence of the different stalk rots varies throughout the Midwest as well as from season to season. The reasons for these variations are not completely known. Disease development appears to be favored by dry weather in early summer followed by ample rainfall for two or three weeks after silking in August and September. Anything that interferes with carbohydrate synthesis—such as leaf damage caused by diseases, hail, equipment, or insects—predisposes stalks to infection.

Unbalanced fertility may also affect the severity of stalk rots. These rots commonly occur where soils contain a large amount of organic matter, are very high in available nitrogen, and are low in potassium. Thick plant populations increase the incidence of stalk rots and stalk lodging, especially when plants are under stress from a lack, or imbalance, of nutrients or water.

**Diplodia stalk rot**

**Symptoms.** When immature plants are infected with Diplodia stalk rot, the leaves suddenly die and turn a dull grayish-green somewhat as if they had been injured by frost. The stalk dies a week or 10 days later. The lower part of the stalk turns from green to tan or brownish. The diseased portions of a stalk are easily crushed and break readily in wind and rain. When a diseased stalk is split, the pith is shown to be deteriorated and brown, although the water-conducting strands (vascular bundles) are left intact (see back cover).

After the stalks have died, numerous pycnidia—fruiting bodies of the Diplodia fungus—appear as raised black dots just beneath the surface of the lower internodes (Fig. 2). The pycnidia cannot be scraped off with the thumbnail.

**Cause.** Diplodia stalk rot is caused by *Diplodia maydis* (*D. zeae*). The fungus produces thousands of microscopic spores in small, black, flask-shaped pycnidia that develop on corn plants in the fall or spring following infection. After spores are mature, they ooze from the pycnidia during warm, moist weather. Wind currents carry the spores to healthy plants, where they infect the stalk or ear.

Infection usually starts at the base of plants, spreading into the stalk and roots. Sometimes it occurs at the nodes between the base of the plant and the ear. Although the Diplodia fungus spreads some distance within the stalk, it does not invade the entire plant, and it seldom spreads from the base into the ear (page 11).
Control. Since otherwise high-yielding hybrids vary widely in stalk-rot resistance, it is important to choose a hybrid with strong stalks, particularly if a field has a history of stalk rot. Locally adapted, full-season hybrids are generally more resistant than earlier maturing plants. Resistance to northern leaf blight is being incorporated into commercial hybrids, and may indirectly reduce some stalk-rot damage in the future.

Stalk rotting and breakage tends to increase when corn is grown in heavy stands. The stalks are reduced so much in diameter that a relatively low level of rot development will weaken them to the breaking point. Some hybrids suffer more from plant competition than others. In fields with a history of stalk rot, it may be desirable to reduce plant populations. If heavy stands are planted, it is especially important to choose hybrids that can withstand stalk rot.

The possibility of stalk rot is increased in infertile soil or soil that is low in available potassium while being excessively high in nitrogen. Balanced soil fertility does not eliminate stalk rot, but applying the proper kinds and amounts of fertilizer, according to soil and tissue tests, often minimizes stalk rotting and breakage.

Do not delay harvest beyond the safe moisture level regardless of equipment used, for stalk rot is progressive until harvest. Fields with a high percentage of early-ripened plants should be harvested first, for the early ripening may be due to stalk rot. If disease is developing rapidly, and a picker-sheller and corn dryer are available, it pays to harvest early before severe lodging occurs.

Your state agricultural experiment station, cooperative extension service, or a reliable seed dealer should be consulted about locally adapted, full-season resistant hybrids.

Gibberella stalk rot

Symptoms. Some of the symptoms of Gibberella are similar to those of Diplodia: Leaves on early-infected stalks suddenly turn a dull, grayish green, while the lower stalks soften and turn tan or brownish (see back cover). When Gibberella-infected stalks are split, however, they generally show a pink to reddish discoloration rather than the brown typical of Diplodia. When infection is severe, Gibberella causes a more complete breakdown or shredding of the pith than does Diplodia.

Gibberella can best be identified by its perithecia (small, round, black fruiting structures), which develop on diseased stalks. These differ from the pycnidia of Diplodia in two ways: The Gibberella perithecia develop on the surface of the stalk rather than under the surface (Fig. 3). And they may be easily scraped off with the thumbnail. Also, they are often less numerous than the Diplodia pycnidia.

Cause. Gibberella zeae is the fungus causing Gibberella stalk rot. It has both asexual and sexual spores. The asexual spores, or conidia, are produced on the pinkish-white mold “threads” (mycelium) that grow from diseased plant parts in warm, moist weather. When in the asexual stage, the organism is known as Fusarium graminearum. Sexual spores (ascospores) are produced in the small, black, flask-shaped perithecia that develop on the surface of diseased cornstalks in the late fall or spring following infection. Ascospores are released from the perithecia during warm, moist weather. Wind currents carry them to infect ears or stalks.

Control. Measures are the same as for Diplodia stalk rot.

Fusarium stalk rot

Symptoms. This rot is difficult to distinguish from Gibberella stalk rot. Fusarium rot, however, is more widespread, occurring throughout the entire Midwest and southward into tropical areas. Rotting commonly affects the roots, the base of the plant, and the lower stalk nodes (Fig. 4). Root and stalk rot normally becomes evident soon after pollination and increases in severity as the plant matures.

Reddish lesions, premature ripening, and stalk breakage are the same as in Gibberella stalk rot.

Cause. Fusarium stalk rot is caused by two closely related fungi, Fusarium moniliforme and F. moniliforme var. subglutinans. The fungi are very common in seed and may occur in all parts of the growing corn plant throughout the season. They are inactive in stalk tissues, however, until the plant approaches maturity or is injured. Then plants of susceptible hybrids start to deteriorate. The extent of decay depends primarily upon hybrid susceptibility and, to some extent, environment.

Both fungi produce an abundance of asexual spores (conidia) that appear as a powdery, cottony-pink mold growth on the leaf sheaths and at nodes. Sexual stages may also occur, but are not as common in the Midwest as in more tropical areas. In their sexual stages, the
Fusarium stalk rot has caused the shredding of this lower stalk node. (Fig. 4)

Charcoal rot is characterized by numerous black sclerotia which infest the interior of the stalks (top) and are visible just beneath the stalk surface (bottom). (Fig. 5)

organisms are known as Gibberella fujikuroi and G. fujikuroi var. subglutinans and closely resemble the sexual stage of G. zeae. The sexual spores (ascospores) are produced in small, bluish-black, flask-shaped peri­thecia on the surface of old cornstalks and are released during warm, moist weather.

Control. Measures are the same as for Diplodia stalk rot.

Charcoal rot

Charcoal rot occurs mostly in the hot, dry areas of the Midwest. It is favored by a lack of moisture between tasseling and denting time.

Symptoms. Charcoal rot first attacks the roots of seedlings and young plants, producing brown, water-soaked lesions that later turn black. When the plant approaches maturity, the disease spreads into the base of the plant and the lower internodes of the stalk. Infected stalks may often be recognized by grayish streaks on the surface of low internodes and by the large numbers of minute black bodies (sclerotia) that are always present on the vascular strands in the interior of shredded stalks (Fig. 5). These sclerotia, which are round to irregular in shape, may be so numerous that they make the rot look grayish-black. Lodging may be severe in certain seasons.

Cause. Macrophomina phaseoli (Sclerotium bataticola) is the causal organism. The fungus is composed of several strains differentiated on the basis of sclerotal size and the presence or absence of black, flask-shaped pycnidia that contain spores. Apparently the strains attacking corn do not form pycnidia. Instead, the fungus is disseminated through the sclerotia and also overwinters in these bodies. The fungus has a wide host range including sorghum, soybeans, and several other crops.

Control. Little is known about the relationships of soil fertility to charcoal rot or the relative resistance of inbred lines and hybrids. Otherwise, the same control measures should be used as for Diplodia stalk rot. It is also helpful to irrigate during dry periods after tasseling and to grow corn in long rotations with crops that are not natural hosts of the fungus.

Pythium and bacterial stalk rots

These minor diseases become damaging only in local areas. Bacterial rots in particular are rare and limited. Both types of rot are favored by extended periods of hot, damp weather. They are most likely to be found in poorly drained river-bottom fields when the air is calm and humid; or in fields where overhead irrigation is used.

Symptoms. These diseases are generally first apparent when plants suddenly fall over in midsummer. Usually a single internode above the soil line is the only
part of the stalk that is rotted (Fig. 6). The diseased area is tan to dark brown, watersoaked, soft (or slimy), and collapsed. The stalks are not broken off completely, and affected plants may remain green for a week or more because the vascular strands remain intact.

Causes. Pythium stalk rot is caused by the fungus *Pythium aphanidermatum* (*P. butleri*), which thrives at high temperatures. Unlike other stalk-rotting fungi, it can attack young, vigorously growing plants before silking.

Bacterial stalk rots are caused by one or more species of *Erwinia* and *Pseudomonas*. Like *Pythium*, the bacteria can attack young plants. (See also “Bacterial leaf blight and stalk rot,” page 18.)

Control. Inbred lines differ in resistance to these rots. Otherwise, no specific control measures are known.

### EAR AND KERNEL ROTS

Corn is susceptible to a number of ear and kernel rots, especially when rainfall is above normal from silking to harvest. In humid areas, these rots occur in almost every field in every season. Losses are increased by insect and bird damage to the ear and by lodging of stalks so that ears touch the ground. Corn ears that are well covered by husks and those that mature in a downward position have less rot than ears with open husks or those that mature upright.

Although ear and kernel rots reduce yield, quality, and feeding value of the grain, they are of less general economic importance than stalk rots.

**Diplodia ear rot or dry rot**

Diplodia is the most common ear rot in most of the Midwest. In some years it has caused losses of 20 percent or more in individual fields. Wet weather from silking to maturity is ideal for infection, particularly if the early summer has been relatively dry.

Both nutritive value and palatability to hogs are reduced in rotted ears.

**Symptoms.** Ears are apparently most susceptible from silking until about 3 weeks later. Husks of early-infected ears appear bleached or straw-colored, in contrast to the green of healthy ears. When infection occurs within 2 weeks after silking, the entire ear becomes grayish-brown, shrunken, very lightweight, and completely rotted (see inside front cover). Such ears stand upright with the inner husks stuck tightly to one another and to the ear because of the Diplodia fungus growing between them. When ears are this badly infected, black pycnidia of the causal fungus are often found on the husks and sides of kernels (Fig. 7).

Ears that are infected later usually show no external signs of disease. When the husks are opened, however, a white mold is seen growing between the kernels, and the kernel tips are discolored. Part or all of the ear may be rotted. In very late infections, the white mold may not be visible between the kernels. Ears sometimes appear healthy until after shelling, when the brown germs and dead kernels become evident. Infection usually begins at the ear base, progressing toward the tip, but may sometimes start at an exposed ear tip.

**Cause.** This rot is caused by *Diplodia maydis*, the same fungus associated with Diplodia stalk rot and seedling blight (pages 7 and 8).
A widely distributed rot, Nigrospora is present to some extent every year. Damage is most severe when normal plant growth is checked by stalk or root rot, leaf
blights, hail, insects, cold, drouth, or root injury. Corn grown on poor soil appears to be more susceptible than that raised on fertile soil, possibly because lack of proper nutrition causes premature dying.

**Symptoms.** Affected ears are lightweight, and kernels are slightly bleached with whitish streaks, poorly finished, and easily pressed into the cob. Chaff of yellow hybrids is often brown or chocolate-colored instead of a normal bright red. In white hybrids the chaff is pale yellowish or gray. Close examination of infected ears shows large numbers of speck-sized, jet black spore masses scattered in the shredded pith of the cob and on the tip ends of the kernels (Figs. 7 and 8). Shanks, bases, and cobs of badly infected ears are often shredded, particularly when ears are picked mechanically or shelled. Many diseased ears are knocked to the ground.

Pound for pound, Nigrospora-rotted corn has almost the same nutritional value as healthy grain.

**Cause.** The causal fungus is *Nigrospora oryzae (Basisporium gallarum)*, which overwinters on old plant refuse in the field. It is frequently present with stalk-rotting fungi, and may help destroy stalk tissue.

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**Gray ear rot**

Although gray ear rot is widely distributed over the eastern part of the Corn Belt, it has rarely been severe. The disease is favored by extended periods of wet weather during the first several weeks after silking.

**Symptoms.** In its early stages gray ear rot resembles Diplodia because of the grayish-white mold that develops on and between the kernels, usually starting near the base of the ear. In early infections, husks are tightly stuck to the ear and bleached.

In advanced stages, gray ear rot is easily distinguished from Diplodia. The ear is dark slate-gray to black, instead of grayish-brown as in Diplodia; the mold on the rotted ears is a darker gray; and very small black specks (sclerotia) are often scattered through the cob. Severely infected kernels have slate-gray to black streaks or specks under the pericarp (Fig. 7).

Early-infected ears are shriveled and mummified by harvest time (Fig. 9). Because of their light weight they remain upright. When the shank and butt are rotted, the ear breaks off.

**Cause.** The disease is caused by *Physalospora zeae (Macrophoma zeae)*. The perithecia of the sexual stage and pycnidia of the asexual stage occur in large lesions on corn leaves and occasionally on the tassel neck or under the sheath of the uppermost leaf. Perithecia and pycnidia may develop in the same lesion. They are black and buried in the leaf tissue. 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 are found in rotted ears and kernels. The sclerotia are resistant to extremes of environment and serve as a means of survival and propagation of the fungus.

**Control.** No control for gray ear rot is known other than the use of adapted hybrids. Since it closely resembles Diplodia ear rot as to time and place of infection on the corn plant, resistance to the two diseases may be closely correlated.

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**Cladosporium (Hormodendrum) kernel rot or ear rot**

This rot is common in some years, especially in the more northerly corn-growing areas of the Midwest. Damage is most severe where corn is prematurely frosted, or where harvesting is delayed until late fall or early winter. Late-maturing hybrids that are high in moisture when killing frosts occur are most commonly attacked.
Symptoms. Dark, greenish-black blotches or streaks form on the kernels, usually over most of the ear (Fig. 7). The black discoloration shows up first where the kernels are attached to the cob. Later the blotches extend upward on the kernels, but seldom reach the crown. Further damage develops in storage after harvest.

Cause. This kernel rot or ear rot is caused by the fungus *Cladosporium herbarum* (*Hormodendrum cladosporioides*). The fungus may invade the crowns of kernels damaged by growth cracks.

Control. No control is known other than to grow locally adapted hybrids. Where possible, fields should be harvested as early as practical after the grain is mature.

Minor ear rots

Several other ear and kernel rots of corn have been described. However, these are limited to certain areas, occur very rarely, and are of little economic importance. Like the major ear rots, they are most common in wet seasons.

**Physalospora ear rot** is confined largely to the southern third or half of the Corn Belt and is similar in some respects to gray ear rot (page 13). Severely infected ears are completely covered by a dark brown to black felty mold (Fig. 10). Mildly infected ears may have a few kernels that are blackened near the base, where most infections begin. The causal fungus, *Physalospora zeicola*, has both a sexual and an asexual stage; the latter is called *Diplodia frumenti*. The asexual spores develop in pycnidia on cornstalks infected by the fungus.

**Penicillium ear rot** is occasionally found, primarily on ears injured by ear-feeding insects or by other causes. The typical powdery, green or blue-green mold grows on and between the kernels, which are often bleached. Damage usually occurs at the tip of the ear. The causal fungus is most frequently *Penicillium oxalicum*, but occasionally other species have been isolated from diseased ears. The same fungi cause a storage rot known as “blue-eye” (page 15).

**Aspergillus ear rot** is another disease that is comparatively rare. A powdery mold, usually black, grows on and between the kernels. Damage is most common at the tip of the ear. *Aspergillus niger* is the fungus most commonly associated with this ear rot, although other species have been isolated from diseased ears. Some of these species, such as *A. glaucus* and *A. ochraceus*, cause a greenish-yellow or tan mold. Aspergillus species cause serious damage to stored corn.

Severe infections of Physalospora ear rot, shown in picture at left, cause a dark brown to black felty mold over the entire ear. (Fig. 10)
Storage rots may develop on cribbed ear corn or binned shelled corn if the moisture content of the kernels is above 12 or 13 percent and the air is warm enough for fungi to grow. Storage rots reduce both the feeding value and the market grade of corn. Badly rotted corn is worthless for seed or feed. Occasionally certain rot-producing fungi will form toxins and hormones that seriously affect livestock.

On ear corn, the first external symptom is typical mold growth on and between kernels and at their base. However, the interior of the kernel may be damaged before mold is visible from the outside (see inside front cover). The germ or embryo is often killed or discolored. One storage rot called “blue-eye” is characterized by a bluish-green germ (see inside front cover). With other rots, the mold may be blue, green, tan, white, black, or pinkish-red.

When storage rots develop in shelled corn, the kernels often cake together to form a crust, usually at the center and top of a bin. Mold growth is often extensive, and infested bins have a musty odor. If aeration is inadequate, spoilage of the surface grain may be intensified as moisture migrates to the upper layers.

Cause. Some 25 or more different species of fungi are known to cause storage rots. Several species of *Aspergillus* and *Penicillium* are the most common storage-rot fungi and frequently are referred to as typical storage molds. These are the *Aspergillus glaucus* and *A. flavus* groups, *A. candidus*, *A. niger*, *A. ochraceus*, *A. versicolor*, *Penicillium rugulosum*, *P. palitans*, *P. oxalicum*, and *P. chrysogenum*. Fungi causing ear and kernel rots in the field usually do not cause storage rots.

No one storage mold attacks corn over a wide range of moistures and temperatures. At least one species of *Aspergillus* can grow slowly on and in corn with a moisture content of 12 to 13 percent. Other fungi grow at moisture contents above 13 percent. They work like a “bucket brigade” at a fire. Each fungus works within rather narrow limits. When these limits are reached, another fungus takes over. Ear-rotting fungi are common and destructive in storage at moisture contents of 18 percent or more.

All storage molds give off heat and moisture, which in turn are used by their successors to accelerate rotting of the grain. The higher the temperature and moisture content, within limits, the faster the rotting. Insects are often common in spoiled grain, taking advantage of and contributing to the heat and moisture given off by the molds.

Control. Ordinarily ear corn is in little danger from storage rots if it is harvested at a moisture content below 20 to 25 percent and is stored in well-ventilated covered cribs. In some seasons, when the weather is too wet for proper maturing and drying, ears may become moldy in the field. Such corn should be artificially dried to a moisture content low enough to stop mold growth.

Storage molds can be kept under control in shelled corn if the grain is dried to a moisture content of 12 percent or less. During bin storage, the grain should be probed frequently for “hot spots,” which indicate spoilage is going on. When hot spots or a crust of moldy grain is found, the following measures should be taken: (1) Remove the rotted and moldy grain. (Moldy corn is considered unsafe for all breeding animals. Otherwise, it may be fed with caution. Mixing it with sound corn reduces the risk. This is especially advisable for cattle and hogs being finished for market.) (2) Check the moisture content of the remaining corn. (3) Turn this corn (or stir it mechanically) and thoroughly mix it to redistribute moisture and allow heat to escape.

Fans can sometimes be used to move small amounts of air through the grain to help maintain a uniform temperature and prevent “wet” spots. For this treatment to be effective, initial moisture content and temperature of the grain cannot be very high. Relative humidity and temperature of the outside air must also be relatively low.

Corn of 25 to 30 percent moisture may be safely stored in airtight silos or other structures that are free of air leaks. Respiration of molds and grain soon uses up the oxygen, halting the growth of harmful fungi. The corn may contain some yeast fungi, however, which, together with the high moisture content of the grain, make it suitable only for feed. According to research tests, this corn has a high feeding value.

Kernel disorders are not actually diseases, but they are important to corn pathology because they rupture or weaken the seedcoats, providing opportunities for fungi to invade the kernels.

**Popped kernel** appears as an irregular break in the seedcoat over the kernel crown (Fig. 11). The kernels look like partially expanded popcorn kernels. This disorder is more common on a few inbred lines than on hybrids grown by farmers.

**Silk-cut.** Despite its name, silk-cut has nothing to do with the silk of the ear. It appears as a horizontal cut or split in the pericarp over the sides of the kernel (Fig. 12). Apparently it affects only certain inbred lines and their combinations in hybrids.
"Popped kernels" appear as irregular breaks in the seedcoat over the kernel crown. (Fig. 11)

Silk-cut causes breaks in the seedcoat that make the kernels susceptible to mold infections. (Fig. 12)

LEAF DISEASES

Certain leaf diseases of corn have increased in economic importance in the Midwest since 1940. In several years one or more of these diseases were severe. The increase in economic importance has almost coincided with the use of hybrid corn. This does not mean, however, that hybrid corn is necessarily less resistant to certain leaf blights than open-pollinated corn.

At least two reasons may account for the apparent relationship between some leaf blights and use of hybrid corn: (1) During the late 1930’s and early 1940’s, weather conditions during several years favored the development of both northern corn leaf blight and southern corn leaf blight in the eastern Corn Belt. It just happened that this was when hybrid corn was being introduced into the area. (2) Hybrid corn is genetically much more uniform than open-pollinated corn. Consequently, when a disease attacks, all plants tend to react alike. Open-pollinated corn, although not highly resistant, is much more heterogeneous, and the degree of disease resistance 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 dews favors the spread and development of leaf blights caused by fungi. The genetic makeup of the plants also affects the severity of the diseases. Soil fertility does not seem to have much effect.

Northern corn leaf blight

Northern corn leaf blight is found throughout the Midwest. It may occasionally become locally severe in the northern and central Corn Belt during humid, moist growing seasons. Where leaf blight is severe, ears may be immature and chaffy. The time when disease first appears is determined largely by weather conditions. In some years it may be found before silking. Under less favorable conditions — such as hot, dry weather — there may be no trace of it.

The earlier leaf blight appears, the more it reduces yield. If disease becomes well established before or shortly after silking, grain yield may be reduced by 30 percent or more. In addition to grain losses, feed value of fodder is lowered and plants are predisposed to stalk rot.

Symptoms. Northern corn leaf blight is recognized by long, elliptical, grayish-green to tan spots on the leaves. When fully developed, the spots may be 1½ inches wide and 6 inches long (see inside back cover). These lesions appear first on the lower leaves. The disease progresses upward until, in severe cases, nearly all leaves of a plant are heavily infected. The plant appears dead and gray, as though injured by frost or drouth. In damp weather the fungus produces tremendous numbers of dark-colored spores on the surface of the lesions. These appear as a gray or black “fuzz.” Sometimes they are arranged in target-like zones. Ears are not infected, although lesions may form on the husks. Since kernels are not attacked, the possibility of distributing the disease by seed is remote.

Cause. Helminthosporium turcicum (Trichometasphaeria turcica) is the causal fungus. It is believed to overwinter in infected corn leaves, at least in the southern part of the Corn Belt. During the following summer spores are formed on old lesions. Wind currents or splashing rains carry the newly formed spores to growing corn leaves. If moisture is present, the spores germinate and penetrate the leaves, thus establishing the disease. The fact that air currents may carry spores
for miles during the summer may account for isolated epidemics in the northern part of the Corn Belt.

Germination and penetration of the leaves take place in 6 to 18 hours when water is on the leaves and the temperature is 65° to 80° F. Spots show up 7 to 12 days after infection. Successive crops of spores form on the leaf lesions and spread to progressively higher leaves on the plant. Under favorable conditions for the disease, the entire corn plant may be prematurely killed.

Besides corn, the fungus also attacks sorghum, sudangrass, Johnsongrass, teosinte, and a few other grasses. Cross inoculations from these hosts indicate that physiologic races exist within the fungus. No races, however, have been found among isolates from corn. This makes the work of the corn breeder and plant pathologist less complicated, because they do not need to breed for resistance to different races.

Control. Resistant hybrids offer the most effective and lasting means of control. It is now possible to get resistant hybrids that are adapted to much of the Midwest; and others with different adaptations are being developed. Hybrids with a much higher level of resistance can be expected shortly. Seed treatment and crop rotation are not effective as controls.

Southern corn leaf blight

Since southern corn leaf blight thrives at slightly higher temperatures than northern leaf blight, its occurrence in the Corn Belt is restricted to about the southern half.

Symptoms. Lesions range from minute specks to spots ½ inch wide and 1½ inches long. They are oblong, parallel-sided, and grayish-tan to tan (see inside back cover). Occasionally they have a dark brown to purplish margin. Dent corn ears are not infected; however, the ear tips and silks of sweet corn may be attacked during a severe epidemic.

Cause. Southern leaf blight is caused by the fungus Helminthosporium maydis (Cochliobolus heterostrophus). No specialized races of the fungus are known, although the disease has been reported to attack teosinte and several other grasses, as well as wheat, barley, and oats.

Control. Like northern corn leaf blight, southern corn leaf blight is best controlled by growing resistant hybrids. Many hybrids that resist northern corn leaf blight are also highly resistant to southern leaf blight. Resistance is usually directly proportional to the number of resistant inbreds used in making up the hybrid. Seed treatment and crop rotation are not effective control measures.

Bacterial wilt or Stewart’s disease

This disease is widespread over much of the Midwest, especially the southern half of the Corn Belt. It is much more severe on sweet corn than on dent corn.

Bacterial wilt, unlike northern and southern corn leaf blights, does not require damp weather and heavy dews for spread and development. The bacteria that cause this disease overwinter in corn flea beetles. When the small, oval, black adult beetles come out of hibernation in the spring, they feed on young corn plants and the bacteria enter the corn plant through the wounds, thus starting infections on the leaves. During the growing season the beetles continue to spread the disease from infected plants to healthy ones, frequently migrating from south to north.

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, large numbers of beetles commonly survive, start infections, and spread the disease during the following growing season. Cold winters reduce the number of beetles, so that there is usually little early infection and the disease does not spread over a wide area.

Symptoms. Susceptible hybrids of sweet corn and popcorn wilt rapidly, resembling plants suffering from lack of water. Severely infected seedling plants may be killed. Infected plants that survive are stunted and may produce no ears. Leaves often have long, irregular streaks that are first pale green to yellowish and later become dry and brown (see inside back cover). A white tassel often develops prematurely. Chocolate-brown cavities may form in the stalk pith of severely infected plants (Fig. 13). Bacteria spread through the vascular strands of such plants and pass through the cob into the kernels. Infected kernels may spread the disease to new areas.

Dent or field corn is generally much more resistant than sweet corn. The disease does not usually spread through an entire plant of field corn, except in a few very susceptible inbred lines that develop the same symptoms as sweet corn.

The characteristic symptoms of field corn appear on leaves as long, irregular, pale-greenish streaks that turn yellow, dry, and become straw-colored (Fig. 14). This is known as leaf blight and it generally appears after tasseling. These dead, dry lesions are sometimes covered with secondary fungi, which are often assumed to cause the disease. The long streaks always originate at the feeding wounds of corn flea beetles. These feeding injuries are readily seen if the leaf is held up to the light. Where the disease is severe, much of the leaf area may be destroyed, yield is reduced, and plants become more susceptible to stalk rot. Dent corn kernels are rarely affected and then only where disease is very severe on susceptible inbred lines.

Cause. Bacterial wilt is caused by the bacterium Xanthomonas stewartii (Bacterium stewartii).

Control. The most practical control is to use resistant hybrids. Golden Cross Bantam was the first resistant hybrid sweet corn. It is widely adapted over most of the eastern Corn Belt. Many other sweet corn
Chocolate-brown cavities may form in the stalk pith of plants severely infected with bacterial wilt. (Fig. 13)

On leaves, bacterial wilt causes streaks that eventually die and become straw-colored. (Fig. 14)

varieties are now available that combine resistance to bacterial wilt with high yield and quality. But even resistant sweet corns are susceptible in the one- to three-leaf stage. In both sweet and field corn, early and short inbred lines appear more susceptible than late and tall inbreds.

In dent corn, a positive correlation may exist between resistance to the late leaf-blight phase of this disease and resistance to northern corn leaf blight. Several inbred lines have good resistance to leaf blight.

Spraying or dusting seedling plants with an insecticide, such as carbaryl (Sevin) or DDT, to kill corn flea beetles helps somewhat to keep the disease from spreading. Adequate levels of potassium in the soil tend to minimize the disease, while high levels of nitrogen predispose plants to bacterial wilt. Seed treatment and spraying plants with fungicides have no effect on control.

Minor leaf diseases

Several fungi and bacteria attack corn leaves but are of little importance, because they rarely occur, do little damage, or only infect certain susceptible lines.

Bacterial leaf blight and stalk rot occurs in some areas of the Midwest. The disease seems to be favored by hot (85° to 95°), showery weather. It has been of minor economic importance.

The disease may appear in localized areas in the field. Leaf lesions range from small elliptical spots to narrow stripes nearly as long as the leaf (Fig. 15). These lesions often merge and affect most of the leaf width. They are first olive-green and oily or watersoaked, later becoming tan and dry—sometimes with a reddish-brown margin. Badly diseased leaves shred easily, especially after wind and driving rain.

Stalk rot usually occurs just above the point where the ear is attached, causing a dark brown rot and shredding of the pith (Fig. 16). As rot progresses, the tops of the plants die. Early-infected plants are dwarfed and often develop multiple ears that are usually sterile and may rot (Fig. 17).

The causal bacterium is Pseudomonas alboprecipitans. It also attacks the leaves of foxtail grasses and Sudangrass.

Because this disease is of minor importance, no control measures have been developed. Sweet corn seems to be slightly more susceptible than dent corn. Popcorn is most susceptible.

Purple sheath spot is widespread, but apparently causes no measurable damage. Purplish-brown, irregular spots of varying sizes become conspicuous on the leaf sheaths, usually after silking (Fig. 18). Beneath these spots the inner leaf sheath is discolored and some of its tissue may be broken down. Several secondary fungi and bacteria feed on the debris that collects behind the leaf sheaths. Inbred lines differ considerably in their reaction to purple sheath spot.
Lesions due to bacterial leaf blight often merge, covering most of the leaf. Badly infected leaves shred easily. (Fig. 15)

Bacterial stalk rot causes shredding and darkening of stalk. (Fig. 16)

Multiple ears, usually sterile, frequently result from early infections of bacterial leaf blight and stalk rot. (Fig. 17)

Purple sheath spot appears as irregular, purplish-brown spots of varying sizes, usually after silking. (Fig. 18)

At first dark green, lesions due to Holcus spot become dry and tan with a reddish margin. (Fig. 19)
Common corn rust is almost universal where corn is grown. Rust generally causes little damage in the United States, although severe infection has reduced yields, especially of sweet corn. In the Midwest, rust often appears soon after silking, but in some years it may appear much earlier. Cool, humid weather favors disease development.

Symptoms. Common rust is recognized by small, oval to elongate pustules (Fig. 21), which are at first cinnamon-brown, becoming brownish-black as the corn matures. The pustules may appear on any aboveground parts of the plant, but are most abundant on the leaves, being scattered over both surfaces.

Common smut or boil smut is found wherever corn is grown. Losses from common smut in the Midwest are highly variable, ranging from a trace up to 6 percent or more in localized areas, and even approaching 100 percent in some individual fields of sweet corn. It is doubtful whether losses in grain yield exceed 2 percent over very wide areas. The number, size, and location of smut galls on the plant affect the amount of yield loss. Large galls on or above the ear are more destructive than galls below the ear. Galls resulting from detasseling are usually small and generally cause little damage.

The relationship between weather conditions and the amount and severity of common smut is not clear. Dry weather generally favors smut; and the disease is more prevalent in the western Corn Belt than in the more humid eastern part. It is not known, however, if the dry, windy weather common in the western area predisposes plants to infection or simply provides better means for spread of the fungus.
Brown spot lesions start as small spots, later merging to form large blotches. (Fig. 20)

The small pustules caused by corn rust are at first cinnamon brown, then turn brownish-black. (Fig. 21)

In early stages of corn smut, white membrane covers the galls. (Fig. 22)

When membrane breaks, dark interior of galls is revealed. (Fig. 23)

This nearly barren ear is the result of maize dwarf mosaic. (Fig. 24)
Maize dwarf mosaic (MDM) is the newest disease of corn in the Midwest, having been discovered in 1962. At present, it is widely distributed in bottomland fields close to rivers and other bodies of water. It is most serious in areas where Johnsongrass is a common weed. In 1964 Ohio suffered an estimated state-wide loss of 5 million bushels of corn, valued at $5.8 million. Illinois, Indiana, Kentucky, Missouri, Arkansas, Tennessee, and other central states have reported losses of 50 to nearly 100 percent in certain fields.

The disease usually reappears in the same fields or general location in succeeding years. Many fields showing a trace or light infection one year are severely damaged in following years. Sweet corn appears to be more susceptible than dent corn.

**Symptoms.** The disease first appears in the youngest leaves as an irregular, light and dark green mottling; or as elongate, light green blotches (flecks) and interrupted stripes (see inside back cover). Corn plants showing this mosaic pattern are usually stunted and bushy because of bunching of the upper internodes. As infected plants get older the mosaic often disappears and young leaves become more yellowish. About a third of the young leaves on many hybrids develop streaks and blotches of a dull to brilliant red or reddish-purple (see inside back cover). Excessive tillering and multiple ear shoots may develop as the disease progresses. Severely diseased plants are partly or totally barren (Fig. 24) and may die prematurely. The disease apparently increases susceptibility to root and stalk rots.

**Causes.** *Ustilago maydis* (*U. zeae*) is the causal fungus. It attacks only corn and the closely related teosinte. The black spores that make up most of the sooty galls are easily blown long distances by the wind. The spores germinate in water at about 50° to 95° F. Infection occurs by means of spores germinating directly from a germinating chlamydospore or developing after fusion of opposite mating types. The corn smut fungus causes host cells to increase in size and number, forming galls. Eventually the galls are entirely converted to a black, powdery, spore mass.

The time interval between infection and the formation of mature galls varies from 1 to 3 weeks or more. Spores formed in the first smut galls may germinate and infect the same or other plants. Galls form and spores disseminate more or less continuously through the summer. The smut fungus overwinters as spores in crop refuse, manure, and possibly the soil.

When animals eat “smutty” stalks, leaves, and ears, the spores remain alive during passage through the animal’s alimentary canal and are carried in the manure. Spore masses are killed by the acids in silage.

**Control.** The most effective control measure is to plant hybrids with some resistance. No hybrid is completely resistant, however, and herbicides may lower what resistance there is. Other measures are to avoid mechanical injuries to the plant when cultivating or spraying, protect the plants against corn insects, and follow a well-balanced soil fertility program based on soil tests. In the home garden, the number of spores can be reduced by removing the galls before they rupture. Seed treatment is not effective.
most lasting method of control. A few hybrids that are adapted to the Corn Belt and have a reasonably high degree of tolerance or resistance are now available. In hard-hit areas farmers are already growing these hybrids. More hybrids that are adapted and highly resistant should be available shortly. Resistant sweet corn varieties probably won’t be developed for several years.

Because Johnsongrass appears to be the major wintering host of the MDM virus(es), many states are pushing vigorous programs to eradicate this weed. Destroying Johnsongrass probably slows down the spread of the disease to nearby corn fields.

Applications of insecticides or fungicides, time of planting, and crop rotation have little or no effect on control, but raising the fertility level may decrease yield loss if infection occurs.

**DOWNY MILDEW (CRAZY TOP)**

Downy mildew or crazy top is widespread but sporadic over the Midwest. It is seldom prevalent enough to cause much damage, although losses of at least 60 percent have been reported in parts of some fields. The disease occurs only where soil becomes flooded or waterlogged sometime between germination of the corn kernels and the time when seedlings are 6 to 10 inches tall.

**Symptoms.** Instead of forming a normal tassel, the floral parts continue to grow and appear as a bushy mass of small leaves (Fig. 25). No pollen is produced, since the tassel is completely deformed. Ear formation may also be checked, causing the ear shoots to be numerous, elongated, and barren. In severely infected plants, no ears or tassels are formed at all; stunting is pronounced; leaves are narrow and strap-like; and suckering is excessive (Fig. 26). Common corn smut (page 20) often occurs on these abnormal leafy growths.

**Cause.** *Sclerophthora macrospora* (*Sclerospora macrospora*) is the fungus causing downy mildew. It attacks not only corn but also a large number of wild grasses, where it probably survives in the absence of corn. Little is known about the way infection occurs, but it is likely that swimming spores (zoospores) in flood waters penetrate the seedlings.

**Control.** The most direct control method is to provide adequate soil drainage. Little is known about the relative resistance of inbred lines and hybrids. Seed treatment has no effect in control.
flask-shaped fungus is composed of interwoven bodies too small to be seen with a compound microscope, recognizable by the symptoms they produce in infected hosts. Fungi found notably in the rusts and smuts. Some spores are very light and can be blown hundreds of water, insects, animals, man, and machinery. When conditions are favorable, the spore germinates to produce a hyphal tube that later develops into a new fungus body. Many fungi multiply by forming spores at the ends of, within, or on specialized hyphae. The spores are microscopic bodies that function like the seeds of higher plants and are carried by water, wind, man, insects, animals, and machinery. A spore landing on a plant under the proper conditions (usually moderate temperature and a film of moisture) can produce a new fungus body. Many fungi produce both sexual and nonsexual (asexual) spores. The way in which the sexually formed spores are produced is the basis for classification of fungi into three of their main groups: Phycymycetes, Ascomycetes, and Basidiomycetes. Sexually produced spores have not been found in the fourth main group, the Fungi Imperfecti. No spores are known for some fungi, which have been classified in a fifth group, the Mycelia Sterilia. A single thread or filament that constitutes the body (mycelium) of a fungus. It may be divided into cells by cross walls or be one long cell. Some hyphae are specialized for producing spores, penetrating host tissues, overwintering, or trapping nematodes. A localized area of diseased tissue. Spots, cankers, blisters, pustules, and scab are lesions. Any fungus with conspicuous, profuse, or woolly growth (mycelium or spore masses). Occurs most commonly on damp or decaying matter and on the surface of plant tissue. The mass of interwoven threads (hyphae) making up the vegetative body of a fungus. The mycelia of fungi show great variation in appearance and structure.

Glossary

**Ascospore**—A sexually produced fungus spore borne in an ascus. The ascus, in turn, is contained in a fruiting body, of which there are two types—perithecia and apothecia.

**Bacterium** (pl. bacteria)—A one-celled, microscopic organism that lacks chlorophyll. Bacteria reproduce by simple fission or dividing in half. Some have whiplike flagella that may aid them to swim. Bacteria are widely distributed in air, soil, water, bodies of living plants and animals, and dead organic matter.

**Chlamydospore**—A thick-walled, asexual spore formed by the modification of a fungus hypha. The term is also applied to the spores (teliospores) produced by smuts.

**Conidium** (pl. conidia)—An asexual type of fungus spore formed from the end of a special spore-bearing hypha.

**Fungus** (pl. fungi)—A low form of plant life that, lacking chlorophyll and being incapable of manufacturing its own food, feeds on dead or living plant or animal matter. The body of a fungus consists of delicate, microscopic threads known as hyphae, many of which form branched systems called mycelia often evident to the naked eye. The mycelia, which may form inside or on the surface of the plant host, have different branching habits and structures that help to identify the fungus. Many fungi multiply by forming spores at the ends of, within, or on specialized hyphae. The spores are microscopic bodies that function like the seeds of higher plants and are carried by water, wind, man, insects, animals, and machinery. A spore landing on a plant under the proper conditions (usually moderate temperature and the film of moisture) can produce a new fungus body. Many fungi produce both sexual and nonsexual (asexual) spores. The way in which the sexually formed spores are produced is the basis for classification of fungi into three of their main groups: Phycymycetes, Ascomycetes, and Basidiomycetes. Sexually produced spores have not been found in the fourth main group, the Fungi Imperfecti. No spores are known for some fungi, which have been classified in a fifth group, the Mycelia Sterilia.

**Hypha** (pl. hyphae)—A single thread or filament that constitutes the body (mycelium) of a fungus. It may be divided into cells by cross walls or be one long cell. Some hyphae are specialized for producing spores, penetrating host tissues, overwintering, or trapping nematodes.

**Lesion**—A localized area of diseased tissue. Spots, cankers, blisters, pustules, and scabs are lesions.

**Mold**—Any fungus with conspicuous, profuse, or woolly growth (mycelium or spore masses). Occurs most commonly on damp or decaying matter and on the surface of plant tissue.

**Mycelium** (pl. mycelia)—The mass of interwoven threads (hyphae) making up the vegetative body of a fungus. The mycelia of fungi show great variation in appearance and structure.

**Nematodes** (also called nemas, roundworms, or eelworms)—Generally microscopic tubular animals usually living free in moist soil, water, and decaying matter or as parasites of plants and animals. Responsible for many plant diseases. Nematodes that cause plant disease pierce the cells of a plant with a stylet and suck up juices. Nematodes also (1) provide wounds by which other plant pathogens may enter, and (2) transmit disease-producing organisms.

**Perithecium** (pl. perithecia)—A flask-shaped fungus fruiting body that contains sac-like membranes (asci) in which spores (ascospores) are produced. The spores are expelled or otherwise released through an opening at the top.

**Pycnidium** (pl. pycnidia)—A flasklike fungus fruiting body containing nonsexual spores (conidia). It is formed on the surface or more or less embedded in the tissue of the host; often it opens by a pore. The spores are commonly extruded in mass or in long coils through the pore.

**Sclerotium** (pl. sclerotia)—A small, compact, resting form of a fungus. It is composed of an interwoven mass of mycelial threads with a hard outer rind. Sclerotia are generally dark-colored, are more or less round or flat, and vary greatly in size. They may remain viable in the soil, in plant refuse, or in seeds for many years and can germinate or bear fruiting bodies that infect new plants under favorable conditions of temperature and moisture.

**Spore**—A part of a fungus corresponding to the seed of higher plants. A microscopic, one- to many-celled body serving to reproduce and disseminate a fungus. Spores may be either nonsexual (asexual), formed directly from vegetative hyphae or in special fruiting structures (e.g., pycnidia); or sexual, formed from a union of two cells representing a difference in sex. Some, called resting spores, have thick walls that enable them to survive unfavorable growing conditions. Some spores are very light and can be blown hundreds of miles by the wind. Others are transported easily by water, insects, animals, man, and machinery. When conditions are favorable, the spore germinates to produce a hyphal tube that later develops into a new fungus body.

**Teliospore**—A thick-walled resting spore of a fungus, found notably in the rusts and smuts.

**Virus**—Submicroscopic, filterable, infectious agents (bodies) too small to be seen with a compound microscope. Viruses have characteristics of both living and nonliving matter. They are large nucleoproteins having a high molecular weight and the capacity to multiply (replicate) and act like living organisms when in specific plant or animal cells. They are usually recognizable by the symptoms they produce in infected hosts.

**Zoospore**—A motile, sexually produced fungus spore.
Large lesions — northern corn leaf blight; small lesions — southern corn leaf blight.

Bacterial or Stewart's wilt on sweet corn. Field corn is usually much more resistant than sweet corn or popcorn.

From top to bottom, maize dwarf mosaic on Johnsongrass, sudangrass, sorghum, and corn.

Maize dwarf mosaic causes red blotching on leaves of many hybrids at tasseling time.

A field infested with maize dwarf mosaic. Note the difference in height between susceptible and resistant lines of corn.
Eighty-five percent of the corn in this field has lodged because of stalk rots.

Diplodia stalk rot.  Gibberella stalk rot.