Stalk Rot of Corn

J. J. CHRISTENSEN and
R. D. WILCOXSON

MONOGRAPH NO.
3

PUBLISHED BY
THE AMERICAN
PHYTOPATHOLOGICAL
SOCIETY

Stalk Rot of Corn

J. J. CHRISTENSEN and
R. D. WILCOXSON

Department of Plant Pathology and Physiology
Institute of Agriculture, University of Minnesota, St. Paul

MONOGRAPH NO. 3

Published for
The American Phytopathological Society
at The Heffernan Press Inc., Worcester, Mass.
1966

Arrangements for this Monograph were made in 1963 by a previous Committee of the Society. Editing and arrangement for publication were by the 1965-66 Committee on Phytopathological Monographs and Reviews:

K. F. Baker, C. W. Boothroyd, J. A. Browning,
P. M. Halisky, and J. D. Menzies.

This and preceding monographs for sale by the Business Manager, The American Phytopathological Society.

ACKNOWLEDGMENTS

We are indebted to N. A. Anderson, K. F. Baker, E. E. Banttari, C. W. Boothroyd, A. J. Hooker, H. G. Johnson, B. W. Kennedy, M. F. Kernkamp, and T. Kucharek for reading the manuscript and for making constructive suggestions. We are also grateful to our many colleagues throughout the world who sent unpublished data, pictures, and manuscripts concerning stalk rot of corn. The work could not have been done without the efficient help of our secretaries, whom we thank: Grace Aschenbach, Charlotte Patoile, Shirley Anderson, Cathleen Larson, Darlene Durant, and Suzanne Rooney.

The survey of literature for this monograph was concluded in August 1964.

J. J. Christensen died 20 June 1964.

Paper No. 1222, Miscellaneous Journal Series, Minnesota Agricultural Experiment Station.

Contents

Introduction

Stalk rot of corn (*Zea mays* L.) is of world-wide significance. The following are some of the many reports of stalk rot from different parts of the USA: Arkansas (283), Delaware (192), Florida (81), Illinois (22, 169, 251, 353), Iowa (76, 210), Kansas (149, 206, 207), Kentucky (368, 370), Maryland (116), Michigan (7), Minnesota (43), Missouri (30), Nebraska (70, 111, 182, 183), New Jersey (37, 48), New York (26), North Carolina (153), Ohio (178, 312), Oklahoma (175), Pennsylvania (94, 155), South Carolina (10), Texas (2, 405, 406), Virginia (93, 277), West Virginia (177).

It has also been reported from many countries of the world: Argentina (32, 139, 301), Australia (84, 223), Bulgaria (33), Canada (158, 200, 381), Costa Rica (102), Colombia (326), Egypt (290, 294, 295, 296, 297), El Salvador (3), France (212, 226), Guatemala (307), Italy (59, 174), India (114, 268), Israel (157, 371), Kenya (190), Manchuria (225), Mexico (39, 232, 233, 279, 280, 358, 400), Nicaragua (57), Puerto Rico (311), Rhodesia (292), Romania (265, 302), Russia (104, 248, 282, 324), South Africa (18, 19, 36, 92), Venezuela (264). Many people regard stalk rot as being probably the most important and destructive disease of corn in the world.

Stalk rot is a disease complex caused by several different species of fungi and bacteria. It is of variable importance from region to region and season to season; occasionally it becomes epidemic over wide areas. Severity of stalk rot varies greatly as temperature, rainfall, soil drainage, soil type, available nutrients, and other conditions change and interact. The variety of corn planted, and agricultural practices such as the use of sound seed, time of planting, crop sequence, application of fertilizers, and plant populations, have a marked influence on severity of infection. Mechanical injuries and insect damage also tend to increase the severity of stalk rot.

Development of desirable modern corn hybrids demands that considerable attention be given to the incorporation of lodging and stalk-breakage resistance, particularly in countries where mechanical harvesters are in common use. This problem is greatly complicated because plant breeders have also been emphasizing bigger and heavier ears, or more ears per stalk, to increase yields. There has also occurred a widespread improvement in cultural practices, involving dense plant populations and the liberal use of fertilizers. All of these developments produce greater seed yields and this greatly taxes the strength of the corn stalk. Rot weakens stalks and accounts for most of the broken stalks and much of the lodging. Stalk rot must, therefore, be given due consideration in any modern corn improvement program.

Stalk rot of corn has been known for over 60 years, but during the early part of the 20th century damage was usually attributed primarily to seedling blight, root rot, and ear rot. No one knows when stalk rot of corn first became a destructive disease. It is unfortunate that early workers seldom distinguished clearly between root rot, crown rot, and stalk rot. Many of these studies probably involved all three types of rot, as well as seedling blight. It is clear from the literature and from numerous photographs, however, that crown rot and root rot frequently involved the basal portion of the stem (Fig. 2G). Recent studies indicate that these rots may be closely interrelated. They will, therefore, be discussed in this publication as they relate to stalk rot.

More definite early information is available on ear rot and stalk rot caused by *Diplodia zeae* (Schw.) Lev. than on any other single organism. This may be attributed to the fact that *Diplodia zeae* was for a long time considered the primary cause of crown rot, ear rot, and stalk rot of corn in the corn belt of the USA.

In 1834 Schweinitz described *Diplodia zeae* on corn, but is was not until 1906 that it was recognized as causing an important disease of corn (35, 111). About that time *Diplodia*-infected corn was considered as a possible cause of illness in horses in Nebraska. This led to a detailed study of the organism by Heald et al. (112), who were the first to make a pathogenicity test. Infections were obtained by placing inoculum on the silk and the husks and by puncturing the husk and stem. Mature pycnidia were produced about 3 weeks after inoculation. The pycnidia, spores, and cultures of the fungus were described and illustrated. They concluded that there was little or no transmission of the fungus from one plant to another during the same season.

In 1909, Burrill and Barrett (35) described the symptoms and the life history of *Diplodia zeae* in considerable detail. The organism was grown on many nutrient media, and the production of pycnidia noted. They obtained infection from spores taken from corn stalks that were 1 and 2 years old. They also concluded that the spores were disseminated at least 350 yards.

Smith and Hedges (330) obtained proof in greenhouse tests that *Diplodia zeae* entered the plant through the roots. The mycelium was found in root, stem, and cob tissue. They concluded that the fungus in the soil invaded the roots and then passed upward into the stem, cob, and finally the kernels. This type of systemic infection was doubted by Durrell (76, 77) and others (169). McNew (201) concluded that crown rot and basal stem rot arose from infected mesocotyl. At present the nature of stalk infection is still controversial, but the evidence indicates that *Diplodia zeae* enters the stalk through various avenues.

Moore (227) in 1896, and Peters (254) in 1904, called attention to a widespread disease of cattle and other animals known as the "stalk rot" disease. This disease occurred chiefly in the fall and early winter when cattle were feeding on cornstalks in the field. Peters (254) suggested that the disease might have been caused by a *Fusarium* sp., and Sheldon (323), working with Peters, named the fungus *Fusarium moniliforme* Sheldon. Valleau (368) concluded, after making numerous isolations from corn stalks, that *Fusarium moniliforme* was the primary cause of root and stalk rot of corn in Kentucky and

other central and southern states. This fungus is known to be a common cause of stalk and ear rot of corn.

In 1914, Pammel (237) described the *Fusarium* disease of corn in Iowa. Then in 1915 and 1916 he and his co-workers (238) pointed out that in many parts of Iowa stalks were broken at the lower nodes or near them. In many cases stalks were lying on the ground, many were barren, the pith of diseased corn was soft and partially destroyed, and the tissues were brownish or reddish in color. They stated that *Fusarium* diseases were likely to be the most important problem in the corn growing area in Iowa in certain years. Today *Fusarium* spp., particularly *Fusarium graminearum* Schwabe and *Fusarium moniliforme,* are considered among the most destructive fungi on corn, causing stalk rot, seedling blight, root rot, and ear rot.

In this publication consideration will be given to major pathogens that cause stalk rot, as well as to many of minor importance. Organisms that primarily cause foliage disease, although they occasionally cause a limited amount of stalk rot, will not be discussed. The economic importance, geographical distribution of the pathogens, ecological factors influencing infection and development of pathogens, host ranges, methods of creating artificial epidemics, inoculation techniques, development of resistant varieties, and nature of resistance will be emphasized.

For many years it was assumed that *Diplodia zeae, Gibberella zeae* (Schw.) Petch, and *Fusarium* spp. were the primary causes of stalk rot, but now it is fairly well known that many other pathogens may be destructive in certain regions. Rather than review the history of each of these pathogens separately, the historical information will be included in the various sections that follow.

Losses

The world production of corn in 1962 was about 7 billion bushels (367). The USA, with an annual production of between three and four billion bushels, is the principal corn-producing country of the world. Certain states, like Iowa and Illinois, often produce more than 500 million bushels a year, and production in other states ranges upward from a few hundred thousand bushels. A yield loss from stalk rot of only 7.5% could, therefore, cause a loss of more than $500,000,000.

In most of the corn-growing states the losses are frequently much higher than 7.5%; sometimes they are reported as high as 10-20%. Roane (277) estimated the loss caused by stalk rot in Virginia to be 15-20% of the crop. In Ohio, Williams and Schmitthenner (394) estimated direct loss at 10-15%, plus an additional loss due to unharvested ears, ear rots, and harvesting difficulties. DeVay et al. (63) considered stalk rot the most destructive disease in Minnesota. They estimated the loss at 10%, which amounted to about $35,000,000. Fields with as much as 80% broken stalks were common, particularly in the southwestern part of the state. They found that many plants were severely rotted, even though there were no conspicuous signs of the disease on the plant surface. Stalk rot is the most destructive disease of corn in Iowa. According to Worf and Foley (399) yield reductions of 8 to 16% are not uncommon, and losses up to 25% have been reported. As early as 1915 Pammel et al. (238) reported that 5 to 50% of the corn stalks were broken because of stalk rot. Fields with 90% broken stalks were also reported. Durrell (76) found that the percentages of stalks infected by *Diplodia zeae* varied from 4 to 47%. He estimated the reduction in yield from *Diplodia* alone to be about 10%. Manns and Adams (192) put the corn crop loss in Delaware at 15% for the year 1920. Five to 50% of corn stalks were blown over or broken, and some died prematurely.

From 1930 through 1939, McCallan (196) gives the mean loss due to root rot, stalk rot, and ear rots of corn as 9%, with a range of 4 to 16% for all corn harvested in the United States. McKeen (198, 199) reported that corn root and stalk rots reduced the potential yield of corn 50 to 60% in Ontario, Canada.

Losses within a given state may vary considerably over a period of years. This is well illustrated by work in Illinois. In 1930 Koehler and Holbert (161) presented data on losses due to five pathogens; the losses varied from 6 to 35%, depending on the pathogen. In 1925 Koehler et al. (160) estimated the loss from stalk rot at 1% or more during the 7 years from 1917 through 1923. Some fields had as much as 70% of the plants with stalk and root rot. Hooker and Britton (129, 130) in 1959 reported that percentages of infected stalks in standing corn varied from 18.5% to 82.5%, with an average of 52.6%. In 1960, Koehler (169) considered the average annual loss in yield from stalk rot to be 7 to 10%. Hooker and Britton (130) in 1962 measured an average loss due to premature plant killing over a 2-year period at 8.6%, or a value loss of more than $70,000,000. This did not take into consideration additional indirect losses.

Broken stalks result in large losses because many ears are missed by mechanical pickers, and consequently they are not harvested. Ears on infected stalks also tend to drop off readily during harvesting operations. Ears which come into contact with moist ground frequently become moldy. Moldy ears are of poor quality and may be unfit for feed. In some years losses from broken stalks are minimized by pasturing livestock in the field.

It is hard to determine the actual losses from stalk rot because there apparently is no place where corn is completely free from the disease. The greatest losses probably are indirect and generally overlooked. Even when the damage is obvious, root rot, lodging, poor pollination, poor genetic types, and weather are often blamed.

Stalk rot perhaps causes the greatest damage when it develops in the basal portion of the stalk, particularly in that part which extends below the ground, because here it involves the destruction of the tissues to which many of the roots are attached.

Under certain conditions, much of the water and nutrient taken up by roots does not reach the above-ground part of the plant. This results in barren stalks, smaller ears, or premature drooping of ears, and plants appear to mature early. This damage is most apparent on rolling land and in drier regions, particularly during periods of drought in the later part of the growing season. The potential loss is graphically illustrated in Fig. 1F.

The reduction in yield from the use of infected seed has usually heen attributed to loss of stand (76, 161, 209, 269). Sometimes, however, considerable loss may result from subnormal plants grown from infected seed, or as a result of crown infection originating from infested soil (120).

When determining the losses caused by stalk rot of corn, it is important to consider not only such losses as reduced weight and quality of shelled corn, but also the expenditure of time, labor, and capital in growing and harvesting the crop. According to Hooker and Britton (129, 130), if one could save the cost of cultivation and land occupied by diseased corn plants, the net return per acre of corn grown in Illinois could be increased approximately 28%.

Damage caused by stalk rot may also result in losses in crops grown the year after corn. This is especially true with soybeans (Fig. 1G). Seeds from dropped ears and from ears on broken stalks not picked up by mechanical harvesters may produce volunteer plants. These may be so abundant that a soybean field may have the appearance of a corn field. In such a field, losses in soybean yield not only result from competition, but if volunteer corn is removed before the soybeans are harvested, considerable labor is necessary to get such a field ready. If the corn is not removed, it greatly interferes with the efficient harvesting of the soybeans. If the beans and the immature corn are stored together the moisture content of the beans may be raised and thus increase the danger that the soybeans will deteriorate in storage.

Damage caused by stalk-rotting organisms is quite evident when stalks produce small ears, when the stalks and shanks are broken, or when the plants are killed before the kernels are in the hard-dough stage. Stalk rot is not always so conspicuous. It may reduce the yield of apparently healthy plants. Infected plants frequently develop no obvious external symptoms,

Another important loss that is indirectly related to stalk rot is that which results from seedling blight and scab in wheat and barley. Infected corn stalks are an important source of inoculum of *Gibberella zeae* and *Fusarium* spp. which cause seedling blight and scab of small grains. It is a widely held opinion that wheat and barley production decreased in importance in southern Minnesota after corn became extensively grown, largely because of the scab problem.

Even today there is little or no definite information on the total amount of damage caused hy stalk and basal stem rot. There is some experimental data on direct yield loss in a few states on a limited number of hybrids, but much more is needed under different environmental conditions. Koehler (169) found that stalk rot reduced yield 6-10 bushels per acre in hybrids grown in areas where stalk rot was severe in comparison with the same hybrids in areas where stalk rot was slight. Hooker and Britton (129, 130) obtained an 18% lower yield from rotted but standing stalks and a 27% lower yield from plants both lodged and rotted than from adjacent and apparently healthy corn plants. During a 3-year study Michaelson and Christensen (216) inoculated two hybrids with *Gibberella zeae* and *Diplodia zeae* by means of the toothpick method. The yield of the inoculated plants was reduced 6.8% by *Gibberella zeae* and 9.7% by *Diplodia zeae*. Michaelson (217) used the same method and demonstrated that stalk rot reduced the yield of shelled corn by 2 to 22%, depending on the pathogen, the hybrid, the plant parts inoculated, the number of infections per plant, the time of inoculation, and the season. Wilcoxson (385), using the toothpick technique and *Fusarium graminearum* and *Diplodia zeae*, inoculated 14 hybrids that were about equally susceptible to natural infection. In a 3-year test, the yields were reduced as much as 17%. There was some tendency for rot in the lower internodes to cause the largest reduction in yield. Losses were greater when the hybrids were inoculated 9 to 10 weeks before the harvest, rather than 5 to 6 weeks before harvest.

Studies have shown that necrotic lesions in single internodes may interrupt physiological processes and thus may cause severe reduction in yield. Wilcoxson and his associates (181, 386) found that yield of grain was significantly reduced when necrosis in the second internode involved 50% or more of the tissue, whereas necrosis of less than 50% of the internode tissue caused no significant reduction in yield.

Wilcoxson (385) also noted that in many instances stalk rot was severe and loss was relatively large without any outward sign of the disease. This often introduces another large error in estimating the amount of damage caused by stalk rot.

The location and number of the lesions in the stalk may influence the amount of loss in yield. Inoculation above the shank node reduced the yield about 13% per plant, as did inoculation of lower internodes. Although one rotted internode reduced yield considerably, several lesions per stalk caused even greater yield reduction (218, 385). Troyer (360) measured yield reduction in five hybrids growing in Illinios as 2.3% when *Diplodia zeae* was inoculated into one internode, and 5.4% when inoculations were made in three internodes. Koehler (169) also secured greater losses when multiple inoculations were made.

Reduction in yield may differ considerably from year to year, even when the same hybrids are inoculated with the same fungus isolate (218, 385). For example, Michaelson (218) recorded 5.5% loss in yield for Minnhybrid 607 in 1951, but in previous years the loss ranged from 5.4% to 21.5%.

Stalk rot was caused by several fungi in France, but that caused by *Colletotrichum graminicolum*

(Ces.) Wils. was particularly destructive (212, 214). In Egypt *Cephalosporium maydis* Samra, Sabet, and Hingorani sometimes causes severe losses, infection sometimes reaching 25% in some fields (298).

Bacterial stalk rot of corn is sometimes destructive in certain regions and countries. Rosen (283 to 287) was the first to report that bacteria could cause severe stalk rot and damage to corn. He reported losses as high as 30% in some regions in Arkansas. In general, bacterial stalk rot of corn in the USA is not economically important, but it has been reported to be destructive in Egypt (290) and India (114). Hingorani et al. (114) reported that bacterial stalk rot caused from 15% to 33% damage in certain American varieties of corn grown in India.

Losses may also result from feeding infected plant tissue to animals. Billings (20) and Moore (227) called attention to frequent and widespread outbreak of a disease among cattle known as "corn-stalk disease." This disease occurred when the cattle were feeding on corn stalks in the field. Although Moore made numerous attempts to determine the possible cause of the "corn-stalk disease," his results were inconclusive. He did, however, isolate a bacterium from corn similiar to the one described by Burrill (34), which causes stalk rot of corn, and this bacterium was fatal to certain animals that were inoculated with a pure culture. During Moore's time, fungi, with exception of *Ustilago maydis* (D.C.) Cda., were apparently not suspected to be disease-producing agents of animals and, therefore, were ignored.

Peters (254) suggested in 1904 that a serious disease of animals, especially horses, cattle, and swine, feeding on corn stalks, was probably due to *Fusarium* sp. Hunt (136) isolated several species of *Fusarium* but concluded that *Fusarium moniliforme* was the dominant species present in the stalks.

Diplodia zeae (111) was reported in 1906 to be very common in corn in certain parts of Nebraska, and was considered to be the cause of poisoning of horses in Kansas (108). Feeding tests with moldy corn as an exclusive grain ration for 2 months, however, gave negative results. Smith and Hedges (330) suggested that *Diplodia zeae* might be the cause of the so-called corn-stalk disease so prevalent among cattle in the west. They also indicated that *Diplodia zeae* might be associated with the so-called pellagra which caused many deaths among poor people following the consumption of moldy corn and hominy. Mason (194) reported that pellagra may be due to *Nigrospora* spp.

The farmers in Africa, according to Bijl (19) and Mitchell (224), thought that *Diplodia zeae* caused paralysis and death of stock fed infected cobs of corn. Sheep were reported to be particularly susceptible. Evans (92) also found in Africa that all cobs contained *Diplodia zeae;* later *Fusarium* spp. were also isolated from other specimens. From certain other localities *Fusarium* spp. were the prevalent organisms. He noted that the diseased ears made very undesirable beer.

Bijl (19) fed ears of corn infected with *Diplodia zeae* to a limited number of animals for several weeks and observed no ill effects. He also grew the organism on crushed maize for weeks and then fed it to cattle without any ill effect. Extracts from cultures were injected into mice and rats without harmful effects.

Mayo (195) found that "staggers" of horses in Kansas was due to the animals consuming corn infected with *Aspergillus glaucus* Link. This fungus is not a primary cause of stalk rot, but it and its relatives commonly grow on organic materials such as improperly stored feed.

Christensen and Kernkamp (44) found that ground kernels from ears which were artificially inoculated with *Gibberella zeae* in early milk stage were very toxic to swine. In fact, the animals would not eat the corn unless it was mixed with other feed. Extract from diseased grain also proved toxic, hut not cultures grown on grain mixture. Sippel (325) reported that moldy corn caused rapid death of swine.

It is apparent from the above review that there is much to be learned about the effect of microorganisms on animals. All feeds are infested with many different species of microorganisms, including many plant pathogens. More work should be done to learn how these organisms influence the growth, longevity, productivity, and reproduction, as well as the psychology and behavior of animals.

Pathogens, Symptoms, and Distribution

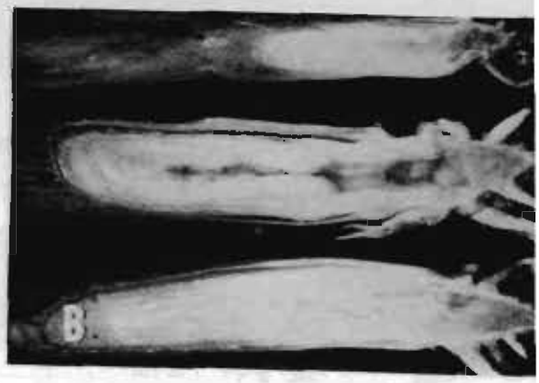
Almost all major pathogens that cause stalk rot of corn are found in virtually every country and region where corn is grown. A few pathogens are destructive, at least as far as is known, only in certain regions. Many of the pathogens have been isolated from all parts of the corn plant: roots, crown, or hasal part of the stem below the soil level, seeds, leaves, and stalks. By far the greatest number of isolations have been made from seedlings and kernels. Many of the secondary invaders are also world wide in distribution.

Many investigators have proved that certain species

——→

Figure 1. **A.** Corn stalks showing light brown to black lesions caused by *Gibberella zeae*. Similar lesions are also produced on corn stalks by other pathogens. **B.** Crowns and stalks of young corn plants with internal necrosis caused by *Bacterium stewartii*. **C.** A field of corn near Redwood Falls, Minnesota, showing about 90% of the plants lodged because of stalk rot. **D.** Plants which died prematurely (left) because of inoculation with *Diplodia zeae*. Living plants (right) were not inoculated. **E.** Seedling blight of corn at 25° C when plants were inoculated with isolates of *Fusarium graminearum* from Missouri = 9, Minnesota = 8, New York = 7, and disease free = 6. **F.** Corn with severe stalk rot (upper) and healthy (lower) showing the reduction in ear size due to stalk rot. **G.** Volunteer corn in a field of soybeans in southern Minnesota. **H.** Corn plants showing extensive injury caused by corn root worm (left). (Photos A, B, and D. courtesy of B. Koehler (169), University of Illinois; F and H, courtesy of H. G. Johnson, University of Minnesota.)

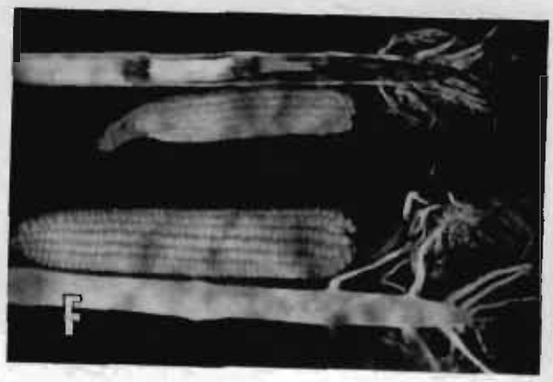

A


B


C

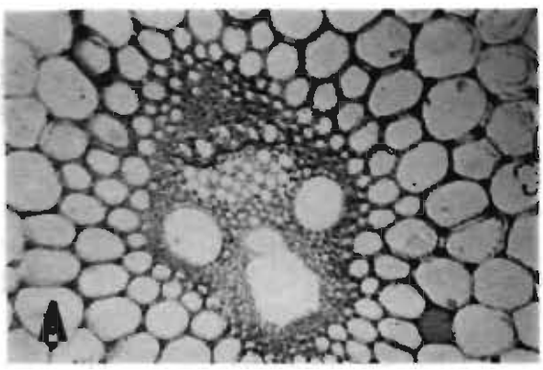

D

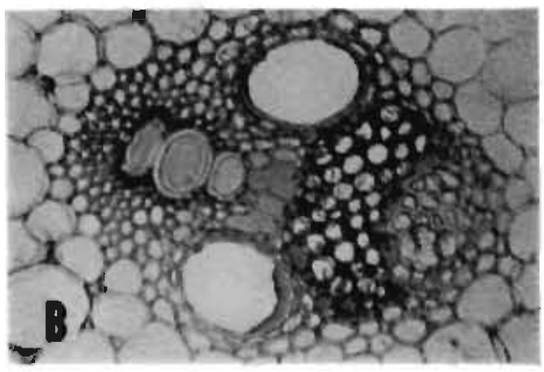

E
6
7
25
8
9

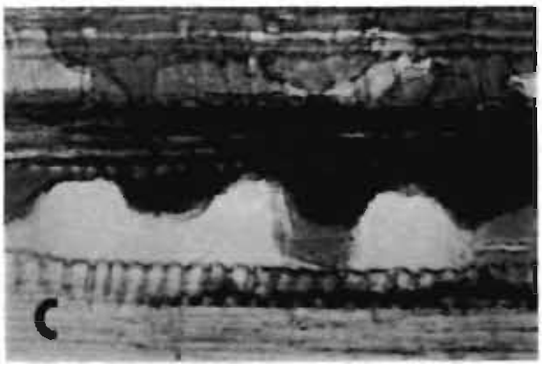

F

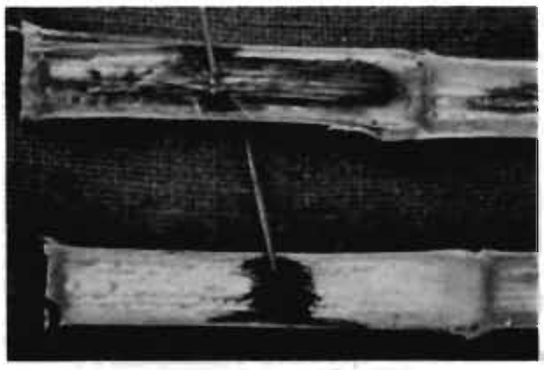

G

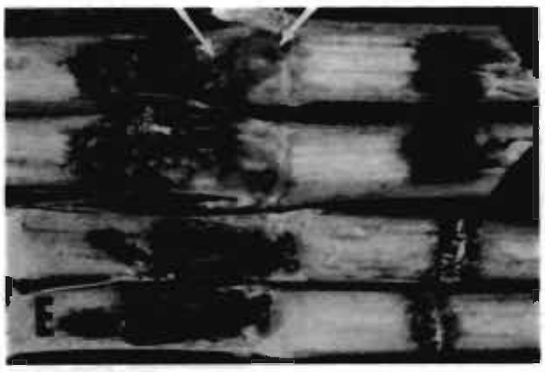

H


A

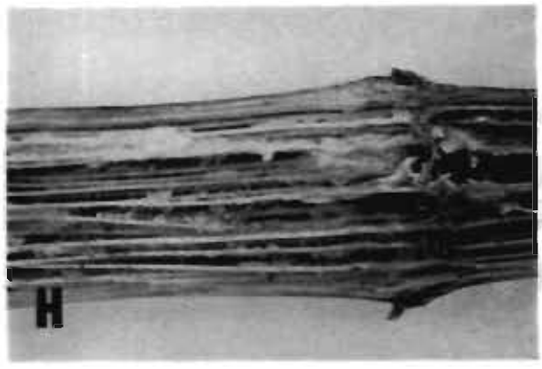

B


C




E


F


H

of fungi can cause severe stalk rot when plants are inoculated. Haws (109) proved that at least 11 strains of *Fusarium* cause stalk rot. Young (402) produced severe stalk rot with a *Penicillium* sp. and a small-spored *Helminthosporium* sp. Williams and Willis (395) did it with *Collectotrichum graminicolum*.

Manns and Adams (192) found that many pathogens were carried internally in the kernels, where seed treatment could not reach them. They found *Gibberella zeae, Fusarium moniliforme, Diplodia zeae,* and *Cephalosporium* sp. present in seed in a very high percentage of the samples from 21 states. *Fusarium moniliforme* was the prevalent species isolated. They considered *Cephalosporium sacchari* Butler an active corn root and stalk parasite.

Porter (266, 267) isolated fungi from corn nodes collected in 20 different states. *Fusarium* spp., *Diplodia zeae, and Penicillium* spp. were among the most common fungi. Some of the *Penicillium* spp. undoubtedly were saprophytes. It is, of course, possible that secondary invaders may help to break down the stalk tissue and thus contribute to stalk rot and stalk breakage.

Peterson (255, 256) isolated many species of fungi in 7 genera from corn stalks in New Jersey. Sixteen of these species caused stalk rot when the organisms were artificially introduced into the stalks of the susceptible lines of corn. A species of *Trichoderma* was the most aggressive, even more so than isolates of *Gibberella zeae* and *Fusarium moniliforme*. Furthermore, certain isolates of *Fusarium* spp., *Curvularia* spp., and *Helminthosporium sativum* Pam., King, and Bakke were about as pathogenic as *Gibberella zeae*. Certain isolates of *Mucor* and *Penicillium* also caused rot.

Wood (398) made over 700 isolations from roots and basal portions of stems of corn of 24 single-crosses, during two growing seasons in Ohio. More than 70% of the isolates were unidentified species of *Fusarium*, 20% were *Fusarium graminearum*, 2% *Trichoderma* spp., less than 1% were *Diplodia zeae*, and the remainder were unidentified.

Gibberela zeae and Fusarium spp.—In the absence of fruiting bodies there is no definite symptom on the stalk or root by which one can differentiate infection by *Gibberella zeae* and *Fusarium* spp. from that by *Diplodia zeae*. The initial symptoms produced by these pathogens appear soon after pollination as light brown to almost black lesions near the lower nodes (Fig. 1A). Lesions caused by *Gibberella zeae* and *Fusarium* spp. may sometimes have concentric rings, and sometimes they may also extend away from the node as a long streak. *Fusarium moniliforme* often attacks plants earlier in the season than do the other pathogens, and it is often associated with injuries. Lesions produced by *Fusarium moniliforme* frequently have less distinct margins than have those produced by *Gibberella zeae* or *Diplodia zeae*. When badly infected stalks are cut open, pink-colored mycelium and stalk tissue may indicate the primary pathogen to be *Gibberella zeae* or a species of *Fusarium*. Isolation techniques should be used with observed symptoms to determine the identity of the pathogen.

If conditions are favorable for rapid development of parasites shortly after silking, the plant may be killed prematurely. Such plants wilt suddenly and present a more or less frosted appearance (Fig. 1D). The leaves eventually become dry and shatter easily. Plants killed prematurely not only yield poorly, but the ears are chaffy and lack proper luster.

Gibberella zeae is one of the most important stalk-rot pathogens of corn. Its distribution is world wide (13) and it attacks and multiplies on a great many different kinds of plants. In the United States it appears to be most common on corn in the cooler growing areas of the corn belt, and along the Atlantic seaboard. It has been reported by several investigators to be the most destructive of the stalk-rot organisms in New York, New Jersey, Minnesota, Pennsylvania, and Canada (26, 37, 63, 380, 381).

The prevalence of *Gibberella zeae* varies tremendously from year to year. Even in regions where *Diplodia zeae is usually the predominant organism, Gibberella zeae* occasionally becomes the more destructive pathogen. In most years it is common in the corn belt (63, 168). Koehler and Boewe (168) reported a survey of the relative prevalence of *Gibberella zeae* and *Diplodia zeae* as causal agents of stalk rot in Illinois from 1946 through 1956. *Gibberella zeae* was more prevalent than *Diplodia zeae* in 1946 and 1955, and in 3 other years the pathogens were virtually equal in prevalence. For the 11-year period the average percentage of infected stalks for *Gibberella zeae* was 26 and for *Diplodia zeae* was 31. The percentages of *Gibberella zeae* infected stalks ranged from 18 to 37, whereas *Diplodia zeae* varied from 18 to 53. *Gibberella zeae* is also one of the most important pathogens to cause seedling blight and root rot of corn, and this may be a factor in its widespread importance as an incitant of stalk rot.

Fusarium moniliforme is widely distributed throughout the world on corn, and is perhaps best known as a cause of ear rot. However, it is a common

---

Figure 2. **A, B.** Cross sections of vascular bundles near stalk rot lesions developing in the pith of a corn plant inoculated with *Fusarium graminearum*. Note in A, the dark walls of the parenchyma cells and the accumulation of dark substances in the intercellular spaces, and in B, the necrosis and accumulation of dark substances in the phloem, vessels, tracheids, and protoxylem lacunae. **C.** Longisection of a vascular bundle near a stalk-rot lesion developing in the pith of a corn plant inoculated with *Fusarium graminearum*. Note the dense dark substance deposited in the vessels. **D.** Corn stalks inoculated with *Fusarium graminearum*, showing the type of lesion that develops in a susceptible (upper) or a resistant plant (lower). **E.** Necrosis of corn stalk tissue when corn borer larvae were present (upper two internodes) or absent (lower two internodes). Larvae hatched from eggs placed in cavities cut in the first and second internodes above the ground. **F.** Stalk rot that developed in corn injured by hail. **G.** Stalk rot of the lower portion of a corn stalk. The rot apparently spread into the stalk from the roots. **H.** Radial section of a corn stalk with severe stalk rot which originated at the nodes.

stalk-rot organism (158, 168, 200, 277, 352), and may also cause premature death of corn plants. In fact, Koch and Murwin in Canada (158) considered it more virulent than *Gibberella zeae*. Roane (277) concluded that *Fusarium moniliforme* in certain years was a major cause of stalk rot in Virginia, Boosalis (unpublished data) concluded that it is the major pathogen involved in the rotting of corn stalks in the irrigated regions of western Nebraska. However, Koehler (169) considered it of minor importance in comparison with *Diplodia zeae* and *Gibberella zeae*. Valleau (368) indicated that *Fusarium moniliforme* was a primary cause of root and stalk rot of corn in Kentucky and other central and southern states. Leonian (177) considered it a primary cause of seedling blight. Manns and Phillips (193) reported it to be just as virulent as *Gibberella zeae*. Porter (266, 267) reported that *Fusarium moniliforme* invades the stalks shortly after pollination, but that it was usually not destructive. Although Ullstrup (364) considered it a secondary invader, Young (402) demonstrated that *Fusarium moniliforme* caused severe stalk rot when inoculated into the plant by the toothpick method. Others have obtained similar results. Although many workers consider *Fusarium moniliforme* to be a wound parasite on older plants, there is good evidence that it often is found in older plants that were infected during the seedling stage (169).

Foley (99) doubted that *Fusarium moniliforme* is primarily a secondary invader. He isolated the organism from kernels, roots, leaf sheath, stalk, and axillary buds. It was most frequently isolated from the leaf sheath, and from nodes more frequently than internodes. It becomes much more common on these sites as the plants mature.

Kingland (155), working in Pennsylvania, found *Fusarium moniliforme* to be the most prevalent fungus in 1955, but in the next 2 years *Fusarium moniliforme* Sheld. var. *subglutinans* Wr. and Rg. was the most common. Niederhauser (232) and Ullstrup (364) reported that this disease is more abundant in dry seasons, especially in the parts of North America where dry weather usually prevails during the growing season. *Fusarium moniliforme* apparently will develop in a drier environment than will either *Gibberella zeae* or *Diplodia zeae*.

Fusarium moniliforme var. *subglutinans* was observed by Edwards (84) in Australia. He found perithecia on old corn stalks and proved the fungus to be pathogenic on seedlings and corn stalks. This organism was found on old corn stalks in New Jersey by Ullstrup (364). The distribution of *F. moniliforme* var. *subglutinans* in the USA is not known. According to Ullstrup (363) it may have been overlooked or mistaken for *Gibberella fujikuroi* (Saw.) Wr., or perhaps it was recently introduced into this country. In recent years it has been shown to be fairly common in the USA and other parts of the world (155, 158, 169, 178, 229, 311, 348, 372).

Fusarium moniliforme and *Fusarium moniliforme* var. *subglutinans* have been used in this manuscript, rather than *Gibberella fujikuroi,* because ascigerous stages have seldom been seen by most investigators. It is not, furthermore, always clear whether the various authors have made a serious attempt to distinguish between the two organisms. The use of the conidial names seems reasonable and proper according to international rules for nomenclature (173). The ascigerous stage of these two fungi has been reported from several states and from other parts of the world (85, 277, 363).

Pammel et al. (238) were, in 1915, the first to call attention to the important role of *Fusarium* spp. in causing stalk rot. Since then these fungi have been isolated from corn throughout the world. Artificial inoculation has proved that they cause severe seedling blight, root and stalk rot of corn. There always is the possibility that some isolates were confused with *Fusarium graminearum* or *F. moniliforme*.

Holbert and Hoffer (123, 124) considered *Gibberella zeae* the most harmful organism associated with root, stalk, and ear rots. Porter (266, 267), DeVay et al. (63), Littlefield (179), Chiang and Wilcoxson (40), and Peterson (256) found *Fusarium* spp. more common than *Gibberella zeae* or *Diplodia zeae*.

Diplodia spp.—Symptoms on stalks cannot easily be distinguished from those produced by *Gibberella zeae* or species of *Fusarium,* and the problem has been discussed in the section dealing with *Gibberella zeae*. It is possible, however, to judge the importance of *Diplodia zeae* in stalks if the rot spreads through the shank into the ear. This happens frequently with *Diplodia zeae;* the ear becomes covered with white mycelium and the rot spreads from the base of the ear to the tip.

Of four species of *Diplodia* which occur on corn—*Diplodia zeae* (Schw.) Lev., *D. macrospora* Earle, *D. frumenti* Ell. and Ev., and *D. tubericola* E. and E., (19, 79, 80, 82)—*D. zeae* is by far the most common species in the USA. According to Hoppe (131) *Diplodia zeae* is more aggressive than *Diplodia macrospora* when the ears of corn are artificially inoculated. *Diplodia macrospora* is prevalent in the eastern part of the USA. *Diplodia zeae* occurs in the USA to some extent almost throughout the range of corn. It is most common and destructive in the central part of the corn belt, and least prevalent in northern and eastern parts and drier areas of the USA. It is not common in Canada.

Hoppe (132 to 135) found *Diplodia zeae* present on damaged kernels of corn coming to the market from all the corn-growing regions in the USA. Porter (267) found *Diplodia zeae* in the nodal tissue of corn stalks from 24 states in the eastern half of the USA, whereas several other investigators rarely or never found *Diplodia zeae* associated with stalk rots in Virginia, New York, and Ontario, Canada (26, 37, 277, 380, 381). Damage from *Rhizoctonia zeae* Voorhees might easily be confused with that caused by *Diplodia zeae* according to Voorhees (374).

Cephalosporium spp.—Cephalosporium acremonium Cda. is not uncommon on corn in the United States, sometimes causing considerable damage in some states. Although the damage is not always ap-

parent as stalk rot, *Cephalosporium acremonium* is often classed as a stalk-rotting organism (273). Although some consider it a weak pathogen (1, 107, 273), Taylor (352) obtained good infection with *Cephalosporium acremonium* by inoculation. Recently, Koehler (169) also stated that it was an active parasite. Harris (107) found that *Cephalosporium acremonium* apparently filled the vascular bundles with a gum-like substance; he considered it an active though weak pathogen.

Black-bundle disease is caused by *Cephalosporium acremonium*, and primarily attacks vascular bundles, but the pathogen also causes stalk rot and certain other symptoms that are similar to those induced by other stalk-rotting organisms. The disease is usually not apparent during the first half of the growing season, but as the season progresses, reddish coloration occurs on the leaves and stalks, and there is frequently excessive tillering. Barren stalks or small ears are common. The rot which may develop is usually limited and is not a good diagnostic character. The symptom most common is conspicuously blackened vascular bundles which may extend through several internodes and nodes.

Cephalosporium maydis causes a rot of the lower portion of the plant in Egypt (298, 299). It differs from other species in the shape and size of conidia and conidiophores, the color and type of colony, temperature requirement, and is the only one that causes wilt of maize. In Egypt, the disease is widespread, and sometimes 25% of the plants in a field are affected. The first symptom of the late wilt caused by *Cephalosporium maydis* is a moderately rapid wilting of the corn plant just prior to tasseling or shortly before maturity. The wilting progresses from the lower to the upper portions of the plant. The leaves are at first dull green, then they turn yellow and eventually become dry. The vascular bundles in the stalk are reddish brown, and eventually the nodes turn this color. In advanced stages the lower internodes become dry, shrunken, and hollow. Frequently a wet rot develops in the lower part of stems infected with *Cephalosporium maydis*. This is induced by the invasion of secondary fungi and bacteria.

Nigrospora spp.—*Nigrospora oryzae* (B. and Br.) Petch and *N. sphaerica* (Sacc.) Mason have both been reported on corn (194). Standen (337 to 340) found great differences in size of spores and cultures among isolates, however, and concluded that it was most difficult to separate the two species. He referred to all Iowa collections as *Nigrospora oryzae*.

Nigrospora oryzae has been known as *Basisporium gallarum* Moell., and the rot caused by the fungus has been known as *Basisporium* dry rot (33, 194, 226). It is most common on ears, husks, and shanks, but also attacks stalks. The lesions appear on stalks as the plant approaches maturity, and most infection occurs on the lower internodes. The lesions are black and shallow, and not necessarily associated with a node. When the tissue from a lesion is placed in a moist chamber the fungus sporulates profusely. Usually ear rot is not evident until harvest, and then the presence of the fungus is manifest by the presence of black spores on the ears. Shanks are weakened by the fungus, and thus the ear is easily knocked from the plant. Exposure of the plants to early frost or low temperatures predisposes the stalks to attack. Usually the fungus is not of great importance on stalks, although epidemics have been reported (78, 169, 272, 303, 304). *Nigrospora oryzae* is found in many different parts of the world: USA (11, 78, 337), Bulgaria (33), France (226), Romania (302 to 304), and South America (340). It has also been reported on tomato (270).

Sclerotium bataticola Taub.—This is the sclerotial and mycelial stage, and *Macrophomina phaseoli* (Maubl.) Ashby is the pycnidial stage of the fungus that causes charcoal rot of corn. There are sterile and fertile forms (12, 105). It was first reported on corn in California in 1932 by Mackie (188), and it is most important in the USA in the warmer and drier areas (119, 175, 182, 317, 319, 405, 406). It also occurs in India (355), Egypt (294), and Argentina (32, 301).

Charcoal rot first becomes apparent as the corn plant begins to mature, although seedlings may be infected (319). Once infection is well established, it progresses rapidly. The disease is usually more or less limited to the lower part of the stalk, but it may extend into much higher internodes. The pathogen causes premature ripening and often results in stalk breakage at the crown. The surface of the diseased lower nodes and internodes usually turns gray and then dark brown, and eventually innumerable small black sclerotia are produced beneath the epidermis. When the diseased stalk is cut open, the pith is usually disintegrated, leaving only the vascular strands intact and virtually covered with small black sclerotia. The disease can be identified readily by the innumerable sclerotia on the vascular strands. The roots are frequently invaded and contain sclerotia in the diseased tissue. Although there are other stalk-rotting organisms present, the presence of *Sclerotium bataticola* is not difficult to detect (Fig. 3C, D).

Pythium spp.—Although *Pythium* spp. are better known as causes of seedling blight, they also cause severe stalk rot (169). Branstetter (30) was one of the first to call attention to these organisms as corn pathogens, and his report was soon followed by others (67 to 69, 143, 271, 369). These investigators proved pathogenicity, studied taxonomy, and measured genetic variability and physiological capacity of the various species involved. More recently, sources of resistance have been sought (89, 127, 320).

Stalk rot due to *Pythium* was first noticed by M. T. Jenkins in Virginia in 1940, according to Elliott (90). She found that *Pythium butleri* Subr. was the pathogen, and determined that it was most virulent in hot moist climates and that some lines of corn were resistant. *Pythium* stalk rot is most prevalent in fields near river bottoms or in fields flooded by rivers (90, 219, 370). Since 1940, *Pythium* stalk rot has been found in a number of different areas: Virginia (90), Iowa (Hooker unpublished), Kentucky (370), Indiana (365), Venezuela (264), Canada (199).

A number of species have been reported on roots and stalks of corn: *Pythium aphanidermatum* (Eds.)

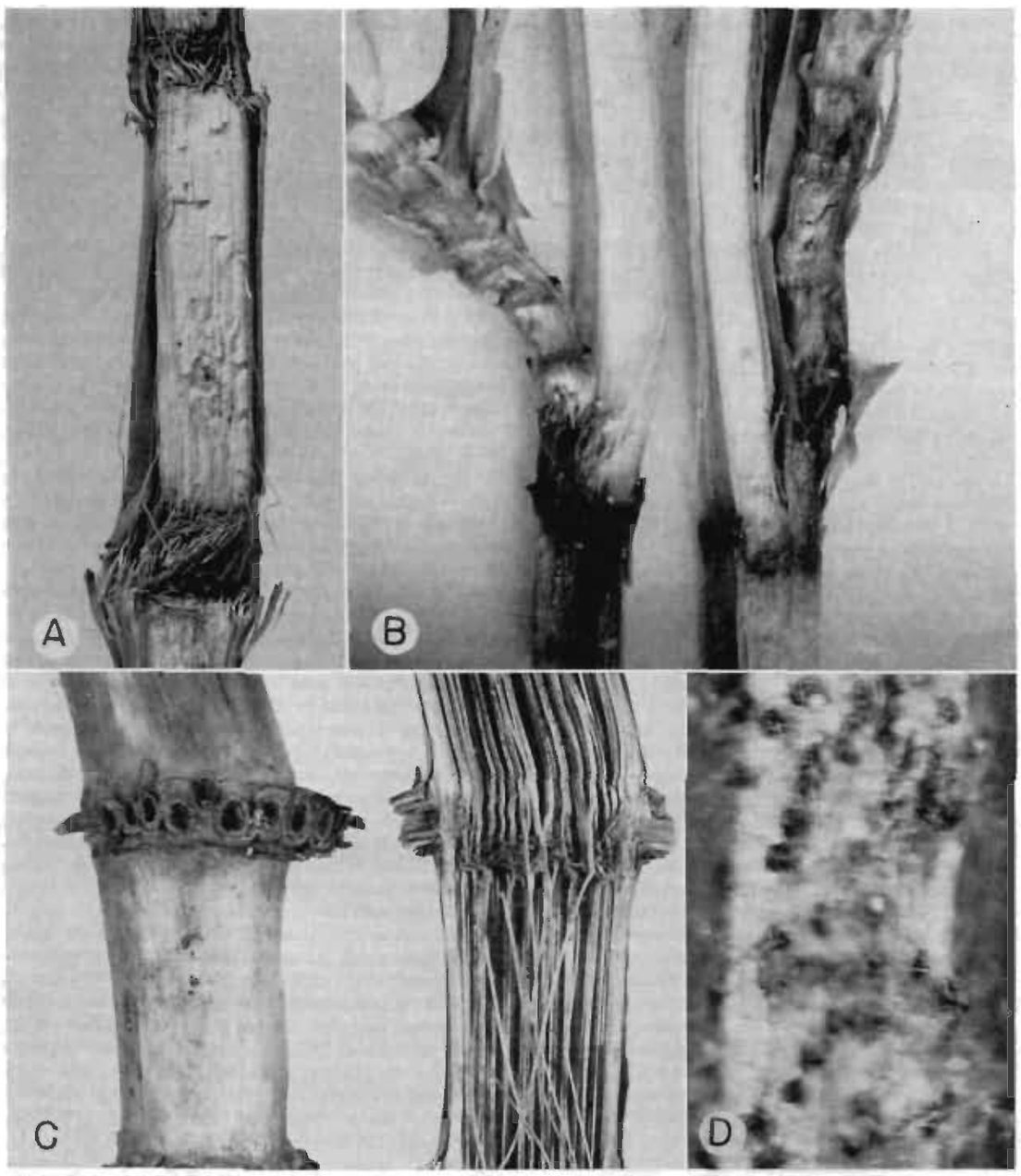


Figure 3. **A.** Nodes of a corn plant disintegrated by fungi. Note that the internodal tissue has not yet decomposed. **B.** Corn plants with stalk rot in the nodes and the shanks. **C.** Charcoal rot of corn, showing the light-colored surface of a lower infected internode and the internal appearance of a badly rotted stalk. **D.** A single vascular strand from C, showing sclerotia. (Photos C and D, courtesy of T. D. Wyllie, University of Missouri.)

Fitz. (264), *P. butleri* (68, 90, 169), *P. arrhenomanes* Drechs. (67, 143), *P. debaryanum* Hesse, *P. graminicolum* Subr., *P. irregulare* Buis., *P. paroecandrum* Drechs., *P. rostratum* Butl., and *P. splendens* Braun (69, 127, 320).

Pythium stalk rot usually becomes obvious shortly before or after corn has tasseled. The rot usually extends only a few internodes above the ground and develops rapidly. Infected stalks become soft and fall over while still green, and may remain turgid for several days. Elliott (90) reported that inoculated tissue may collapse in 4 or 5 days after inoculation, and then the plants fall down. There is usually a twisting of the stalk (Fig. 4), as happens with bacterial stalk rot (Fig. 5). In fact, *Pythium* and bacterial stalk rot are often confused. Bacteria are often associated with

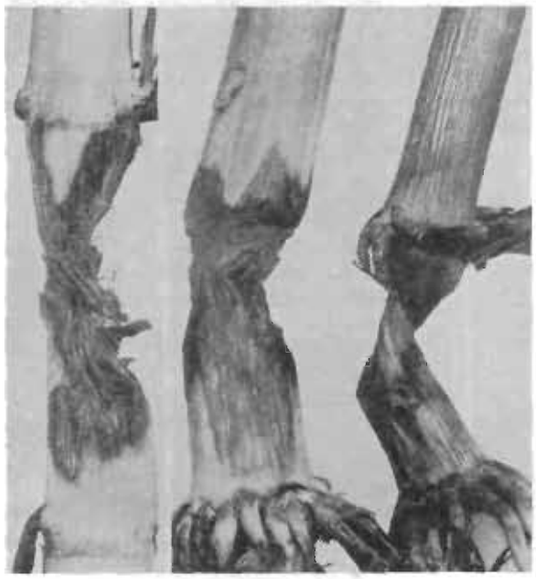


Figure 4. Stalk rot caused by *Pythium butleri*. The left stalk has been opened to show the extent of the necrosis. (Courtesy of University of Illinois (169).)

Figure 5. Stalk rot caused by *Erwinia dissolvens*. (Courtesy of University of Illinois (169).)

Pythium rot (23). The infected areas are water soaked, with brown tissue at the margin, and usually with a sharp line of demarcation between them. The rind of the stalk, the epidermis, lignified cells of the hypodermis, and the inner cells become softened and disorganized, leaving only brown vascular bundles intact. The vascular bundles apparently continue to function for several days after the plant has fallen to the ground.

Elliott (90) states that plants about 1 meter tall are most susceptible. Young (401) and others (128, 169) have shown that plants are very susceptible when inoculated after silking. McKeen (199) found *Pythium arrhenomanes* present on seedling plants as well as in crowns and basal stem portions of older plants during most of the growing season.

Bacteria.—Bacterial stalk rot of corn was first described by Burrill (34) in 1889. The rot was most prevalent on low fertile ground. He reported the rot to be most common on roots and basal portions of stalks. The infected plants became stunted and chlorotic, and the severely infected plants died. Burrill's description of the disease is not the same as that now attributed to bacterial pathogens. Bacterial root rot also occurs (91).

Rosen (283, 285 to 287), between 1919 and 1926, described a root and stalk rot caused by *Phytomonas dissolvens* Rosen, now called *Erwinia dissolvens* (Rosen) Burkh. Taxonomy has also been studied by Waldee (375, 376). Stalk rot has been reported from several states and countries: Nebraska (70), Illinois (23, 219), West Virginia (341), Arkansas (283), California (8, 9), Italy (174), Russia (282), Israel (371), and Canada (200). In 1960 one variety of corn, Virginia hybrid 42, when grown in Russia, had up to 20% bacterial stalk rot (282). Severe bacterial stalk-rot damage has been reported by Rosen as well as by Kelman et al. in the USA (153), by Sabet in Egypt (290, 296), by Ludbrook (186) in Australia, and by Hingorani et al. (114) in India.

Another type of bacterial rot of corn caused by *Phytomonas lapsa* Ark has been reported from California by Ark (8, 9). Whether or not this organism differs from the one described by Rosen has been questioned. Johnson et al. (148) also described a bacterial leaf-spot and stalk-rot pathogen, *Pseudomonas alboprecipitans* Rosen, which occurred in several southern and central states of the USA. Symptoms were similar to those described by Rosen.

Boewe (22) reported that a bacterial stalk rot apparently caused either by *Erwinia carotovora* (Jones) Holland or *E. dissolvens* has been observed in Illinois since 1922. Although at the time of his report it had been prevalent, in recent years especially in southern Illinois, it was of minor importance.

Stewart's disease of corn, caused by *Bacterium stewartii* E.F.Sm., is primarily known as a leaf disease. The pathogen, however, also causes a limited amount of stalk rot (Fig. 1B). It produces cavities in the stalk which are caused by the collapse of tissue. The lesion is at first chlorotic, but eventually it is necrotic and may even be confused with lesions induced by other pathogens. Severe leaf infection by *Bacterium stewartii* is known to predispose plants to stalk rot caused by *Diplodia zeae* and *Gibberella zeae*, and likely by certain other organisms.

In 1930 Prasad (268) reported a bacterial stalk rot of corn attributed to *Erwinia dissolvens* near Pusa, India, but it apparently was not again reported in India until about 1959, when it was considered to be destructive on several varieties of corn in the principal corn-growing areas of India. Hingorani et al. (114) thought that the pathogen was similar if not identical to *Erwinia carotovora* f. sp. *zeae* previously described in Egypt by Sabet (290).

A bacterial stalk rot of corn was first reported in Egypt by Samra in 1953 (297). Then in 1954 Sabet (290) identified the pathogen as *Erwinia carotovora* (Jones) Holland f. sp. *zeae* Sabet. Sabet (292) considered the bacterium causing stalk rot of maize in

Southern Rhodesia as *Erwinia carotovora* f. sp. *zeae*. He also suggested that this organism may also be the cause of top rot of maize in Australia (186). Stakman (336) has indicated that this organism might be present in Mexico; at least symptoms are similar, and a bacterium is also present in rotten plants in Mexico.

Bacteria are also commonly associated with many of the pathogens that cause stalk rot. They are particularly common with *Pythium* spp. and *Cephalosporium* spp. Most workers consider them to be secondary invaders, but it is likely that they are far more important in the development of stalk rot than is generally recognized.

Bacteria generally cause rather conspicuous rotting of the stalk. The affected stalk usually topples over, twisting as it falls (Fig. 5). The severe twisting of the stalk is one of the best means of recognizing the disease in the field. The bacteria attack young as well as adult plants. The basal internodes of the stalk disintegrate and become a soft mass, thus causing the stalk to fall over. The rot is mostly restricted to the interior of the stalk, although it may also occur on the outer portions. The pith tissue is often reduced to a slimy mass and frequently emits an unpleasant odor. Some assume that this foul odor is due to secondary invaders. The rot may progress up the stalk to a limited extent and may involve the leaves, sheath, ear shank, and husk.

The infected areas are at first water-soaked, they often become brown to blackish, then dry and die. Also the affected stalks usually break between either the first or second internode. Kelman et al. (153) noted that when overhead irrigation is used, plants frequently collapse at the fourth and fifth internodes. They also reported that rot progressed rapidly and that plants collapsed 3 to 5 days after inoculation.

Ludbrook (186) in Australia described a bacterial disease of corn known as top rot. The pathogens were not named. This disease made its appearance about a month before the corn tasseled. The stem apex, immature tassel, and bases of the topmost leaves were involved in a soft wet rot which emitted an offensive odor. When the stalks were split longitudinally a gray or brown water-soaked rot of the parenchyma was observed to extend downward from the apex of the stem. The ear and shank were also attacked. The apical growth was arrested and no tassel or grain developed.

Phaeocytosporella zeae Stout.—*P. zeae* attacks the basal portion of the stalk. The symptoms resemble *Diplodia* stalk rot. Pycnidia, like those of *Diplodia zeae*, break through the epidermis when they become mature. Pycnidia are elongated, not round like *D. zeae*. The ostiole is long in *P. zeae*, whereas it is round in *D. zeae*. In addition, the spores of *P. zeae* are nonseptate and oval in shape. Not much is known about the damage produced (169). It has been reported from the USA (52, 169, 346) and France (212), but it is of little economic importance.

Pyrenochaeta terrestris (Hansen) Gorenz, Walker, and Larson.—*P. terrestris* occasionally causes root and stalk rot of corn (52, 145, 169). Although it [illegible] of plants, it is not considered an important pathogen of corn. *P. terrestris* attacks primarily the basal portion of the stalk, particularly the part below the soil. The lesions, especially those below the soil surface, appear as dark brown blotches. As the plant approaches maturity, the dark blotches often become blended with reddish areas. Identification is difficult without making appropriate isolation (169).

Secondary Organisms.—It has been repeatedly pointed out that many species of fungi and bacteria belonging to many genera can be readily isolated from corn with stalk rot (63, 109, 256). Most of these organisms are usually considered to be secondary invaders, although some can cause stalk rot of living plants if injected into the stalks. Some of the more common genera represented are: *Alternaria, Aspergillus, Botrytis, Cephalosporium, Chaetomium, Colletotrichum, Cladosporium, Curvularia, Fusarium, Helminthosporium, Mucor, Penicillium, Physalospora, Rhizoctonia, Rhizopus, Spicaria,* and *Trichoderma.*

It is well known that *Penicillium* spp. cause moldy corn and seedling blight. Christensen (unpublished data) and Young (402, 403) clearly demonstrated that *Penicillium oxalicum* Currie and Thom caused severe stalk rot of corn which resembled that incited by *Diplodia zeae* and *Gibberella zeae*. It is, of course, a common secondary invader of corn. *Penicillium* spp. have frequently been isolated from mature corn stalks and from injuries in living stalks. Whether or not they all can enter living uninjured stalks is not definitely known. *Penicillium oxalicum* can kill stalk tissue in advance of the mycelium. Perhaps by this method it may also be a primary pathogen in the stalk.

Although *Trichoderma* spp. have been isolated from corn stalks, they have usually been considered saprophytes. In New York, however, they may cause severe stalk rot. Haws (109) found certain isolates which were more pathogenic than many of the *Gibberella zeae* isolates. The rot was similar to that caused by *G. zeae* hut it was somewhat drier. The decay also has a tendency to progress to the outer cortical region of the stalk and the nodes. There is no information about natural infection of corn with *Trichoderma* spp.

Several species of *Helminthosporium* have been isolated from corn and proved to cause stalk rot. Koehler and Boewe (168) isolated *H. carbonum* Ullstrup from both green and dead stalks. Haws' (109) results indicated that it causes more severe stalk rot than *Fusarium moniliforme*. Koehler and Boewe (168) consider *Helminthosporium carbonum* to be associated with senescence of the corn plant. Corn was not infected when sprayed with *H. sativum* Pam., King, and Bakke (41) but subsequent work by Christensen (unpublished) has established that *H. sativum* is pathogenic to corn when injected into stalks (Fig. 6). It is worth noting that, according to Robles (281), not all races of *H. sativum* attack corn. Young (402, 403) demonstrated that *Helminthosporium* sp. caused severe stalk rot when the corn plants were injected with the fungus at about the time of silking. Peterson (256) found that certain isolates

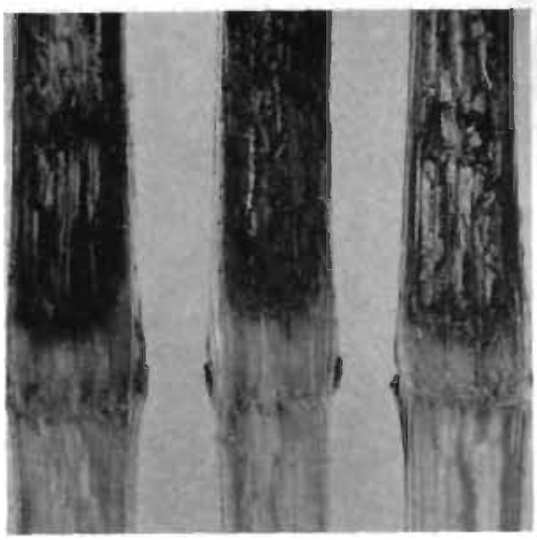


Figure 6. Stalk rot caused by inoculation with *Helminthosporium sativum*.

of both *Helminthosporium sativum* and *Curvularia* spp. were about as pathogenic as *Gibberella zeae*. *Helminthosporium turcicum* Pass. is a common leaf pathogen of corn and is reported to cause stalk rot under some conditions (51, 281, 403).

Although *Colletotrichum graminicolum* is usually a leaf parasite rather than a stalk-rot pathogen, it does cause severe stalk damage in certain regions. Williams and Willis (395) considered it a common pathogen of corn in Ohio. Fifty per cent of their isolates from rotten stalks yielded *C. graminicolum* in 1961. The organism readily caused stalk rot when introduced into stalks shortly after pollination. In Europe it is usually considered one of the most destructive stalk-rot pathogens of corn (25, 214). It also infects sorghums, grasses, and other cereals (17, 66).

Physalospora zeicola E. and E. has been shown to be pathogenic on corn by Eddins and Voorhees (82), but its economic importance has not been established. These authors also investigated its taxonomic and physiologic relationships.

Genetic Variation of Pathogens

It is well established that cultures of fungi and bacteria may vary in virulence, particularly over a period of many months. This is to be expected since the genetic constitution of microorganisms is not static, but dynamic. Genetic changes are induced by mutation, somatic recombination, heterocaryosis and dissociation, and hybridization and segregation. It has been clearly demonstrated that most species of plant pathogens, including those that cause stalk rot, consist of numerous races, strains, and biotypes that differ in their parasitic capabilities and many other characters (42).

Bijl (19) studied variability of many different cultures of *Diplodia zeae*, and observed striking differences in growth on different media, and also pronounced variation in production of pycnidia, as well as in their shape and size. The spores also differed greatly in number, size, and septation. Pycnidia varied in size from 177-217 μ to 400-540 μ depending upon the substrate. The size of the spores varied from 8-11 μ to 23-33 μ, and they also varied in shape, curvature, and number of septa.

Several others have also shown that there is marked variability in cultures of *Diplodia zeae* derived from single spores. Isolates respond differently to environment, and differ in pathogenicity on corn (310, 402, 403). The work of Hoppe (131) proved that *Diplodia zeae* consisted of many races or biotypes which differed markedly in physiological and biochemical characteristics. He obtained 21 different races from 21 isolates derived from 21 ears of corn obtained from different parts of the USA. Schroeder (310) isolated three distinct isolates of *Diplodia zeae* from a single ear of corn. Two were from single spores isolated from the same pycnidium. The three isolates differed in many characteristics: pycnidial production, color of mycelium, growth rate, ability to utilize nitrogen or cereal extracts, compatibility response, as well as pH, thiamine, and biotin requirements. It is significant that the two isolates from the same pycnidium differed strikingly in many characters. The optimum temperature for growth of one isolate was 25°C and for the other 30°C. The differences in biochemical requirements among races may be helpful in explaining why races of *Diplodia zeae* also differ in parasitic capabilities.

Isolates of *Diplodia zeae* also differ in temperature requirements for growth and perhaps for pathogenicity. This may help to explain why Young et al. (404) obtained great differences in pathogenicity of three isolates of *Diplodia zeae* in Oklahoma, Missouri, and Minnesota. Such pronounced differences are not restricted to *Diplodia zeae*. It is common in most species of stalk-rotting organisms. When one considers the marked differences that exist among the biotypes of a given species, it is no wonder that many workers disagree on morphological characters used to distinguish the different species that cause stalk rot.

The extent of genetic differences within a species is well illustrated by *Gibberella zeae*. Eide (87), Ullstrup (362), Covey (49, 50), Haws (109), and others have proven that *Gibberella zeae* consists of many biotypes that differ in many characters, including pathogenicity. The most work on differences among isolates of *Gibberella zeae* is presented by Haws (109). He studied the genetic differences of 36 isolates of *Gibberella zeae* from 17 states of the United States, from three provinces of Canada, and from Japan and the Netherlands. The isolate from Japan was from wheat, and the rest were from corn. All of the isolates grew between 3 and 33°C. Twenty of the isolates had optimum growth at 27, 11 at 24, and two grew best at 30°C. This difference in temperature helps to explain why different optima have been reported for *Gibberella zeae*. Covey (49, 50) and Haws (109) each showed that isolates of *Gibberella zeae* differed greatly in temperature requirement for pathogenicity on corn seedlings.

Thirty-three isolates of *Gibberella zeae* tested by

Haws (109) differed greatly in pathogenicity on two single-cross hybrids. The disease ratings varied from 0.9 (23% of the internodal tissue rotted) to 3.53 (87% of the internodal tissue rotted). This range also occurred among 13 isolates from New York. The three isolates from Canada varied in pathogenicity from a disease index of 1.38 (35% of the tissue rotted) to 3.19 (80% of the tissue rotted). The isolate from Japan gave a disease index of 2.9 (73% of the tissue rotted) and the Netherlands isolate produced a disease index of 2.3 (58% of the tissue rotted). Sometimes the so-called disease-resistant hybrid was more susceptible to certain isolates than the susceptible hybrid: the reverse reaction also occurred. Sometimes the reaction of the varieties was about the same to the different isolates.

There were striking differences in the production of perithecia formed among the isolates studied by Haws (109). Twelve of 36 isolates of *Gibberella zeae* failed to produce perithecia on corn stalks. In general, there was an association between perithecial production on corn and tomato fruit, and pathogenicity. Although four isolates that produced an abundance of perithecia on corn and tomato also caused severe stalk rot, the isolates that failed to produce perithecia were usually weakly pathogenic on corn. Tomato is an excellent material on which to produce perithecia (56). There were also tremendous differences in the number of conidia produced among 21 isolates tested. There was no correlation between sporulation and ability to cause stalk rot of corn (109). Haws (109) found that average optimum sporulation for 21 isolates was 27° C. Andersen (4, 5) reported that most rapid and abundant formation of macroconidia occurred between 28 and 32° C.

There is, unfortunately, a tendency to draw sweeping conclusions from tests involving one or a few isolates of a pathogen based on the reaction of relatively few host plants, sometimes on one or two inbred or hybrid varieties of corn. The conclusions would be far more reliable if the results were based on many isolates obtained from widely different sources, and if a large number of inbreds and hybrids derived from diversified parental stock were included in such tests. Such tests should be continued over a period of years.

Much remains to be accomplished before we shall understand the genetic variability of stalk-rot pathogens. As can be seen from our presentation in this paper, very little has been done.

Host Range

We will not list the hosts for the various pathogens, but merely indicate that these organisms attack many plants. Most of the organisms that cause stalk rot of corn attack a great many species of plants. Some of these pathogens attack species involving many genera and families of plants. MacInnes and Fogelman (187) proved that *Gibberella zeae* attacked not only gramineous hosts, but many vegetable crops. This is also true for species of *Fusarium* that attack corn. *Pythium* spp., *Colletotrichum graminicolum*, and *Helminthosporium* spp. also have wide host ranges. *Sclerotium bataticola*, according to Boewe (24), has a very wide host range; in Illinois it has been reported on 41 host plants, including 15 families and 36 genera. *Diplodia zeae* occurs on dent, flint, sweet, pop, and flour corns, and on teosinte. The extent of the host range has not been studied in detail. Teosinte is susceptible to virtually all pathogens that attack corn. *Erwinia carotovora* attacks many species of cultivated crops belonging to many genera and families. *Erwinia carotovora* f. sp. *zeae* is known to attack bajra, jowar, and tobacco. It would not be surprising if it, like *E. carotovora*, is found to attack a large number of unrelated plants.

Sources of Inoculum

Only general remarks may be made about sources of inoculum, and they apply to the stalk-rot diseases caused by fungi and bacteria. The entire subject should be critically studied as a comprehensive program concerned with ecology of these important organisms.

Although stalk rot is caused by many pathogens, many of them live from one season to another on or in seed, in soil, and in plant debris. Since many attack a variety of hosts, they may live over on the remains of many species of plants. They may overwinter as fruiting bodies, spores, mycelium, and sclerotia. There is little information on factors influencing growth and persistence of various stalk-rot pathogens in the soil. Although several workers have obtained viable conidia of *Diplodia zeae* from pycnidia in host tissue which had remained in soil for several seasons, the ability of the stalk-rotting pathogens to exist as free-living entities in soil and to invade growing plants has never been clearly demonstrated. It has been shown repeatedly that infection readily occurs from naturally and artificially infested soil. It also has been well established that several organisms grow well in sterilized soil. Certain ones grow well in compost, field soil, and peat soil, but not in sand. It is generally assumed that natural infection from the soil arises from infected plant parts. In non-sterilized soil and in culture, the pathogens are greatly inhibited by other organisms.

Gibberella zeae, *Fusarium moniliforme*, *Diplodia zeae*, and *Fusarium* spp. can overwinter as dormant mycelium in seed, as spores on the outside of the seed, on diseased corn roots, stalks, shanks, and husks, and also in ears left in the field. According to Burrill and Barrett (35) and Holbert et al. (122) pieces of diseased stalks infected with *Diplodia zeae* for one or two years have been found in late summer almost covered with black cirri of spores which germinated readily. Some pieces of stalk almost 3 years old also bore pycnidia with viable spores. *Diplodia zeae* can survive in old stubble and trash even though plowed under for several seasons (76). The fungus did not grow in sterilized sand free of organic matter; however, it grew well in sand to which organic matter was added. It also grew well in field soil. Durrell (76) thought that *D. zeae* grew as a saprophyte, and that this helped survival in soil.

Corn seed is often the source of infection of seedlings, and perhaps also of stalks. Reddy and Holbert (273) found that *Cephalosporium acremonium* was

seed borne, and that diseased plants grew from infected seed. Harris (107) found that infected seed did not yield diseased plants. Holbert et al. (120, 121) said that plants susceptible to root and stalk rots, and grown from infected seed, produced much lower yields of marketable ears and higher percentages of nubbins, as well as rotted and chaffy ears, than plants grown from disease-free seed, and resistant to infection. These characteristics are now known to be associated with infected stalks. There is evidence that infected seed is a source of infection for seedling blight and for stalk rot. This, however, has been a disputed question for at least 50 years.

Seedling blight of corn caused by *Gibberella zeae* and *Fusarium* spp. is frequently prevalent during periods of cool weather when the plants grow slowly. It is often a destructive disease of corn in the northern part of the US corn belt. Thus not only does seedling blight cause loss in stand, but the plants may be weakened and thus predisposed to root, crown, and stalk rot. *Diplodia zeae* is favored by higher temperatures. This may be one of the reasons why *D. zeae* is not as destructive as *Gibberella zeae* in northern parts of the corn growing regions.

Smith and Hedges (330) thought *Diplodia* infection was systemic, whereas Durrell (76) and Clayton (47) concluded that *Diplodia* did not spread into the stalks from infected seed. McNew (202) presented experimental evidence that crown infection and basal stem rot resulted from infected seed. Koehler and others (160, 161) concluded that incidence of stalk breakage was no higher in plants grown from infected seed than from nearly disease-free seed. It has also been pointed out (34, 366) that only a small quantity of seed has been infected with *Diplodia zeae* in recent years. Seed is not generally infected with *Gibberella zeae*, yet much of the stalk rot is caused by this fungus. Koehler (169) doubts that there is any association between stalk rot caused by *Fusarium moniliforme* and seed infection. It is fairly well established that there is a high association between inbred seed infected with *Cephalosporium acremonium* and black-bundle disease. *Cephalosporium maydis* is soil borne and perhaps also seed borne. Bacteria are suspected of living overwinter on corn seed and in the field on debris of infected plants.

Gibberella zeae produces ascospores, macroconidia, and chlamydospores. More or less spherical and purple to black perithecia are borne on the surface of the diseased parts of the corn plants, as well as many other species of plants such as wheat and barley. On corn, perithecia form on stalks and stubble, usually in late summer and early fall, but on plant refuse they develop virtually throughout the growing season. The perithecia may also mature throughout the season, but on corn stalks, at least in the northern regions, perithecia usually do not mature until moist cool weather prevails, usually during the spring. Many asci usually develop within the perithecia. The asci normally contain 8 ascospores, which are hyaline, long-oval in shape, and have three septa. The ascospores are 20 to 30 × 3 to 5 μ in size, and they may be shot 15 to 20 cm into the air. They may be exuded from the perithecia during relatively warm humid conditions.

Gibberella zeae may also produce large quantities of conidia on corn-stalk debris in the spring. They are slightly hyaline, have pointed ends, are 30 to 60 × 4 to 6 μ in size and usually have three to five septa. Some isolates of the fungus may produce abundant macroconidia; some produce chlamydospores. Isolates of *Gibberella zeae* also differ in ability to form perithecia.

Pycnidia of *Diplodia zeae* first appear as small dark specks under the epidermis of stalks in late summer and fall, but usually mature during the next spring and summer. When the weather is moist, the pycnidia break through the rind of the stalks, and large numbers of spores ooze out in long cirri, sometimes several cm long, consisting of numerous two-celled spores held together in a water-soluble matrix. The pycnidia are usually most abundant during late summer on the infected basal portions of the stalks. On segments from these stalks the following year, they are most abundant during the spring and summer. *Diplodia zeae* produces two kinds of conidia. The common type, which has been known for about 50 years, are straight to slightly curved, cylindrical to elliptical with round ends, dark brown in color, two-celled (rarely three-celled) and 20-33 × 5-7 μ. In most cases one cell is slightly narrower than the other. Johann (144) reported that thread-like conidia were produced by *D. zeae;* these conidia are 25-35 × 1-2 μ in size; they are not common. Very little is known about the dissemination of the conidia. They are undoubtedly disseminated by rain, wind, and perhaps by insects.

Mode of Infection

Although it is well known that many pathogens cause severe seedling blight of corn, the relationship of seed infection and seedling blight to stalk rot is not definitely known. Some consider infected seed and diseased seedlings as the primary source of infection for stalk rot, others do not. Others find that the roots and crowns serve as avenues for invasion of stalks. Still others think that most stalk infection, especially by major pathogens, is local, the pathogen entering via rudimentary ears, nodes, and leaf tissue (Fig. 2H, 3A and B). The importance of these various sites of infection has also been questioned repeatedly. Since many pathogens are involved, all of them surely do not enter the stalk in the same way. There are probably many diverse methods by which pathogens enter the stalk.

Smith and Hodges (330) claimed that infection caused by *Diplodia zeae* was systemic, the fungus traveling through the plant into the ear. This has been discredited by many workers. Bijl (18) obtained infection by inoculating young plants just below the ground line; he did not think that *Diplodia zeae* was systemic. Holbert and Hoffer (123, 124) illustrated rows of broken corn stalks which they attributed to the planting of diseased seed because stalks in adjacent rows, derived from healthy seed, were erect. Raleigh (269) suggested that *Diplodia zeae* infection might spread from mesocotyl to above-ground parts

of the stalk. Holbert et al. (122) reported that *Diplodia zeae* invaded the mesocotyl.

When McNew (203, 204) began his detailed study on crown-rot infection, it was generally thought by plant pathologists that, if infected seedlings did not die, the general activity of the pathogen stopped. McNew's critical studies proved that at least *Diplodia zeae* continues its parasitic development once the mesocotyl becomes infected, even after the adventitious root system is established. His work also demonstrated that whenever *Diplodia zeae* is present in corn refuse on the soil, it may attack the mesocotyl and later spread into the crown in a manner similar to that from infected seed. He concluded that invasion was not systemic, and did not usually extend far beyond the crown unless the growing conditions were exceptionally poor for the corn plant.

According to McNew, most of the initial infection for stalk infection occurs during the seedling stage. The fungus in the seed or in the soil invades the mesocotyl or the crown of the seedling. The progress of the fungus is slow until about the time of pollination, when the quiescent fungus becomes active again, and the lower internodes of the stem are invaded. McNew considered seed infected by *Diplodia zeae* to be the major source of infection, but this is not in accord with Ullstrup (366), who found no close correlation between *Diplodia*-infected kernels and *Diplodia* stalk rot.

McNew was never able to isolate *Diplodia* from lesions on the mesocotyl until at least 10 days after the seed was planted in infested soil. Most of the infection occurred much later, usually several weeks. From 90-115 days after the soil was infested with *Diplodia zeae,* McNew found from 20 to 70% of the basal portion of the stem infected. The lesion usually occurred at the junction of the mesocotyl and soil, but occasionally infection also occurred above this point.

When the fungus is seed borne, it enters the mesocotyl through wounds caused by the emergence of seminal roots. From infested soil, entrance into the crown is also through the mesocotyl. In most cases the fungus is usually well established on the base of the crown by midseason. Later the lesion may extend upward, but it does not usually involve the entire crown nor extend above the ground until after pollination. There is little evidence of extensive external injury until the plants begin to mature (Fig. 2G).

Diplodia zeae has never been observed to reach the ears from infested soil. McNew (204) stated, however, that *Diplodia zeae* progresses 4-6 internodes above the ground when plants are grown under unfavorable conditions, but usually not more than two internodes when conditions are favorable for the host.

Blaak (21) found that the endodermis in the mesocotyl often provides a barrier against fungus penetration into the stele. He also found that *Fusarium* spp. were present in the tissue near adventitious roots on the stalk, and in leaf scars of young leaves long before stalk rot develops.

Durrell (76) inoculated corn plants in the field by [illegible] around the crown, but obtained no invasion of stalks. McNew (204) accounted for these negative results on the basis of taking the notes too early in the season (the latter part of August). Also the extent of crown invasion depends upon the time of initial infection, variety of corn, the pathogen, and environmental conditions. McNew produced crown infection by infesting steamed soil with infected crowns and stalks which had overwintered in the field.

McNew (204) stated that treatment of clean seed did not reduce percentages of crown-infected plants. Treatment of infected seed increased materially the percentages of crown infection over those not treated This is explained on the basis that the treatment inhibited the fungus without killing it; the embryo is not killed, and as the seedling grows, it is invaded by the fungus that has been dormant. Thus the percentage of crown rot usually increased because the seedling did not die. *Diplodia* crown infection of field corn varied from 14 to 52% in central Iowa from 1930-1937. McNew (204) stated that practically every corn plant grown from *Diplodia*-infected seed developed crown infection at maturity. In Ohio, Wood (398) observed the development of root rot and basal stem rot throughout the growing season on many hybrids. He observed no basal stem rot 6 weeks after planting, but 4 weeks later the symptoms were apparent. He suggested that the mesocotyl was the avenue of entrance for fungi causing basal stem rot. He did not think that the aerial portion of the crown served as an entrance for stalk-rotting organisms.

Much of the recent work indicates that stalk-rot infection arises from the roots, mesocotyl, and crown. Craig and Hooker (54) secured stalk root of dent corn in the greenhouse by infesting the soil with *Diplodia zeae* when the plants were 8 weeks old. The pathogen progressed into the stalk by way of infected roots. Twelve weeks after soil infection (about 6 weeks after silking) the roots of the plants had large brown spots that extended to the junction with the stalk. The outer surface of these stalks was discolored, and when the stalk was opened, dark discoloration was visible in the basal crown tissues, as well as in the rind and the first two internodes. The internodal vascular bundles were brown but the parenchyma tissue was not discolored.

Local infection of the corn stalk has been known for a long time. Burrill and Barrett (35) were unable to obtain infection of leaf sheath and stalk except by stab inoculations. Heald et al. (112) produced infection on stalk, husk, and silk. Durrell (74, 75) called attention to sheath blotches produced by many organisms. He reported that prior to flowering of the corn plant, the ligules of the leaf sheath tightly clasp the stalk and thus prevent material from slipping down inside the sheath. Furthermore, if the material did drop down, the rapid elongation of the stalk would lift it up again. At the time of pollen production, the stalk has ceased to elongate, and the ligules begin to loosen and expose the cavity between the sheath and stalk. About this time the pollen, soil, and various kinds of spores accumulate in these cavities. Moisture, an important factor, is readily held in these cavities. During wet weather they fill with water and

usually remain so for days after the rain. Even in dry weather the inside of the sheaths is frequently moist. Durrell (76) states that this water may contain sugars from the corn plant. These sheath cavities become an ideal chamber for germination and growth of diverse organisms. The pathogens present may invade the corn plant; on the sheath they cause blotches, and at the base of the sheath they attack the node. The invaded part, usually called the outer rind, turns brown and is water-soaked. The fungi spread mostly up the stalk, although sometimes downward as well. This has been demonstrated repeatedly in inoculated plants. A large percentage of ear infections occur through the shank.

Durrell (76) emphasized the importance of local infection. His isolation tests indicated a much higher percentage of plants with the second and third nodes infected than plants infected only at the first node. He also recorded many plants with infection only at the third, fourth, or fifth nodes. The percentage of nodes infected above the fourth node decreased rapidly. Rarely did he find the seventh, eighth, and ninth nodes to be infected with *Diplodia zeae*. There was a higher percentage of nodes infected than internodes, and upper internodes and nodes were freer of *Diplodia zeae* than the lower ones. Healthy shanks can also be found on plants with infected nodes. The crown and first internode above the ground were less infected than those immediately above. Furthermore, in infected stalks there are gaps of healthy tissue that intervene between diseased areas. These results all support the idea that infection is local.

Durrell (76) explained the greater percentage of infection at the lower part of the plant by two facts: the leaf sheaths are older and more readily invaded, and the moisture in the leaf sheath cavities is more constant. He observed less infection when the lower leaves were stripped off early in the season, indicating the important role of the sheath cavity in infection. McNew (204) thought that the unusual high incidence of infection at the lower nodes that Durrell (76) obtained and attributed to the loosening of the leaf sheath and relative high humidity near ground level was in part due to progressive invasion from the mesocotyl.

According to Koehler et al. (160), invasion of nodes may occur with *Gibberella zeae, Diplodia zeae,* and *Fusarium moniliforme* from spores that have fallen between the stalk and leaf sheath. Durrell reported that spores of *Nigrospora oryzae* germinate and grow in the cavity in the presence of pollen grains. There is evidence that many fungi do enter the stalk via nodes, rudimentary shoots, and injuries, or via roots and mesocotyl, and some pathogens enter both ways. Koehler (169) stated that occasionally *Diplodia zeae* entered the stalk via the root below the ground. He found no indication that stalks became infected through the mesocotyl.

There is good evidence that *Fusarium moniliforme* can infect through the underground part of the corn plant. Some think it sometimes travels up throughout the plant. This is based on the fact that it can be isolated from both the underground part and the above ground part of the plant during the growing season. It is true, however, that the frequency of isolation of the pathogen increases tremendously as the season progresses. Some indicate that it is local infection, others think it more or less systemic (99). Since it is a good wound parasite, it may cause infection when introduced into the plants; it most likely infects the stalk like *Gibberella zeae* and other species of *Fusarium,* via the nodes.

Fusarium moniliforme apparently infects stalks through seedlings, as well as by infection through nodes. Some workers think that most, if not all, infection occurs through the seedling, and consider the fungus more or less systemic. Others believe that it commonly infects the stalks through the shoots and wounds. There is fairly good evidence that a high percentage of stalks are infected early in the season. In the USA it is the most common organism isolated from the corn plant throughout the growing season. According to Koehler (169), *Fusarium moniliforme* attacks the stalks earlier in the season than does *Gibberella zeae* or *Diplodia zeae*. Over a 2-year period he found many of the stalks infected by late July. Although most *Fusarium* lesions were below ground level, some were at nodes near the ground.

Although *Fusarium moniliforme* may penetrate the stalk directly, its entrance is greatly facilitated by wounds. It is the most common pathogen isolated in injuries caused by insects (169). Internodal cracks produced by various agents are often invaded by *Fusarium moniliforme*. Roane (277) concluded that *Fusarium moniliforme* enters the node from the infected leaf sheath. His observations indicated that a wet rot first developed at nodes, and then progressed into the internodes. The cortex, especially at the nodes, was progressively weakened until the stalk broke.

McKeen (200) concluded that the common basal stalk rot caused by fungi usually originates from diseased corn roots, but may also arise in tunnels caused by the corn borer. Foley (94) indicated that corn borers did not tend to increase the amount of stalk rot caused by *Gibberella zeae*. Although insect tunnels served as portals of entry of stalk-rot organisms, they were not nearly as important as roots.

Bacterial infections apparently occur through insect injuries, cracks on the stalk, mechanical injuries, and injuries caused by emergence of brace roots. Infection may also occur through stomata.

Very little is known about the mode of infection of other stalk-rot pathogens. *Gibberella zeae* and species of *Fusarium* are thought to behave about like *Fusarium moniliforme* and *Diplodia zeae,* though there is little evidence to support this opinion. *Nigrospora oryzae* attacks the plant both when it is a seedling and when mature (78); and seed treatment with fungicides helps to prevent seedling infection (272). *Sclerotium bataticola* apparently attacks mature plants in the field, and appears to enter through the underground portions of the plant. Seedlings can be infected, but this method is probably not important in the field because high temperatures are required for the fungus to attack the plant (182). *Cephalosporium acremonium* is largely seed borne, and the disease arises each year from infected seed (273).

Reddy and Holbert (273) also indicate that some control can be achieved by seed treatment. The fungus is frequently isolated, however, from sites of insect injury such as corn-borer tunnels (169, 350, 352). Since these injuries are common on corn, their importance in the development of the black-hundle disease should not be overlooked. Nothing is known about entry of *Pythium* spp. into stalks of corn. Perhaps it enters through roots but many lesions develop 6 to 10 inches above the ground with green tissue above and below the lesion (Hooker, unpublished).

According to Koehler (169) the decreasing order for entry of fungi into stalks is: through nodes at junction of leaf sheath; through junction of roots above the ground level and to some extent below the ground; through insect wounds; directly through the epidermis of the stalk below and above soil level; and through shoot buds. This, of course, is not in accord with many investigators who claim that most of the infection results through infected seed and infested soil. It is most likely that infections occur readily by diverse methods, depending on the pathogen, region, season, source of seed, cultural practices, and environment throughout the growing season and late fall. One seldom finds a plant with severe stalk rot, but without root rot and basal stem rot. Basal stalk rot in Minnesota may start in roots or crown, and spread into the lower portion of the stalk, but much of the rot higher on the stalks arises from nodal infection and injuries of diverse types.

Factors that Influence Stalk Rot Development

Moisture and Temperature.—In some regions, such as in Minnesota, *Diplodia zeae, Gibberella zeae,* and *Fusarium moniliforme* are present to some extent each year, and often they may occupy the same stalk. In certain years *Diplodia zeae* may be the primary cause of stalk rot, and in other years it may be one of the other pathogens. In other regions *Cephalosporium* spp. or bacterial pathogens predominate, and in still other areas *Sclerotium bataticola* is destructive. Pathogens thus vary in their environmental requirements for development of stalk rot.

Temperature, rainfall, and humidity have a marked influence on infection and development of stalk rot. Some diseases are favored by high temperature and excess moisture during a relatively short period. Free moisture on the surface of the corn plant is not essential for germination of spores, since moisture accumulates between the leaf sheath and the stalk. Also there is a tendency for moisture to accumulate in wounds of diverse types. Most soils contain enough moisture to permit pathogens to enter plants through underground parts.

Durrell (76) emphasized that development of *Diplodia zeae* is most destructive in regions of heavy rainfall in late summer or during the late growing season. Certain other conditions must, however, accompany high precipitation: a) sufficient nutrients in the corn plant; b) rapid growth of tissue; c) the leaf sheath must become loosened. The presence of water is then essential between the leaf sheaths and the stalks in order to permit the germination of spores and the growth of the organisms.

Although moisture is one of the most important factors, temperature also plays a significant role. Stevens (342) indicates that species of *Diplodia* generally grow well at high temperatures. Togashi's book (359) should be consulted for information about temperature requirements of various pathogens, including those that cause stalk rot. According to Durrell (76), minimum temperature for growth (dry-weight basis) of *Diplodia zeae* lies between 10 and 15° C, the maximum between 35 and 40° C, and the optimum between 28 and 30° C. There is evidence that in certain years temperature is not as significant as precipitation. In years of high rainfall *D. zeae* was most destructive in Iowa. Durrell concluded that "the relation of soil temperature to *Diplodia* infection is not very direct."

It is well known that most lots of corn seed sown at low temperatures are most subject to seedling blight by many organisms. Dickson and Holbert (65) have attributed this to physiological changes within the tissue of young seedlings. It is also known that high temperatures may predispose corn seedlings to attack by *Pythium* spp., *Penicillium oxalicum,* and *Nigrospora oryzae.* Chilling predisposes plants to stalk rot caused by *Nigrospora oryzae,* according to Smith and Holbert (327). Reddy (272) found that lots of corn seed that germinated readily at low temperatures (below 11° C) were injured very little by *Nigrospora oryzae,* whereas seed of corn that required a higher temperature for germination was usually severely injured by the organism. The reverse is true for *Gibberella zeae* and certain species of *Fusarium* (64).

Workers in Minnesota, Missouri, and Oklahoma exchanged single-crosses of corn, and isolates of *Diplodia zeae* and *Gibberella zeae.* Tests with *Gibberella zeae* were discontinued after one year, but experiments with *Diplodia zeae* were made in 1954, 1955, and 1957 (404). Tests in Oklahoma were successful only in 2 years because of drought. The same isolate of *Diplodia zeae* was used in each state during 1954, 1955, and 1957. Each corn entry was inoculated by inserting toothpicks infested with *Diplodia zeae* into the second internode above the ground 1 to 2 weeks after tasseling. The isolates were generally most pathogenic in states where they originated. Furthermore, all isolates were more pathogenic in Missouri and Oklahoma than in Minnesota, the infection being distinctly most severe in Oklahoma. When the weather was hot and dry in 1955, the isolates of *Diplodia* from Missouri and Oklahoma were more pathogenic in Minnesota than the isolate from Minnesota. The single-cross hybrids generally were more resistant in Minnesota than in Missouri or Oklahoma, although resistance varied with the isolate, hybrid, location, and year of test. In growth studies, the Minnesota isolate made slightly better growth at 12°, but at 30 and 36° C it made the poorest growth of the three isolates. The striking results obtained by this three-state cooperation indicate the urgent need of more extensive work on a regional basis. It also indicates that cooperative tests must be repeated for several years.

Unusually hot dry weather during the active grow-

ing period of corn favors the development of charcoal rot (182, 406). It is generally concluded that an abundance of rain over several weeks beginning about the time of pollination is conducive to stalk rot caused by *Diplodia zeae, Gibberella zeae,* and *Fusarium* sp. (76, 158, 366). Ullstrup (365, 366) notes this to be particularly true if weather is unusually dry in June and July. There are indications that *Diplodia zeae* is favored by somewhat higher temperatures than *Gibberella zeae.*

It is well known that the rot due to *Diplodia zeae* and *Gibberella zeae* may differ considerably in severity in different years in a given state and in different places in the same state in a given year (251, 252), thus indicating unequal influence of environmental conditions on these fungi. According to Koehler (169), this yearly variation is more apparent in the stalk rot that causes premature drying and stalk breaking than for rots that do not become evident until the corn is mature. Michaelson (218) found more stalk rot in corn artificially inoculated with *Diplodia zeae* and *Gibberella zeae* on comparatively dry soil than on soil saturated with water for 3 weeks after inoculation. We have repeatedly observed much more premature killing of plants inoculated by the toothpick method with both *Diplodia zeae* and *Gibberella zeae* in dry hilly soil in Minnesota than on relatively wet soil in a depression of the same field (Fig. 1D).

Covey (49, 50) found that isolates of *Gibberella zeae* differ greatly in their ability to cause seedling blight at different temperatures. An isolate from New York was not pathogenic to corn at 25° C, whereas an isolate from Missouri was highly pathogenic. A Minnesota isolate was only moderately virulent at 25° C (Fig. 1E). In culture, the Minnesota and Missouri isolates grew at 35° C, while the New York isolate did not grow above 30° C. All three isolates grew equally well at 10° C. It seems likely that isolates of *Gibberella zeae* and others might differ strikingly in their ability to attack stalks at specific temperatures. Much work needs to be done with pathogenicity of isolates at different temperatures. Peterson (257) observed that certain pathogens could attack corn seedlings at relatively high temperature. The work by Young et al. (404) indicates that isolates of *Diplodia zeae* differ with respect to their temperature requirements.

Charcoal rot, caused by *Sclerotium bataticola,* is generally not destructive on corn or other crops, except in regions where hot and moderately dry weather prevails in late summer and early fall, i.e., Nebraska, Oklahoma, and Texas. Corn apparently becomes susceptible to this pathogen later than to *Diplodia zeae* or *Gibberella zeae.* Charcoal rot develops best at about 37° C. According to Koehler (165), either high soil moisture or low soil temperature inhibits charcoal rot. In irrigated areas, the disease can be prevented by maintaining high soil moisture. This is a good example of a disease limited in distribution by temperature and moisture.

Stalk rot in Minnesota was least destructive over a period of years during comparatively cool growing seasons. Melhus and Durrell (208) reported severe stalk rot in Iowa in years of high summer temperatures, and low incidence of the disease in relatively cool summers. The amount of stalk breakage was not always closely associated with the degree of stalk rot. In 1963 in Minnesota there was thus considerable necrosis of nodes and internodes, but very little breaking of the stalks. This is attributed to the calm and relatively dry and warm fall with few strong winds, permitting early harvesting of the corn.

Michaelson (218) obtained much more stalk rot in corn grown in the greenhouse at a temperature of about 30 than at 18° C when the plants were inoculated with *Diplodia zeae* and *Gibberella zeae.* These results were reversed when seedlings were inoculated. It is significant that corn stalks inoculated with *Diplodia zeae, Gibberella zeae, Penicillium oxalicum, Helminthosporium* spp., and *Fusarium moniliforme* developed considerable rot over a wide range of temperatures during late August and early September in Minnesota. There is a real need for controlled experiments in this field.

Bacterial stalk rots are most prevalent and destructive in regions with high rainfall during the growing season, or when plants are grown under irrigation and on land subject to flooding. All authors agree that the disease is favored by high temperatures. Hingorani et al. (114) give the optimum temperature for growth of *Erwinia carotovora* f. sp. *zeae* as 30 to 35°, the minimum as 5° and the maximum as 40° C. Sabet (293) found that moisture-temperature requirements for infection of corn seedlings with *Erwinia carotovora* f. sp. *zeae* varied with the soil texture. Optimum conditions for infection were 35° C and 70% moisture in light loam soil. In lighter soils infection occurred at lower temperatures and higher moisture levels than in heavier soils.

A corn plant requires an enormous amount of water; it has been estimated that a single plant will remove about 50 gallons of water from the soil during the growing season. One acre of corn with 16,000 plants will use about 800,000 gallons of water in a season. Necrosis of the stalk, especially when a considerable portion of basal portions of the stem is involved, will disrupt the pathway of transport of water, nutrients, and other compounds essential for growth. Diseased plants are thus apt to die prematurely whenever drought and other unfavorable conditions arise.

Michaelson (218) showed that less stalk rot developed in the field in corn stalks inoculated with *Diplodia zeae* and *Gibberella zeae* when the plants were growing on wet soil, than on relatively dry soil. The plots were flooded with 7 to 10 cm of water about 2 weeks before inoculation. This is in accord with other tests we made several years ago. The plants inoculated on dry plots died 2 to 3 weeks after inoculation, whereas those on wet ground remained green almost as long as the noninoculated plants.

McNew (203) found that *Diplodia zeae* scarcely grew at a soil moisture content above 70% water-holding capacity, but it invaded the corn plant readily at 90%. Although the fungus grew best at 50% soil moisture, the host was not invaded as severely as at 90%. At 30%, *Diplodia zeae* did not progress far up

the crown, even though it was capable of growth at soil moisture below this level. McNew's results (203) indicated that light crown infection at low soil moisture was nearly as injurious as very severe infection at high soil moisture. Any deviation in soil moisture from optimum for the growth of corn rendered the plant subject to injury by crown infection. Reduction in growth may also occur as a result of late crown infection. This apparently results in some loss of adventitious roots, and discoloration of xylem indicates that water transportation might be hampered. He also showed that poorer development of infected plants was associated with decreased water consumption. The transpiration ratio of all plants infected with basal stem rot was more than that of noninfected plants.

Soil Fertility.—There is a great deal of information about the influence of soil fertility on yield and lodging in corn (171, 185). It is generally agreed that high soil fertility and adequate plant populations result in greatest yields. It is also agreed that nitrogen will generally increase lodging, whereas potassium will reduce it. Stalk strength is obviously influenced by these chemicals. There is also considerable evidence that soil fertility sometimes greatly influences the susceptibility of corn to stalk rot. This evidence has been accumulating ever since 1930 when Koehler and Holbert (161) reported that *Diplodia* stalk rot was more prevalent when corn was grown on highly productive soils, though Smith and Holbert came to an opposite conclusion (327).

Many workers now agree that stalk rot is more severe when nitrogen is in excess in relation to potassium (6, 94, 100, 116, 161, 165, 171, 234, 235, 247, 354). According to these workers, nitrogen tends to increase stalk-rot severity and potassium tends to decrease it. We have found in Minnesota that nitrogen may interact in some seasons with phosphorus on certain corn hybrids infected with different pathogens to produce more severe stalk rot. In some seasons, however, nitrogen has not influenced stalk rot at all. Spencer and McNew (333) found that excess nitrogen and deficient potassium greatly increased bacterial wilt in sweet corn. Phosphorus at low levels resulted in necrotic lesions, and at high levels resulted in dwarfing and necrosis. Other bacterial diseases of stalks should be studied to determine whether soil fertility will greatly influence them.

Most workers have not reported phosphorus as an important factor affecting development of stalk rot. In a greenhouse study, Thayer and Williams (354) found that phosphorus decreased severity of stalk rot and concluded that a high level of phosphorus would protect corn against the disease. We have found in field experiments, however, that the development of stalk rot in relation to phosphorus is about as complicated as in the case of nitrogen. Our work indicates that the response to phosphorus fertilization of field plots varies with the season, the corn hybrid, and the pathogen used for inoculating the plants. In some experiments it also interacted with nitrogen and potassium to increase stalk rot. At no time did it reduce severity of the disease though it sometimes had no effect at all.

Nearly all the workers cited have indicated that stalk rot is reduced in severity when there is adequate potassium fertilization. We have found in Minnesota that the influence of potassium on stalk rot may also be variable. We have noted that potassium, alone or together with phosphorus, may either increase or decrease stalk rot when certain hybrids are inoculated with some fungi in certain seasons. We have found that it usually neither increases or decreases stalk rot severity when plants are inoculated.

It is noteworthy that Younts and Nusgrave (407) and Koehler (169) concluded that potassium chloride fertilizer decreased stalk rot, but that this was not true when potassium sulfate or potassium metaphosphate was used. It was suggested that the decrease in disease was from applying chlorine and not from applying potassium. Much more work of this type needs to be done not only with potassium, but with nitrogen and phosphorus as well.

Hoffer and Carr (117) found that an accumulation of aluminum and iron in the corn plant rendered the stalks more subject to invasion by stalk-rot organisms. They also noted that application of phosphate and lime reduced the severity of stalk rot. Koehler et al. (160) found that, although lime did not influence the percentage of broken stalks, it greatly decreased the percentage of leaning stalks. They obtained no consistent results when they experimented with rock phosphate, acid phosphate, bone meal, sodium nitrate, or potassium sulfate.

Certain workers suggested that mineral deficiencies and nodal accumulation of iron and aluminum compounds caused symptoms similar to those associated with stalk rots. One of the characteristics of the growing corn plant is the brown to purple discoloration of the plate of vascular tissue at the nodes. In such cases all the nodes are often more or less uniformly discolored.

Some of the nodes discolored by the toxic compounds are free of microorganisms, but frequently various microorganisms can readily be isolated from them. Young plants may also have brownish lesions on the roots from which no organism can be isolated. However, as the season advances, these roots become thoroughly infected with microorganisms.

Hoffer and Krantz (118) found that the roots of corn were weakened and rendered more susceptible to root-rotting pathogens as a result of the accumulation of iron and aluminum compounds at the nodes of the stalk. The accumulation of these compounds, especially when the potassium was low in the soil, was thought to interfere with normal translocation of nutrient from leaves to the roots. As much root infection and nodal discoloration usually occurs long before the silking period of the corn, the compounds predisposed the plants not only to root infection but to nodal rot and stalk rot. Excessive accumulation of aluminum and iron may occur when these metals are available in subtoxic concentration. These metals, especially aluminum, accumulate in the plates of vascular tissue at the nodes of stalks and shanks, and in the scutellum of the kernels. These toxic metals have a tendency to plug the vascular bundles, and thus interfere with normal function of the plant. Nodal

disintegration begins in the absence of microorganisms in the tissues. Corn plants differ greatly in the quantities of aluminum and iron salts absorbed. The application of lime and phosphate to soil containing subtoxic amounts of iron and aluminum salts will help prevent excessive amounts of these compounds in the plant.

Otto and Everett (235) reported differences in stalk rot in corn hybrids growing in fertility plots though it was known that corn hybrids differed in their ability to utilize nutrients (172). We have also obtained similar results in Minnesota experiments. Future workers might well take this important interaction into account when they design experiments.

The various studies on the influence of soil fertility on stalk rot have been made with naturally occurring stalk rot or with stalk rot resulting from inoculation with *Diplodia zeae*, *Fusarium graminearum*, *Fusarium moniliforme*, *Pythium* spp. or *Gibberella zeae*. None of the studies have attempted to show that the response to fertilizer might vary because of the pathogens, however. We have inoculated the plants with *D. zeae* and *F. graminearum*, and have found in some seasons that certain of the fertilizers resulted in stalk rot being more severe when some corn hybrids were inoculated with one of the pathogens but not when inoculated with the other.

It is possible that some confusion about the influence of soil fertility on susceptibility to stalk rot may result from different methods of judging stalk rot. Those workers who have used lodging, or the resistance of stalks to squeezing between the fingers, as the measure of stalk rot may have confused resistance to disease with inherent stalk strength. Although stalk rot often weakens stalks, it is not the only factor that may produce such an effect. We have found that the influence of fertilizer on the size of necrotic lesions in stalks inoculated with various fungi may not be related to the influence of fertilizer on the strength of the stalk. In fact, although lesions are large, the stalks may be resistant to lodging, and vice versa, due to the fertilizers.

It can be readily seen that many factors other than soil fertility influence stalk rot development, and in any one season or field these other factors might be of greater importance than soil fertility. Furthermore, these various factors may interact to produce disease. Much more work will be needed before the relation of soil fertility to stalk rot development will be understood. It is likely that it will never be possible to control the disease by manipulating soil fertility, because of the many interacting factors.

Relations among Microorganisms.—With certain exceptions, it is often very difficult to determine the cause of stalk rot in the field. *Pythium*, *Sclerotium*, and bacterial rots are among the easiest to identify, although they may be associated with other organisms which increase the difficulty of diagnosis. As the plant approaches maturity, the identity of certain pathogens in the field may become more apparent because of the fruiting of the pathogen. The abundant fruiting of *Diplodia zeae* and *Gibberella zeae* may be a good indication of the causal organism, and production of pinkish coloration on and in the stalk and nodes may also give an indication that *Fusarium* spp. are involved. Just because an organism is fruiting on the basal portion of a stalk does not necessarily indicate that it is the sole, or even the primary cause of the rot. It may be necessary to isolate from the lesions. There may be several separate infections, and certain pathogens may be more or less systemic (96, 99). In various parts of the world, if not in all parts, it is not justifiable in most cases to assume that only one pathogen is involved in an epidemic of stalk rot. Porter (267), DeVay et al. (63), Littlefield (179), Haws (109), and others have shown that many kinds of organisms cause stalk rot and can often be isolated from a small piece of necrotic tissue.

Standard isolation techniques are usually employed in isolating pathogens from corn stalks. If the rind of the stalk is not removed, the infected part should be put in running water for 1 hour or longer to remove foreign organisms and soil. The material is then cut into small pieces and thoroughly washed in sodium hypochlorite (0.5% available chlorine) containing a trace of detergent, or in a weak alcohol solution for 1 to 2 minutes. The tissues are then transferred directly to nutrient media without washing in sterile water. Acidified or non-acidified nutrient media may be used as desired. Several media probably should be used, especially in earlier studies.

DeVay et al. (63) made isolations over a 3-year period from the margin of rotted tissues in living corn stalks collected in 200 locations in Minnesota. *Fusarium* spp., particularly *F. graminearum*, were isolated from 98% of the samples. In another test, they made isolations from samples of rotted stalks obtained from 109 locations in Minnesota. *Fusarium* spp. again predominated. Other organisms, such as *Diplodia zeae*, *Trichoderma* spp., and *Alternaria* spp. were common, and 12 other species were also occasionally isolated. Bacteria were especially common in 1956. They concluded that many of these organisms weakened the stalk and thus contributed materially to stalk breakage. Many more detailed isolation studies should be made at different times of the year and at many different regions. Small pieces of infected tissue should also be used in making isolations from corn stalks in order that essential information may be obtained on the role of associated microorganisms in relation to stalk rot.

The association of fungi and other microorganisms in the production of stalk rot has not been thoroughly studied. Many investigators apparently are of the opinion that only a few species of fungi actually are the primary cause of stalk rot. The role of many so-called secondary invaders has, therefore, been more or less overlooked, or they are usually considered to be unimportant saprophytes. Few have recorded all the different fungi and bacteria isolated from infected tissue. In recent years it has been demonstrated that many of these so-called secondary invaders cause stalk rot when injected into the healthy stalk (38, 256). They may even cause a distinct type of stalk rot, as reported in Egypt, where secondary organisms associated with *Cephalosporium maydis* cause rot known as late wilt (294). There is also the possibility that they may play an important ecological role in the

development of various microorganisms in the stalk. Penetration of numerous organisms might thus be affected when all these organisms are present in the cavity between the leaf sheath and stalk, in insect tunnels, and in hail-damaged tissues. They might also interact to play a very important role in connection with antibiosis or metabiosis, and they may also be involved in synergistic effects. The possible effects that microorganisms might have on each other is well illustrated by work done with *Diplodia zeae* and *D. macrospora*. Stevens (344) has shown that *D. macrospora* is largely restricted to the southeastern USA, and suggests that this might be a result of a restrictive temperature requirement for growth, and inability to compete with *Diplodia zeae*. Hoppe (131) has shown that *D. macrospora* cannot compete with *Diplodia zeae* when both fungi are placed in a single ear of corn. Wilson (396) found that *D. macrospora* grew well on a number of different media when the media had first been staled by *Diplodia zeae*. Apparently *Diplodia zeae* secretes a biotin-like substance that enables *D. macrospora* to utilize substrates on which it will not ordinarily grow (156, 343, 396).

Isolation from stalks by Porter (267), Chiang and Wilcoxson (40), DeVay et al. (63), Littlefield (179), Ho (115), Tijerina (358), and others have shown that many organisms are commonly associated in stalks, roots, and crowns of infected plants. Bacteria are particularly common. McKeen (199) has emphasized that bacteria appear to be constantly associated with *Pythium* root rot and basal stalk rot which arises from root and crown infection. Elliott (90) indicated that bacteria normally follow the *Pythium* sp. in stalk-rot infection. Inoculations made with many of these organisms showed that they can cause stalk rot. Thus Haws (109) found that *Trichoderma* spp. caused more severe stalk rot than did isolates of *Gibberella zeae*, and that *Fusarium* spp. and *Penicillium* spp. (particularly *P. oxalicum*), caused severe stalk and ear rots. Unfortunately only a few tests of this type have been made.

Several investigators have mixed inocula before injection into the corn plant. Reece (274) mixed inocula of seven pathogens known to cause stalk rot prior to inoculation by spraying, but the comparative effect in relation to a single pathogen, unfortunately, was not apparent because the individual organisms were not studied. The rot that developed on the sprayed plot differed, however, from that resulting from inoculation by the toothpick method with *Diplodia zeae* and *Gibberella zeae*. Michaelson (218) inoculated the same internode of corn at the same point, and about 5 cm apart, with *Diplodia zeae* and *Gibberella zeae*, using the toothpick method. The amount of rot was about the same whether the fungi were inserted into the same opening or alone. More rot developed when the toothpicks were separated by about 5 cm, as the two infected areas soon coalesced. There was no indication that the two pathogens inhibited or stimulated each other. He concluded that rot induced by *Gibberella zeae* did not influence the development of rot induced by *Diplodia zeae* in another part of the stem, and vice versa. The tests involved only single isolates of each species; the results might have been different had other isolates been used. For instance, Borlaug (28) found that a mixture of races of *Fusarium lini* Bolley greatly reduced the percentage of wilted flax plants.

Hoppe (131) demonstrated interspecific aversion between *Diplodia zeae* and *D. macrospora*. He found that there were many strains of *Diplodia zeae*, and that certain strains inhibited the others. Thus, if two or more strains were introduced into one plant, only one strain could be isolated. Ho (115) concluded that the association of two active pathogens causing root rot of corn generally increased stalk rot. The combination of a pathogen with a nonpathogenic strain or a saprophyte sometimes decreased the severity of stalk rot.

Tijerina (358) reported an antagonistic relationship in Mexico between a *Helminthosporium* sp. and a *Gliocladium* sp., and between a bacterium and a *Helminthosporium* sp. when sterile and non-sterile soil were infested at the time the corn seeds were planted. It is interesting that the amount of infection was reduced on some hybrids, not on others.

Edwards (86) obtained indications that *Gibberella fujikuroi* and *G. fujikuroi* var. *subglutinans* were antagonistic toward *Trichoderma viride* Pers. ex Fr. Since much infection by *G. fujikuroi* occurs early in the life of seedlings, and there are many reports that all parts of the corn plant are infected, it would be highly desirable to ascertain if *Fusarium moniliforme* is inhibitory toward other organisms. It has been reported several times that *Diplodia zeae* and *F. moniliforme* are rarely isolated from the same necrotic lesion.

Bacteria, *Pythium* spp., and *Fusarium moniliforme* were isolated in Canada from necrotic parts of stalks. Those three organisms were often found in close association. According to McKeen (200), all stalk-wilting pathogens eventually produce the same disease syndrome. *Diplodia zeae* and *Gibberella zeae* were rarely found.

There is practically no information about the influence of other diseases on the development of stalk rots. Several workers (125, 169) have reported that bacterial blight and northern leaf blight increased the amount of stalk rot, and as lines resistant to these diseases are made available (141, 167), more definite information on this subject should become available. Pendleton et al. (252) correlated the amount of stalk rot and the loss of leaf area from northern leaf blight. The increase in stalk rot when leaves are diseased may not simply be due to loss of leaf area, but toxic substances may be released from the diseased leaves which would render the stalks more susceptible to rot. Michaelson (217, 218) reported that smutted plants were more susceptible to stalk rot than were plants free of smut, but we have been unable to confirm this observation. We used hybrids, however, that were different from those used by Michaelson. Peterson (257) failed to find an association between smut susceptibility and seedling blight caused by *Fusarium moniliforme* and *Gibberella zeae*.

Plant Maturity.—As stated previously, corn does not generally become susceptible to stalk rot until about silking time. This has been demonstrated many

times for such pathogens as *Diplodia zeae, Gibberella zeae*, and *Fusarium* spp. (128, 218). At about that time physiological changes occur, the plants become much more vulnerable to attack, and the rot progresses much more rapidly in the tissue. Stalks of corn still alive and growing are much less subject to attack by secondary invaders or primary pathogens. Green corn plants approaching maturity, but still quite succulent when killed by frost, are also readily invaded by stalk-rot organisms that normally cannot enter healthy growing plants.

As early maturity renders plants more susceptible, any factor that hastens maturity of corn renders the plant more subject to stalk rot. Early planting of corn in a zone adapted to a particular variety thus tends to increase the amount of stalk rot and breakage of stalks. Likewise, foliage diseases also may cause corn to mature rapidly, and thus render the plants more subject to stalk rot. It is known that corn borers, grasshoppers, chinch bugs, hail, and drought may cause premature dying of plants, and thus affect the rate of rot development.

The amount of stalk rot induced by artificial inoculation with *Diplodia zeae* and *Gibberella zeae* varies greatly with date of planting when the plants are inoculated at comparable stages of growth of the corn plant. The amount of rot resulting from natural infection may also vary considerably. It is usually much more severe in early-planted corn. Early-maturing varieties are as a rule much more severely attacked than are late-maturing varieties planted at the same location and time. If these early varieties are planted late in the season, however, less rot develops on them, and late-maturing varieties planted early in the season develop more rot than when planted at the regular time. Grikenko (104) found that corn sown early and artificially inoculated had twice as much infection in the fall as corn sown about 2 weeks later.

In comparing stalk rot, as determined by stalk breakage when hybrids are grown in their proper maturity zones, it cannot be definitely stated that early-maturing varieties are always more subject to rot than the late varieties. If all varieties, irrespective of maturity, are grown in a given maturity zone, however, the early-maturing varieties will be most severely attacked. The exceptions to this rule probably depend on the location of the test and the predominant pathogens, as well as inherent differences in resistance. Late-maturing corn grown in Minnesota may show little or no rot, as measured by stalk lodging. This has been demonstrated repeatedly over a 7-year period (258 to 263, 361).

For one year Koehler (160) reported that the percentage of broken stalks increased with late planting, but in two other years the reverse was true. The cause for this reversal is not known; perhaps the period of rot development came when there was heavy rain or drought.

Nigrospora oryzae causes stalk rot and predisposes corn to broken stalks, especially when the plants are growing in or exposed to conditions unfavorable to the host. As with most other stalk-rotting organisms, most of the apparent infection occurs near the ground. The infection becomes most prevalent as the corn plant approaches maturity, although infection may occur before pollination. When plants are exposed to light frost or to temperatures near freezing, the plants become susceptible to infection. Stalks killed by low temperatures also soon become infested with saprophytes.

Although barren stalks are often attributed to stalk rot, there are apparently many factors that cause them. Burtt-Davy (36) considered barren stalks a hereditary character, and Garber et al. (101) studied the inheritance of a gene conditioning barrenness in an inbred line of corn. They also studied physiological effects of lack of ears on corn plants. Hunt (136) attributed barren stalks to environment. The percentage of barren stalks varied with the season; if barrenness was due to genetic abnormality, such plants would tend to eliminate themselves. He attributed barren stalks to lack of soil fertility and to disease. Selby (313) said that root rot caused both dwarfing and barrenness of stalks. Russell (288) reported more barren stalks in dry than in wet years, due to lack of available nutrients.

Reddy and Holbert (273) emphasized that there was a strong association between barren plants and infection with *Cephalosporium acremonium*. Durrell (76) was not sure that barrenness of stalks was associated with *Diplodia zeae*, because planting of clean seed produced as many barren stalks as did planting of diseased seeds.

Jugenheimer (150) made extensive studies of barren stalks. He caused barrenness by covering the ears with parchment bags before silking. The barren plants were artificially inoculated with *Diplodia zeae* about 6 inches above the ground line. He studied the number of internodes rotted and the horizontal spread of the rot in the cortex. His results indicated that barren plants were more susceptible to pith rot than normal plants. There was a tendency toward more resistance to cortex rotting in both the inbreds and single-crosses. Plants that had had their leaves clipped were more susceptible to stalk rot than were barren plants. Several workers are not in accord with Jugenheimer; Hoadley (116) agrees with Holbert et al. (125) that ear formation decreased resistance to stalk rot. Pappelis (240) saw little effect of ear removal on severity of stalk rot.

Plant Population.—Large plant populations per acre generally make plants more prone to stalk rot. First, the stalks of plants grown under crowded conditions are smaller in diameter and, therefore, less stalk rot is required to weaken the stalks to the breaking point. Although some workers have also indicated that the plants are more subject to stalk rot, the amount of data on this subject is limited. Some hybrids apear to be better competitors than others when crowded (171, 347).

Wilcoxson and Covey (388) tested the relative susceptibility of corn at 5,000, 12,000, and 25,000 plants per acre. Single- and double-cross hybrids were inoculated 10 days after tasseling with *Fusarium graminearum* and *Diplodia zeae* by the toothpick method. The amount of necrosis was estimated at 28 days after inoculation. The test was condueted for 2 years at Rosemount and one year at Lamberton,

Minnesota. The necrotic lesions produced by both pathogens increased as the plant population increased. For each pathogen in the test the differences due to plant populations, the corn hybrids, and the interactions of these two variables were statistically significant. These results indicate that the corn was predisposed to stalk rot in the denser population. Additional experiments are needed on susceptibility of corn grown at different population densities involving more varieties grown under different environments.

Koehler (160) found virtually no difference in percentages of broken stalks among plants growing in hills of two or three plants. However, the percentages of living plants were increased about 8% in plants growing three per hill over plants growing two per hill.

Injuries.—Removal of functional leaves from a corn plant increases the severity of stalk rot (125, 217, 218). It is well known that hail, insects, and fungus and bacterial pathogens may cause severe defoliation or leaf injury of corn plants and an increase in severity of stalk rot. A number of workers have demonstrated that partial defoliation of corn stalks either by clipping or by leaf-blighting organisms will increase the amount of stalk rot when the plant is artificially inoculated with *Diplodia zeae* or *Gibberella zeae*.

Michaelson (218) showed that cutting the leaves off about half way from the tip 2 weeks before inoculation with *Diplodia zeae* or *Gibberella zeae* increased the severity of stalk rot. Breaking the leaves had much less effect. Clipping off the leaves on the day of inoculation also had little or no effect. Koehler (166) also obtained more rot when the leaves were cut 2 weeks before silking than on the day of inoculation. Clipping of leaves also increased the size and number of lesions on the stem.

Koehler (166) reported marked increases in the amount of natural infection by *Diplodia zeae* and *Gibberella zeae* when the leaves were clipped before silking. The number of barren stalks was also increased by such treatment. Clipped plants inoculated with *Gibberella zeae* and *Diplodia zeae* yielded less and had a higher percentage of broken stalks. Pappelis (244, 245) found that clipping leaves and roots increased susceptibility to stalk rot caused by *Gibberella zeae* and *Sclerotinia bataticola*. Jugenheimer (150) removed the tip third of each leaf at the time of inoculation and concluded that rot resulting from natural infection was about the same on clipped and non-clipped plants. In some tests the amount of stalk rot was less on plants with clipped leaves. Hoadley (116) also reported that defoliated plants were more resistant than vigorous plants. The cause for these conflicting results is not definitely known; perhaps the environment or variety of corn were deciding factors.

There is considerable evidence that senescence and chemical change in the pith result in the plant becoming susceptible to stalk rot. Leaf damage of various kinds thus increases severity of stalk rot, and prevention of ear development decreases susceptibility. Several investigators have reported that resistance to stalk rot is associated with a high carbohydrate level in the stalks (55, 169, 213). Most of the data clearly indicate that any factor which tends to increase sugar levels in corn stalks reduces the severity of infection, and vice versa. Craig and Hooker (55) studied 12 inbred lines of dent corn that differed greatly in susceptibility to stalk rot. They found that a high sugar level and a high pith density were associated with resistance to stalk rot. The downward trend in sugar level after silking was positively associated with senescence and susceptibility to stalk rot. The decrease in sugar was accompanied by a decrease in the number of living cells in the pith tissue.

Holbert et al. (125) found that artificially induced or naturally occurring chilling and freezing of corn stalks, shanks, and ears increased the severity of rot caused by *Diplodia zeae*. This suggests that such treatment might decrease the carbohydrate level or kill cells in the plant, although they did not actually determine the factors which stimulated the growth of the fungus. Detasseling and defoliation of immature corn plants for green fodder are common agricultural practices by Egyptian farmers. Similar practices sometimes occur in Mexico and other countries. This increases the infection by stalk-rot fungi.

The establishment of certain fungi may be influenced by severity of injuries. Even if secondary invaders do not penetrate deeply into the nodes and stem, they could easily cause sufficient decay to render the stalks subject to stalk breakage. This certainly appears to be true of wounds caused by hail and insects, and the same may also be true of nodal infection when the plant begins to mature. Eldredge (88) gave a good review of the extent of hail damage in the corn belt, and its damaging effect on plants. The amount of hail and the stage of plant development are both important. Durrell (76) claimed that hail increased the prevalence of stalk rot. Littlefield (179) called attention to the extent and type of hail damage in Minnesota. In 1963 he pointed out that in 17 counties in Minnesota alone more than 150,000 acres of corn were damaged to some extent by hail. He showed that hail injuries created avenues for the entrance of pathogens and saprophytes, and greatly increased the amount of stalk rot. He followed the progress of the rot and isolated many fungi at varying intervals from tissues surrounding hail injuries. The predominant species were *F. moniliforme* and other *Fusarium* spp. Results clearly indicate that injuries of various types tend to increase the prevalence and severity of stalk rot (Fig. 2F).

Insects.—Insects aid in the development of stalk rot in several ways. They may carry inoculum into the tissues, or provide an opening through which pathogens enter the stalk. Movement of the insect through the stalk tissue distributes pathogens within the stalk. Frass deposited within tunnels and between the stalk and the leaf sheath furnishes an excellent medium for rapid growth of both parasites and saprophytes. Injuries caused by insects weaken the plants and render them more susceptible to attack by microorganisms. Holbert et al. (125) indicate that damage by chinch bug greatly increased the rate of stalk rot development.

The widespread European corn borer has increased the damage caused by stalk rot and has com-

plicated development of hybrid corn resistant to stalk rot (7). Christensen and Schneider (45, 46, 308) found that certain lines and varieties resistant to stalk rot developed considerably more rot when injured by the corn borer (Fig. 7A, B). The injury caused by the corn borer is usually conspicuous, and damage caused by microorganisms associated with the insect is frequently overlooked or in many instances attributed to the borer. Studies at Minnesota indicated that damage by corn borer cannot be separated from damage caused by microorganisms. At St. Paul on 1 October, 1948, 84% of internodes with borer injuries had conspicuous stalk rot, and at Waseca, Minnesota, stalk rot was present in 90% of the internodes infested by the European corn borer. The insects frequently infest the shank, and 93% of infested shanks at Waseca contained rot.

A plant may sometimes contain several insect tunnels in nodes, internodes, shanks, and ears. Whenever these injuries are associated with rots, severe damage results. The damage attributed to the European corn borer may be partly due to rots that follow insect infestation. Microorganisms belonging to many genera were isolated not only from the frass and insect but from discolored tissue surrounding insect tunnels in the internodes. *Fusarium* spp. were the predominating fungi isolated, although *Diplodia zeae* and *Gibberella zeae*, and other parasites and saprophytes, also were present. The species isolated, and their relative abundance varied with locality, climatic condition, and developmental stage of the host (45, 46). Fungi were isolated from larvae throughout the year, but were less prevalent in inactive larvae collected from diseased stalks in the winter; the prevalence of bacteria remained about the same during late summer, fall, and winter. Fungi were readily isolated from infested portions of the corn stalks throughout the fall and winter. All the organisms isolated from the insects were also isolated from frass and diseased plant tissue (Fig. 8A, B, C).

Gibberella zeae and *Diplodia zeae*, as well as other organisms, may progress rapidly within the internode; however the node greatly retards their spread. The corn borer frequently attacks the node directly or tunnels through it, and thus aids in the rapid spread of the fungi within the plant. Laboratory tests showed that larvae of the corn borer during late summer and early fall are usually infested both internally and externally with diverse species of fungi. The European corn borer weakens the plant, consequently the weakened tissues are more readily subject to attack by certain stalk-rotting parasites and by saprophytes. According to Roberts (278), the European corn borer and the southwestern corn borer provided avenues of entrance to stalk-rot pathogens, and greatly increased the amount of stalk rot in Okla-

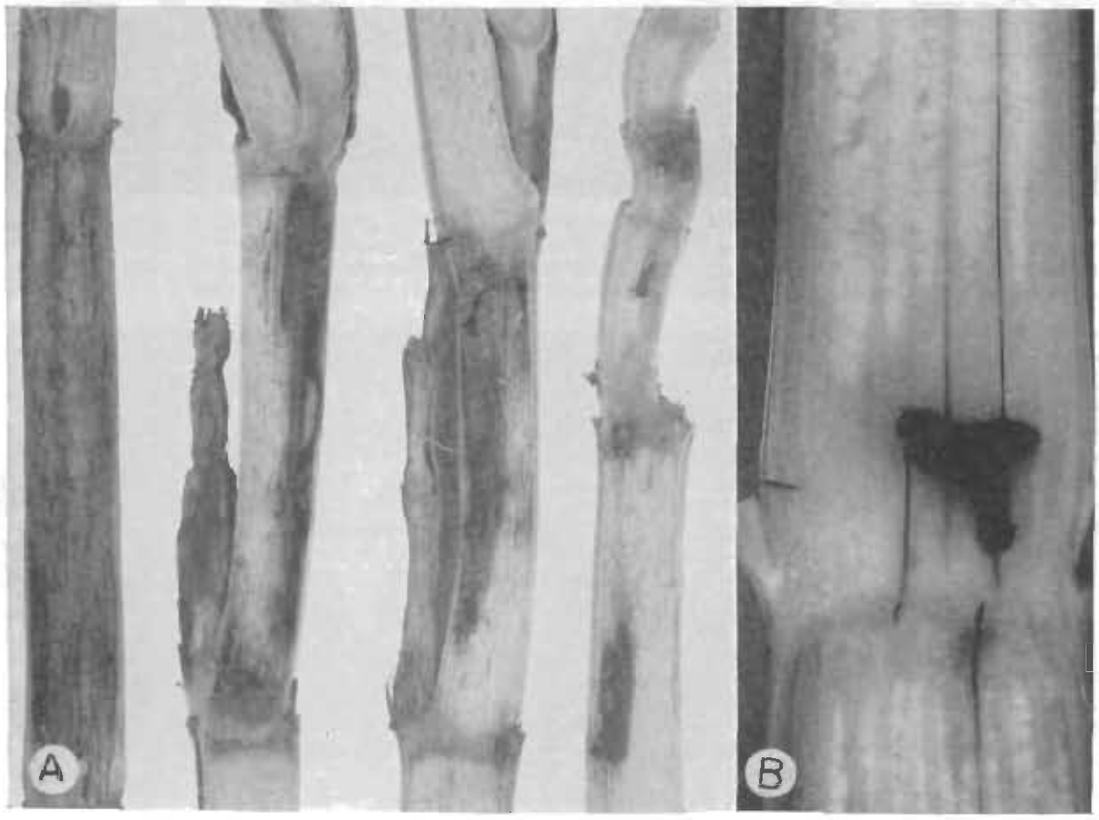


Figure 7. **A.** Stalk rot associated with injury of corn stalks by European corn borer. **B.** The margin of an injury by European corn borer showing the necrotic tissue and dark vascular bundles extending from the necrotic area.

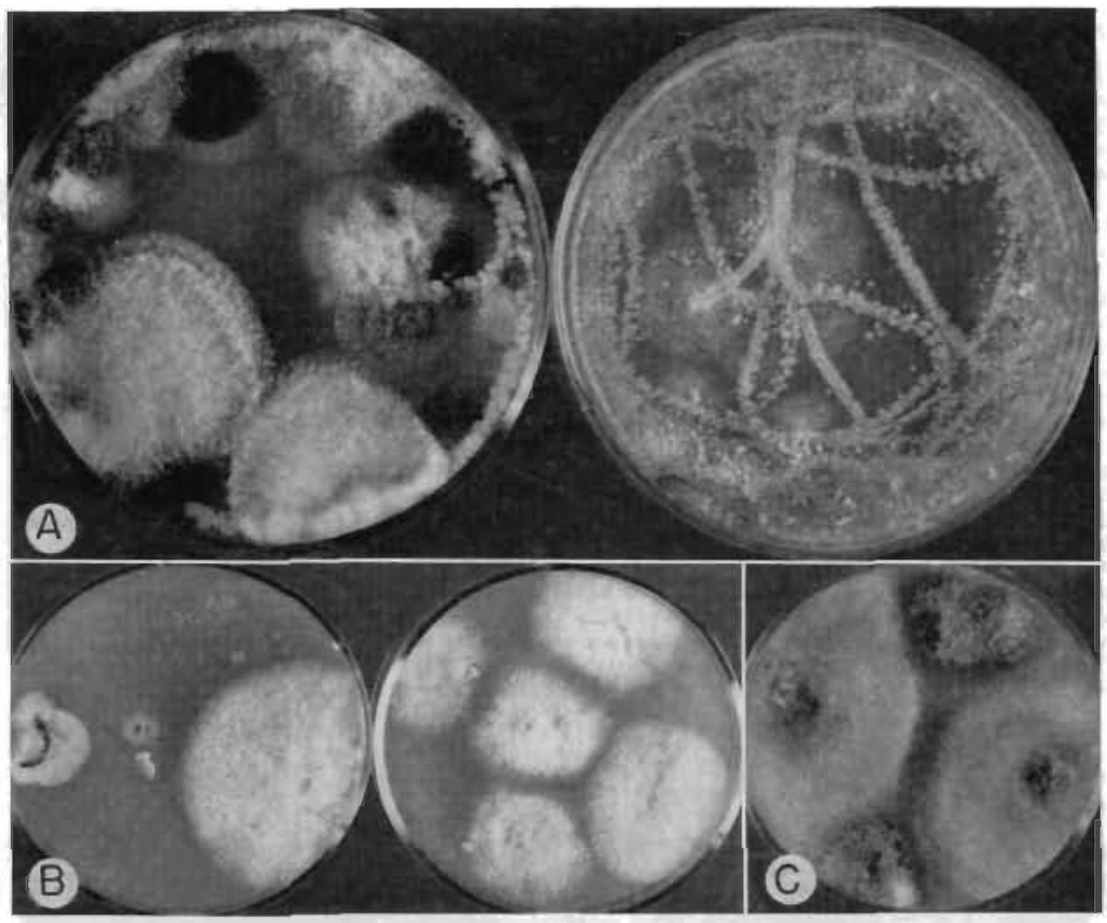


Figure 8. **A.** Fungus cultures that developed after European corn borer larvae crawled over the surface of potato-dextrose agar. **B.** Cultures of yeast and *Fusarium* spp. that grew from European corn borer larvae that had been killed and surface-disinfected. **C.** Cultures of fungi, mostly species of *Fusarium* and *Trichoderma*, that grew from clumps of frass from larvae of the European corn borer.

homa. He indicated that the southwestern corn borer greatly facilitated the ingress of *Diplodia zeae* through the stalk; when the stalks were split open about 7 weeks after inoculation, they were all infested with the insect. Isolation from the frass of these insects yielded cultures of many fungi and bacteria, with *Diplodia zeae* being dominant. Other investigators have found that *Diplodia zeae* rarely occurs in tissues wounded by insects.

Koehler (169) made isolations from tissue associated with European corn borer. The most common fungi isolated were *Fusarium moniliforme*, *Cephalosporium acremonium*, *Mucor* spp., *Penicillium* spp., and *Trichoderma* spp. Although *G. zeae* was isolated from tissues injured by the borer, *Diplodia zeae* was not. He emphasized that *D. zeae* infection is usually not materially increased by mechanical injury. Although Koehler (251) once reported that *Diplodia zeae* infection followed injury caused by corn borer, he later assumed this to be incorrect. He was of the opinion that infection by *Diplodia zeae* and *Gibberella zeae* started at nodes, and that when the fungus reaches the insect tunnel it spreads faster than it would through sound tissue. Haws (109) also reported that wounds made by the corn borer were not important as portals of entry for stalk-rotting organisms. This is contrary to evidence obtained at Minnesota and Oklahoma where it has been shown that these fungi gain direct entrance through insect wounds and tunnels. At least in certain areas insect injuries are of importance.

From rotted stalks in the vicinity of corn borer tunnels, Taylor (351, 353) isolated the following organisms most frequently: *Fusarium moniliforme*, *Cephalosporium acremonium*, *Diplodia zeae*, *Gibberella zeae*, and *Nigrospora oryzae*. Others less prevalent were *Mucor* and *Rhizopus* species.

Messiaen et al. (214) concluded that, although *Colletotrichum graminicolum* in France penetrates corn directly, it also gains entrance through tunnels produced by the corn borer. According to Savulescu and Rayss (303), the angoumois grain moth is a vector for *Nigrospora oryzae*, but Reddy and Holbert (273) did not consider it of any importance.

Southern corn root worm, the northern corn root worm, and other soil insects injure roots (Fig. 1H, 9A), and are often associated with root rot, lodging, and basal stalk rot of corn. Control of these insects may help reduce the amount of lodging and stalk rot. Summers (349) exposed corn plants to *Pythium graminicola*, to larvae of southern corn root worm, and combinations of both; all caused reduced root and top development. The fungi most prevalent in secondary roots and tissues in the three lower internodes of the maize stalk were *Pythium graminicola*, *Gibberella* and *Fusarium* spp., and other species of *Pythium* (Fig. 9B).

Michaelson (218) made wounds in corn stalks simulating mechanical damage of the European corn borer. After treating the surface of the corn stalks with 70% ethanol, a horizontal hole about 6 mm in diameter was cut with a sterilized cork borer into about the middle of the stalk. The hole was covered with grafting wax or tape to prevent contamination. Then inoculations were made with *Gibberella zeae* and *Diplodia zeae* in the internode above the hole and below the same hole. There was no more rotting or injury than in the non-injured internodes. This type of injury did not predispose the tissues to stalk-rot infection.

Chiang and Wilcoxson (40) introduced egg masses of the European corn borer, inoculum of *Fusarium graminearum*, or both, into artificial insect tunnels in corn stalks of many varieties. Putty was used in 1959 to plug the tunnels to prevent drainage of fluid from the tissue, but insect establishment was thereby considerably reduced. They were plugged with cotton in 1960. When it was desirable to produce tunnels aseptically, a portion of the stalk was first washed with 70% ethanol and then drilled with a flamed bit. Egg masses in the black-head stage, averaging about 20 eggs per mass, were placed on paper discs about 12.5 mm in diameter and were inserted into artificial tunnels. *Fusarium graminearum* was increased on autoclaved wheat kernels and a single infected kernel was placed in the tunnel. The influence of the European corn borer in the development of necrotic tissue was very pronounced 4 weeks after treatment. The amount of necrotic tissue in the stalks infested with corn borer larvae was always more extensive than when the stalks were not infested (Fig. 2E). The following organisms were isolated from necrotic lesions: *Fusarium graminearum*, *Fusarium moniliforme*, *Diplodia zeae*, and species of *Penicillium*, *Aspergillus*, *Alternaria*, *Rhizopus*, yeasts, and bacteria. *Fusarium graminearum* in the tunnel with the corn borer larvae favored rapid development of the larvae.

Large quantities of frass may be deposited within insect tunnels or in the space between the leaf sheath and the stalk. Frass is an excellent substrate for microorganisms, and they undoubtedly increase on it. Some fungi, such as *Penicillium oxalicum*, can kill host tissue in advance of penetration by producing toxic substances as they grow on frass or other substrates (58). Many organisms considered to be saprophytes or weak pathogens may in this way become involved in the development of stalk rot. As shown above, many of these organisms are commonly isolated from rotten tissue as well as from insects.

Nematodes.—Data on the effect of nematodes on the severity of stalk rot are meager. Edmunds (83) found that soil fumigation for the control of nematodes not only reduced root rot during the early stages of the growth, but reduced the incidence of stalk rot from about 56% to about 27%. In this case the presence or absence of stalk rot was determined by squeezing the stalk by hand.

Miller and his associates (220, 221, 222) found an apparent relationship between the number of nematodes and root rot. Graham and Holdeman (103) also showed a direct correlation between nematode buildup and root injury in corn. There is good evidence that nematodes often increase the amount of root rot in the presence of pathogens on diverse crops. Since many of the stalk-rotting pathogens may enter through the roots, it seems probable that certain

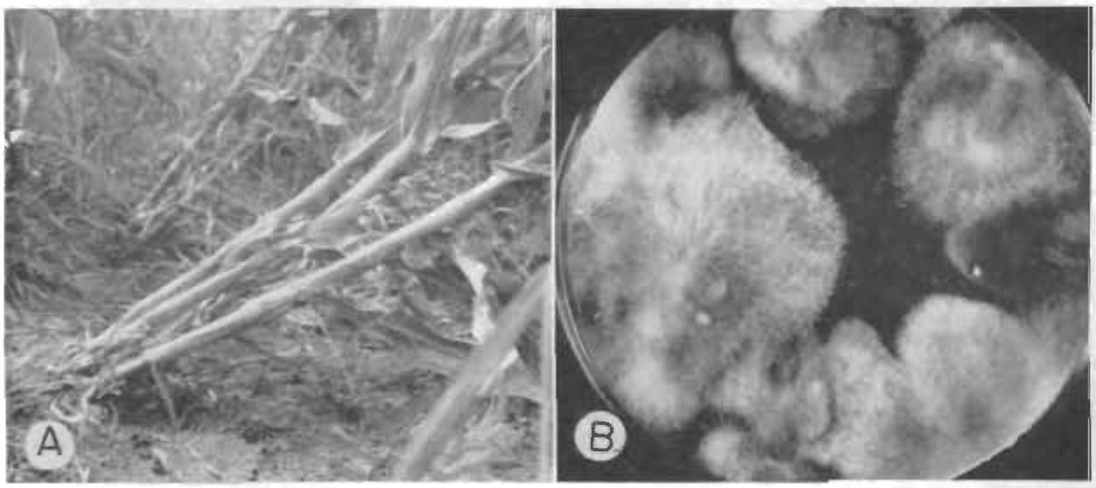


Figure 9. **A.** Leaning corn plants, due to root damage by northern corn root worm and associated root rot. **B.** Fungi isolated from roots of plants shown in A. The fungi are species of *Fusarium* and *Trichoderma*.

nematodes should also help to establish these fungi in the corn plant.

Inoculation Procedures

Production of Inoculum, and Inoculation.—A rapid, simple, and reliable technique for inoculation of corn is of primary importance in testing inbred and hybrid corn for stalk-rot resistance, and would also be of great value in studying the inheritance of resistance. Such a method would permit the use of fewer plants.

Heald et al. (112) in 1909 were the first to inoculate corn stalks with a fungus. Since then inoculation of stalks has been used extensively in testing corn for resistance to stalk rot. It has made possible the elimination of vast numbers of susceptible inbreds in the early generations. It also makes possible the testing of some plants with two or more races of a species, or different species of pathogens in the same or different plants. Inoculation has been used successfully with many fungus pathogens and with two or more bacteria.

Some of the more common methods used to inoculate corn stalks are presented here. Smith and Holbert (328) inserted an awl into the part of the plant to be inoculated, and the opening was filled with a spore suspension. Smith, Hoppe, and Holbert (329) used a somewhat similar technique: the internode was punctured with a steel needle and then a water suspension of inoculum was injected into the puncture. Ivanoff (138) described a needle inoculation method which has been extensively used for *Diplodia* inoculations by Koehler (169) in Illinois, as well as by workers in other states. The needle has a side opening through which the inoculum is ejected by gravity; the outflow is regulated by an air valve or the size of the needle. Use of a needle with the opening in the side rather than at the end prevents clogging of the needle. Although the stalks can be inoculated quickly by these injection techniques, there is usually a surplus of inoculum delivered each time. Also, the needle is usually inserted at an angle from above which leaves an opening that admits water during rainy weather, and many other organisms may be washed into the wound. This method would be undesirable if different isolates or species of fungi are placed in different sites on the same stalk.

The toothpick method of inoculation developed by Young (401) and Koehler (165, 169) is now used extensively. Since special care must be used in preparing the material, a short description of the method is given. Round wooden toothpicks about 6 cm long are used. They must be boiled several times (about 1 hr each time) in tap water to remove toxic substances that inhibit the growth of fungi. After each boiling they are thoroughly washed in fresh tap water. When the toothpicks are thoroughly dry they are packed into glass jars (about 150-200 per jar), and enough potato-dextrose broth is added to thoroughly moisten the toothpicks and leave a slight excess in the bottom of the jar. The jars of toothpicks are sterilized immediately after the broth is added, and inoculated with broth cultures of the pathogens or with bits of agar cultures. The fungi rapidly cover the toothpicks, and inoculum is ready for use in about 1.5 weeks (Fig. 10A, B).

To inoculate plants with toothpicks, the desired internode is opened with a punch and the toothpick is inserted into the hole. No special precautions are taken against surface-borne contaminants. A punch for making the holes in the stalks can be made by driving a nail into a short wooden handle or by filing off a regular ice pick. The punch should be about 15 mm long, and the diameter should be relatively small. The punch should not pass through the stalk, and the hole should be slightly smaller than the toothpick which should fit tightly in the stalk (Fig. 10C, D). The toothpicks are often inserted upwards at an angle, presumably to reduce secondary infection. If two different isolates or species are used per stalk, it is highly advisable that they be placed at least two internodes apart. The technique can be used for fungi that do not normally sporulate in culture. The toothpick method is considered one of the most rapid and feasible means of inoculation. With practice, one person can easily inoculate from 500 to 700 plants per hour; on several occasions more than 1,000 stalks have been thus inoculated. Also one can readily inoculate the same plant at different internodes with several species of fungi, or races of the same species, without obvious contamination. A relatively uniform amount of inoculum can be delivered to each plant since the fungi grow relatively uniformly over the toothpicks. Toothpicks also serve to mark the sites of inoculation. Because of this, untrained people can be used in the field, and their work readily supervised.

Wernham (378, 379) modified the toothpick technique. His method, called the "pipe-cleaner" technique, consisted of cutting sterile pipe cleaners into pieces 2.5 cm in length, and soaking them in a spore suspension. An awl was used to make an opening for the insertion of pieces of pipe cleaners infested with inoculum. With a team of three, one making the holes, and two inserting the pipe-cleaner plugs, between 350 and 400 stalks per hour can be inoculated.

Another method consists of making holes in the stalk and then inserting agar cultures of whole grains of cereals. Others have used cork borers to remove a portion of stem and then have returned the plug of tissue after inoculations were made. Still others have left the hole open or covered it with certain materials. Williams and Menon (389) concluded that, for a large-scale field test, the cork borer method is the most desirable, chiefly because of ease of preparation of the inoculum and rapidity of inoculation procedure. They state that by the cork-borer method only 5 to 10 seconds are required per inoculation. Thus 350 to 700 plants per hour could be inoculated.

Semeniuk et al. (314) placed inoculum of *D. zeae* in the husks of ears and behind the leaf sheath and obtained infection, but results were so variable that the method has not been used for evaluating factors affecting disease development.

Spraying of the entire plant at intervals with a spore suspension has been used by a few workers. Reece (274) made use of this method on rather extensive field tests at St. Paul and Waseca, Minnesota, and the method was used for many years on a co-

Figure 10. **A.** Three-week-old cultures of *Diplodia zeae* (two left jars) and *Fusarium graminearum* (two right jars), growing on toothpicks. **B.** Toothpicks covered with *Diplodia zeae* (left) and *Fusarium graminearum* (right) ready for inoculation. **C.** Illustration of how toothpicks bearing inoculum are held in one hand and a punch in the other, just before a hole is punched in the corn stalk and a toothpick inserted. **D.** Necrotic lesions in a stalk resistant (left) and susceptible (right) to stalk rot. Note the larger lesions in the internodes higher on the plants.

operative project between the Departments of Plant Pathology and Botany, and of Agronomy and Plant Genetics, University of Minnesota. Usually a mixture of seven pathogens was used for inoculum: *Diplodia zeae, Gibberella zeae, Fusarium moniliforme, Helminthosporium carbonum, Penicillium oxalicum, Pythium butleri,* and *Nigrospora oryzae.* All seven species caused stalk rot when injected into the stalks at about silking time. The stalks were sprayed four times at about 10-day intervals beginning at silking stage. There is no evidence to suggest how effective the spray method is, but Reece did obtain good infection in his experiments.

A simple and easy way to produce an abundance of inoculum for spraying plants consists of growing the organisms separately in glass jars containing a mixture of sterile wheat and oats, and a small piece of potato. The pathogens can readily be washed from the grain with water and this suspension will serve as inoculum. The inoculum can also be produced in large quantities in flats, then dried and stored at low temperatures (309, 379).

Several investigators (38, 169, 389) have compared the effectiveness of the various methods of inoculation. All agree that good infection results whenever the inoculum is inserted or injected into the stalk. Results from spraying plants are not as clear cut. Whenever methods are compared, sufficient controls

must be used. It frequently happens that injured plants that are not inoculated develop a certain amount of stalk rot. The rot is presumably caused by microorganisms present on the stalk surface which find their way into the stalk through injuries. Injuries made for inoculating purposes should thus be no larger than necessary.

Most workers have confined their attention to inoculation of plants in the field. Michaelson (215) found, however, that sections of stalk tissue could be inoculated in the laboratory, and essentially the same disease reaction was obtained as when the same inbred lines or hybrids were inoculated in the field or greenhouse. He pointed out that this method provides for uniform control of environment during the test period.

Age of Plant Inoculated.—Numerous investigators have reported that stalk rot is a disease of corn approaching maturity (76, 125, 146, 161, 204, 223). There are several pathogens, however, that may gain entrance early in the life of the plant, e.g., *Diplodia zeae* and *Fusarium moniliforme.* Michaelson (218) made stalk inoculations with *Gibberella zeae* and *Diplodia zeae* at weekly intervals beginning 7 July, and ending 8 September. He obtained infection on all dates, but the earlier inoculations were much less effective than the later ones. He also found that the infection from early inoculation did not become as extensive as did infection from later inoculation, even if there was a longer period for rot development. This was true for both *Diplodia zeae* and *Gibberella zeae.* He showed that infection could occur long before pollination, and the fungi remain more or less dormant until the silking period. Michaelson also found that, although stalks of corn were susceptible to stalk rot over long periods of time, the period of greatest susceptibility began several weeks before pollen production and continued until the dent stage of maturity. This was true for both *Diplodia zeae* and *Gibberella zeae.* The data obtained by Michaelson (218) suggest that, once the plant has reached a certain stage of maturity, tbe resistance does not change. Sprague (334) and Jugenheimer (150) concluded that more stalk rot developed when inoculations were made at silking than at a later date. Sprague (334) stated that the greatest disease severity occurred when the plants are inoculated at the time of pollination. Hooker (128) made similar tests. He inoculated stalks with *Diplodia zeae* at intervals of 1 to 4 weeks after silking, and obtained similar results. The greatest amount of rot occurred after the earliest inoculation. In susceptible inbreds, the rot continued to develop for weeks, whereas in the resistant lines, very little spread occurred after the first week.

Koehler (169) also found that inoculation with *Diplodia zeae* and *Gibberella zeae* just before silking was as effective as at any later date. Inoculation 3 weeks before pollination gave poor results as compared with later inoculation. Koehler (169) indicated that inbred reactions differ when inoculated at different times. Line Ky. 27 thus developed less rot from *Diplodia zeae* and *Gibberella* zeae when inoculation was made about 4 weeks after silking, whereas with inbred P8, rot developed equally well on different dates of inoculation. In Illinois, inoculations with *Diplodia zeae* for measuring hybrid reaction were not successful when made at or shortly after anthesis for early-maturing hybrids, about 1 week after anthesis for medium-maturing hybrids, and about 2 weeks after anthesis for late-maturing hybrids (Hooker, unpublished).

There is considerable evidence that corn is very resistant to *Gibberella zeae, Diplodia zeae,* and other fungi until about silking. This is not true for *Pythium* sp. and for certain bacteria. In these cases the vigorous plants are most susceptible, at least to natural infection.

Results indicate that the exact timing of inoculation is not critical, hence one can inoculate on the same date a group of hybrids or inbred lines that differ as much as 10 days in the time when pollination occurs. This assumes that the amount of rot is used as a basis of measuring resistance, and not premature dying of the stalk.

Site of Inoculation.—There is considerable evidence that rot severity depends on the internode in which the inoculation is made, but there is no general agreement on the exact internodes to inoculate in order to obtain the most efficient results, though most workers inoculate internodes below the ear. Some report that rot develops faster and more extensively in the upper internodes than the lower ones (Fig. 10D). They point out that rot develops well in the first internode, but that this internode is shorter and often enveloped by brace roots, hence more difficult to inoculate and to split open. The second internode above the ground is usually the first elongated internode, and is most frequently used.

Hooker (128) inoculated four inbreds in the first, second, third, fourth, and fifth elongated internodes. All the inbreds did not react alike to the pathogens at the different internodes. Thus in the susceptible line, Os 420, the rot was equally severe in the five inoculated internodes. In the resistant B14, rot was least severe in internodes one and two, but it increased progressively in the next three internodes, and in the fifth internode the rot was as severe as in the susceptible inbred. Cappellini (38) in New Jersey obtained progressively greater amounts of rot with the distance up the stalk. In one year, Koehler (169) obtained significantly more stalk rot in the first elongated internode than in the third and fifth internodes. In other tests, he inoculated a moderately susceptible variety with *Diplodia zeae* in the first, third, and fifth internodes, and the amount of rot was about the same in all three internodes. In another test, he obtained more rot in the fourth internode than in the first. He indicated that the rot in the lower internode might have affected the rot in the fourth node. This, however, would not appear likely when one considers the work of Michaelson (218). Pappelis (243) found that inoculation with *Gibberella zeae* or *Diplodia zeae* in the first and fourth internodes above the ground gave significant correlations in the amount of rot in the two internodes. He concluded that it would be possible to study stalk rot by double inoculations of single plants with one or two fungi.

Our work in Minnesota clearly indicates that stalk

rot becomes progressively more severe in the internodes from the bottom to the top of the plant (Fig. 10D). This is clear with both *Diplodia zeae* and *Fusarium graminearum* inoculated into hundreds of lines of corn. The difference is more difficult to detect in susceptible plants than in more resistant ones, and the difference is most easily detected when the lowermost inoculated internode is compared with the highest inoculated internode. Often the difference in the amount of rot in adjacent internodes is slight. Disease ratings may, for this reason, be accurately made even though the same internode is not inoculated on each plant.

Differences in susceptibility among internodes may be due to differences in carbohydrate content. De Turk et al. (60) found consistently, in two single-cross hybrids, less carbohydrate and lower total sugars in the lower part of the stalk than in the middle parts of normal plants. Also important is the degree of tissue hydration and number of living cells (240, 246, Hooker unpublished).

Scoring Disease Development.—Splitting the stalk open and observing the rot is the most reliable method of determining both the amount of stalk rot and whether or not it resulted from natural infection or inoculation. It may not be the most commonly used method, but for many tests it is certainly the most precise and reliable method.

Although the amount and extent of the rot is recorded in several ways, it is usually rated on a numerical scale (169, 274, 401). One of the most common methods is to estimate the percentage of tissue decayed in an internode (Fig. 11). Whenever the rot extends beyond the internode inoculated, the total rating given is equal to the sum of the disease rating of the internodes infected. According to Barnes (16), Boothroyd and his associates used the same rating scale, but used half-unit increments from 0.5 to 4 to correspond to percentages ranging from 12.5 to 100% of the damaged internodes. With either system a higher numerical rating indicated that rot had spread into adjacent internodes.

Koehler (169) and others (38, 151, 379) measured both the extent of discolored pith and the number of rotten internodes. The area of the rotten tissue was considered in relation to the internodes. Decimals indicated the rot within and beyond a single internode. Hooker (128) used a disease scale from 1 to 6,

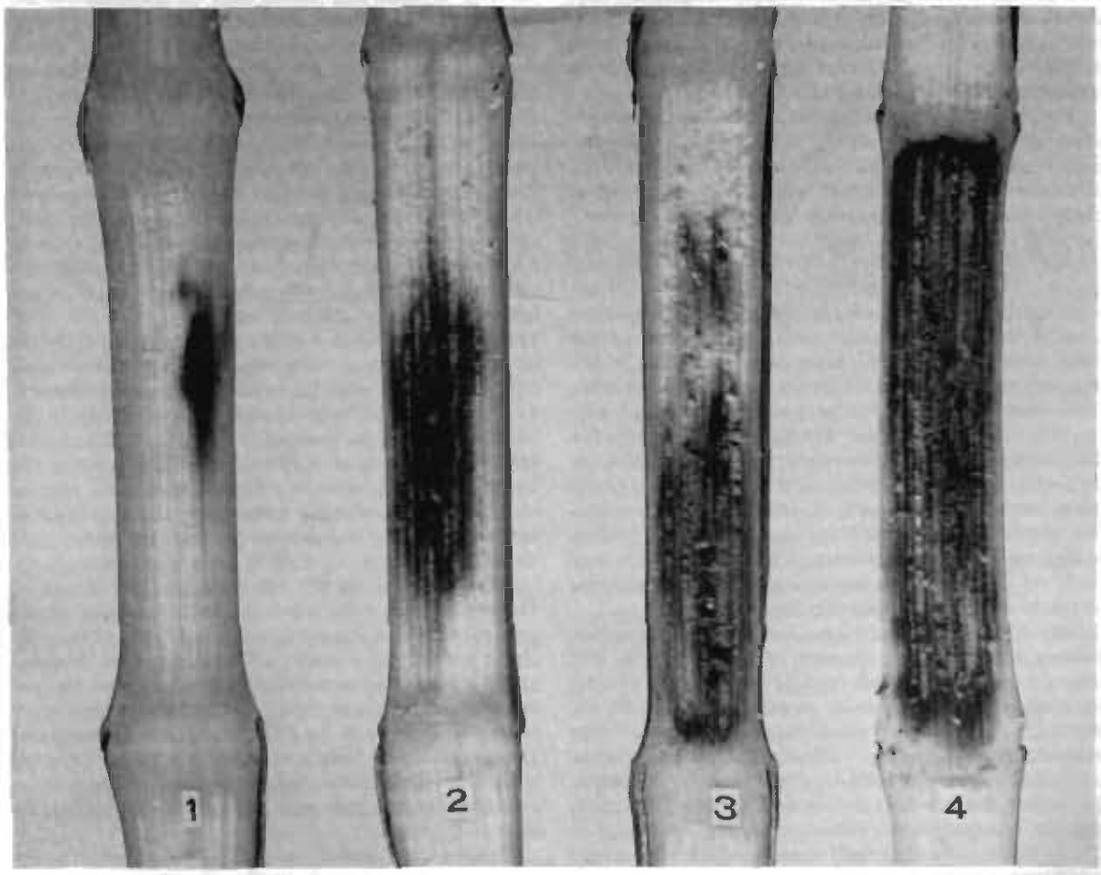


Figure 11. Necrotic lesions in corn stalks inoculated with *Fusarium graminearum* which had been growing on toothpicks. These lesions are in the second internode above the ground: 1 = less than 25% of the internode tissue necrotic, 2 = 26 to 50% of the tissue necrotic, 3 = 51 to 75% of the tissue necrotic, 4 = 76 to 100% of the tissue necrotic.

1 to 4 if rot was confined to one internode, 5 if the rot had spread into adjacent internodes, and 6 if the plant was killed. De Vay (62) used the 1 to 4 system for estimating stalk rot, but used "R" for resistance (if less than one-fourth of the tissue of the internode was rotted), "MR" for moderate resistance (involving one-fourth to one-half of the tissue of an internode), "MS" for moderately susceptible (involving from one-half to three-fourths), and "S" for susceptible (if the entire internode was decayed or if rot extended into adjacent internodes). Regardless of the method used to estimate the amount of rot, all have given excellent differential results. These estimation methods have a decided advantage, since much less time is required for evaluation than when the necrosis is measured with a ruler.

Some workers take into consideration a number of different symptoms when determining the susceptibility of corn to stalk rot. These symptoms include the amount of fruiting by the pathogen, firmness of the stalk, and amount of stalk discoloration. Koehler (169) found an association in Illinois between the average size of the lesion on the stem and the number of infected nodes. He also noted an association between both nodal infections caused primarily by *Gibberella zeae* and the spread of stalk rot induced by inoculation with *Diplodia zeae,* and premature death of plants. Reece (274) also obtained similar results in extensive tests on nodal infection.

The extent or rate of rot in cortical tissue is also sometimes used to measure severity of stalk rot. Smith and Holbert (327, 329) concluded that the spread of cortical infection was a good measure of determining the reaction of stalks to *Diplodia zeae.*

Evaluating Stalk Rot, and Selecting for Resistance

A number of authors have repeatedly called attention to the difficulty of evaluating stalk rot in the field. Some workers are primarily interested in the visible amount of rot, others are concerned with the total damage as it affects harvesting, yield, and subsequent crops. From an agronomic viewpoint, the development of varieties with good standability is perhaps the most valuable approach for obtaining plant improvement. Some, therefore, resort to selecting plants that do not lodge and have few broken stalks; such plants are assumed to have resistance to stalk rot. Such data obviously depend on the pathogen involved and the climatic conditions.

The time when final data are recorded may influence materially the amount of rot observed, and also the ease with which varietal differences can be determined. Although most workers record stalk-rot notes 3 to 4 weeks after inoculation, there are tremendous differences in this respect. The time may vary from 1 to 10 weeks, in some cases even longer. It is not uncommon to record natural infection shortly after maturity. Koehler (169) and Hooker (127) concluded that final data on stalk rot should not be taken until 3 to 4 weeks after inoculation. This may, of course, be too late if one is dealing with bacteria and *Pythium.* In other instances waiting too long may result in the conclusion that all plants are susceptible. Early hybrids may thus be dead when notes are taken, and death accelerates decay of the stalks. Data resulting from inoculation of plants should be taken before the plants die. The preponderance of evidence indicates that notes on most kinds of stalk rot should be taken about 4 weeks after inoculation. For medium and late maturing inbreds and hybrids this time interval may be increased (Hooker, unpublished).

Selection in the field, without the aid of inoculation for stalk rot resistance, is a slow process, and reliable selection can only be made after several years of testing in many localities. It would be highly desirable if some simple test, perhaps of chemical analysis or physiological reaction, could be used to eliminate the vast majority of material. This would reduce the amount of field testing, and hasten selection of resistant material. So far very little progress has been made in this field.

Production of Fruiting Bodies.—Production of fruiting bodies by the pathogen is sometimes used to determine that stalk rot has occurred, and to identify the pathogen in the field. *Sclerotium bataticola* can thus usually be identified and the prevalence and severity of natural infection may be estimated in the field by the presence of sclerotia and by the dark discoloration of the basal portion of the stem. This is true at least in certain seasons and regions. *Diplodia zeae* usually produces pycnidia, but it is usually 2 to 3 weeks after infection before fructification begins, if it begins at all in that growing season. *Gibberella zeae* may or may not produce the ascigerous stage during the current growing season. Some isolates may not produce the perithecial stage. Furthermore, many diseased areas of the stalk may harbor fungi that do not produce conspicuous sporulation on the plant surface. Production of fruiting bodies sometimes is not a reliable index of stalk rot.

Relation of Seedling Blight to Stalk Rot.—Most of the organisms that cause seedling blight also cause stalk rot. It has been important to ascertain whether or not inbred and hybrid corns react similarly to the same organism in the seedling stage and in adult plants. As there may be pronounced differences in the tissues involved, there is no good reason to assume that plants of different ages react similarly to the same pathogens. It is not surprising, therefore, that there is no general agreement on this question.

Koehler et al. (159) reported that perithecia of *Gibberella zeae* were much more abundant on stalks grown from seed susceptible to scutellum rot than on stalks grown from sound seed. Reddy and Holbert (273) found that susceptibility to scutellum rot was correlated with susceptibility to *Cephalosporium acremonium.* Holbert et al. (121) concluded that plants grown from seed harvested from corn plants infected with *Diplodia zeae* developed more ear rot than when the plants came from seed of plants free of *Diplodia zeae.*

Reilly (275) found no association between seedling blight caused by *Penicillium oxalicum* and stalk rot of adult plants caused by *Gibberella zeae.* There was, however, a high correlation between seedling blight and stalk rot caused by *Diplodia zeae.* Semeniuk

(316) failed to show an association. Hooker (126) also failed to find an association between seedling blights and stalk rots caused by *Diplodia zeae* or *Gibberella zeae*. Young (403) found that differences in reaction of inbreds to seedling blight and stalk rot were rather pronounced. Inbred Minnesota C. 11, for example, was very susceptible to *Diplodia* seedling blight, but one of the most resistant to stalk rot caused by the same isolate. Inbred Minnesota C. 14 was, conversely, one of the most resistant to seedling blight, but most susceptible to *Diplodia* stalk rot. Blaak (21) states that, in general, the results of numerous workers are inconclusive in respect to a correlation between seedling blight and stalk rot caused by *Gibberella zeae*. Semeniuk (319) found corn seedlings were susceptible to *Sclerotium bataticola* in greenhouse trials. He also noted that some inbred lines and hybrids were more resistant than others. He suggested that seedling reactions might be useful for selecting resistant plants, but we know of no instance where this has been done. Resistance of seedlings to *Pythium* root necrosis has been reported by several people (127, 271, 320).

Inoculation of Stalks.—Inoculation of stalks by diverse methods is used extensively in selecting for resistance to stalk rot (Figs. 2D and 10D). Plants found to be resistant when inoculated may not necessarily be resistant to natural infection; there are many exceptions. Zuber et al. (409) found an inbred line resistant to stalk rot when inoculated with *Gibberella zeae*, but Koehler (169) reported it susceptible to natural infection in Illinois. In attempting to explain these differences one should not forget that there are many pathogens as well as biotypes of pathogens.

Despite the fact that natural infection and inoculation do not always give the same result, many investigators have reported statistically significant correlations between infection resulting from natural and artificial inoculation (150, 169, 334). Sprague (334) and Jugenheimer (152) reported progress in breeding for resistance to stalk rot on the basis of artificial stalk inoculations. Whenever a large number of inbred and hybrid lines are to be evaluated, it is usually possible to eliminate most of the susceptible lines simply by inoculating them with the desired pathogens. It should be remembered, however, that inoculation does not indicate any morphological resistance the host may possess.

Whitney and Mortimore (381, 383) concluded that root rot was always associated with stalk rot. They suggest that root-rot reactions might be used as a basis of evaluating stalk-rot resistance. Hooker (126) found an association between root rot and basal stalk rot. He favored the estimation of root rot on the basis of severity of stalk rot because of the comparative ease of observing disease development. McNew (204) also obtained a high correlation between natural infection and artificial inoculation. Sprague (334) found a close relationship between *Diplodia* stalk rot and the percentage of stalk breakage. Zuber et al. (409) obtained no significant correlation between stalk-rot resistance and lodging and disease rating for *Diplodia zeae* and *Gibberella zeae*, respectively. Smith et al. (329), using inoculation, noted a correlation between the spread of rot in the pith and the rind, between natural infection and the percentage of broken stalks, between the spread in pith and natural infection, between pith spread and broken stalks, between the spread in the cortex and natural infection, and between cortical spread and the percentage of broken stalks. On the other hand, Troyer (360) obtained a negative correlation between the mean stalk strength of 193 inbreds and the percentage of plants with stalks broken below the ear.

Sprague (334) secured a close correlation between infection induced by inoculating with *Diplodia zeae* at the base of stalks and that obtained by natural infection with *Gibberella zeae*. Andrew (6) and Koehler (165), on the other hand, concluded that inoculations with *Diplodia zeae* were unreliable when selecting for resistance to *Gibberella zeae*. Koehler (169) stated that the spread of rot resulting from inoculation with *Diplodia zeae* and *Gibberella zeae* was correlated with premature death of corn plants induced by natural infection.

Koehler (169) states that, in comparative tests, *Gibberella zeae* is less pathogenic than *Diplodia zeae*. This has also been our frequent experience. It is not always true however, and as Koehler points out, natural infection caused by *Gibberella zeae* sometimes is just as severe as that caused by *Diplodia zeae*. The reasons for this are not definitely known, but the numerous races or biotypes of *Gibberella zeae* and *Diplodia zeae* could perhaps account for these differences.

Sprague (334) and Wernham (380) expressed a widely held opinion that hybrids resistant to *Diplodia zeae* are also usually resistant to *Gibberella zeae*. Hooker (126) presents data to support the opinion. Young (403), Reece (274), Semeniuk (315), Koehler (169), and Kochler and Boewe (164), however, have reported that certain inbred lines and varieties do not react similarly to these pathogens. Koehler (169) found a big discrepancy between the response of one inbred and a single-cross hybrid which appeared resistant to natural infection but highly susceptible when inoculated with *Diplodia zeae* and *Gibberella zeae*, respectively. He also reported outstanding differences between the reactions of inbreds and hybrids to *Diplodia zeae* and *Gibberella zeae*. Jugenheimer (150, 151), however, concluded that inoculation was far superior to natural infection in selection of resistant corn strains.

Barnes (16) inoculated inbred and hybrid corn at the same stage of maturity by the toothpick method. He concluded that, although inoculation appeared practical, one should question the advisability of relying too much on this method, because in several instances he found a lack of positive correlation between natural infection and infection by inoculation among certain varieties. This was also found by Semeniuk (315). The reasons for these inconsistent results may be found in differences of inoculum, temperature, and fertilizer. Precautions are necessary when selecting hybrids, and the decision should not be based on one year's results.

Prematurely Dead Plants.—In some regions, especially in Iowa and Illinois, premature death of corn

plants is one of the important types of damage caused by stalk rot (Fig. 1D). Koehler (169) thought that premature dying of plants was one of the best methods for estimating the comparative severity of stalk rot in a field. As corn matured, the percentage of prematurely killed plants usually increased. He obtained a high correlation between premature dying, and both diseased nodes per stalk resulting from natural infection, and the amount of rot that developed from artificial inoculation with *Gibberella zeae* and *Diplodia zeae*.

A group of investigators at Illinois (73, 250 to 253) studied stalk rot prevalence of some hybrids at the same location in 2 years, and at two locations in the same year over a period of 11 years. They found that premature death of the plants was an excellent method to evaluate stalk rot.

It is important to remember that the amount of premature killing varies tremendously from one region to another and apparently depends on time of infection, climatic condition, amount of moisture, topography of the land where the corn is grown, and severity of foliage diseases. Premature death may result not only from natural infection but also from infection after inoculation. According to Koehler (166, 169), premature dying occurs less frequently in plants inoculated with *Gibberella zeae* than with *Diplodia zeae*. Neither can premature death of plants be used as a criterion of infection unless there is a relatively high percentage of killed plants. Data must be taken before plants die as a result of maturity. By harvest time early varieties in a given locality are likely to have a higher percentage of killed plants than late-maturing hybrids. Data should be taken, therefore, on plants of about the same stage of maturity. It may be desirable to use several categories of maturity in rating the extent of premature killing (150). If the land is rolling, plot replication is most important.

Determination of Stalk Strength.—Firmness of the rotted areas of a stalk has been used to indicate the severity of infection (234, 235). This method consists of pinching the stalks 1 or 2 feet above the ground and giving them a vigorous push at the same time. The firmness is sometimes combined with visual observation to arrive at the disease rating. The amount of tissue softening within the rotted area may vary with the pathogens, environment, and whether in hybrids or inbreds. This is a simple and practical method. Its chief fault apparently lies in the fact that corn stalks of certain hybrids may harbor considerable rot which is sufficient to cause severe losses in kernel yield, yet the stalks are perfectly solid when squeezed between the fingers.

Attempts have recently been made to determine precisely the strength of stalk tissues by measuring the pressure needed to crush them. Rind thickness and crushing strength appear to be correlated, and vary greatly with various environmental factors. Thompson (356, 357), Zuber and Grogan (408), McRostie et al. (205), Loesch et al. (184), and Foley (98) have applied these procedures to tissues infected with stalk-rot pathogens and have found that less pressure was needed to crush tissue from internodes with rot, or from susceptible plants, than was needed to crush tissue from healthy or resistant plants. It seems likely that much could be learned about stalk strength if more of this type of work were done. The modern techniques for determining strength of wood might be useful.

Relation of Stalk Breakage to Stalk Rot.—The development of corn hybrids with improved standability has been a major objective in corn improvement for many years. Standability of a corn plant refers to its ability to remain upright in the field when the plant has reached full maturity and tissues have died. Poor standability usually includes root breakage, stalk lodging, and stalk breakage, and usually involves disease and insect damage. There may, of course, also be inherent structural weaknesses in plants. A secondary factor is wind strength; if stalks are rotted, the number of lodged or broken plants increases with wind velocity, especially late in the season. Most of the plants in entire fields might thus be broken (Fig. 1C).

Broken stalks are sometimes used to estimate the amount of stalk rot, and are also used by plant breeders in selecting for stalk-rot resistance. Broken stalks are obviously a good character to indicate to breeders whether or not the stalks are resistant to stalk rot, because broken stalks may make harvesting difficult, and delay harvesting.

The development of high-yielding hybrid corn that is resistant to the lodging and stalk breakage caused by plant pathogens is one of the most difficult problems encountered in breeding desirable agronomic hybrids. These characters are associated with many variables, including physiological and morphological traits, plus resistance to plant pathogens and insects.

The role of stalk-rot organisms in breakage of stalks has long been recognized. Pammel et al. (239) considered that much stalk breakage is the result of stalk rot. Since then many workers have reached the same conclusion.

Different methods have been used in the field to determine standability of corn under natural conditions: pushing the stalk, hitting it gently with a board paddle, kicking the base of the stalk, comparing the number of stalks fallen and lodged with the number standing. Pulling devices have been used to determine relative root anchorage (106). These are helpful methods, but they do not necessarily give definite information on severity of stalk rot in relation to standability. Some corn with good standability may actually be susceptible to stalk rot, and thus sustain considerable reduction in yield (385).

Durrell (77) used a mechanical procedure for breaking the stalks and found that rot reduced the breaking strength of stalks about 50%. He attributed this stalk weakness to assimilation of cellulose and lignin by stalk-rotting organisms. Since then, many have reported that microorganisms account for much of the stalk breakage and lodging.

Zuber and Grogan (408), employing a mechanical test for evaluating stalk strength, concluded that crushing strength of a stalk was correlated with stalk lodging and rind thickness. Loesch et al. (184) later concluded that the thickness of rind was not affected

by infection with *Diplodia zeae,* and that plants with thicker rinds had stronger stalks, and the rot did not significantly influence the amount of lodging in such plants.

Koehler et al. (160) concluded that *Diplodia*-infected seed reduced yields about 30% and increased leaning of plants by 10%. They attributed leaning and broken plants to rotting of primary roots and mesocotyl, which resulted in underdevelopment of the secondary root system. Planting of seed infected with *Cephalosporium acremonium* also increased the percentages of broken stalks. Inoculating seeds with *Gibberella zeae* increased the percentage of leaning plants. There was no significant increase, however, in percentages of broken stalks from the use of seed naturally infected with *Diplodia zeae, Gibberella zeae,* or *Fusarium moniliforme.* Durrell (76) also reported that no relationship was evident between diseased seed and breakage of plants at the first few nodes above the ground. Jugenheimer (150) concluded that there was an association between the amount of stalk rot and lodging in inbred lines. Others have reached similar conclusions. Since stalk rot is not necessarily the primary cause of all lodging, many investigators refer only to stalk breakage, and place lodging in a specific category. Nelson (230) recognized stalk rot as a cause of lodging, but thought that lodging and disease should be treated separately. He pointed out that stalks may be weakened when in dense populations. Others have emphasized that large populations often induce spindly stalks.

Koehler et al. (160) emphasized that throughout the United States corn seed is often internally infected with *Fusarium moniliforme,* yet there is no indication that percentages of leaning plants are increased by sowing such infected seed. This is rather surprising, as *F. moniliforme* often causes severe stalk rot.

Although the percentage of broken stalks is not always correlated with severity of stalk rot, it is frequently used to estimate the amount of stalk rot. There may be many factors that influence the amount of stalk breakage in a region, such as physiological and morphological characters of the plant, variation in soil type, fertility, moisture, and temperature, amount of wind at maturity, insects, and heritable weakness as expressed under certain conditions. Stalk rot usually does not cause breakage of stalks until the plant approaches maturity. The earlier the infection develops, usually the greater the amount of stalk breakage, and the longer mature plants with stalk rot are left in the field, the greater the amount of stalk breakage. This is particularly true if there are frequent rains, followed by relatively warm weather and strong winds.

There is a tendency for stalks of certain hybrids and inbreds to break primarily at definite places on the stalk. Of course, the point of breakage is dependent on the plant part attacked and the pathogen involved. Stalks attacked by bacteria and *Pythium* nearly always break near the ground line. In healthy plants, internodes and nodes near the ground are usually much stronger than those higher up on the stalk.

According to Foley (97), more than 95% of the stalks broke near the node in the lower portion of an internode. The node at which breakage occurred varied with the hybrid. Certain ones nearly always broke at the third node above the ground, others broke with about equal frequency at the third, fourth, or fifth node. He observed no increase in percentage of stalk breakage through artificial inoculation of the stalk, although he thought that much of the stalk breaking was related to stalk rot. Corn plants with extensive decomposition of stalk parenchyma usually had the highest percentage of broken stalks. He observed that many broken stalks were not badly rotted, yet severely rotted stalks were not always broken. Young (403) and Wilcoxson (385) found that inoculation of stalks with *Diplodia zeae* above the ears reduced yield in the absence of broken stalks. There is fairly good evidence that certain hybrids and inbreds have good standability, although the internal tissue is very susceptible to internal rot by *Diplodia zeae* and *Gibberella zeae.* During a 7-year period of testing commercial varieties in Minnesota (258 to 263, 361), there was no obvious association between yield and percentages of broken stalks. Thus, in 1961 some varieties with 20 to 40% stalk breakage outyielded those with 15% or less, and vice versa. Similar results were also apparent in other years. It should be emphasized, however, that on small trial plots all the ears are picked by hand, and such tests do not necessarily represent a fair comparative yield trial when the same varieties are mechanically harvested. In tests of the latter type, the amount of broken and lodged stalks and dropped ears might materially alter the harvested yield.

Resistance to *Pythium* stalk rot and bacterial stalk rot is often based on the percentage of plants that topple over at the ground line. In this instance stalk breakage appears to be entirely adequate and characteristic for the diseases.

Resistance

Resistance of corn to stalk rot involves many physiological, morphological, and perhaps functional characteristics, which are in turn influenced by many factors. As far as we know, no inbred or hybrid of corn is immune to stalk rot. Some are considered highly resistant to certain pathogens, at least to certain biotypes of the pathogen. Certain hybrid varieties are now grown in the corn belt that appear to be fairly resistant to stalk rot. How long these will remain resistant is unknown, and whether they will be resistant in other areas remains to be seen.

Artificial epidemics are usually more comparable from year to year than natural infections. It is, therefore, highly desirable to have an efficient, convenient, and reliable method of producing an artificial infection. In recent years much progress has been made on techniques for testing lines and varieties of corn. There is no mutual agreement on the method of selecting for resistance. Pammel et al. (239) were perhaps the first to suggest that development of varieties resistant to stalk rot was the desirable method for controlling this disease. Since then many investigators have reported pronounced variation in inbreds and hybrids. Jugenheimer (150) found that there was

a tremendous variation among inbreds that were exposed to natural infection. In 1934, the infection ranged from a disease rating of 1 (very resistant) to 4.9 (very susceptible). In 1935 differences were almost as great. The differences in disease ratings in crosses ranged from 1.4 to 4.4. Koehler and Boewe (164) found that during the severe epidemic of stalk rot in 1946, the worst on record, all susceptible inbreds and single-crosses were completely killed, and all the stalks were broken as early as September. Certain inbreds and crosses had relatively little internal rot, and were either resistant or moderately resistant. The difficulty of obtaining resistant hybrids may be illustrated by the work of Wiser et al. (397), who reported that inbreds vary tremendously in their resistance to ear rot; some transmit a high degree of resistance, whereas others impart susceptibility to their progenies. Russell (289) stated that ability of an inbred to contribute to stalk resistance can be measured satisfactorily only by testing hybrid combinations in the field. Taylor (352) found that, when inbreds were artificially inoculated with *Cephalosporium acremonium*, they were more susceptible than the hybrids.

Resistance to basal stalk rot and stalk rot involves resistance to several pathogens. While resistance to one or more pathogens has been frequently reported, there is no evidence that any variety is resistant to all stalk-rotting organisms. Varieties considered resistant to *Diplodia* stalk rot are very susceptible to bacterial stalk rot in Egypt (294).

Sprague (334) stated that reaction of corn to *Diplodia zeae* provided a measure for resistance to stalk rot in general. There can be no question that a great many inbreds and hybrids can be eliminated by testing either with *D. zeae* or *Gibberella zeae*, but varieties resistant to one pathogen are not necessarily resistant to other pathogens, and resistance in one part of a plant does not insure resistance in other tissues (126). Young (403) and Reece (274) found that certain inbreds and hybrids reacted differently to different pathogens. Some were susceptible to *Gibberella zeae*, but strikingly different in their reaction to *Diplodia zeae*. DeVay et al. (62) inoculated more than 100 inbreds and hybrids with *Diplodia zeae* and *Gibberella zeae* on toothpicks, and the disease rating was based on amount of necrosis of the stalk 3 weeks after inoculation. They found tremendous differences in the degree of susceptibility. Some inbreds were resistant to *Gibberella zeae*, but highly susceptible to *Diplodia zeae*. The reverse also occurred infrequently.

Wood (398) obtained marked differences in reaction of 24 single-cross dent-corn hybrids to root rot and basal stem rot in Ohio when they were grown in soil previously cropped to corn. All 14 crosses except one involving the two parental inbreds, Ill.A and WF9, had more than 50% diseased roots. The other 10 crosses with the inbred, Oh45, had a lower disease rating than did other crosses.

Open-pollinated corn varieties also differ in susceptibility to stalk rot. Some think that open-pollinated corn is more susceptible than hybrids derived from inbreds of a given variety. All open-pollinated varieties are obviously made up of a population of plants different from one another not only in agronomic characters, but in disease reaction to different pathogens. The plants in hybrid populations are obviously more or less uniform in genetic constitution, including disease reaction. All disease reactions including stalk rot often appear much more uniform and conspicuous, therefore, in hybrid varieties than in open-pollinated varieties.

Sidorov et al. (324) tested 38 inbreds, hybrids, and varieties of corn for resistance to *Diplodia zeae* in Russia. They reported marked differences in susceptibility. Flint corns were generally more resistant than dent corns, and certain inbreds from Argentina and the USA were resistant. Sansom (300) reported that *Diplodia zeae* was prevalent in Rhodesia. All the local varieties were susceptible to stalk rot, but improvement has been made by hybridization and selection. Elliott (90) found varieties of corn resistant to *Pythium* sp. The Egyptian workers considered many local varieties resistant to bacterial stalk rot (294).

In important corn-growing areas of the USA there are hundreds of hybrid varieties of corn available for planting. These varieties differ greatly in many characteristics, including their tendency to lodging and stalk breakage, and their susceptibility to internal necrosis which may reduce grain yield without breakage of stalks. The performance of hybrids in particular regions or states can, fortunately, be obtained from the various experiment stations (71, 72, 231, 258 to 263).

Some hybrids yield better than others when infected with either *Diplodia zeae* or *Gibberella zeae* (218, 385, 387); this suggests a certain amount of tolerance to stalk-rot infection. There is a need for much more testing of hybrids for this valuable character under different field conditions.

According to Sprague (334), there are marked differences in frequency of genes for resistance to stalk rot in different varieties. In one variety the incidence of genes for resistance is low, but much higher in a synthetic variety made up of many stiff-stalked inbreds. It should be remembered that stiff-stalked varieties are not necessarily resistant to stalk rot.

Smith et al. (329) found hybrids of resistant and susceptible inbred lines to be fairly resistant to cortical spread of *Diplodia zeae* when artificially inoculated. This indicated that dominant factors imparted resistance to *D. zeae*. Jugenheimer (150) studied resistance to 412 inbreds, 93 single-cross, and 63 top-cross hybrids over a period of years. He found that 24 top-crosses were decidedly more resistant than the 24 inbreds involved. The rotted internodes of inbreds had an average disease rating of 3.2, and top-crosses only 2.2.

Young (403) compared the pathogenicity of 6 species of fungi: *Diplodia zeae*, *Gibberella zeae*, *Fusarium moniliforme*, *Penicillium oxalicum*, *Pythium butleri*, and *Helminthosporium* sp., on eight inbreds and four single-crosses of corn. Tests were made on both seedlings and adult plants. On the seedlings, *Gibberella zeae* was most virulent, followed in order of decreasing virulence by *Helminthosporium*, *Pythium butleri*, *Diplodia zeae*, *Fusarium moniliforme*, and *Penicillium oxalicum*. Differences among

the pathogens were statistically significant. All inbreds were susceptible to *Gibberella zeae* and *Helminthosporium* sp. The inbreds were generally susceptible to the other four pathogens, although in some cases an inbred was susceptible to one pathogen and resistant to another, and vice versa. With the adult corn, Young (403) likewise found differences in the reactions of the adult plants to the six pathogens. McKeen (199) found that certain inbreds were susceptible to *Gibberella zeae* and resistant to *Pythium* sp., and vice versa.

Jugenheimer (150) studied six inbreds and nine single-crosses. The average *Diplodia* disease rating for the susceptible inbreds was 5.6 (amount of pith rotted in the internodes), and the resistant inbred averaged 3.1, whereas the single-crosses averaged 3 5. Resistance in crosses thus approached the resistant parental inbreds. In the matter of cortex rot similar results were obtained. He also found that some crosses were less resistant than the parental inbred line. He concluded that, although resistance to stalk rot was complex and partially dominant, it appeared to be due to many factors. He also concluded that some inbreds were more potent in transmitting resistance than others. Taylor (352) studied the inheritance of corn to stalk rot caused by *Diplodia zeae*. He concluded that inheritance was complex, and reported that the F_1 hybrids between moderately resistant and susceptible inbreds approached the resistant parent. The F_2 hybrids and backcrosses were intermediate between the two original parents.

Sprague (334) concluded, on the basis of inoculations with *Diplodia zeae*, that resistance of the F_1 was intermediate between two parental inbreds, one susceptible and the other resistant. The F_2 had a little more resistance than the F_1, while backcrosses tended to approach the reaction of their recurrent parent.

Reece (274) studied the inheritance of stalk-rot reaction in the field to two specific organisms, *Diplodia zeae* and *Gibberella zeae*, and also to a mixture of stalk-rotting organisms, including *Diplodia zeae*, *Gibberella zeae*, *Fusarium moniliforme*, *Helminthosporium carbonum*, *Penicillium oxalicum*, *Nigrospora oryzae*, and *Pythium butleri*. He studied inheritance of stalk-rot resistance between two crosses grown at St. Paul and Waseca, Minnesota, for 2 years. The type of inheritance was quantitative in nature. He also obtained a high correlation between stalk-rot reactions of F_3 and F_4 plants to general field infection and to *Gibberella zeae* inoculations, but this was not true for *Diplodia zeae* inoculations. He found highly significant differences among F_3 and F_4 lines to *Diplodia zeae* and general field stalk rots.

Zuber et al. (409) obtained good agreement between the reaction of inbred parents, F_1 and F_2 progenies, and backcrosses when inoculated with *Diplodia zeae* and *Gibberella zeae*. They concluded, therefore, that the reaction of the inbreds gave as much information on resistance as the F_1 and F_2 crosses.

Hooker (unpublished) has studied in Illinois the inheritance of resistance to *Diplodia zeae* in numerous inbred lines through inoculation of inbred, F_1, F_2, and backcross populations. The data indicate that resistance is a quantitative character, and inherited on a multiple-factor basis. Heritability values were quite high. Although reacting similarly as lines, some inbred lines were more effective than others in transmitting resistance in hybrid combinations.

Most workers agree that resistance is inherited in a quantitative manner and that the degree of resistance in hybrids is usually proportional to the number of resistant inbreds used in the cross. There is a strong tendency for corn hybrids to respond similarly to the stalk rot caused by different pathogens. The hybrid plant, whether a single- or double-cross hybrid will tend usually toward resistance or susceptibility, depending upon the genetic makeup of the parental material. It is, of course, important to choose parents adapted to a particular region.

According to Koehler (169), hybrids once considered resistant have been discarded because of their susceptibility. It is reasonable that this should happen, because stalk rot is a complex disease involving many pathogens which consist of many races and biotypes of differing parasitic ability.

Since seedling blights and ear rots are often related to stalk rot, some information about resistance to these diseases will be presented. Holbert et al. (122) showed that strains of corn differed in their resistance to root rot when the kernels were inoculated with *Gibberella zeae* before planting. McIndoe (197) studied the inheritance of seedling blight due to *Gibberella zeae*. He found that crosses between resistant and susceptible inbreds gave different results in the F_1 generation. The hybrids were sometimes resistant and sometimes susceptible. Two parents intermediate in susceptibility sometimes produced resistant progeny; crosses between two very resistant parents sometimes produced progeny that were intermediate in resistance. Hayes et al. (110) obtained variable results and concluded that reaction of seedlings to *Gibberella zeae* was of little value in a corn-breeding program. Blaak (21) obtained somewhat similar results. Barnes (14), however, found that seedlings from parents resistant to stalk rot were much less severely infected than those seedlings from parents susceptible to stalk rot. He concluded, however, that many unrelated crosses should be made before definite conclusions are drawn regarding the relationship of seedling resistance to stalk-rot resistance. Such a test should be made with a number of different species and isolates of pathogens.

Young (403) found that the resistance of a cross to seedling infection was always more or less equal to the more resistant parent. Although in some cases this relationship held true for the reaction to stalk rot, there was no general or consistent relationship between reactions of parents and progeny. Since inbreds did not usually react the same to seedling blight and stalk rot, he concluded that the same genetic factors do not control the reaction to both seedling blight and stalk rot. The disease reactions of certain inbreds were, moreover, specific to only certain races or biotypes of the pathogen. Tests for resistance should, therefore, not be based on single isolates but on many isolates from different sources.

Smith and Trost (332) obtained no correlation between the amount of *Diplodia zeae* ear rot in inbred lines and their progenies. Wiser et al. (397) concluded that inheritance of corn ear-rot resistance was quantitative. The inbreds which have essentially similar resistant reactions to ear rot differed widely in their ability to transmit the resistance. Savulescu and Rayss (303) reported that *Nigrospora oryzae* did not attack Romanian maize. *Zea mays* var. *vulgata*, but attacked severely *Z. mays* var. *dentiformis* grown on the Danube plains.

Holbert et al. (121) concluded that corn resistant to ear rot was also resistant to root and stalk rot. Koehler and Holbert (161) reported that resistance to ear rot was dominant.

Since inbred and hybrid corn varieties differ greatly in resistance to stalk rot, it is important to know the nature and cause of these differences. For plant breeders to make rapid progress and to utilize inbred lines most effectively and efficiently, they should understand the nature of resistance. It would be extremely important to learn whether there is a generalized type of resistance against the major stalk-rotting organisms. It would be desirable to learn why one inbred or hybrid is resistant to *Diplodia zeae* but susceptible to *Gibberella zeae*, and vice versa. In recent years many attempts have been made to account for differences in susceptibility among corn varieties. These studies involved physiological, biochemical, and morphological tests.

It is well established that corn stalks inoculated at about silking time are much more susceptible than when the plant is growing rapidly. Corn stalks at this time are more susceptible in the upper internodes than in those near the ground. This difference in resistance tends to disappear in 2 or 3 weeks. Although early-maturing varieties tend to be more susceptible than late varieties, sometimes the reverse is true. These differences in resistance and susceptibility suggest that physiological changes and differences must occur in the host. Studies on the differences in resistance among inbreds and hybrid crosses which are in the same maturity class also indicate that these differences are primarily physiological. It is true, however, that certain types of standability may be morphological rather than physiological; standability of a variety is not necessarily an indication of resistance to rot within the stalk.

There are conflicting reports on the morphological nature of resistance. Durrell (76, 77) indicated that resistant inbred lines contained more lignified tissue, especially in the lower nodes, than did susceptible inbreds. This was determined by recording with a spring balance the pressure required to break the stalk. It required about twice as much pressure to break the stalk of a healthy node as a diseased one. Hunter and Dalbey (137) observed an association between anatomical structure of inbred lines and the tendency to lodge. Loesch et al. (184) concluded that rind thickness was not affected by *Diplodia zeae* infection, but that the crushing strength of the stalk in lodged and susceptible crosses was reduced. On the basis of histological studies, Magee (189) concluded that resistance to stalk breakage probably involved both morphological and physiological factors. Stiff-stalked inbred lines generally had greater stalk diameter, thicker cell walls in the epidermis, fewer vascular bundles in the rind, and a higher percentage of sheath per bundle than the weak-stalked inbreds. Certain weak- and strong-stalked inbreds, however, did not differ morphologically. Boothroyd (27) determined the stalk diameter, thickness of rind, number of vascular bundles in the rind and the pith of inbreds with poor and good standability, but found no association between these characters and standability of corn. Hoadley (116) failed to find any indication that stalk-rot resistance was associated with mechanical barriers.

Fusarium moniliforme is retarded in the corn seedling by the endodermis of the radicle (373). Blaak (21) made extensive histological studies of tissues of primary and secondary roots, mesocotyl, and coleorhiza. This work involved 18 varieties of corn differing in resistance to stalk rot. He concluded that there was no correlation between the histological properties and resistance to the seedling blight caused by *Gibberella zeae*. He concluded that standability of corn was associated with a high number of vascular bundles in the fourth node above the soil. Even when the plants were partly rotted by *Gibberella zeae*, the number of vascular bundles appeared to give strength to the stalks.

Johann and Dickson (146) found that certain inbred and hybrid lines contained a substance in the stalks which, when extracted and introduced into a culture medium, retarded the growth of several organisms. This test involved 11 inbreds and five single-crosses that differed markedly in susceptibility to stalk rot. Extracts obtained before the silking period of plant maturity, from all material, retarded the growth of *Diplodia zeae*, *Gibberella zeae*, and *Nigrospora oryzae* in a nutrient culture. They found that the inhibitory substance was less abundant as the plant approached maturity: the decrease in the amount of the substance was slower in the resistant stalk than in the susceptible one. This was related to the greater susceptibility of the plants as they matured. Defoliation and the prevention of pollination did not modify the growth-retarding effect of the substance. Whitney and Mortimore (382) obtained a crude ether extract from two-months-old corn plants which proved to be inhibitory to *Gibberella zeae* and *Fusarium moniliforme*. At first they thought that the high degree of stalk-rot resistance in young corn plants might be due to a high level of this compound, but later considered it unimportant when they found about equal amounts in resistant and susceptible hybrids. Barnes (15, 16), using *Gibberella zeae* as an assay organism, concluded that there is an inhibitory substance in resistant stalks that is present in significantly higher concentrations than in susceptible stalks at the early stages of kernel development. After silking, the toxic substance decreased in all inbreds and crosses. He considered the substance to be fungistatic rather than fungicidal to *Gibberella zeae*. His results indicated the substance, 6-methoxy benzoxozolinone, did not occur in a free state in the corn plant, but existed as a glucoside precursor. He postulated that the invading fungus

caused an irritation of the host cell which stimulated the release of the antifungal compound.

Pappelis (240, 241) found that, at the end of the growing season, the spongy pith tissue of lodged and stalk-rot-susceptible varieties was dead, whereas similar tissue from non-lodged, but resistant varieties, was alive. He suggested that living cells continuously form a phenolic compound, and when wounded they release sufficient toxic substance to inhibit the invading fungi.

Foley (95) reported that cellulase occurred in corn plants susceptible and resistant to *Fusarium moniliforme*. He suggested that resistant plants might contain a cellulase inhibitor.

Barnes (15, 16) also isolated an indole-containing compound from the corn stalks which stimulated the growth of *Gibberella zeae*. Substances that stimulate growth of fungi might, therefore, he as important as inhibitory substances. Taylor (351) obtained similar results. He found that the plant juice from resistant plants supported the growth of *Diplodia zeae* somewhat better than juice from susceptible plants. He also incorporated juices from nodes and internodes into agar and grew *Diplodia zeae* on them. These gave no indication of differences in resistance among inbred lines or parts of host involved.

Andrew (6) thought that hybrid corn resistant to one agent might be susceptible to another, and this might be due to different chemicals present in the various hybrids.

Considerable evidence has been obtained that the level of sugar in the stalk may greatly influence the severity of stalk rot. Dickson (64) and Dickson and Holbert (65) reported that high pentose sugar in corn seedlings renders them susceptible to blight; whereas, corn high in hexose sugars was resistant. Cell walls of the highly resistant inbreds were more highly suberized than those of susceptible inbreds. Plants, when grown at higher temperatures, 24° C or above, were more highly suberized (249). Peterson found no relationship between sugar content of plants and resistance to seedling blight (257).

Morris (228) and Sayre et al. (305) found that total sugar in corn stalks reaches a peak about one week before fertilization of ears, and then decreases with maturity. These changes were in concentration of sucrose rather than in the level of free reducing sugars. They also found that removing the ears from the stalks caused 3 to 5% increase in sucrose over that of fruiting stalks. Barren plants are usually resistant to stalk rot. The removal of some of the leaves of the corn plant reduced sucrose content of the stalks; this explained the increased susceptibilty to rot when plants were defoliated.

Holbert et al. (125), from experiments in which pollination was prevented and which involved partial defoliation, concluded that low carbohydrate content of the stalk increased susceptibility to *Diplodia* stalk rot. De Turk and associates (60, 61) found that, during September, susceptible hybrids had less total sugar in stalks and shanks than did the more resistant crosses. Differences in sugar contents were also greater, or at least as great, in the lower part of the stalk as higher up. They also found that removing about 30% of the leaves during 10 days after pollination increased susceptibility to *Diplodia* stalk rot. There was an equal amount of reducing sugar in both the resistant and susceptible varieties. Messiaen (213) found in France that hybrid corn was more resistant when the sugar content was higher than 59% in the basal portion of the stem. Craig's studies (53, 55) indicated that sugar content of susceptible inbreds decreased the first 4 weeks after pollination, whereas it increased in resistant inbreds.

Zuber et al. (409) found a high correlation between stalk rot caused by *Diplodia zeae* and *Gibberella zeae*, and high nitrogen content of the stalk. There was a negative correlation with content of cellulose, crude fiber, lignin, and ash. They believed that infection of stalks by both *D. zeae* and *G. zeae* might interfere with translocation of substances as well as with their elaboration and utilization.

The mode and nature of action of fungi are important as they relate to available food in the plant. Durrell (76) pointed out that *Diplodia zeae* was an omnivorous feeder and grew well on most standard media; it even grew well and fruited profusely on pure cellulose agar. Since it can readily utilize cellulose, it is easy to understand why it grows well on the nodes and weakens the stalk. Because *D. zeae* grew on a great variety of substances he concluded that it produced many different enzymes.

Stevens and Wilson (343) reported that *Diplodia zeae* produced a biotin-like substance, but *Diplodia macrospora*, a less common species of *Diplodia* on corn, did not. Both species require the biotin-like substance for growth. Apparently the corn plant provides this vitamin for *Diplodia macrospora*, and a similar situation may prevail for other more important pathogens.

Kent (154) found that *Diplodia zeae* produced a substance during normal growth which inhibited its own growth and retarded spore germination. McNew (202) had previously shown that an extract of old *Diplodia zeae* cultures not only inhibited the fungus in culture but also prevented seedling blight. It is also possible that substances released by fungi may make corn more susceptible to stalk rot. Wilcoxson and Sudia (384) found that gibberellic acid made corn seedlings more susceptible to seedling blight.

Johann (142) showed that *Penicillium oxalicum* produced lesions on seedlings of corn in advance of the hyphae. By killing the tissue in advance of growth, the fungus paved the way for its ingress and perhaps that of other fungi. According to McNew (204), *Diplodia zeae* also produced lesions on the mesocotyl of corn in advance of the mycelium. Roberts (278) noted marked discoloration of vascular tissue in corn stalks by *Diplodia zeae* in advance of the mycelium. Similar discoloration of vascular bundles is also caused by *Gibberella zeae*, *Fusarium* sp., and *Cephalosporium* sp. The killing of cells and tissues in advance of the fungus may be important in the physiological damage to the host.

Craig and Hooker (54) made histological examinations of adventitious roots and stalks infected with *Diplodia zeae*. They observed browning and deposition of granular material in the roots. The vessel ele-

ments of the infected roots and stalks were occluded with a lignin-like material, as indicated by staining with phloroglucinol. In many cases there apparently were no hyphae present in the occluding substances. They believed that the most damaging aspect of the infection was the plugging of the vascular system. Johann and Dickson (146) reported that discoloration of tissue by fungi was darker in resistant than in susceptible tissues. The spread of *Diplodia zeae* was limited to the dead cells. Also the inherent susceptibility to stalk rot was associated with the prevalence of dead parenchyma cells in the stalk pith. Pappelis (240) and Pappelis and Smith (241, 246) found that stalks with whitish pith tended to be susceptible to *Diplodia zeae*. Cells containing air were low in specific gravity, non-living, and more susceptible to attack. Pith with high density had a high percentage of living cells and was associated with resistance to stalk rot. Craig (53) and Craig and Hooker (55) obtained similar results on specific gravity of pith tissue in relation to susceptibility to stalk rot. There also was a tendency for the specific gravity to diminish with time after silking. The data indicate an inverse association between specific gravity and susceptibility to stalk rot. Wernham (380) recorded a positive association between susceptibility to stalk rot caused by *Gibberella zeae* and the percentage of dry pith in the stalk internodes. More attention should be given to this association, as Pappelis has made the same observation in connection with *Diplodia zeae* and *Gibberella zeae* (242, 246).

Bijl (19) stated that the mycelium of *Diplodia zeae* is confined to the cell lumen, and the hyphae grew from cell to cell through the pits in the cell walls. Hyphae were never observed to bore directly through the cell wall. Pearson (249) studied the parasitism of *Gibberella zeae* on seedlings.

Although stalk rot of corn has been studied extensively during the past 50 years, little is known about what happens to cells as necrosis develops. The size and color of the lesions are usually described along with the observation that the pith disintegrates, leaving only the vascular bundles and the sclerous outer tissues of the stalk (169, 200). Darkened vascular bundles (Figs. 7B, 12) extending from the necrotic lesion have been mentioned by several workers (54, 76, 204, 217, 273), but no explanation of their origin has been given.

It is difficult to study the development of stalk-rot lesions occurring naturally on plants in the field because one does not know where lesions will be and they are often large before they are discovered. Roberts (278) and Littlefield and Wilcoxson (180, 181) studied this problem by following the development of lesions on inoculated plants. They think that the development of lesions resulting from natural infection and inoculation is similar. Roberts (278) found that the necrosis spreads upward and downward from the point of inoculation; in 2 weeks the necrotic area reached the nodes from the point of inoculation located midway in the internode. Some necrotic vascular bundles also passed through the node and may extend as far as the fourth internode above the inoculation site. The intensity of the coloration of the pith tissue and bundles may vary, even with a single pathogen, from light to dark brown, and there may be a dark band that encircles the entire rotted area. In a few days the infected pith tissue of the inoculated internode begins to deteriorate, and gives rise to the dry rot so characteristic of *Diplodia zeae*. Roberts found that the discolored cells surrounded the bundles, extending from one bundle to another. These cells collapsed and were irregular in shape. The parenchyma cells outside this band were only slightly discolored, and some were not necrotic at all. Hyphae of *Diplodia zeae* were observed to be intracellular adjacent to the darkened band of pith tissue. They were also observed to a limited extent in cells slightly discolored. The number of hyphae varied from a few strands to a ball of mycelium. Dark material in the infected cells increased in intensity as the amount of mycelium increased. Wherever discoloration of the pith cells occurred, the intercellular spaces were filled with a dark-colored substance. Necrosis within the bundle was usually confined to the xylem and phloem tissues; the phloem was more intensely discolored than the xylem. The hyphae were observed in the lumen of the sieve tubes in the phloem, and the vessels in the xylem were often plugged with a mass of the dark brown hyphae of *Diplodia zeae*. The hyphae observed in the phloem were much smaller than those found in either the pith or xylem. Necrosis

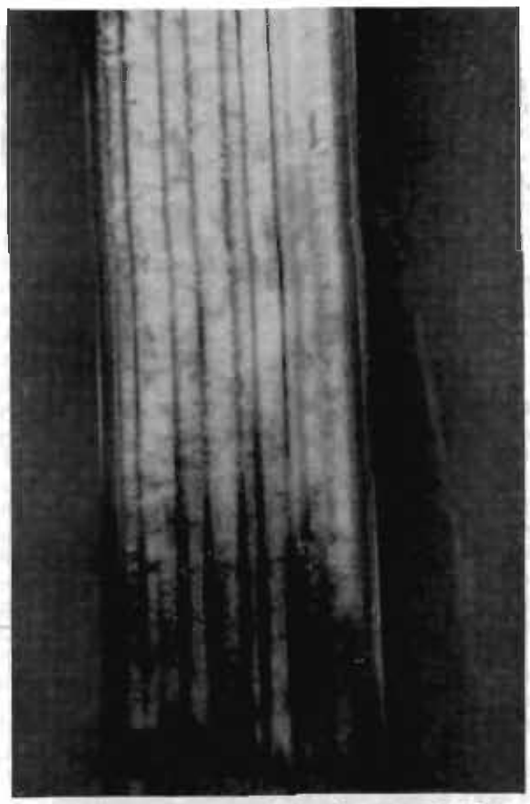


Figure 12. Dark vascular bundles extending from a stalk-rot lesion produced by inoculating with *Fusarium graminearum*.

of bundles did not decrease as they passed through the node. After a few weeks, deterioration frequently had begun in the internode above the one inoculated. Necrotic bundles often extended to the fourth internode above the inoculated internode. *Diplodia zeae* was isolated from the bundles in the third internode above the point of inoculation. Furthermore, most of the upper internodes were necrotic at the end of 4 weeks. Strands of necrotic tissue extending from the site of the inoculation into the node and the next internode are not uncommon. These strands were associated with chlorotic streaks in the rind tissue. Five weeks after inoculation, the rind was almost completely necrotic, and quickly lost rigidity. Sporulation was abundant 4 weeks after inoculation.

Results of Littlefield and Wilcoxson (180, 181) were similar to those just mentioned, but present more details on lesion development. They studied two stalk-rot susceptible inbred lines and two resistant lines inoculated in the second internode above the soil with *Fusarium graminearum* during anthesis on 9 August, 1961. Inoculated internodes were collected 3, 6, 9, 12, and 15 days after inoculation, and fixed in formalin-alcohol-acetic acid solution. Free-hand sections of the internodes were made with a razor blade, and mounted in glycerin-jelly or lactophenol. The sections were made across the central portion of the necrotic area close to the point of inoculation (the A zone), across the margin of the necrotic lesion (the B zone), across the tissues outside the necrotic area but containing darkened vascular bundles (the C zone). In this paper the different parts of the lesion will be referred to by the A, B, or C designation. Except where specified, all sections were taken from the central (pith) vascular system rather than from the peripheral (rind) vascular system. None of the sections was stained, and all were cross sections except where it was necessary to have longitudinal sections. Approximately 22,000 sections were examined. The development of lesions in the several inbred lines was similar. The lesions in resistant lines differed from those in susceptible ones only in size, not in the way they developed.

The first noticeable change in the tissues of zone A was darkening of cell walls in the pith surrounding vascular bundles and the occluding of intercellular spaces of the pith with an amber to brown substance of dried resinous appearance (Fig. 2A). Walls of the sieve tubes and companion cells also began to darken. About 10% of the bundles had vessels and tracheids that were partially to completely occluded. Six days after inoculation the lesions were usually large enough to be divided into the A, B, and C zones. On the sixth day, the walls of pith cells surrounding the vascular tissue in zone A were very dark, and heavy intracellular occlusions had begun to form. Phloem cell walls were more intensely discolored than on the third day and, in about 50% of the bundles, sieve tubes and companion cells were occluded. The protoplast collapsed in many of the sieve tubes. The vessels were occluded in about 10% of the bundles. Nearly all phloem in zone C bundles was normal except that a few cell walls had darkened. About 25% of the darkened bundles were occluded (Fig. 2B and C). By the ninth day after inoculation the discoloration and occluding of the pith parenchyma was much more intense than on the sixth day. Essentially all vascular bundles in zone A had heavily discolored and occluded phloem, and about 50% had occlusions in the vessels. The occlusion and discoloration of the pith cells in zone B was much less intense than in zone A. The phloem cell walls in zone B were discolored, but there was less plugging than in zone A. Nine days after inoculation none of the sieve tubes or companion cells of zone C contained occlusions, but most had discolored walls. Approximately 25% of the bundles had partially occluded vessels. Pith cells appeared healthy in zone C. Twelve days after inoculation the occlusion and discoloration of parenchyma cells surrounding the vascular bundles was very pronounced. Occlusion of xylem and phloem in zone A was less prevalent than at 9 days. This might have been due to translocation of the substance into the B zone where about 25% of the bundles had occluded phloem, and 25-40% occluded xylem. In zone B the pith discolorations were less intense than in zone A. In zone C, 12 days after inoculation, no phloem occlusions were found, and less than 50% of the darkened bundles had discolored phloem cell walls. About 50% of the darkened bundles contained occluded vessels, and all were surrounded by healthy pith cells. The tissues infected for 15 days were essentially the same as those infected for 12 days.

To identify chemically the substance occluding the tissues, histochemical studies were made. Tests for lignin, lipids, proteins, and starch were all negative. Resorcin blue stained the majority of the occlusions on which it was tested, but analin blue did not. When the tissues were placed in ruthenium red, the occlusions in xylem were stained in more than 90% of the bundles. The occlusions in phloem and pith tissues were, however, not stained. Solubility tests were also made to further characterize the occluding material. The substance was insoluble in hot water, ethanol, formalin-alcohol-acetic acid solution, saturated copper oxide ammonia, and 1% NaOH. The substance thus appeared to be pectin or a substance containing pectin. It did not appear to be callose, lignin, lipid, protein, or starch.

Sections were also studied from lesions of 10 plants inoculated with *Diplodia zeae* since Craig and Hooker (54) reported finding lignin in occluding substances occurring in xylem of plants infected with that pathogen. Some sections were treated with ruthenium red, a stain for pectic compounds, and others with phloroglucinol, a stain specific for lignin. The occluding material in the xylem was stained with ruthenium red, but in the phloem and pith it was not. None of the substance was stained with lignin stain. When sections of tissue from lesions were placed in pectinase to determine whether the enzyme could remove the occluding substances, the pectinase failed to remove any occlusions in the cells, and it caused no dissolution of the parenchyma tissues located in necrotic lesions, particularly older lesions. In healthy tissues, pith immediately around the vascular bundles was dissolved and all of the bundles were left free of

the parenchyma tissue. In 2-day-old necrotic lesions about 80% of the vascular bundles were separated from the parenchyma tissue. In 4-day-old lesions practically none of the parenchyma tissue was dissolved by pectinase, and only one or two per cent of the vascular bundles were separated from the pith cells. At this time large quantities of the dark-colored materials were present in the pith cells. The parenchyma cells surrounding the dark vascular bundles extending out of the necrotic areas were frequently dissolved hy the pectinase, but if the cell walls had begun to darken or if the dark substances had begun to accumulate, the pectinase did not affect the tissues. In all sections, when necrotic or healthy tissue was treated with pectinase, the phloem partially to completely disintegrated. In healthy tissue treated with pectinase, the parenchyma and the phloem tissues disintegrated, and the vascular hundles were left free.

Littlefield and Wilcoxson (181) also studied the development of necrosis in plants naturally infected with stalk-rot fungi. Naturally occurring stalk rot usually is not detected in Minnesota until after mid-September. The stalks selected for sectioning had dark lesions on the nodes, were beginning to soften slightly, or were quite soft when squeezed hetween the thumb and fingers. The sections were made with a razor blade and mounted in glycerine-water solution. The most noticeable macroscopic characteristic of the tissue was the disintegration of pith parenchyma surrounding the vascular bundles. The disintegration of the parenchyma tissue began immediately around the vascular bundles in the outer areas of the central vascular system, and the parenchyma around the more internally located bundles later began to disintegrate. Most of the parenchyma in the stalk gradually disintegrated, and finally produced an outer shell (the peripheral vascular system) with loose vascular bundles of the central vascular system extending between the nodes without supporting ground parenchyma (Fig. 2G and H). The result of this decay was a loss of stalk strength and an increased susceptibility to lodging. All bundles in the central vascular system were free from discoloration and no occluding substances were present in any of the tissues. The phloem had completely disintegrated in all bundles observed. Blackened bundles were present only in the necrotic areas of the peripheral vascular system of these naturally infected plants. Approximately 5% of the bundles in the necrotic peripheral vascular system had xylem vessels partially to completely occluded. The occluding substance appeared to be similar to that described from inoculated plants, and it also stained with ruthenium red. There was darkening of cell walls and accumulation of intercellular brown substances in the sclerenchymatous tissue making up the nonvascular tissue of the rind. The most consistent effect of natural infection observed in peripheral vascular bundles was the complete disintegration of phloem tissue. Mycelium was often observed in the area formerly occupied hy phloem.

It has often been questioned whether the lesions in corn stalks resulting from inoculation are essentially like those resulting from natural infection. The evidence by Roberts (278) and Littlefield and Wilcoxson (180, 181) indicates that the development of lesions in both naturally-infected and inoculated plants is essentially the same process. In both kinds of plants, the pathogen generally first acted on the parenchyma cells surrounding the vascular bundles, then the phloem cell walls were either discolored or disintegrated, and finally many xylem cells were occluded with a dark colored substance. When lesions were found in the pith of naturally infected plants, the plants were senescent; the phloem and parenchyma cells had disintegrated, and there was no occlusion of xylem. The senescent cells were not capable of forming the dark substance before they disintegrated. The differences noted may also result because lesions in inoculated plants are not usually more than 15-30 days old, whereas in naturally infected plants they may be much older.

The importance of pectic-enzyme activity in the development of lesions should be investigated further. The growth of *Fusarium graminearum* and *Fusarium moniliforme* on pectin in vitro, and the similarity of healthy tissues treated with pectinase and those in naturally infected plants, suggest that pectinase is involved in the formation of lesions. As indicated by staining reactions, pectin was present in the occlusions in xylem of inoculated plants, although the occluding material was not hydrolyzed by pectinase. The pectin component of the occlusions may have been chemically or physically united with other components in such a way as to render them resistant to the action of the pectic enzyme. A similar resistance to pectinase in lesions produced hy tobacco-mosaic virus was reported hy Weintraub and Ragetli (377). While in vitro studies indicated that pectinase caused the disintegration of tissues within the stalk and presumably decreased stalk strength and increased lodging, more work is needed before it will be understood how enzymes may be important in the development of necrotic lesions in corn stalks.

Control

The control of stalk rot should be a major objective in the corn-producing areas of the USA, if not of the world, because of the tremendous loss each year. Although production of corn for grain without stalk-rot losses is unlikely at present, damage has been reduced. No one of the procedures discussed here will, by itself, control stalk rot, hut it is widely held that various combinations of these procedures often produce good results (147, 169, 331).

Planting Sound Seed.—Many investigators attributed slow and unthrifty growth of corn to the influence of seed-borne pathogens (162). Jebel et al. (140) found tbat root rot, ear rot and stalk rot, premature death, and broken shanks were usually less pronounced in plots planted with seed from ears without internal cob discoloration. The differences were, furthermore, greater on poor than on good soil. These observations suggest that control of stalk rot, in general, must also involve control of seedling blight and root rot. The selection of sound disease-free seed is therefore necessary. This is sometimes difficult because several pathogens, such as *Fusarium monili-*

forme and *Cephalosporium acremonium*, are carried internally in the seed. Seed may be infected internally with several fungi without showing external symptoms.

Holbert et al. (122) concluded that many seeds have only small infections and are most difficult to detect, even by a germination test. Such seed may produce plants that are severely infected with root rot and stalk rot.

Seed Treatment.—All seed corn should be treated with a fungicide that kills the parasites rather than with one that merely delays infection. Seed treatment may also prevent, to some extent, infection by pathogens in the soil, such as *Gibberella zeae, Fusarium* sp., and *Cephalosporium maydis*. There is fairly good evidence that seed treatment tends to delay mesocotyl invasion when the seed is planted in infested soil, and that this may reduce crown rot and basal stem rot (29, 163).

Kernels injured during processing are more apt to become infected that are sound seeds. Treatment will protect such seed against infection. It is practically impossible, in the USA, to obtain seed of commercially grown hybrids that has not been treated. This practice has undoubtedly produced more uniform stands of healthy corn.

Chemical Control.—In contrast to the developments in chemical treatment of seed, there has been practically no work to control stalk rot with chemical treatment of soil or foliage (318, 322). If infection through the roots is a common means of stalk invasion, spraying to protect plants against stalk rot would be fruitless. There is good evidence, however, that stalk rot may also result from local infection of the stalks. In regions where infection occurs through the nodes or leaf sheaths, spraying with fungicides at appropriate intervals might be helpful. There is real need for studies on effect of spraying with fungicides in different geographical regions.

Britton and Hooker (31) applied two protective fungicides to corn plants at weekly intervals, beginning one week before silking, hut failed to reduce the amount of stalk rot. They indicated that it was possible that the two fungicides used were non-toxic to *Diplodia zeae* and *Gibberella zeae*, the cause of most of the stalk-rot infection in Illinois. It is also possible that much of the infection occurred prior to the beginning of fungicide application. They thought that their experiments indicated that little, if any, infection occurred through the basal nodes.

We tried to control stalk rot in 1961 by treating soil with chemicals. We selected a number of fungicides reported to be effective against soil-borne pathogens, and applied them to the soil in different amounts just before seed was planted. Some of the plots were also fumigated with methyl bromide several weeks before seeding. The experiment was located in a region where stalk rot has been a continuing problem for many years, and included both resistant and susceptible hybrids. None of the chemicals resulted in control of the disease.

It is possible that chemical control might be developed if new approaches were made. Bacterial diseases are controlled with antibiotics, and Sabet (291) indicated they may have some promise in control of these diseases on corn. McNew (202) indicated that a filtrate from old cultures of *Diplodia zeae* prevented seedling blight when infected seeds were soaked in it. Perhaps the substance would be effective if it were sprayed onto stalks.

Crop Rotation and Sanitation.—Crop rotation is often suggested as a possible method for controlling stalk rot (66, 169, 335). Ullstrup (364) probably expressed the current opinion held by most persons interested in growing corn: "Crop rotation is probably more beneficial in improving soil fertility and tilth than in reducing corn diseases. Destruction of disease harboring corn refuse is hardly practical where many thousands of acres are grown." Rotation of crops and sanitation, nevertheless, have been suggested many times and perhaps more attention should be given to the problem (113, 159, 170, 236, 393). Such control measures might he useful in some parts of the world, or when biological control metbods are to be preferred. In regions with considerable rainfall, especially in those regions in which the soil is not frozen a long time, decay is rapid and antibiotic action should be greater than in northern regions. In addition to possible effects on pathogens, rotations may improve plant growth and thus help to offset some of the effects of disease (321).

Crop rotation has little effect on stalk rot incited by *Pythium* spp. No differences in stand were obtained when various lines of corn which differed in susceptibility to seedling blight were planted in a crop sequence at Rosemount, Minnesota. There was some evidence that soil previously planted with some of the crops in the sequence tended to reduce spore germination of *Fusarium graminearum* and *F. moniliforme*, but this did not influence disease (191).

There is very little experimental data on the effect of crop rotation in relation to stalk rot of corn. Since certain studies indicate that stalk rot of corn may arise from root and crown infection, crop rotation might well affect the amount of stalk rot. Some investigators think that, when corn is grown on the same ground year after year, the pathogen increases in abundance. Although this is logical, there is inadequate experimental data to substantiate it. The continuous planting of corn for two or more years is becoming more common in the corn belt of the USA. In some fields corn has been grown continuously 20 or more years, with little indication that disease is any more severe because of it. The relationship of crop succession to stalk rot, stalk breakage, as well as yield, is very important, especially as crop rotations become shorter or are abandoned.

Williams and Schmitthenner (392, 393) studied the effect of crop rotation over a 7-year period, and found that root and stalk rot was most severe in plots planted continuously to corn for 7 years, and was least in plots following soybeans. The crop-sequence study involved corn, oats, wheat, alfalfa, and soybeans. Isolations from infected stalks yielded mostly *Fusarium roseum, F. cerealis*, and a smaller amount of *F. moniliforme* and *Diplodia zeae*. The average yield of corn over the 7-year period showed a negative correlation with the amount of root and stalk rot.

Corn yields at the end of the seventh year of continuous corn were about two-thirds of the yield of the 2-year corn plot and only half of the yield of the plot preceded by soybeans. Although it is not known how rotation affects the incidence of the rot, it has a marked effect on fungus populations (390, 391). Antagonism may alter the amount of inoculum of root- and stalk-rotting organisms.

Durrell (76) suggested long rotations for control of *Diplodia* root and stalk rot. A long rotation is essential for best results, as the organism can live on old corn stalks in the field for at least 3 years. He stated that attempts should be made to hasten the decay of stalks by covering the material well during plowing. Clayton (47) found that crop rotation helped to control *Diplodia* ear rot. Whether it helped control stalk rot was not stated. Koehler (169) and others (66) think that crop rotation helps to control stalk rot.

Richardson (276) in Canada found that the corn crop grown in soil infested with root pathogens was influenced materially by the preceding crop. The best control was obtained when soybeans immediately preceded maize. Corn following timothy was most severely infected, even more severely than when maize followed maize.

Wilcoxson and Covey (387), between 1955 and 1962, found no reduction in the size of necrotic lesions in stalks of many different hybrids inoculated with *Diplodia zeae* and *Fusarium graminearum* in crop-sequence plots. They found, however, that stalk breakage and lodging varied greatly from year to year, and in some years some of the hybrids lodged more following some crops. It appeared impossible to predict which crop would favor the breaking of corn stalks when corn was planted in any single year. Boothroyd et al. (26) reported that stalk rot in corn following corn was no more prevalent than when corn followed grain or a legume. Bijl (19) claimed that the spores of *Diplodia zeae* in Africa came from old infected plant debris left in the field. The destruction of all diseased material would, therefore, reduce the amount of infection the following season. A system of crop rotation which excludes maize for 3 or 4 years from or near the infected fields of corn would further assist in controlling the disease. He stated that it is generally held that stalk rot is especially prevalent and destructive in fields where corn has been planted several years in succession. To reduce the amount of inoculum, the land must be well plowed or else the infected material may prove a serious factor, as the spores formed on it will be capable of infecting the succeeding crop. Different systems for preparing soil for crops will influence how crop debris is decomposed and how the soil itself is changed (176, 306). The complete removal, or plowing under of all the corn debris should help reduce root and stalk rots. Koehler and Holbert (161) in 1930 encouraged the removing or plowing under of corn refuse, even when only a single farm was involved. They emphasized, however, that sanitation would be most effective if practiced on an extensive scale. This is logical, as spores produced on old plant refuse may be carried by the wind to the new corn crop. They emphasized that if the refuse is covered by soil, spores cannot get into the air and cause infection. They may have overlooked the fact that after each farm operation some of the undecayed stalks, roots, and ears are returned to the surface. Corn refuse in soil can also induce root rot and crown rot, as certain of the stalk-rotting pathogens can readily live in soil for 1 year, and some for 2 or more years.

A few workers think that sanitation has little value in controlling stalk rot. Parker and Burrows (247) concluded that leaving crop residues on the soil surface decreased the amount of root and stalk rot. We have seen no increase in stalk rot on plots to which extra corn debris has been added during the past 6 years. Many remedies for control of stalk rot encourage the practice of sanitation. Such practices include clean plowing, ensiling of corn, or burning of all corn refuse, and some also suggest burning of *Fusarium*-infected straw of other cereals. The burning of any plant refuse is not considered good agronomic practice, and probably should not be recommended.

When corn fodder or ears are fed to animals, the pathogens may remain alive in unconsumed plant parts. The manure containing the plant parts may, therefore, harbor the pathogen, and should be turned well under when the soil is plowed. Bijl (19) stated that spores of *Diplodia zeae* were capable of germination after having passed through the digestive tract of mice, but germination was greatly reduced after 12 hours in the digestive tract. Spores which passed through a canary were dead. The presence of bacteria in excreta could inhibit the growth of fungi or perhaps kill them. Stiemens (345) found that when spores of *Diplodia zeae* and *Gibberella zeae* were fed to cattle, spores of the former were not viable when recovered from the manure, and those of *G. zeae* were not recovered at all, apparently being digested. No data are available which indicate how long the spores can persist in barnyard manure. It is possible that they may even multiply there; it is also possible that urea and microorganisms may kill them. Studies are needed in this area.

Use of a Balanced Fertilizer Program.—Over a period of years it has been repeatedly observed that stalk rot is much more destructive in a field than in one nearby, although the same hybrid is grown in each. The reason for this is obscure. The availability of nutrients could account for the differences. It is well established that plants in soils containing a large amount of available nitrogen or organic matter are more subject to attack by *Pythium* spp., *Diplodia zeae*, and *Gibberella zeae*, especially when potassium is low. Whenever large amounts of nitrogen fertilizer are applied, therefore, a proper amount of potassium should be added. Thrifty plants are especially susceptible to infection by bacteria and *Pythium*. See other parts of this paper for a more complete discussion of the problem.

Control of Diseases and Insects.—In other portions of this paper the influence of insects and diseases on severity and abundance of stalk rot has been considered. If insects were controlled by the use

Figure 13. Corn stalks which have been bent over below the ear to hasten drying of the plants to gain some control of stalk and ear rots as well as insects, birds, and storm damage. (Photo courtesy of D. J. Welhausen, The Rockefeller Foundation.)

of resistant hybrids, chemicals applied to soil or foliage, or other control measures, the control of stalk rot should be made easier in some instances.

Resistant Hybrids.—The most logical method for controlling stalk rot is to grow resistant hybrids which are adapted to the region desired. This has not always been possible, but growers in certain areas are now able to obtain hybrids with published records of performance. Minnesota growers, for example, may obtain hybrids with a record of less than 10% of the plants lodged at harvest time (258 to 263). This is also possible in other states (251). Growers should contact their agricultural experiment stations for the best information available for their region. These performance trials provide an excellent record of the progress in development of stalk-rot-resistant hybrids.

Miscellaneous Control Measures.—Early harvest is highly desirable as it is well known that the longer standing corn is exposed to weather, the more stalk breaking is likely to occur. Overmature corn often develops severe breakage and lodging, and mature plants often break during strong winds, especially when accompanied by rain, snow, or sleet. The artificial drying of harvested corn permits earlier harvesting than formerly, and growers have found that such a practice definitely reduces loss from stalk breakage and lodging.

Melhus (211) stated that *Diplodia* ear rot in Guatemala was most severe in wet regions, and control consisted of bending over the stalk below the ear at about maturity (Fig. 13). This exposed the ear to drying conditions, accelerated the drying of ears, and thus retarded the growth of the pathogen. This practice also prevented water from accumulating between the husk and kernels. The practice also helped to prevent lodging and stalk breakage. It requires too much hand labor, however, to be important in the USA or other areas where labor is expensive.

Corn should not be planted in cold areas, as this predisposes seedlings to disease.

It is possible that stalk rot may vary in severity because of physical properties of the soil, although Boothroyd et al. (26) obtained as much stalk rot on sandy loam soil as on clay loam. Water relations are quite different in soils of different types, and this may also affect stalk rot (293). Bacterial stalk rot may be controlled by growing corn in cool dry climates where soil is alkaline or neutral in reaction. Moist warm air favors the disease (293).

Unusually dense plantings should be avoided because stalk rot and stalk lodging tend to be more severe in such situations. At present, growers are wise to use those plant populations which experience has proved will provide maximum yield.

Literature Cited

1. Al-Ani, H. Y. 1957. Association of *Cephalosporium acremonium* Corda with the black-bundle disease of corn. Iowa State Col. J. Sci. 31:349-350. (Abstr.)
2. Altstatt, G. E., and P. A. Young. 1944. Incidence of charcoal rot in north and east Texas. Plant Dis. Reptr. 28:899-900.

3. ANCALMO, O. 1961. Enfermedades del maíz en El Salvador, p. 65-72. *In* Séptima Reunión Centroamericana, Proyecto Cooperativo Centroamericano para el Mejoramiento del Maíz. 108 p.
4. ANDERSEN, A. L. 1948. The development of *Gibberella zeae* headblight of wheat. Phytopathology 38:595-611.
5. ANDERSEN, A. L. 1948. The relation of pH to sporulation and growth of *Gibberella zeae* on agar and in liquid media. Phytopathology 38: 1. (Abstr.)
6. ANDREW, R. H. 1954. Breeding for stalk-rot resistance in maize. Euphytica 3:43-48.
7. ANDREWS, E. A., and R. L. JANES. 1950. Stalk rot of corn in Michigan during 1949. Plant Dis. Reptr. 34:207-208.
8. ARK, P. A. 1940. Bacterial stalk rot of field corn caused by *Phytomonas lapsa*. Phytopathology 30:1. (Abstr.)
9. ARK, P. A. 1941. Persistence of *Phytomonas lapsa* on seed of field corn. Plant Dis. Reptr. 25:202.
10. ARNDT, C. H. 1943. Fungi affecting corn stalks, leaves, and sheaths in northwestern South Carolina in 1943. Plant Dis. Reptr. 27:562.
11. ARZBERGER, E. G. 1913. The cob-rot of corn. Ohio Agr. Exp. Sta. Bull. 265:69-82.
12. ASHBY, S. F. 1927. *Macrophomina phaseoli* (Maubl.) comb. nov. The pycnidial stage of *Rhizoctonia bataticola* (Taub.) Butl. Brit. Mycol. Soc. Trans. 12:141-147.
13. ATANASOFF, D. 1920. *Fusarium*-blight (scab) of wheat and other cereals. J. Agr. Res. 20:1-32.
14. BARNES, J. M. 1957. The influence of nitrogen, phosphorus and potassium on the severity of corn seedling blight. M.S. thesis, Cornell Univ.
15. BARNES, J. M. 1959. Extraction and bioassay of an antifungal substance from inbreds and hybrids of corn differing in susceptibility to *Gibberella zeae*. Phytopathology 49:533. (Abstr.)
16. BARNES, J. M. 1960. Investigations on stalk rot of corn caused by *Gibberella zeae*. Part I. A comparison of two methods of evaluating the severity of stalk rot in several corn varieties. Part II. Aspects of the biochemical nature of stalk rot resistance. Ph.D. thesis, Cornell Univ.
17. BATES, G. R. 1960. Branch of Botany, Plant Pathology, and Seed Testing. Minist. of Agric. Rhodesia Nyasaland 1958-1959.
18. BIJL, VAN DER, P. A. 1914. Preliminary investigation on the deterioration of maize infected with *Diplodia zeae* (Schw.) Lev. Roy. Soc. of South Africa. Trans. 4:231-239.
19. BIJL, VAN DER, P. A. 1916. A study of the dry rot disease of maize caused by *Diplodia zeae*. Bull. Union S. Africa Dept. of Agr. Sci. 7:60.
20. BILLINGS, F. S. 1889. Original investigations of cattle diseases in Nebraska. Nebraska Agr. Exp. Sta. Bulls. 7, 8, 9, and 10.
21. BLAAK, G. 1957. Histological studies of seedlings and older plants of several varieties of corn infected by *Gibberella*. M.S. thesis, Cornell Univ.
22. BOEWE, G. H. 1949. Bacterial stalk rot of corn in Illinois. Plant Dis. Reptr. 33:342-343.
23. BOEWE, G. H. 1949. Rosen's bacterial stalk rot vs. Elliott's *Pythium* stalk rot of corn. Plant Dis. Reptr. 33:441.
24. BOEWE, G. H. 1963. Host plants of charcoal rot disease in Illinois. Plant Dis. Reptr. 47:753-755.
25. BÖNING, K., and F. WAILNER. 1936. Welke, Fusskrankheit und andere Schädigungen an Mais durch *Colletotrichum graminicolum* (Ces.) Wils. Phytopathol. Z. 9:99-110.
26. BOOTHROYD, C. W., H. J. OTTO, and J. M. BARNES. 1955. Stalk rot of corn in New York, 1954. Plant Dis. Reptr. 39:380.
27. BOOTHROYD, C. W. 1962. Histological structure of corn stalks in relation to field resistance to stalk rot. Phytopathology 52:726. (Abstr.)
28. BORLAUG, N. E. 1945. Variation and variability of *Fusarium lini*. Minnesota Agr. Exp. Sta. Tech. Bull. 168.
29. BRANSTETTER, B. B. 1922. Treatment of seed to control root and stalk rots. Phytopathology 12: 30. (Abstr.)
30. BRANSTETTER, B. B. 1927. Corn root rot studies. Missouri Agr. Exp. Sta. Res. Bull. 113.
31. BRITTON, M. P., and A. L. HOOKER. 1963. Failure to control corn stalk rots with above ground applications of protectant fungicides. Plant Dis. Reptr. 47:470-471.
32. BRUNI, O., H. J. SAVOIA, and E. F. GODOY. 1961. Podredumbre de la raiz y podredumbre basal del tallo del maíz, p. 255. *In* Actas de la Va Reunión Latinoamericana de Fitotecnia. Tomo II. Instituto Nacional de Tecnologia Agropecuaria. Republica Argentina Secretaria de Estado de Agricultura y Ganaderia. 599 p.
33. BUBAK, F., and P. KOSAROFF. 1911. Einige interessante Pflanzenkrankheiten aus Bulgarien. Centralbl. Bakt. II. 31:495-502.
34. BURRILL, T. J. 1889. A bacterial disease of corn. Illinois Agr. Exp. Sta. Bull. 6:165-175.
35. BURRILL, T. J., and J. T. BARRETT. 1909. Ear rots of corn. Illinois Agr. Exp. Sta. Bull. 133:63-109.
36. BURTT-DAVY, J. 1914. Maize, its history, cultivation, handling, and uses, with special reference to South Africa. Longmans, Green and Co. London. 831 p.
37. CAPPELLINI, R. A. 1956. Stalk rot of corn in New Jersey, 1955. Plant Dis. Reptr. 40:244.
38. CAPPELLINI, R. A. 1959. A comparison of techniques and sites of inoculation in field corn artificially inoculated with *Gibberella zeae* (Schw.) Petch. Plant Dis. Reptr. 43:177-179.
39. CERVANTES, J. 1954. Enfermedades del maíz en México. p. 312-317. *In* Primera Reunión Centroamericana. Proyecto Cooperativo Centroamericano para el Mejoramiento del Maíz. 465 p.
40. CHIANG, H. C., and R. D. WILCOXSON. 1961. Interactions of the European corn borer and stalk rot in corn. J. Econ. Entomol. 54:850-852.
41. CHRISTENSEN, J. J. 1922. Studies on the parasitism of *Helminthosporium sativum*. Minnesota Agr. Exp. Sta. Tech. Bull. 11.
42. CHRISTENSEN, J. J., and J. E. DEVAY. 1955. Adaptation of plant pathogen to host. Ann. Rev. Plant Physiol. 6:367-392.
43. CHRISTENSEN, J. J., and J. E. DEVAY. 1956. Stalk rot of corn. Minnesota Farm and Home Sci. 13(2):3, 6.
44. CHRISTENSEN, J. J., and H. C. H. KERNKAMP. 1936. Studies on the toxicity of blighted barley to swine. Minnesota Agr. Exp. Sta. Tech. Bull. 113:1-28.
45. CHRISTENSEN, J. J., and C. L. SCHNEIDER. 1948. Corn borer aggravates stalk and ear rot. Minnesota Farm and Home Sci. 6(1):6-7.
46. CHRISTENSEN, J. J., and C. L. SCHNEIDER. 1950. European corn borer (*Pyrausta nubilalis*) in

relation to shank, stalk, and ear rots of corn. Phytopathology 40:284-291.
47. CLAYTON, E. E. 1927. *Diplodia* ear-rot disease of corn. J. Agr. Res. 34:357-371.
48. COOK, M. T. 1918. Diseases of grains and forage crops. New Jersey Agr. Exp. Sta. Circ. 102.
49. COVEY, R. P. 1959. Studies on the seedling blight of corn caused by *Fusarium graminearum*. M.S. thesis, Univ. Minnesota.
50. COVEY, R. P. 1959. The effect of 3 isolates of *Fusarium graminearum* and of herbicides on seedling blight of corn. Phytopathology 49:537. (Abstr.)
51. COX, R. S., and E. A. WOLF. 1955. A crown rot of sweet corn caused by *Helminthosporium turcicum*. Phytopathology 45:291-292.
52. CRAIG, J., and B. KOEHLER. 1958. *Pyrenochaeta terrestris* and *Phaeocytosporella zeae* on corn roots. Plant Dis. Reptr. 46:622-623.
53. CRAIG, J. 1960. Physiological, chemical and morphological plant factors in *Zea mays* L. associated with *Diplodia* stalk-rot reaction. Ph.D. thesis, Univ. Illinois.
54. CRAIG, J., and A. L. HOOKER. 1961. *Diplodia* root and stalk rot of dent corn. Phytopathology 51: 382-385.
55. CRAIG, J., and A. L. HOOKER. 1961. Relation of sugar trends and pith density to *Diplodia* stalk rot in dent corn. Phytopathology 51:376-382.
56. CROZIER, J. A., and C. W. BOOTHROYD. 1959. Tomato a new suscept of *Gibberella zeae* (Schw.) Petch. Plant Dis. Reptr. 43:446-447.
57. CUEVAS, O. 1961. Enfermedades mas importantes del maíz en Nicaragua. p. 64. *In* Séptima Reunión Centroamericana. Proyecto Cooperativo Centroamericano para el Mejoramiento del Maíz. 108 p.
58. CURRIE, J. N., and C. THOM. 1915. An oxalic acid producing *Penicillium*. J. Biol. Chem. 22:287-293.
59. CURZI, M. 1929. A serious new disease of maize. Rend. accad. Inazl. Lincei, Ser. 6a, 10:306-308 (Rev. Appl. Mycol. 9:174-175).
60. DETURK, E. E., E. B. EARLEY, and J. R. HOLBERT. 1937. Resistance of corn hybrids related to carbohydrates. Illinois Agr. Exp. Sta. Ann. Rept. 49:43-45.
61. DETURK, E. E. 1939. Chemistry, disease, and cold injury of corn related. Illinois Agr. Exp. Sta. Ann. Rept. 50:62-64.
62. DEVAY, J. E., R. P. COVEY, and D. B. LINDEN. 1957. Methods of testing for disease resistance in the corn disease nurseries at St. Paul and comparisons of 110 lines of corn for resistance to diseases important in the North Central region. Plant Dis. Reptr. 41:699-702.
63. DEVAY, J. E., R. P. COVEY, and P. N. NAIR. 1957. Corn diseases and their importance in Minnesota in 1956. Plant Dis. Reptr. 41:505-507.
64. DICKSON, J. G. 1923. Influence of soil temperature and moisture on the development of seedling-blight of wheat and corn caused by *Gibberella saubinetii*. J. Agr. Res. 23:837-870.
65. DICKSON, J. G., and J. R. HOLBERT. 1926. The influence of temperature upon the metabolism and expression of disease resistance in selfed lines of corn. J. Amer. Soc. Agron. 18:314-322.
66. DICKSON, J. G. 1956. Diseases of field crops. Second ed. McGraw-Hill Book Company, Inc., New York. 517 p.
67. DRECHSLER, C. 1928. *Pythium arrhenomanes* n. sp., a parasite causing maize root rot. Phytopathology 18:873-875.
68. DRECHSLER, C. 1934. *Pythium butleri* and *P. aphanidermatum*. Phytopathology 24:7. (Abstr.)
69. DRECHSLER, C. 1936. *Pythium graminicolum* and *Pythium arrhenomanes*. Phytopathology 26:676-684.
70. DUNCANSON, H. B. 1892. A bacterial disease of corn. *In* Pubs. Nebraska Acad. Sci., No. 2, p. 21-23.
71. DUNGAN, G. H., J. G. BIGGER, A. L. LANG, B. KOEHLER, and R. W. JUGENHEIMER. 1946. Illinois hybrid corn tests 1945. Illinois Agr. Exp. Sta. Bull. 517.
72. DUNGAN, G. H., J. G. BIGGER, A. L. LANG, B. KOEHLER, and R. W. JUGENHEIMER. 1947. Illinois hybrid corn tests 1946. Illinois Agr. Exp. Sta. Bull. 521.
73. DUNGAN, G. H., A. L. LANG, J. H. BIGGER, B. KOEHLER, and O. BOLIN. 1939. Illinois corn performance tests 1938. Illinois Agr. Exp. Sta. Bull. 450.
74. DURRELL, L. W. 1920. The purple sheath spot of corn. Phytopathology 10:54-55. (Abstr.)
75. DURRELL, L. W. 1920. A preliminary study of the purple leaf sheath spot of corn. Phytopathology 10:487-495.
76. DURRELL, L. W. 1923. Dry rot of corn. Iowa Agr. Exp. Sta. Res. Bull. 77.
77. DURRELL, L. W. 1925. A preliminary study of fungous action as the cause of down corn. Phytopathology 15:146-154.
78. DURRELL, L. W. 1925. *Basisporium* dry rot of corn. Iowa Agr. Exp. Sta. Res. Bull. 84.
79. EDDINS, A. H. 1930. Dry rot of corn caused by *Diplodia macrospora* Earle. Phytopathology 20: 439-448.
80. EDDINS, A. H. 1930. A new *Diplodia* ear rot of corn. Phytopathology 20:733-742.
81. EDDINS, A. H. 1930. Corn diseases in Florida. Florida Agr. Exp. Sta. Bull. 210.
82. EDDINS, A. H., and R. K. VOORHEES. 1933. *Physalospora zeicola* on corn and its taxonomic and host relationships. Phytopathology 23:63-72.
83. EDMUNDS, J. E. 1963. The relationship of soil fumigation with D-D for the control of *Pratylenchus penetrans* (Cobb 1917) Chitwood and Oteifa 1952, to subsequent development of root and stalk rot of corn. M.S. thesis, Cornell Univ.
84. EDWARDS, E. T. 1933. A new *Fusarium* disease of maize. Agr. Gaz. N. S. Wales 44:895-897.
85. EDWARDS, E. T. 1935. Studies on *Gibberella fujikuroi* var. *subglutinans* the hitherto undescribed ascigerous stage of *Fusarium moniliforme* var. *subglutinans* and on its pathogenicity on maize in New South Wales. N. S. Wales Dept. Agr. Sci. Bull. 49:1-68.
86. EDWARDS, E. T. 1940. The biological antagonism of *Gibberella fujikuroi* and *Gibberella fujikuroi* var. *subglutinans* to *Trichoderma viride*, with notes on the pathological effects of the latter fungus on maize. J. Australian Inst. Agr. Sci. 6:91-100.
87. EIDE, C. J. 1935. The pathogenicity and genetics of *Gibberella saubinetii* (Mont.) Sacc. Minnesota Agr. Exp. Sta. Tech. Bull. 106.
88. ELDREDGE, J. C. 1935. The effect of injury in imitation of hail damage on the development of the corn plant. Iowa Agr. Exp. Sta. Res. Bull. 185.
89. ELLIOTT, C. 1942. Relative susceptibility to *Py-*

thium root rot of twelve dent corn inbreds. J. Agr. Res. 64:711-723.

90. ELLIOTT, C. 1943. A *Pythium* stalk rot of corn. J. Agr. Res. 66:21-39.
91. ESSARY, S. H. 1917. Bacterial root-rot of corn. Bull. of Plant Disease Survey No. 3. Sept. 15, p. 52.
92. EVANS, I. B. POLE. 1912-13. Union of South Africa, Department of Agriculture Report with Appendices, Appendix 8: Union Dept. of Agr., Div. Plant Pathol. and Mycol. p. 169-183.
93. FENNE, S. B. 1946. Corn stalk rots cause severe damage in Virginia in 1945. Plant Dis. Reptr. 30:62.
94. FOLEY, D. C. 1955. The effect of fertility on stalk rot of corn in Pennsylvania. Ph.D. thesis, Pennsylvania State Univ.
95. FOLEY, D. C. 1959. The presence of cellulase in corn stalks infected with *Fusarium moniliforme*. Phytopathology 49:538. (Abstr.)
96. FOLEY, D. C. 1959. Systemic infection of corn by *Fusarium moniliforme*. Phytopathology 49:538. (Abstr.)
97. FOLEY, D. C. 1960. The response of corn to inoculation with *Diplodia zeae* and *Gibberella zeae*. Phytopathology 50:146-150.
98. FOLEY, D. C. 1962. Stalk deterioration of plants susceptible to corn stalk rot. Phytopathology 52:10. (Abstr.)
99. FOLEY, D. C. 1962. Systemic infection of corn by *Fusarium moniliforme*. Phytopathology 52: 870-872.
100. FOLEY, D. C., and C. C. WERNHAM. 1957. The effect of fertilizers on stalk rot of corn in Pennsylvania. Phytopathology 47:11-12. (Abstr.)
101. GARBER, R. J., R. B. DUSTMAN, and C. R. BURNHAM. 1936. Yield and composition of eared and earless maize plants in a selfed line segregating barren stalks. Agron. J. 28:85-91.
102. GAROFALO, O. A. 1961. Informe preliminar sobre una nueva enfermedad del maíz y enfermedades mas comunes en Costa Rica. *In* Séptima Reunión Centroamericana. p. 63-64. Proyecto Cooperativo Centroamericano para el Mejoramiento del Maíz. 108 p.
103. GRAHAM, T. W., and Q. L. HOLDEMAN. 1951. Nematode injury to tobacco, cotton and corn in relation to populations of root-knot and meadow nematodes. Phytopathology 41:14. (Abstr.)
104. GRIKENKO, G. 1962. Diplodioz vskhodov Kukuruzy. Kukuruza 7:55-56. (Rev. Appl. Mycol. 41:709.)
105. HAIGH, J. C. 1928. *Macrophomina phaseoli* (Maubl.) Ashby. The pycnidial stage of *Rhizoctonia bataticola*. Trop. Agr. (Ceylon) 70:77-79.
106. HALL, D. M. 1934. The relationship between certain morphological characters and lodging in corn. Minnesota Agr. Exp. Sta. Tech. Bull. 103.
107. HARRIS, M. R. 1936. The relationship of *Cephalosporium acremonium* to the black-bundle disease of corn. Phytopathology 26:965-980.
108. HASLAM, T. P. 1910. Meningo-encephalitis. Kansas Agr. Exp. Sta. Bull. 173.
109. HAWS, C. L. 1961. Studies of strain relationships of *Gibberella zeae* (Schw.) Petch. M.S. thesis, Cornell Univ.
110. HAYES, H. K., I. J. JOHNSON, and E. C. STAKMAN. 1933. Reaction of maize seedlings to *Gibberella saubinetii*. Phytopathology 23:905-911.
111. HEAID, F. D. 1906. New and little-known plant diseases in Nebraska. Science 23:624.
112. HEALD, F. D., E. M. WILCOX, and V. W. POOL. 1909. The life-history and parasitism of *Diplodia zeae* (Schw.) Lev. Nebraska Agr. Exp. Sta. Ann. Rept. 22:1-7.
113. HLRR, L. J. 1957. Soil mycoflora associated with continuous cropping of corn, oats, and wheat. Ohio J. Sci. 57:203-211.
114. HINGORANI, M. K., U. J. GRANT, and N. J. SINGH. 1959. *Erwinia carotovora* f. sp. *zeae*, a destructive pathogen of maize in India. Indian Phytopathol. 12:151-157.
115. HO, WEN-CHUN. 1944. Soil-inhabiting fungi attacking the roots of maize. Iowa Agr. Exp. Sta. Res. Bull. 332.
116. HOADLEY, A. D. 1942. A study of the nature of resistance of dent corn to *Diplodia zeae* (Schw.) Lev. stalk rot. Ph.D. thesis, Univ. Maryland.
117. HOFFER, G. N., and R. H. CARR. 1923. Accumulation of aluminum and iron compounds in corn plants and its probable relation to root rots. J. Agr. Res. 23:801-823.
118. HOFFER, G. N., and B. A. KRANTZ. 1951. Deficiency symptoms of corn and small grains, p. 59-105. *In* Hunger Signs in Crops, 2nd ed., Amer. Soc. Agron. and Nat. Fert. Assoc. Washington, D. C. 390 p.
119. HOFFMASTER, D. E., J. H. MCLAUGHLIN, W. W. RAY, and K. S. CHESTER. 1943. The problem of dry rot caused by *Macrophomina phaseoli* (*Sclerotium bataticola*). Phytopathology 33: 1113-1114. (Abstr.)
120. HOLBERT, J. R. 1926. Physiologic and morphologic differences in certain inbred strains of corn varying widely in disease resistance. Ph.D. thesis, Univ. Illinois.
121. HOLBERT, J. R., W. L. BURLISON, H. H. BIGGAR, B. KOEHLER, G. H. DUNGAN, and M. T. JENKINS. 1923. Early vigor of maize plants and yield of grain as influenced by the corn root, stalk and ear rot diseases. J. Agr. Res. 23:583-629.
122. HOLBERT, J. R., W. L. BURLISON, B. KOEHLER, C. M. WOODWORTH, and G. H. DUNGAN. 1924. Corn root, stalk, and ear rot diseases, and their control through seed selection and breeding. Illinois Agr. Exp. Sta. Bull. 255.
123. HOLBERT, J. R., and G. N. HOFFER. 1920. Corn root and stalk rots. Phytopathology 10:55. (Abstr.)
124. HOLBERT, J. R., and G. N. HOFFER. 1920. Control of the root, stalk, and ear rot diseases of corn. U.S. Dept. Agr. Farmers' Bull. 1176.
125. HOLBERT, J. R., P. E. HOPPE, and A. L. SMITH. 1935. Some factors affecting infection with and spread of *Diplodia zeae* in the host tissue. Phytopathology 25:1113-1114.
126. HOOKER, A. L. 1956. Association of resistance to several seedling, root, stalk, and ear diseases in corn. Phytopathology 46:379-384.
127. HOOKER, A. L. 1956. Correlation of resistance to eight *Pythium* species in seedling corn. Phytopathology 46:175-176.
128. HOOKER, A. L. 1957. Factors affecting the spread of *Diplodia zeae* in inoculated corn stalks. Phytopathology 47:196-199.
129. HOOKER, A. L., and M. P. BRITTON. 1959. High losses caused by cornstalk rots. Illinois Res. 1:17.
130. HOOKER, A. L., and M. P. BRITTON. 1962. The effects of stalk rot on corn yields in Illinois. Plant Dis. Reptr. 46:9-13.

131. HOPPE, P. E. 1936. Intraspecific and interspecific aversion in *Diplodia*. J. Agr. Res. 53:671-680.
132. HOPPE, P. E. 1938. Relative prevalence and geographic distribution of various ear-rot fungi in the 1937 corn crop. Plant Dis. Reptr. 22:234-241.
133. HOPPE, P. E. 1939. Relative prevalence and geographic distribution of various ear-rot fungi in the 1938 corn crop. Plant Dis. Reptr. 23:142-148.
134. HOPPE, P. E. 1941. Relative prevalence and geographic distribution of various ear-rot fungi in the 1940 corn crop. Plant Dis. Reptr. 25:148-152.
135. HOPPE, P. E. 1942. Relative prevalence and geographic distribution of various ear-rot fungi in the 1941 corn crop. Plant Dis. Reptr. 26:145-149.
136. HUNT, C. A. 1919. Barren stalks not hereditary. Orange Judd Farmer 66:20.
137. HUNTER, J. W., and N. E. DALBEY. 1937. A histological study of stalk-breaking in maize. Amer. J. Bot. 24:492-494.
138. IVANOFF, S. S. 1934. A plant inoculator. Phytopathology 24:74-76.
139. JAUCH, C. 1961. Reseña de patología maicera. p. 239-242. *In* Actas de la Va Reunión Latinoamericana de Fitotecnia. Tomo II. Instituto Nacional de Tecnología Agropecuaria. Repúblíca Argentina Secretaría de Estado de Agricultura y Ganadería. 599 p.
140. JEHLE, R. A., F. W. OLDENBURG, and C. E. TEMPLE. 1927. The relation of internal cob-discoloration to yield in corn. Five years' results. Maryland Agr. Exp. Sta. Bull. 290.
141. JENKINS, M. T. 1957. Evaluation of lines for resistance to *Helminthosporium*. Amer. Seed Trade Assn. Pub. No. 12:7-13.
142. JOHANN, H. 1928. *Penicillium* injury to corn seedlings. Phytopathology 18:239-242.
143. JOHANN, H., J. R. HOLBERT, and J. G. DICKSON. 1928. A *Pythium* seedling blight and root rot of dent corn. J. Agr. Res. 37:443-464.
144. JOHANN, H. 1939. Scolecospores in *Diplodia zeae*. Phytopathology 29:67-71.
145. JOHANN, H. 1943. *Phoma terrestris* in the roots of mature maize plants. Phytopathology 33:526-528.
146. JOHANN, H., and A. D. DICKSON. 1945. A soluble substance in cornstalks that retards growth of *Diplodia zeae* in culture. J. Agr. Res. 71:89-110.
147. JOHNSON, H. G. 1961. Stalk rot and lodging of corn. Minnesota Agr. Ext. Serv., Farm and Home Fact Sheet, Plant Pathology No. 3.
148. JOHNSON, A. G., A. ROBERT, and L. CASH. 1949. Bacterial leaf blight and stalk rot of corn. J. Agr. Res. 78:719-732.
149. JOHNSTON, C. O. 1929. Diseases of corn in Kansas. Kansas State Agr. Bien. Rept. 48:174-191.
150. JUGENHEIMER, R. W. 1940. Resistance to *Diplodia* infection in inbred lines and hybrids of maize. Ph.D. thesis, Iowa State Univ.
151. JUGENHEIMER, R. W., and A. A. BRYAN. 1938. Developing inbred lines of corn resistant to stalk and ear rots. Iowa Agr. Exp. Sta. Ann. Rept. 1937-1938:33-36.
152. JUGENHEIMER, R. W. 1958. Hybrid maize breeding and seed production. FAO Agric. Development Paper No. 62.
153. KELMAN, A., L. H. PERSON, and T. T. HEBERT. 1957. A bacterial stalk rot of irrigated corn in North Carolina. Plant Dis. Reptr. 41:798-802.
154. KENT, G. C. 1940. An inhibitor produced by *Diplodia zeae* (Schw.) Lev. Iowa Agr. Exp. Sta. Bull. 274.
155. KINGSLAND, G. D. 1958. Etiology and epiphytology of stalk rot of corn in Pennsylvania. Ph.D. thesis, Pennsylvania State Univ.
156. KINSEL, K. 1937. Carbohydrate utilization by the corn Diplodias. Phytopathology 27:1119-1120.
157. KNORR, L. C., and W. EBELING. 1961. World citrus problems. II. The Gaza Strip. Plant Protection Bull. 7:115-120.
158. KOCH, L. W., and H. F. MURWIN. 1945. The hybrid corn industry in Ontario: pathological and other problems. Empire J. Exp. Agr. 13: 100-111.
159. KOEHLER, B., J. G. DICKSON, and J. R. HOLBERT. 1924. Wheat scab and corn rootrot caused by *Gibberella saubinetii* in relation to crop successions. J. Agr. Res. 27:861-879.
160. KOEHLER, B., G. H. DUNGAN, and J. R. HOLBERT. 1925. Factors influencing lodging in corn. Illinois Agr. Exp. Sta. Bull. 266.
161. KOEHLER, B., and J. R. HOLBERT. 1930. Corn diseases in Illinois; their extent, nature, and control. Illinois Agr. Exp. Sta. Bull. 354.
162. KOEHLER, B., and J. R. HOLBERT. 1938. Combatting corn diseases in Illinois. Illinois Agr. Exp. Sta. Cir. 484.
163. KOEHLER, B. 1938. Seed treatment tests with crown-injured corn. Phytopathology 28:13. (Abstr.)
164. KOEHLER, B., and G. H. BOEWE. 1947. *Gibberella zeae* damage in Illinois in 1946. Plant Dis. Reptr. 31:169-170.
165. KOEHLER, B. 1950. Corn stalk and ear rot studies. Improved techniques in hybrid seed corn production. Rept. 5th Hybrid Corn Ind. Res. Conf. Amer. Seed Trade Assn. Pub. 5:33-46.
166. KOEHLER, B. 1953. Loss of leaf area increases damage from *Gibberella* stalk rot. Phytopathology 43:477-478. (Abstr.)
167. KOEHLER, B. 1955. Correlation between resistance to Stewart's leaf blight and northern leaf blight in corn. Plant Dis. Reptr. 39:164-165.
168. KOEHLER, B., and G. H. BOEWE. 1957. Causes of corn stalk rot in Illinois. Plant Dis. Reptr. 41: 501-504.
169. KOEHLER, B. 1960. Cornstalk rots in Illinois. Illinois Agr. Exp. Sta. Bull. 658.
170. KOMMEDAHL, T., and T. D. BROCK. 1954. Studies on the relationship of soil mycoflora to disease incidence. Phytopathology 44:57-61.
171. KRANTZ, B. A., and W. V. CHANDLER. 1951. Lodging, leaf composition, and yield of corn as influenced by heavy applications of nitrogen and potash. Agron. J. 43:547-552.
172. LANG, A. L., and F. C. BAUER. 1939. Some corn hybrids are more effective users of plant food. Illinois Agr. Exp. Sta. Ann. Rept. 1936-1937: 37-43.
173. LANJOUW, J. [Ed.] 1956. International code of botanical nomenclature adopted by the Eighth International Botanical Congress, Paris, July 1954. Article 59. The International Bureau for Plant Taxonomy and Nomenclature, Utrecht, Netherlands.
174. LANZA, F. 1955. Marciume batterioc del culmo di mais. Italia Agr. 92:77-78.

175. LARSH, H. W. 1943. Corn diseases in Oklahoma. Plant Dis. Reptr. 27:614.
176. LARSON, W. E., F. W. SCHALLER, W. G. LOVELY, and W. F. BUCHELE. 1956. New ways to prepare corn ground. Iowa Farm Sci. 10:215-218.
177. LEONIAN, L. H. 1932. The pathogenicity and the variability of *Fusarium moniliforme* from corn. West Virginia Agr. Exp. Sta. Bull. 248.
178. LIMBER, D. P. 1927. *Fusarium moniliforme* in relation to diseases of corn. Ohio J. Sci. 27:232-248.
179. LITTLEFIELD, L. J. 1964. Effects of hail damage on yield and stalk rot infection in corn. Plant Dis. Reptr. 48:169.
180. LITTLEFIELD, L. J., and R. D. WILCOXSON. 1962. Histological study of cornstalk rot. Phytopathology 52:18. (Abstr.)
181. LITTLEFIELD, L. J., and R. D. WILCOXSON. 1962. Studies of necrotic lesions in cornstalks. Amer. J. Bot. 49:1072-1078.
182. LIVINGSTON, J. E. 1945. Charcoal rot of corn and sorghum. Nebraska Agr. Exp. Sta. Res. Bull. 136.
183. LIVINGSTON, J. E. 1945. Important diseases of corn in Nebraska. Nebraska Agr. Ext. Circ. 1804.
184. LOESCH, P. J., JR., O. H. CALVERT, and M. S. ZUBER. 1962. Interrelations of *Diplodia* stalk rot and two morphological traits associated with lodging of corn. Crop Sci. 2:469-472.
185. LONG, O. H. 1953. Nitrogen and spacing experiments with corn. Tennessee Agr. Exp. Sta. Bull. 232.
186. LUDBROOK, W. V. 1942. Top rot of maize, sweet corn, and sorghum. Australian J. Council Sci. and Ind. Res. 15:213-216.
187. MACINNES, J., and R. FOGELMAN. 1923. Wheat scab in Minnesota. Minnesota Agr. Exp. Sta. Tech. Bull. 18.
188. MACKIE, W. W. 1932. A hitherto unreported disease of maize and beans. Phytopathology 22: 637-644.
189. MAGEE, J. A. 1948. Histological structure of the stem of *Zea mays* in relation to stiffness of stalk. Iowa State Coll. J. Sci. 22:257-268.
190. MAHER, C. 1931. Ear rots and root rots of maize in Kenya and some suggestions for their control. Kenya Colony and Protectorate Department of Agr. Bull. 5:22.
191. MALLIK, M. A. B. 1961. Influence of soil factors on seedling blight of corn. M.S. thesis, Univ. Minnesota.
192. MANNS, T. F., and J. F. ADAMS. 1921. Corn root rot diseases. Delaware Agr. Exp. Sta. Bull. 128.
193. MANNS, T. F., and C. E. PHILLIPS. 1924. Corn root rot studies. J. Agr. Res. 27:957-964.
194. MASON, E. W. 1927. On species of the genus *Nigrospora* Zimmermann recorded on monocotyledons. Brit. Mycol. Soc. Trans. 12:152-165.
195. MAYO, N. S. 1891. Enzootic cerebritis, or "staggers" of horses. Kansas Agr. Exp. Sta. Bull. 24.
196. MCCALLAN, S. E. A. 1946. Outstanding diseases of agricultural crops and uses of fungicides in the United States. Contr. Boyce Thompson Inst. 14:105-115.
197. MCINDOE, K. G. 1931. The inheritance of the reaction of maize to *Gibberella saubinetii*. Phytopathology 21:615-639.
198. MCKEEN, W. E. 1951. A corn root- and stalk-rot complex hitherto known as *Gibberella zeae* stalk rot. Phytopathology 41:26. (Abstr.)
199. MCKEEN, W. E. 1951. Seedling susceptibility of dent corn inbreds to root-rot caused by *Pythium arrhenomanes*. Sci. Agr. 31:475-479.
200. MCKEEN, W. E. 1953. Preliminary studies of root and basal stalk rot of maturing corn in Ontario. Can. J. Bot. 31:132-141.
201. MCNEW, G. L. 1932. Parasitism of *Diplodia zeae* on the crown of the corn plant. Phytopathology 22:18. (Abstr.)
202. MCNEW, G. L. 1935. Preliminary studies on the effect of filtrates from cultures of *Diplodia zeae* upon seedling blight of maize. Iowa State Coll. J. Sci. 9:481-487.
203. MCNEW, G. L. 1935. The prevention of seedling blight of *Zea mays* L. by autotoxins of *Diplodia zeae* (Schw.) Lev. and the relation of mesocotyl infection of crown invasion. Ph.D. thesis, Iowa State Univ.
204. MCNEW, G. L. 1937. Crown infection of corn by *Diplodia zeae*. Iowa Agr. Exp. Sta. Res. Bull. 216.
205. MCROSTIE, G. P., and J. D. MACLACHLAN. 1942. Hybrid corn studies I. Sci. Agr. 22:307-313.
206. MELCHERS, L. E., and C. O. JOHNSTON. 1923. Corn root, stalk, and ear rot disease investigations in Kansas: Report of Progress in 1922. Phytopathology 13:52. (Abstr.)
207. MELCHERS, L. E., and C. O. JOHNSTON. 1924. Second progress report on studies of corn seed germination and the prevalence of *Fusarium moniliforme* and *Diplodia zeae*. Phytopathology 14:45. (Abstr.)
208. MELHUS, I. E., and L. W. DURRELL. 1922. Dry rot of corn. Iowa Agr. Exp. Sta. Circ. 78.
209. MELHUS, I. E., C. S. REDDY, W. P. RALEIGH, and L. C. BURNETT. 1928. Seed treatment for corn diseases. Iowa Agr. Exp. Sta. Circ. 108.
210. MELHUS, I. E. 1934. *Diplodia* dry rot of corn. Iowa Agr. Exp. Sta. Rept. on Agr. Res. p. 75.
211. MELHUS, I. E. 1953. A preliminary study of the diseases of corn and some related hosts in Guatemala. Iowa State Coll. J. Sci. 27:519-536.
212. MESSIAEN, C. M. 1955. Les principales maladies du mais en France et leurs caracteres distinctifs. France Ingenieurs des Services Agricoles Bull. Tech. d'Information 105. 4 p.
213. MESSIAEN, C. M. 1957. Richesue en sucre des tiges de mais et verse parasitaire. Rev. pathol. vegetale et entomol. agr. France 36:209-213.
214. MESSIAEN, C. M., R. LAFON, and P. MOLET. 1959. Nécroses de racines, pourritures de tiges et verse parasitaire du mais. Ann. des Epiphyt. ser. C, 10:441-474.
215. MICHAELSON, M. E. 1951. A laboratory method for testing reaction of corn to stalk-rotting organisms. Phytopathology 41:26. (Abstr.)
216. MICHAELSON, M. E., and J. J. CHRISTENSEN. 1953. Reduction in yield of corn due to stalk rot. Phytopathology 43:479. (Abstr.)
217. MICHAELSON, M. E. 1953. Factors affecting development of stalk rot of corn caused by *Diplodia zeae* and *Gibberella zeae*. Ph.D. thesis. Univ. Minnesota.
218. MICHAELSON, M. E. 1957. Factors affecting development of stalk rot of corn caused by *Diplodia zeae* and *Gibberella zeae*. Phytopathology 47: 499-503.
219. MILLER, P. R. 1949. Stalk rots on corn. Agr. Chem. 4:47.
220. MILLER, R. E. 1962. Plant parasitic nematodes associated with corn roots in New York state. M.S. thesis, Cornell Univ.

221. MILLER, R. E., C. W. BOOTHROYD, and W. F. MAI. 1962. Plant parasitic nematodes associated with corn roots in New York. Phytopathology 52:22. (Abstr.)
222. MILLER, R. E., C. W. BOOTHROYD, and W. F. MAI. 1963. Relationship of *Pratylenchus penetrans* to roots of corn in New York. Phytopathology 53:313-315.
223. MILLIKAN, C. R., and W. V. LUDBROOK. 1943. Maize diseases in Victoria. J. Dept. Agr. Victoria 41:207-212.
224. MITCHELL, D. T. 1920. Poisoning of cattle by *Diplodia*-infected maize. S. African J. Sci. 16: 446-452.
225. MIURA, M. 1921. Diseases of important economic plants in Manchuria (trans. title). S. Manchuria Railway Co. Agr. Exp. Sta. Bull. 2.
226. MOLLIARD, M. 1902. *Basisporium gallarum* n. gen. n. sp. Bull. Soc. Mycol. France 18:167-170.
227. MOORE, V. A. 1896. Cornstalk disease, and rabies in cattle. U.S. Dept. Agr. Bur. Anim. Ind. Bull. 10.
228. MORRIS, V. H. 1931. Effect of barrenness and reduction of leaf area on the sugar content of corn stems. Ohio Agr. Exp. Sta. Bull. 470:23-24.
229. MURILLO, P. G. 1951. Resistencia del maiz al *Fusarium moniliforme* Sheld., p. 52-57. *In* Primera Asamblea Latinoamericana de Fitoparasitología, la, Mexico. D. F., 1950. Trabajos presentados. Mexico. Secretaría de Agricultura y Ganadería, Oficina de Estudios Especiales, Folleto Miscelaneo No. 4.
230. NELSON, C. E. 1958. Lodging of field corn as affected by cultivation, plant population, nitrogen fertilizer, and irrigation treatment. U.S. Dept. Agr. Production Res. Rept. No. 16.
231. NEWMAN, J. E., S. R. MILES, and P. L. CRANE. 1952. Performance of private and open-pedigree corn hybrids in Indiana. Indiana Agr. Exp. Sta. Circ. 380.
232. NIEDERHAUSER, J. S. 1949. Enfermedades del maíz en México. Folleto de Divulgación No. 9. Oficina de Estudios Especiales, S.A. G. México.
233. NIEDERHAUSER, J. S. 1956. Plática sobre enfermedades del maíz. p. 109-121. *In* Reunión Centroamericana. Proyecto Cooperativo Centroamericano para el Mejoramiento del Maíz. 178 p.
234. OTTO, H. J. 1956. The influence of nitrogen and potassium fertilization on the incidence of stalk rot of corn in New York. Ph.D. thesis, Cornell Univ.
235. OTTO, H. J., and H. L. EVERETT. 1956. Influence of nitrogen and potassium fertilization on the incidence of stalk rot of corn. Agron. J. 48:301-305.
236. PAHARIA, K. D. 1956. The effect of cropping sequence on soil microflora in relation to development of root rots of cereals. Ph.D. thesis, Univ. Minnesota.
237. PAMMEL, L. H. 1914. Serious root and stalk diseases of corn. Iowa Agr. 15:156-158.
238. PAMMEL, L. H., C. M. KING, and J. L. SEAL. 1915. Corn stalk and corn root diseases in Iowa. Iowa Agr. Exp. Sta. Circ. 21.
239. PAMMEL, L. H., C. M. KING, and J. L. SEAL. 1916. Studies on a *Fusarium* disease of corn and sorghum. Iowa Agr. Exp. Sta. Bull. 33.
240. PAPPELIS, A. J. 1957. Nature of resistance to *Diplodia* stalk rot of corn. Ph.D. thesis, Iowa State College.
241. PAPPELIS, A. J., and F. G. SMITH. 1960. Nature of resistance to *Diplodia* stalk rot of corn. Phytopathology 50:650. (Abstr.)
242. PAPPELIS, A. J. 1962. Relationship of seasonal changes in pith condition ratings and density to *Gibberella* stalk rot of corn. Phytopathology 52:24. (Abstr.)
243. PAPPELIS, A. J. 1962. Double inoculation for corn stalk rot studies. Phytopathology 52:24. (Abstr.)
244. PAPPELIS, A. J. 1963. Increased stalk rot susceptibility in corn following root and leaf injury. Phytopathology 53:624. (Abstr.)
245. PAPPELIS, A. J. 1963. Corn stalk rot symptoms induced by root injury. Phytopathology 53:624-625. (Abstr.)
246. PAPPELIS, A. J., and F. G. SMITH. 1963. Relationship of water content and living cells to spread of *Diplodia zeae* in corn stalks. Phytopathology 53:1100-1105.
247. PARKER, D. T., and W. C. BURROWS. 1959. Root and stalk rot in corn as affected by fertilizer and tillage treatment. Agron. J. 51:414-417.
248. PASTUSHENKO, L. T. 1962. Pro bakterial'nu steblovu hnyl Sorho, Kukurudzy ta Sudans'koyi travy na Ukraiyini. (On bacterial stalk rot of sorghum, maize, and sudan grass in the Ukraine.) J. Microbiol., Kiev 24:34-39. (Rev. Appl. Mycol. 41:654-655.)
249. PEARSON, N. L. 1931. Parasitism of *Gibberella saubinetii* on corn seedlings. J. Agr. Res. 43: 569-596.
250. PENDLETON, J. W., G. H. DUNGAN, J. H. BIGGER, B. KOEHLER, A. L. LANG, R. W. JUGENHEIMER, and G. E. MCKIBBEN. 1950. Illinois tests of corn hybrids in wide use in 1949. Illinois Agr. Exp. Sta. Bull. 536.
251. PENDLETON, J. W., G. H. DUNGAN, J. H. BIGGER, B. KOEHLER, A. L. LANG, R. W. JUGENHEIMER, and G. E. MCKIBBEN. 1951. Illinois tests of corn hybrids in wide use in 1950. Illinois Agr. Exp. Sta. Bull. 544.
252. PENDLETON, J. W., G. H. DUNGAN, J. H. BIGGER, B. KOEHLER, A. L. LANG, R. W. JUGENHIEMER, and G. E. MCKIBBEN. 1952. Illinois tests of corn hybrids in wide use in 1951. Illinois Agr. Exp. Sta. Bull. 552.
253. PENDLETON, J. W., B. KOEHLER, A. L. LANG, P. E. JOHNSON, and J. H. BIGGER. 1954. 1953 Illinois corn tests. Illinois Agr. Exp. Sta. Bull. 571.
254. PETERS, A. T. 1904. A fungus disease in corn. Nebraska Agr. Exp. Sta. 17th Ann. Rept. p. 13-22.
255. PETERSON, J. L. 1961. Comparative pathogenicity studies of fungi associated with corn stalk rot. Phytopathology 51:578. (Abstr.)
256. PETERSON, J. L. 1961. Studies on the prevalence and comparative pathogenicity of fungi associated with corn stalk rot. Plant Dis. Reptr. 45: 208-210.
257. PETERSON, P. D. 1929. Reactions of selfed lines of corn to seedling blight caused by *Gibberella saubinetii* (Mont.) Sacc. and *Fusarium moniliforme* Sheldon. Ph.D. thesis, Univ. Minnesota.
258. PETERSON, R. H., E. H. RINKE, and J. C. SENTZ. 1959. 1958 Minnesota hybrid corn performance trials. Minnesota Agr. Exp. Sta. Misc. Rept. 28.
259. PETERSON, R. H., E. H. RINKE, and J. C. SENTZ. 1960. 1959 Minnesota hybrid corn performance trials. Minnesota Agr. Exp. Sta. Misc. Rept. 28.
260. PETERSON, R. H., E. H. RINKE, and J. C. SENTZ

1961. 1960 Minnesota hybrid corn performance trials. Minnesota Agr. Exp. Sta. Misc. Rept. 28.
261. PETERSON, R. H., E. H. RINKE, and J. C. SENTZ. 1962. 1961 Minnesota hybrid corn performance trials. Minnesota Agr. Exp. Sta. Misc. Rept. 28.
262. PETERSON, R. H., E. H. RINKE, and J. C. SENTZ. 1963. 1962 Minnesota hybrid corn performance trials. Minnesota Agr. Exp. Sta. Misc. Rept. 28.
263. PINNELL, E. L., A. F. TROYER, and E. H. RINKE. 1957. 1956 Minnesota hybrid corn performance trials. Minnesota Agr. Exp. Sta. Misc. Rept. 28.
264. PONTIS-VIDELA, R. E. 1951. Una podredumbre del tallo del maíz (*Zea mays* L.) en Venezuela causada por *Pythium aphanidermatum*. Agronomía Tropical Maracay 1:13-28.
265. POPESCU, V. 1959 & 1960. Contributii la studiul putregaiului rosu al tulpinilor si stiuletilor de porumb produs de ciuperca *Gibberella zeae* (Schw.) Petch. Date noi cu privire la patogenitatea ciupercii *Gibberella zeae* (Schw.) Petch. (Contribution to the study of red rot of maize stems and cobs due to *G. zeae*. New data on the pathogenicity of *G. zeae*). Lucr. Stiint Inst. Agron. Cluj. 15:183-191; 16:207-211 (Rev. Appl. Mycol. 41:224).
266. PORTER, C. L. 1927. The fungous flora of the nodes of corn. Phytopathology 17:41. (Abstr.)
267. PORTER, C. L. 1927. A study of the fungous flora of the nodal tissues of the corn plant. Phytopathology 17:563-568.
268. PRASAD, H. H. 1930. A bacterial stalk rot of maize. Agr. J. India 35:72.
269. RALEIGH, W. P. 1930. Infection studies of *Diplodia zeae* (Schw.) Lev. and control of seedling blights of corn. Iowa Agr. Exp. Sta. Res. Bull. 124.
270. RAMSEY, G. B. 1922. *Basisporium gallarum* Moll., a parasite of the tomato. Bot. Gaz. 74:325-328.
271. RANDS, R. D., and E. DOPP. 1934. Variability in *Pythium arrhenomanes* in relation to root rot of sugarcane and corn. J. Agr. Res. 49:189-222.
272. REDDY, C. S. 1933. Resistance of dent corn to *Basisporium gallarum* Moll. Iowa Agr. Exp. Sta. Bull. 167.
273. REDDY, C. S., and J. R. HOLBERT. 1924. The black-bundle disease of corn. J. Agr. Res. 27:177-205.
274. REECE, O. E. 1949. Inheritance of reaction to root and stalk rot in maize. Ph.D. thesis, Univ. Minnesota.
275. REILLY, J. J. 1952. Correlation of seedling blight to stalk rot and nature of seedling resistance to *Diplodia zeae*. Phytopathology 42:473. (Abstr.)
276. RICHARDSON, J. K. 1942. Studies on root rot of corn in Ontario. Can. J. Res. 20:241-256.
277. ROANE, C. W. 1950. Observations on corn diseases in Virginia from 1947 to 1950. Plant Dis. Reptr. 34:394-396.
278. ROBERTS, B. J. 1952. The growth of *Diplodia zeae* (Schw.) Lev. in corn stalk tissue. M.S. thesis, Oklahoma State Univ.
279. ROBLES, L. H. 1948. Pathogenicity of isolates of corn root-rot organisms in Mexico. Phytopathology 38:22. (Abstr.)
280. ROBLES, L. H. 1948. Sumario y conclusiones de los experimentos sobre pudriciones de la raiz, de la caña y la mazorca del maíz en México. Oficina de Estudios Especiales de la S.A.G. México. Typed report.
281. ROBLES, L. H. 1949. The pathogenicity of species of *Helminthosporium* on corn. Phytopathology 39:1020-1028.
282. RODIGIN, M. N., and G. V. POLETAEVA. 1961. Steblevaya bakterial naya gnil'. (Bacterial stalk rot) Kukuruza 7:54. (Rev. Appl. Mycol. 41:225.)
283. ROSEN, H. R. 1919. A bacterial root-rot of field corn. Arkansas Agr. Exp. Sta. Bull. 162.
284. ROSEN, H. R. 1921. A bacterial root and stalk rot of field corn. Phytopathology 11:32-33. (Abstr.)
285. ROSEN, H. R. 1921. Further observations on a bacterial root and stalk rot of field corn. Phytopathology 11:74-79.
286. ROSEN, H. R. 1922. The bacterial pathogen of corn stalk rot. Phytopathology 12:497-499.
287. ROSEN, H. R. 1926. Bacterial stalk rot of corn. Arkansas Agr. Exp. Sta. Bull. 209. (*Also in:* Phytopathology 16:241-267.)
288. RUSSELL, C. E. 1919. Barren stalks in corn. Orange Judd Farmer 66:175.
289. RUSSELL, W. A. 1961. A comparison of five types of testers in evaluating the relationship of stalk rot resistance in corn inbred lines and stalk strength of the lines in hybrid combinations. Crop Sci. 1:393-397.
290. SABET, K. A. 1954. A new bacterial disease of maize in Egypt. Empire J. Exp. Agr. 22:65-67.
291. SABET, K. A. 1956. The effects of streptomycin and terramycin, singly and in combination, on the leaf blight disease of maize caused by *Bacterium carotovorum* f. sp. *zeae* Sabet. Ann. Appl. Biol. 44:152-160.
292. SABET, K. A. 1957. A note on the identity of a bacterium causing stalk rot and leaf blight of maize in Southern Rhodesia. Empire J. Exp. Agr. 25:165-166.
293. SABET, K. A. 1957. On the effect of certain environmental conditions on infection with the bacterial root- and stalk-rot disease of maize. Indian J. Agr. Sci. 27:467-474.
294. SABET, K. A., A. S. SAMRA, M. K. HINGORANI, M. G. ABDEL RAHIM, H. A. EL-SHAFEY, I. M. MANSOUR, IKBAL KHALIL, F. A. M. FADL, and NADIA DAWOOD. 1960. Annual report of the project entitled "Stalk and root rots of maize in the United Arab Republic" for the period July 1961 to June 1962.
295. SABET, K. A. 1961. Investigations on root and stalk rots of maize in Egypt (Etiological Studies). Egypt Min. Agr. Tech. Bull. Agr. Ext. Dept.
296. SABET, K. A., A. S. SAMRA, M. K. HINGORANI, and I. M. MANSOUR. 1961. Stalk and root rot of maize in United Arab Republic. FAO Plant Prot. Bull. 9:121-125.
297. SAMRA, A. S. 1953. A bacterial rot of maize in Egypt. Egypt Min. Agr. Res. Comm.
298. SAMRA, A. S., K. A. SABET, and M. K. HINGORANI. 1963. Late wilt disease of maize caused by *Cephalosporium maydis*. Phytopathology 53: 402-406.
299. SAMRA, A. S., K. A. SABET, and M. K. HINGORANI. 1962. A new wilt disease of maize in Egypt. Plant Dis. Reptr. 46:481-483.
300. SANSOM, T. K. 1940. Breeding *Diplodia* resistant varieties of maize. Rhodesia Agr. J. 37:442-444.
301. SAVOIA, H. J., E. F. GODOY, and O. BRUNI. 1950. Memoria de la quinta reunión de Maíz 27 y 28 de Julio de 1950 en la Estación Experimental Pergamino p. 21-27. (Rev. Appl. Mycol. 30: 607.)
302. SAVULESCU, T., and T. RAYSS. 1930. Une nouvelle maladie du mais en Roumaine provoquee par *Nigrospora oryzae* (B. et Br.) Petch. Arch. Roumanes Path. Exper. Microbiol. 3:41-53.

303. SAVULESCU, T., and T. RAYSS. 1931. Contribution à la connaissance de la biologie de *Nigrospora oryzae* (B. et Br.) Petch, parasite de mais. Rec. Trav. Cryptogam. dédiés à Louis Mangin. Muséum Nat. d'Hist. Nat., Paris. p. 233-240.

304. SAVULESCU, T., and T. RAYSS. 1932. Influence des conditions exterieures sur le developpement de *Nigrospora oryzae* (B. et Br.) Petch, parasite du mais en Roumaine. Acad. Sci. (Paris) Compt. Rend. 194:1262-1265.

305. SAYRE, J. D., V. H. MORRIS, and F. D. RICHEY. 1931. The effect of preventing fruiting and of reducing the leaf area on the accumulation of sugars in the corn stem. Agron. J. 23:751-753.

306. SCHAFFER, F. W., and D. D. EVANS. 1954. Some effects of mulch tillage. Agr. Eng. 35:731-734, 736.

307. SCHIEBER, E. 1961. Principales enfermedades del maíz en Guatemala. Séptima Reunión Centroamericana p. 73-76. *Proyecto Cooperativo* Centroamericano para el Mejoramiento del Maíz. 108 p.

308. SCHNEIDER, C. L., and J. J. CHRISTENSEN. 1949. Relation of the Europeaen corn borer to stalk rots of corn. Phytopathology 39:21. (Abstr.)

309. SCHROEDER, H. W., M. G. BOOSALIS, and M. B. MOORE. 1953. An improved method of growing inoculum of plant disease fungi. Phytopathology 43:401-402.

310. SCHROEDER, H. W. 1954. Physiologic variation in cultural races of *Diplodia zeae*. M.S. thesis, Univ. Minnesota.

311. SEGALL, R. H., and J. VELEZ FORTUNO. *Gibberella fujikuroi* the cause of bud rot of corn in Puerto Rico. Plant Dis. Reptr. 39:283.

312. SELBY, A. D. 1910. A brief handbook of the diseases of cultivated plants in Ohio. Ohio Agr. Exp. Sta. Bull. 214.

313. SELBY, A. D. 1918. Root rot of corn widespread in 1918 crop throughout Ohio. Ohio Agr. Exp. Sta. Monthly Bull. 3:313-314.

314. SEMENIUK, G., C. S. REDDY, I. E. MELHUS, W. E. LOOMIS, E. W. LINDSTROM, and A. A. BRYAN. 1939. Disease resistance in corn; nature and methods of measuring. Method of inoculation with *Diplodia zeae* in the field. Iowa Agr. Exp. Sta. Ann. Rept. 1938-1939:59-60.

315. SEMENIUK, G. 1941. Development of *Diplodia zeae* and *Gibberella saubinetii* in maize pith following stalk inoculations. Phytopathology 31:20. (Abstr.)

316. SEMENIUK, G. 1942. Comparative reactions of single crosses of dent maize to *Diplodia zeae*. Phytopathology 32:16. (Abstr.)

317. SEMENIUK, G. 1942. Charcoal-rot of maize, new to Iowa. Iowa Acad. Sci. Proc. 49:256. (Abstr.)

318. SEMENIUK, G., I. E. MELHUS, C. S. REDDY, W. E. LOOMIS, G. R. SPRAGUE, and E. W. LINDSTROM. 1942. Disease resistance in corn; nature and methods of measuring. Iowa Agr. Exp. Sta. Rept. on Agr. Res. Part II: 51-53, 1940; Part II: 56-57, 1941.

319. SEMENIUK, G. 1944. Seedling infection of dent maize by *Sclerotium bataticola* Taub. Phytopathology 34:838-843.

320. SEMENIUK, G., J. R. WALLIN, and I. E. MELHUS. 1947. Root-necrosis resistance in maize. Phytopathology 37:20. (Abstr.)

321. SEMENIUK, G. 1948. Root rot in your corn. Iowa Farm Sci. 3:6-7.

322. SEMENIUK, G. 1948. Study of *Diplodia* dry rot of corn. Iowa Agr. Exp. Sta. Rept. on Agr. Res. 370 p.

323. SHELDON, J. L. 1904. A corn mold (*Fusarium moniliforme n. sp.*) Nebr. Agr. Exp. Sta. 17th Ann. Rept. p. 23-32.

324. SIDOROV, F. F., V. I. POTLAICHUK, and G. G. AIBA. 1962. Otsenka kollektsii samoopylennykh linii po ustoichivosti k diplodiozu. (Assessment of the collection of selfed lines for resistance to diplodiosis) Kukuruza 7:57. (Rev. Appl. Mycol. 41:654.)

325. SIPPEL, W. L. 1953. A disease of swine and cattle caused by eating moldy corn. "Proceedings Book." Amer. Vet. Med. Assoc. Nineteenth Ann. Meeting p. 174-181.

326. SKILES, R. L., C. CARDONA, and O. BARROS N. 1958. Reaccion de lineas de maiz a pudriciones del tallo con *Fusarium* y *Diplodia*, p. 203-204. *In* III Reunion Interamericana de Fitogenetistas, Fitopatologos, Entomologos y Edafologos, Bogota, Colombia, 1955. Colombia Ministerio de Agricultura, Oficina de Investigaciones Especiales, Bogota. 459 p.

327. SMITH, A. L., and J. R. HOLBERT. 1931. Cornstalk rot and ear rot. Phytopathology 21:129. (Abstr.)

328. SMITH, A. L., and J. R. HOLBERT. 1932. *Diplodia* stalk and ear-rot studies of dent corn. Phytopathology 22:24. (Abstr.)

329. SMITH, A. L., P. E. HOPPE, and J. R. HOLBERT. 1938. Development of a differential inoculation technique for *Diplodia* stalk rot of corn. Phytopathology 28:497-504.

330. SMITH, E. F., and F. HEDGES. 1909. *Diplodia* disease of maize. (Suspected cause of Pellagra.) Science n. s., 30:60-61.

331. SMITH, G. M., and G. N. HOFFER. 1921. Three methods of controlling the root, stalk, and ear rots of corn. Phytopathology 11:34. (Abstr.)

332. SMITH, G. M., and J. G. TROST. 1934. *Diplodia* ear rot in inbred and hybrid strains of sweet corn. Phytopathology 24:151-157.

333. SPENCER, E. L., and G. L. MCNEW. 1938. The influence of mineral nutrition on the reaction of sweet-corn seedlings to *Phytomonas stewarti*. Phytopathology 28:213-223.

334. SPRAGUE, G. F. 1954. Breeding for resistance to stalk rot. Amer. Seed Trade Assn. Pub. 9:38-43.

335. STAFFELDT, E. E. 1954. Effects of four crop rotations on soil fungi and corn root necrosis. Iowa State Coll. J. Sci. 28:403-404. (Abstr.)

336. STAKMAN, E. C. 1960. La obligación de la fitopatología en el problema de la alimentación humana. p. 479-501. *In* Memoria del Segundo Congreso Nacional de Entomología y Fitopatologia. Escuela Nacional de Agricultura, Chapingo, Mexico. 502 p.

337. STANDEN, J. H. 1939. Prevalence of *Basisporium gallarum* in arrested axillary shoots and secondary ears of maize. Phytopathology 29:656-657.

338. STANDEN, J. H. 1941. The growth of *Basisporium gallarum* in maize cobs. Phytopathology 31:21-22. (Abstr.)

339. STANDEN, J. H. 1943. Variability of *Nigrospora* on maize. Iowa State Coll. J. Sci. 17:263-275.

340. STANDEN, J. H. 1950. Occurrence of *Nigrospora oryzae* (B. and Br.) Petch in Venezuela, with a note on discharge of spores. Plant Dis. Reptr. 34:157.

341. STANLEY, A. R., and C. R. ORTON. 1932. Bacterial

stalk rot of sweet corn. Phytopathology 22:26. (Abstr.)

342. STEVENS, N. E. 1936. A note on the temperature relations of certain fungi. Mycologia 28:510-513.

343. STEVENS, N. E., and W. E. WILSON. 1941. A biotin-like substance produced by *Diplodia zeae*. Science 93:458-459.

344. STEVENS, N. E. 1943. Distribution of *Diplodia zeae* and *D. macrospora* in the United States. Illinois State Acad. Sci. Trans. 36:107-108.

345. STIEMENS, B. 1939. Survival of fungi in the digestive tract of cattle. S. African J. Sci. 36:220-224.

346. STOUT, G. L. 1930. New fungi found on the Indian corn plant in Illinois. Mycologia 22:271-287.

347. STRINGFIELD, G. H., and L. E. THATCHLR. 1947. Stands and methods of planting for corn hybrids. Agron. J. 39:995-1010.

348. SUDIA, T. W., F. A. WOOD, and R. D. WILCOXSON. 1961. Some effects of alpha irradiation on *Gibberella fujikuroi*. Phytopathology 51:336-337.

349. SUMMERS, T. E. 1952. Destruction of maize roots by *Pythium graminicola* Subr. and *Diabrotica undecimpunctata howardi* Barber. Iowa State Coll. J. Sci. 26:294-295.

350. TAYLOR, G. S. 1952. Stalk-rot development in corn following the European corn borer. Phytopathology 42:20-21. (Abstr.)

351. TAYLOR, G. S. 1952. Nutritive value of cornstalk juice inversely related to stalk rot resistance. Phytopathology 42:467. (Abstr.)

352. TAYLOR, G. S. 1953. Stalk rot development in corn following the European corn borer. Iowa State Coll. J. Sci. 27:265-266. (Abstr.)

353. TEHON, L. R. 1924. A preliminary report on the occurrence and distribution of the common bacterial and fungous diseases of crop plants in Illinois. Illinois Dept. Registr. and Ed., Div. Nat. Hist. Survey 15:173-325.

354. THAYER, P., and L. E. WILLIAMS. 1960. Effect of nitrogen, phosphorus and potassium concentrations on the development of *Gibberella* stalk- and root-rot of corn. Phytopathology 50:212-214.

355. THIRUMALACHAR, M. J. 1953. Pycnidial stage of charcoal rot inciting fungus with a discussion on its nomenclature. Phytopathology 43:608-610.

356. THOMPSON, D. L. 1963. Stalk strength of corn as measured by crushing strength and rind thickness. Crop Sci. 3:323-329.

357. THOMPSON, D. L. 1964. Comparative strength of corn stalk internodes. Crop Sci. 4:384-386.

358. TIJERINA, M. A. 1963. Estudio preliminar sobre la pudrición radicular del maíz en México. Tésis de Maestría en Ciencias Agrícolas, Colegio de Postgraduados, Chapingo, México.

359. TOGASHI, K. 1949. Biological characters of plant pathogens temperature relations. Meibundo Co., Tokyo.

360. TROYER, A. F. 1956. The effect of *Diplodia* stalk rot on some agronomic characteristics of several corn inbreds and hybrids. M.S. thesis, Univ. Illinois.

361. TROYER, A. F., E. H. RINKE, and J. C. SENTZ. 1958. 1957 Minnesota hybrid corn performance trials. Minnesota Agr. Exp. Sta. Misc. Rept. 28.

362. ULLSTRUP, A. J. 1935. Studies on the variability of pathogenicity and cultural characters of *Gibberella saubinetii*. J. Agr. Res. 51:145-162.

363. ULLSTRUP, A. J. 1936. The occurrence of *Gibberella fujikuroi* var. *subglutinuns* in the United States. Phytopathology 26:685-693.

364. ULLSTRUP, A. J. 1943. Diseases of dent corn in the United States. U.S. Dept. of Agr. Circ. 674.

365. ULLSTRUP, A. J. 1949. Stalk rot of corn caused by *Pythium butleri* severe in Indiana. Plant Dis. Reptr. 33:331.

366. ULLSTRUP, A. J. 1955. Diseases of corn. *In* G. F. Sprague [ed.], Corn Improvement. Academic Press, New York. p. 465-536.

367. UNITED STATES DEPARTMENT OF AGRICULTURE. 1963. Agricultural Statistics 1963. U.S. Dept. Agr. 635 p.

368. VALLEAU, W. D. 1920. Seed corn infection with *Fusarium moniliforme* and its relation to the root and stalk rots. Kentucky Agr. Exp. Sta. Bull. 226.

369. VALLEAU, W. D., P. E. KARRAKER, and E. M. JOHNSON. 1926. Corn root rot, a soil borne disease. J. Agr. Res. 33:453-476.

370. VALLEAU, W. D., and S. DIACHUN. 1949. *Pythium* stalk rot of corn in Kentucky. Plant Dis. Reptr. 33:341.

371. VOLCANI, Z. 1958. A strain of *Erwinia carotovora* isolated from rotten maize plants. Ktavim (Rec. Agr. Res. Sta. Rehovot.) 8:217-219. (Rev. Appl. Mycol. 38:514.)

372. VOORHEES, R. K. 1933. *Gibberella moniliformis* on corn. Phytopathology 23:368-378.

373. VOORHEES, R. K. 1934. Histological studies of a seedling disease of corn caused by *Gibberella moniliformis*. J. Agr. Res. 49:1009-1015.

374. VOORHEES, R. K. 1934. Sclerotial rot of corn caused by *Rhizoctonia zeae* n. sp. Phytopathology 24:1290-1303.

375. WALDEE, E. L. 1942. The classification of the cornstalk-rot pathogen. Phytopathology 32:18. (Abstr.)

376. WALDEE, E. L. 1945. Comparative studies of some peritrichous phytopathogenic bacteria. Iowa State Coll. J. Sci. 19:435-484.

377. WEINTRAUB, M., and H. W. J. RAGETLI. 1961. Cell wall composition of leaves with a localized virus infection. Phytopathology 51:215-219.

378. WERNHAM, C. C. 1946. Techniques for inoculating corn with disease producing organisms. Pennsylvania Agr. Exp. Sta. Journ. Series Paper 1315.

379. WERNHAM, C. C. 1949. Techniques for inoculating corn with disease producing organisms. Pennsylvania Agr. Exp. Sta. Prog. Rep. 5:1-16.

380. WERNHAM, C. C. 1959. Cornstalk rot trials in Pennsylvania, 1958. Plant Dis. Reptr. 43:863-870.

381. WHITNEY, N. J., and C. G. MORTIMORE. 1957. Root and stalk rot of field corn in southwestern Ontario. I. Sequence of infection and incidence of the disease in relation to maturation of inbred lines. Can. J. Plant Sci. 37:342-346.

382. WHITNEY, N. J., and C. G. MORTIMORE. 1959. An antifungal substance in the corn plant and its effect on the growth of two stalk-rotting fungi. Nature 183:341.

383. WHITNEY, N. J., and C. G. MORTIMORE. 1961. Root and stalk rot of field corn in southwestern Ontario. II. Development of the disease and isolation of organisms. Can. J. Plant Sci. 41: 854-861.

384. WILCOXSON, R. D., and T. W. SUDIA. 1960. The influence of gibberellic acid on seedling blight of corn. Plant Dis. Reptr. 44:312-313.

385. WILCOXSON, R. D. 1962. Stalk rot in relation to yield of corn. Phytopathology 52:416-418.
386. WILCOXSON, R. D., L. J. LITTLEFIELD, and G. A. BEAN. 1963. Size of necrotic lesions in stalks of *Zea mays* in relation to yield. Plant Dis. Reptr. 47:342-344.
387. WILCOXSON, R. D., and R. P. COVEY. 1963. Crop sequence and stalk rot of corn. Plant Dis. Reptr. 47:960-961.
388. WILCOXSON, R. D., and R. P. COVEY. 1963. Corn plant populations and size of necrotic lesions in stalks. Plant Dis. Reptr. 47:962-963.
389. WILLIAMS, L. E., and S. K. MENON. 1957. A cork borer technique of inoculating corn plants with stalk-rot fungi. Plant Dis. Reptr. 41:111-114.
390. WILLIAMS, L. E., and D. D. KAUFMAN. 1962. Influence of continuous cropping on soil fungi antagonistic to *Fusarium roseum*. Phytopathology 52:778-781.
391. WILLIAMS, L. E., and A. F. SCHMITTHENNER. 1962. Effect of crop rotation on soil fungus populations. Phytopathology 52:241-247.
392. WILLIAMS, L. E., and A. F. SCHMITTHENNER. 1962. Effect of crop rotation on yields of corn, oats, and wheat and on corn stalk rot. Phytopathology 52:757. (Abstr.)
393. WILLIAMS, L. E., and A. F. SCHMITTHENNER. 1963. Effect of crop rotation on yields, stalk rot, and root rot of corn. Phytopathology 53:1412-1414.
394. WILLIAMS, L. E., and A. F. SCHMITTHENNER. 1963. Rotation affects corn stalk and root rot. Ohio Agr. Exp. Sta. Farm and Home Res. 48:67-68.
395. WILLIAMS, L. E., and G. M. WILLIS. 1963. Disease of corn caused by *Colletotrichum graminicolum*. Phytopathology 53:364-365.
396. WILSON, W. E. 1942. Physiological studies on two species of *Diplodia* parasitic on corn. Phytopathology 32:130-140.
397. WISER, W. J., H. H. KRAMER, and A. J. ULLSTRUP. 1960. Evaluating inbred lines of corn for resistance to *Diplodia* ear rot. Agron. J. 52:624-626.
398. WOOD, L. S. 1951. Development of root and stalk rot in certain dent corn single-crosses. M.S. thesis, Ohio State Univ.
399. WORF, G. L., and D. C. FOLEY. 1963. Plant disease control. Corn stalk roots in Iowa. Iowa Agr. Ext. Pam. 301.
400. YERKES, W. D., JR., J. S. NIEDERHAUSER, N. E. BORLAUG, E. MARTINEZ, and J. GALINDO. 1959. Some plant diseases observed in Mexico in 1958. Plant Dis. Reptr. 43:500-503.
401. YOUNG, H. C., JR. 1943. The toothpick method of inoculating corn for ear and stalk rots. Phytopathology 33:16. (Abstr.)
402. YOUNG, H. C., JR. 1943. The pathogenicity of certain fungi, singly and in combination, on the various inbred lines and crosses of corn. M.S. thesis, Univ. Minnesota.
403. YOUNG, H. C., JR. 1949. Resistance in corn to several pathogens causing seedling blights and stalk rots. Ph.D. thesis, Univ. Minnesota.
404. YOUNG, H. C., JR., R. D. WILCOXSON, M. D. WHITEHEAD, J. E. DEVAY, C. O. GROGAN, and M. S. ZUBER. 1959. An ecological study of the pathogenicity of *Diplodia maydis* isolates inciting stalk rot of corn. Plant Dis. Reptr. 43:1124-1129.
405. YOUNG, P. A. 1944. Epidemic of charcoal rot of corn and other crops in East Texas. Plant Dis. Reptr. 28:898-899.
406. YOUNG, P. A. 1949. Charcoal rot of plants in east Texas. Texas Agr. Exp. Sta. Bull. 712.
407. YOUNTS, S. E., and R. B. MUSGRAVE. 1958. Chemical composition, nutrient absorption, and stalk rot incidence of corn as affected by chloride in potassium fertilizer. Agron. J. 50:426-429.
408. ZUBER, M. S., and C. O. GROGAN. 1961. A new technique for measuring stalk strength in corn. Crop Sci. 1:378-380.
409. ZUBER, M. S., C. O. GROGAN, M. E. MICHAELSON, C. W. GEHRKE, and J. F. MONGE. 1957. Studies of the interrelation of field stalk lodging, two stalk rotting fungi, and chemical composition of corn. Agron. J. 49:328-331.