1923

Dry rot of corn

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UMI
DRY ROT OF CORN

by

L.W. Durrell

A Theses Submitted to the Graduate Faculty
for the Degree of
DOCTOR OF PHILOSOPHY

Major subject (Plant Pathology)
[No. 15]

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Signature was redacted for privacy.

Head of Major Department
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Graduate Dean

Iowa State College
1923.
Dry Rot of Corn
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Introduction

Our knowledge of dry rot of corn caused by Diplodia zeae and other fungi is only general and not specific. A critical study of its pathogenic effect on the corn crop has been seriously neglected and as a result some of the symptoms of the disease are imperfectly understood, confused or attributed to other causes such as the so called "root rot". This is certainly an unfortunate condition considering that dry rot has caused an annual loss of 17,000,000 bushels in Iowa during the past three years.

The work of Burrill and Barrett (1) and Heald and Wilcox (2) first established Diplodia zeae as the causal agent of one type of dry rot, and they made valuable contributions to the biology of the causal agent, its manner of growth and overwintering; while the later investigations of van der Bijl (9) threw light on some of the physical reactions of the organism. However, data on some of the most important physical relations of Diplodia zeae as temperature, moisture, growth response, and nutritional requirements have not been considered. Further the important association of the discoloration of the leaf sheaths after flowering with the subsequent dry rot infection and development has been completely overlooked. Only infection through the silks, or as suggested by Smith (7) through the roots has been given consideration and there is a prevailing idea that moldy corn or discolored stalks are the result of organisms working up from the roots or the soil or from diseased seed.

In the same way little or no consideration has been given to the elimination of diseased seed as a means of control and a marked confusion exists as to the identity of the organisms causing and associated with dry rot in the seed and seedling injury.

The unusual prevalence and destructiveness of dry rot in Iowa, coupled with our lack of specific knowledge governing its parasitism and its significant effect on seed corn and field stand as described above, induced the writer to make a detailed study of the disease together with its associated molds.
Part I.

The Biology of Diplodia zaeae

Introduction.

Dry rot of corn caused by Diplodia zaeae has been known since 1834, and study has been given in the past to losses caused by its action on the ears, and to its supposed agency in stock poisoning. Little is known of the distribution of the diseases in Iowa the losses resulting from its attack or of the biology of the fungus. In the following discussion consideration is given to the importance of dry rot as a corn disease. The conditions favorable to its attack and the method of infection of the causal organism, Diplodia zaeae together with suggestions for control.
Pistrelbutjon and Loss

Dry rot caused by Diplodia zeae occurs annually in Iowa but only becomes of serious importance when favorable weather conditions prevail. An epidemic of the disease occurred in 1921 throughout the central and northeastern parts of the state where there was heavy rain during the late summer and early fall. Little dry rot was found in the drier western part of the state along the Missouri River. This epidemic was the first recorded for the state since 1909.

In 1922 dry rot was prevalent in parts of the state, but the distribution differed from that in 1921. The greatest amount occurred southwest of the central portion of the state, the north and eastern sections being quite free of dry rot. Where storms injured the crop during the summer, dry rot was prevalent, particularly was this the case where hail fell.

Although certain areas in the state in 1921 were quite free from dry rot other sections showed 20 percent of the ears partially or wholly destroyed. The badly infected ears were left in the field. It was estimated for the whole state that the loss was four percent or 17½ million bushels. This loss does not take into consideration the decreased yield due to dry rot injury on the stalks and seed corn. The former is very difficult to determine. Its importance may be judged somewhat by its prevalence on the stocks in the field. Counts were made in many different fields in five counties as follows:

<table>
<thead>
<tr>
<th>County</th>
<th>Stalks Infected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Story</td>
<td>47%</td>
</tr>
<tr>
<td>Hamilton</td>
<td>25%</td>
</tr>
<tr>
<td>Webster</td>
<td>31%</td>
</tr>
<tr>
<td>Marshall</td>
<td>11%</td>
</tr>
<tr>
<td>Warren</td>
<td>4%</td>
</tr>
</tbody>
</table>

- Distribution and Loss -

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- Story County 47% of stalks infected.
  Hamilton 25%
  Webster 31%
  Marshall 11%
  Warren 4%
That this amount of Diplodia had a tendency to reduce the yield can hardly be questioned. In fact, subsequent data indicates that under certain conditions Diplodia on the stalks may reduce the yield 10 percent. In addition, the damage to seed corn is readily overlooked by the corn grower. Some idea of the amount of seed corn killed by Diplodia may be had from the following germination data: Of 650 ears collected in the north and east central part of the state, 11 percent were unfit for seed purposes. In another collection consisting of 5,000 ears gathered from representative sections of the state, same the/average, 11 percent, was found unfit for planting. Out of 130 ears collected in the south central part, 7 percent were infected with Diplodia. As this corn was quite representative of the state, it gives a fair index of the distribution and prevalence of D. zeae as affecting seed ears.

The losses in the state from dry rot infection, based on 1921 statistics, may be summed up as follows: An average loss of 4 percent of the crop at harvest, or 17,767,000 bu. of moldy corn left in the field. In addition, eleven percent of the seed ears unfit to plant, approximately 141,499 bu. This latter is a double loss, both as feed and seed. This diseased corn does not sprout and the resulting stand is thereby decreased. In many fields, only a 75 percent stand was obtained which resulted in an abnormal reduction in yield.

Further the dry rot infection on the stalks injures the plant sufficiently to reduce the yield. In bad cases this amounted to about 10 percent.
Infection Studies

Though previous descriptions of dry rot have been made by Burrill and Barrett (1) and by Heald and Wilcox (2) attention was given only to mycelial growth and fruiting on the ears and stalks. The earlier symptoms of the disease on the sheaths and nodes were not mentioned. In other papers by Burrill (5-6) some of these symptoms were associated with a bacterial disease of corn. He writes of these symptoms as follows: "The first indication of the disease in a field..... is the dwarfed condition of the young plants...............In many cases it is upon the lowest ground.... Upon closer observation it is found that young diseased plants besides being smaller..... are uniformly yellowish in color.....The bottom portion of the stalk is likewise affected.......If split longitudinally the inner tissue of this lower part is seen to have a uniform dark color........After midsummer, especially, the disease becomes apparent through discolorations of the sheaths. These leaf sheaths become variously spotted as observed from the exterior. Occasionally there is a reddish color."

Symptoms of dry rot caused by Diplodia zeae.

On leaf sheaths

On the leaf sheaths the fungus produces reddish or purplish spots of varying size and shape, appearing after flowering of the corn plant. These lesions may extend down into the node of the stalk or up the leaf killing and discoloring the midrib. (Fig. (1)).

On the ears

Diplodia zeae readily attacks the ears. The badly diseased ears
Fig. (1) External symptoms of Diplodia zeae on the sheath
may be completely covered with the mycelium giving a white moldy appearance when the husk is removed. This may be accompanied by discoloration, the ear being grey or dirty looking, even brown or blackish. In some cases the fungus may penetrate the husk so as to be readily observed from the outside, often fruiting profusely on the husk; in other instances the fungus may attack only the tip or the butt end of the ear. The latter condition is most prevalent. Often the infection is scarcely visible as a fine weft of mycelium between the kernels at the base of the ear or even this may be lacking and only in shelling the kernels can a fine white film be seen at the base. When the kernels show such symptoms the cob and chaff also have a dusty appearance and the kernels are usually loose.

On breaking an ear badly affected with *D. zeae* the fungus if often found fruiting profusely on the cob. The fruiting bodies appear as small black specks.

On the kernels the fungus may show dusty or dirty grey, Fig. (2), in some instances fruiting on the crowns of the kernels. Sometimes the fungus is not visible and is only detected when the corn germinates through shrunken kernel tips appear to be quite constantly associated with dry rot infection. Fig. (3)

**On germination**

The fungus develops in a few days from the infected kernels as a slightly cream colored cottony mass.

**On stalks**

*Diplodia zeae* attacks other parts of the corn plant besides the
Fig. (2) Flakes of mycelium of D. zeae as found on kernels from diseased ears. This is a quite characteristic appearance of Diplodia infected kernels.
Fig. (3) Kernels badly affected with dry rot as they appear after several days on the germinator
Fig. (4') Corn shank infected with Diplodia. The fungus is fruiting profusely at base of shank.
Fig. (5) External and internal symptoms of Diplodia zeae on the lower nodes of the stalk
Fig. (6) Infected corn node showing pycnidia of Dipodia.
Fig. (7) Spore coils of *D. zeae* being discharged from old corn stubble
ear and sheaths. The shank of the ear is frequently affected Fig. (4) and white wefts of mycelium may be seen on it. More common however, is the appearance of large numbers of fruiting bodies at the shank and nodes. In some cases the breaking of the shank is due to the presence of Diplodia although not always. On the stalk the symptoms are much like those on the shank. Within the sheath, at its base, and around the node may often be found a white growth of the mycelium. This may extend over the internode as well. Infection at the nodes, particularly the lower ones, is often manifested as a water soaked discoloration, Fig. (5).

In general however, Diplodia is most evident on the stalk as fruiting bodies which first emerge as minute whitish, evenly scattered dots, later becoming black. Fig. (6-7) Infection on the stalk may occur at any node but especially on the lower ones and is sometimes accompanied by breaking at the weakened nodes.

Growth Reaction of Diplodia Zeae

Moisture relations

Only a brief survey of the action of Diplodia zeae is necessary to emphasize the importance of its relation to moisture which, in fact, is the determining factor in its growth. The moisture content of the substratum and not the atmospheric humidity limits the development of Diplodia. The fungus grows down from the nodes more rapidly than up, possibly due to the greater moisture supply. Corn, high in moisture, stored in warm weather is quickly overrun with Diplodia zeae, though only slightly infected when harvested. It has been repeatedly observed that seed corn may be infected without showing evidence
Fig 8 Rainfall in Iowa August 1921.
Fig 9 Rainfall in Iowa August 1922.
of Diplodia. When such slight infection is accompanied by high moisture the results are serious.

Though Diplodia is scarcely noticeable or even invisible, such corn when hung on the rack, if not quickly dried may become badly molded. Care should be given to the rapid drying of seed corn particularly if picked early, so as to prevent any growth of Diplodia that may be on the ears. To supplement observations on the spread of dry rot on seed ears, 25 ears were picked before frost and hung in wire racks. On the base of each cob was placed a fragment of Diplodia mycelium. In a week these ears showed a growth of Diplodia an inch or more up from the butt. Twelve other ears were treated the same way, but wrapped up in oil cloth, these became entirely overgrown with Diplodia mycelium.

In the field the relation of moisture to D. zea is very pronounced. A correlation may be noted between the amount of dry rot and increased precipitation. In 1921 this relation showed plainly. The map, Fig. (8) gives the areas of greatest precipitation during the month of August 1921. Heavy rains fell that month, between the 15th and 31st. July and the first part of August were dry. Surveys showed more Diplodia infection on the ears in the areas of heavy rainfall than in the drier sections. In the Western part of the state where the precipitation was light, little dry rot occurred.

In 1922 a similar condition was noted though not so pronounced. Fig. (9) shows the precipitation for August. The prevalence of dry rot on the ears indicates that here again the Diplodia development
coincides with the areas of greatest precipitation. In Hardin county there occurred 2.5 percent of dry rot, in Henry county 4.5 percent and in Marion and Dallas counties 7.5 percent. Passing south through Polk and Warren counties the percentage of dry rot decreased from 10 to 4 percent. In Sioux county 3.5 percent of dry rot was found, this low figure in a relatively wet area may be explained by the fact that this section of the state had its lowest precipitation during early September. The corn was less advanced in this northern locality and the heavy rains in August occurred too early to combine with other factors favoring dry rot.

Although conditions may vary from year to year, the observations of the past two seasons rather indicate that it is the amount of rainfall during August that determines the amount of dry rot. Although the bulk of the precipitation occurred at the end of the growing period in 1921 and 1922 there were times earlier in the season when moisture was apparently sufficient to initiate infection. The inference is, therefore, that certain other conditions must also accompany high precipitation in order to produce an epidemic of dry rot. These other conditions exist annually toward the end of the growing season and are as follows: First there is a maximum of stored food in the corn plant; Second the rapid growth of tissues has ceased and the leaf sheaths have become loosened; Third the loose leaf sheaths afford lodgment for D. zeeae. The presence of water between the leaf sheaths and the stalk at this time initiates infection. When a plentiful water supply is available during this period are seasons when dry rot is prevalent.
Temperature relation

Important as moisture may be in the development of dry rot, temperature also has a significant relation. The effect of temperature on the growth of D. zeae on Pfeffer's solution to which had been added five percent dextrose and one percent peptone. Cultures were made in Erlenmeyer flasks and the inoculum consisted of uniform bits of fungus averaging 0.016 gms.

After inoculation the cultures were held in electrically controlled ovens for a definite time, when the growth was removed, washed, and dried to constant weight.

The growth of D. zeae at different temperatures in grams dry weight was as follows:

<table>
<thead>
<tr>
<th>No. cultures</th>
<th>10°C</th>
<th>15°C</th>
<th>20°C</th>
<th>25°C</th>
<th>30°C</th>
<th>33°C</th>
<th>35°C</th>
<th>40°C</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0 gms</td>
<td>0.745</td>
<td>1.306</td>
<td>1.681</td>
<td>1.949</td>
<td>1.961</td>
<td>1.589</td>
<td>1.298</td>
</tr>
</tbody>
</table>

The above figures indicate that the minimum temperature for growth ranges between 10°C and 15°C, the minimum between 35°C and 40°C and the optimum between 28°C and 30°C. The growth of D. zeae in 153 petri dish cultures under the same conditions gave the same indications as to cardinal temperature. Fig. (10). This temperature, it may be noted, is near the optimum for the growth of corn, which according to Lehenbayer (34) is 34°C.

The relation of the air temperature at the time of dry rot infection does not appear to be as significant a factor in the develop-
Fig. 10 Growth curve of D. zea at different temperatures
ment of the disease as does the amount of rainfall. Yearly weather records show the temperature for the period of Diplodia infection to be very constant and offer ample opportunity for favorable growth of the fungus. The records for rainfall however, show wide fluctuation. Years of high precipitation as 1921 showed prevalence of dry rot.

The relation of soil temperature to Diplodia infection is not very direct. Root infection in growing plants as shown in field tests is practically nil. On the germinating seed and seedling however, there may be some bearing. Slightly infected seed can more readily be injured before the plant establishes roots. Soil temperatures as determined in different parts of the state are as follows:

Mean Soil Temperature 1921.

<table>
<thead>
<tr>
<th></th>
<th>Mitchell County</th>
<th>Story County</th>
<th>Pottawattamie County</th>
</tr>
</thead>
<tbody>
<tr>
<td>May</td>
<td>15.0°C</td>
<td>15°C</td>
<td>17.2°C</td>
</tr>
<tr>
<td>June</td>
<td>22.8</td>
<td>24.5</td>
<td>24.5</td>
</tr>
<tr>
<td>July</td>
<td>26.1</td>
<td>27.8</td>
<td>25.5</td>
</tr>
<tr>
<td>August</td>
<td>21.6</td>
<td>21.6</td>
<td>25.5</td>
</tr>
</tbody>
</table>

The average soil temperature during corn planting in the north, central and even the south part of Iowa, is seen to be near the minimum for the growth of Diplodia.

At Ames over several years the temperatures for May are as follows:

<table>
<thead>
<tr>
<th></th>
<th>1917</th>
<th>1918</th>
<th>1919</th>
<th>1920</th>
<th>1921</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum</td>
<td>14.4</td>
<td>26.5</td>
<td>15.5</td>
<td>16.1</td>
<td>16.7</td>
</tr>
<tr>
<td>Minimum</td>
<td>13.4</td>
<td>11.6</td>
<td>13.8</td>
<td>11.6</td>
<td>13.4</td>
</tr>
</tbody>
</table>

* Figures were obtained from records secured by the Iowa Truck Crop Section of the Experiment Station.
These figures indicate that the soil temperature in Iowa at the time of corn planting and during its early growth is not favorable for Diplodia zeae. The damage to seedlings by this fungus occurs before the seed is planted when the grain is still on the cob. There, during the high temperature of late summer, if moisture is sufficient, the dry rot fungus destroys the endosperm or injures the embryo so that when planted the seed is weak or dead. Some slight growth of the fungus can occur on the planted seed and set up a competition between D. zeae and the seedling for the consumption of the stored food of the endosperm. The low soil temperature however, makes this growth comparatively slow.

Oxygen relations.

Not only is the influence of moisture and temperature on the development of Diplodia zeae important; but also the relation of diminished oxygen supply. In order to determine this relation ten cultures of D. zeae were held in an oxygen free chamber at room temperature for 13 days. The oxygen was removed by action of pyrogallic acid and sodium hydroxide. An equal number of cultures were held at the same temperature for the same length of time in a normal atmosphere. No growth was made on the cultures in oxygen free air though the checks grew profusely. At the end of the 13 days the cultures from one of the oxygen free chambers were removed and exposed to the normal atmosphere. In three days the fungus had completely covered these plates with a heavy growth, while the cultures left in the oxygen free chamber still showed no growth at the end of 18 days. Diplodia is apparently an aerobe.

These results are of particular interest in the light of the relation of Diplodia zeae to ensilage, which is cured under aerobic conditions.
Since Diplodia requires oxygen for its growth and development, it is not a factor of importance in ensilage.

Nutritional reactions

The relation of Diplodia zeae to its source of food supply is of interest not only because of the destruction of the stored food in the corn kernel; but also because of the relation to the available food in other parts of the plant subject to invasion by the fungus. Heald & Wilcox (2) grew D. zeae on glucose agar, glycerine agar, and on steril potatoes and carrots, but obtained no pycnidial formation on these media. The fungus fruited on corn meal agar, steril corn stalks glucose agar, and peptoneless agar acidified with citric acid. Corrosion of starch grains in the corn kernel is also mentioned indicating the use of this substance by the fungus.

The writer has found Diplodia zeae to be an omnivorous feeder and growing well on a number of standard media. It also grows profusely and fruits on sterilized corn stalks, on cornmeal, oats, wheat, oatmeal and bean stems.

In order to obtain a better idea of what food constituents are preferred by the fungus, it was grown on synthetic media to which various carbohydrates and other substances were added. Using Pfeffers solution as a base, solutions of the following sugars were made: sucrose, dextrose, levulose, maltose, and lactose.

Each of these solutions was put in seven flasks and sterilized and then planted with uniform small fragments of mycelium. The cultures were then held at room temperature for eight days, after which the fungus growths were dried to constant weight. The fungus grew well on
these five sugars making the slowest growth however on lactose.

In addition to growing the fungus on sugars, plantings were made on media as described by Craybill and Reed (7). On litmus cream agar a spreading growth and pink color reaction was obtained. Starch was not visibly digested by \textit{D. zeae}, but a good growth and profuse sporulation resulted. On peptone the growth was sparse and spreading with some sporulation. With asparagin and rosalic acid a slight reaction resulted. The dried powdered fungus gave a more pronounced color reaction, indicative of amidase activity. On amygdalin agar a very slight growth was obtained.

Casein is digested by \textit{D. zeae}, the fungus growing and fruiting on the agar agar containing it. Egg albumin supports growth, but is not visibly digested by the fungus. Growth on inulin is very slight. On skimmed milk agar, growth is profuse and the color change indicative of the action of erepsin is present. Pure cellulose agar induces profuse growth and sporulation.

From the above ability of \textit{Diplodia zeae} to use a wide range of food substances is apparent and indicates the production by the fungus of the enzymes lipase, erepsin, amidase, sucrose, maltose, invertase, and cytase and possibly trypsin and amylase.

The utilization of cellulose by the fungus is of particular interest as the penetration and growth in the stalk and weakening of the nodes is thus more readily understood. The survival of the organism in old stubble and trash even though plowed under for several seasons appears probable.

The destruction of cellulose by \textit{D. zeae} also has a bearing on methods of seed testing. In rag doll germinators the fungus readily penetrates
the cloth, attacking kernels of adjoining ears. The placing of paper next to the cloth as in the Huddleston (8) "Modified Ragdoll" offers little more resistance to the fungus. Diplodia zeae readily penetrates the paper and will even grow on wet paper alone.

As there was some question whether the fungus could live and grow in the soil in a saprophytic manner, cultures were made on sterilized field soil sterilized quartz sand and also on both of these to which sterilized organic matter had been added. The cultures were kept moist and held at room temperature. No growth was made on the sand free of organic matter. On both the sand and soil to which organic matter had been added good growth resulted. The fungus invaded the sand and soil and was visible as white patches in the small cavities. On field soil an equally good growth occurred, the fungus apparently getting sufficient food material from the organic matter normally in the soil. This experiment suggests that the dry rot fungus may grow in a purely saprophytic and further emphasizes its ability to survive in the soil.

Infection Studies

In the past, studies of the infection of the corn plant by Diplodia zeae have been limited. Burrill & Barrett (1) were able to show the ability of the fungus to infect the ears, but were unable to successfully inoculate stalks or leaf sheaths except by stab inoculations. Heald and Wilcox (2) however, produced symptoms of the disease on stalk, husk, and on the silks. They considered the latter manner of infection to be the typical means employed by the fungus in
entering the ear. They also record that all ears are infected at the same time, which is a significant observation in the light of the method of infection and conditions governing it as given in the present discussion.

Smith (9) records brief experiments on soil infection and Van der Bijl (10-12) suggests the possibility of root infection by D. zeae. However, infection of the corn plant in its various stages of development and the relation of environmental conditions to the invasion of the fungus has not received attention.

Method of Infection

In an earlier paper (13-14) the writer called attention to the blotching of corn sheaths due to various organisms, later observations have added to the list of agents that may cause a blotching of the corn sheath one of the most prevalent of which is Diplodia zeae. This is particularly significant in the present discussion as it explains the more prevalent method of infection of the corn plant by this fungus. Though Diplodia zeae may enter through the silk the chief method of infection may be described as follows: Prior to the production of flowers by the corn plant the ligules of the leaf sheath clasp the stalk very tight preventing anything from slipping down inside the sheath and if anything did drop down the rapid elongation of the stalks would carry it up again.

After flowering however, the stalk has ceased elongating and the action of the wind on the leaves has by this time loosened the ligule and exposed the cavity of the sheath. Fig. (11). At the same time
Fig. 11 Open corn leaf sheath showing pollen and pollen sacs sticking to sides. The white incrustations of sugar on outside of sheath is indicated with the arrow.
Fig. 12 Pollen and pollen sacs as center of infection with in sheath
Fig. 13) *D. zeae* consuming pollen mass at base of leaf
Fig. 14 Drops of water condensed on stalk, photographed immediately after sheath was cut away—such moisture favors mold growth in sheath cavity.
Fig. 15 Section of corn stalk showing Diplodia infection extending up and down from node, manifest as darkened bundles.
masses of pollen fall and roll down the leaf into the sheath together with such spores as they may carry along or are blown in Fig. (12-13)

In addition to this combination of the spores and the stored food present in the pollen, a third factor of moisture enters. The sheaths of corn are frequently moist inside, even in dry weather the writer has found them holding condensed moisture in droplets Fig. (14), often and in wet weather they stand full of water/remaining that way days after the rain. The past season there were periods when this condition existed to such an extent that the water became charged with sugar from the corn plant which was deposited, on evaporation, as a white crust at the edges of the sheath. (Fig. 14). What better culture conditions for Diplodia spores could be described than the above combination of pollen, sugar, moisture? The fungus first feeds on the food material at hand, when this is exhausted it invades the corn plant, Fig. (1,15) causing on the upper part of the sheath, spots and blotches, attacking the thin walled sheath cells rather than the thick walled cells of the stalk. At the base of the sheath the structure of the outer cells of the node as shown in Figs. facilitates entry, there the fungus invades all tissues confining its action however, chiefly to those cells nearer the outer rind, dis-coloring the parts invaded a brownish water soaked color. This discoloration is a good index of the presence of the fungus as many isolations indicate. The fungus grows down from the node rather than up, though travelling in both directions. Fig. (15). Infection of the ears occurs in the manner above described, infection entering at base of husk. However, a large percent of the infection on the ears is produced by the fungus working up the ear shanks from the diseased nodes.
The relation of sheath invasion is of much significance in Diplodia infection of corn as shown by the following test. Five hills of corn were covered with as many cheese cloth tents and the tassels removed from the stalks before they emerged. None of these plants showed any spotting of the sheaths until they were beginning to die and the tents had become weather beaten, while all surrounding corn was purple with blotches shortly after flowering. Platings from these stalks, nodes and internodes, their entire length, showed no Diplodia or other infection while surrounding uncovered plants were found badly effected. The age of the plant does not necessarily influence infection though as previously mentioned, strong vigorously growing roots do not become infected and during rapid growth infection is excluded from the stem. However, in the later case if the sheath is forcibly opened and D. zea inoculum introduced, the plant will be attacked. Partly because the lower leaf sheaths are older and are the first to open up and allow invasion, and partly because of the more constant moisture near the ground. Diplodia zea is most profusely found on that part of the plant. This perhaps has suggested the idea that the fungus enters the crown and imigrates up the stalk, in some cases to the ear. Where these lower leaves are stripped off early in the season, very little Diplodia infection is found on the lower part of the stalk. In one locality in July, 40 plants were stripped of their three lower leaf sheaths. None of these stalks showed infection up to the fourth node, though eleven out of an equal number of stalks, not stripped were badly infected by D. zea at the lower nodes as indicated by isolations from the inner tissues.
Local character of Infection.

That Diplodia zeae is local and not systemic in its infection, gaining entrance at various nodes and on the ear rather than traveling up from the roots, is indicated by considerable evidence as is shown in the following figures:

Using data from the above described plots, it is interesting to consider the relation of seed, seedlings and stalk infection to subsequent ear infection and also the distribution of moldy ears in the field.

The results are shown in the following table:

Table (1) Number of dry rot ears on stalks grown from Diplodia infected and clean seed.

<table>
<thead>
<tr>
<th>Percent stand</th>
<th>Number of dry rot ears</th>
<th>Percent wt. dry rot ears</th>
</tr>
</thead>
<tbody>
<tr>
<td>57 rows from clean seed</td>
<td>90</td>
<td>55</td>
</tr>
<tr>
<td>57 rows from Diplodia infected seed</td>
<td>64</td>
<td>44</td>
</tr>
</tbody>
</table>

In the above table is given the percent stand resulting from planting seed attacked by D. zeae. It will be seen that where ears attacked by this fungus are planted, a decrease of 26 percent of stand results. If D. zeae followed this up by a systemic invasion of the remaining plots a higher percent of dry rot ears would be expected in the diseased rows. This is not the case however, for approximately the same number of moldy ears occur on all rows whether from diseased or clean seed. Infection on the ears appears to be generally distributed in the field as observations in other plots throughout the state also
indicate. Further the figures in the above table do not suggest that the less vigorous plants which result from diseased seed are any more subject to attack by *D. zeae* than those from vigorous clean seed.

Supplementing the observations recorded in the above table numerous isolations were made from stalks attacked by *D. zeae*. Large numbers of counts were made of diseased nodes on stalks in different fields. In the following tables this data is presented as additional evidence that infection by *D. zeae* is local. Observations and counts on 984 stalks revealed the following:

<table>
<thead>
<tr>
<th>Nodes</th>
<th>1st node</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
<th>5th</th>
<th>6th</th>
<th>7th</th>
<th>8th</th>
<th>9th</th>
</tr>
</thead>
<tbody>
<tr>
<td>% of stalks</td>
<td>58</td>
<td>66</td>
<td>63</td>
<td>47</td>
<td>89</td>
<td>14</td>
<td>5</td>
<td>1</td>
<td>.2</td>
</tr>
</tbody>
</table>

On 454 stalks in another locality the following infection was observed:

The inference to be drawn from the above is that *D. zeae* attacks chiefly the older lower part of the plant up to the first 2 or 3 nodes and rarely infects the younger parts above 5th and 7th node. On the same plants observed in the above data note was made of the relation of moldy and broken shanks to the attached ear, etc., with the resulting generalizations:
Only 12 percent of broken shanks bore Diplodia infected ears.
Only 12 percent of Diplodia ears were on Diplodia infected shanks.
Only 12 percent of Diplodia rotted shanks were broken.
Only 13 percent of Diplodia rotted shanks bore Diplodia infected ears.
46 percent of the Diplodia infected shanks were on stalks.
39 percent of the Diplodia infected ears were on stalks.
53 percent of the broken shanks were on Diplodia free stalks.

Further the frequency of Diplodia rotted nodes below Diplodia rotted shanks was found to be as follows:

<table>
<thead>
<tr>
<th>Node above ground</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
</tr>
</thead>
<tbody>
<tr>
<td>Percentage</td>
<td>22%</td>
<td>26</td>
<td>19</td>
<td>24</td>
<td>17</td>
<td>13</td>
<td>8</td>
<td>2</td>
</tr>
</tbody>
</table>

It is evident from this summary of observations on nearly 1,500 plants that little relation exists between the Diplodia infection on stalk, shank, and ear.

To more completely substantiate the above figures, platings were made from numerous corn stalks, node by node, from the ground up. From one series of tests embodying 1,146 isolations, the following results were found:

8 percent of Diplodia infected nodes bore Diplodia free shanks.
5 percent of Diplodia free nodes bore Diplodia infected shanks.
31 percent of Diplodia internodes between infected nodes were free of Diplodia.
21 percent of Diplodia above infected nodes were free of Diplodia.
8 percent of all ear shanks were free of Diplodia.
50 percent of all cobs were free of Diplodia.
5 percent of nodes at shanks were free of Diplodia.
38 percent of internodes below Diplodia infected shanks were free of Diplodia.
10 percent of nodes below Diplodia infected shanks were free of Diplodia.

A frequency of the occurrence of Diplodia zeae at nodes and internodes in another set of observations is as follows:
A frequency of the occurrence of *Diplodia zeae* at nodes and internodes in another set of observations is as follows:

Table (2) Percent of stalks free of *D. zeae* at nodes and internodes.

<table>
<thead>
<tr>
<th>Node</th>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
<th>4th</th>
<th>5th</th>
<th>6th</th>
<th>7th</th>
<th>7th</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
<td>11</td>
<td>15</td>
<td>18</td>
<td>12</td>
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<td>28</td>
<td>46</td>
<td>12</td>
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</tbody>
</table>

It may be noted in the above tabulations that the greater proportion of the infection by *D. zeae* occurs at the nodes than the internodes, also that the first node is not as frequently infected as the second and that the upper nodes and internodes are more free of *D. zeae* than the lower ones. If the fungus travelled successively from node to internode up the stalk, plateings from node and internode would show the same degree of infection.

It also appears from these figures that clean shanks can be borne on infected nodes and infected shanks on clean nodes and that whereas only 8% of the shanks were free of *Diplodia* 50% of the cobs on these shanks were free. It might be mentioned that the isolations represented in these figures were from stalks that were mature and nearly ready to harvest it which time *D. zeae* is most prevalent, rapidly invading tissue that has ceased to grow.

On greener stalks cut August 27th, the results are somewhat different. From isolations from 50 stalks, made at every node and internode from grind up the following results were obtained:
All sheaths in above stalks were covered with Diplodia sheath spot.

From another lot of somewhat more mature corn from another field the following data was gathered Sept. 1. This field it might be said, had borne corn the previous year and the soil was full of old Diplodia covered stubble. Tissue was plated from every node and internode from ground up to the fifth node using 28 representative stalks.

Table 3

<table>
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<tr>
<th>Stalk number</th>
<th>Crown</th>
<th>Internode #1</th>
<th>Node #1</th>
<th>I^1</th>
<th>N^2</th>
<th>I^3</th>
<th>N^3</th>
<th>I^4</th>
<th>N^4</th>
<th>I^5</th>
<th>N^5</th>
<th>Condition of stalk</th>
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continued
Table (4) contd.

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</tbody>
</table>

x = Diplodia infection.

The outstanding points in these figures are that again the crown and first internode are less affected than those immediately above, also that in most of the stalks attacked gaps of clean tissue intervene between diseased areas. This point would have been evident in the data from the other tables had it been presented in the same way. Another noticeable thing in the above table is that certain stalks are very clean and others quite diseased. It is of particular interest in this connection to point out that the clean stalks were also green and the diseased stalks dry, that is those stalks still live and growing, are less subject to Diplodia attack than those that are on the decline.

Forty stalks, from those plants subjected to root inoculation on July 10th and August 1st, were also plated node by node Sept. 11th and 16th. In these the same relation of green and dry stalks was manifest.
On Seedlings

Aside from the more obvious dry rot on the corn ears the infection of the seedlings is of particular importance. The questions arise what is the relation of the diseased seed to seedling blight, can *D. zeeae* in the soil or on old stubble attack the seedling, and with the first point under consideration vigorous corn was taken from the germinator and transplanted before they were large enough to be injured. Fifty plants were planted in flats over corn stalks which were heavily covered with ripe pycnidia. The cultures were held at 28°C soil temperature for eight days, then dug up. No effect was noticeable on the roots though these surrounded and lay next to the fruiting *D. zeeae* on the old stalks.

Later 46 clean plants were used repeating the above experiment, of these five plants showed rotting of some of the very small roots directly adjoining the Diplodia covered stalk, 15 other plants showed a spot of decay here and there.

Further 31 clean seedlings were planted in flats in which the soil was heavily seeded with Diplodia growing on sterilized oats, and held at 30°C. Out of this lot 10 showed rotted roots and crowns. A similar lot of 30 seedlings were held for the same length of time at 15°C to 20°C. In this case only five plants showed necrotic areas on the roots. The indications are, therefore, that *D. zeeae* can attack young corn roots. However, it must be considered that in the above experiments the amount of infecting material was very great and out of all proportion to what might occur in the field, also the soil
temperature was higher than that occurring in the field, and more favorable.

Carrying the above experiment into the field, special infection plots were arranged in which corn showing 100 percent germination was planted over Diplodia infected stalks, corn kernels, and masses of mycelium. The hills in these plots were planted one foot apart, three kernels to a hill, with the inoculum growing on oats placed below the seed corn. When plants were eight to ten inches high, every second and third hill was dug up and the seedling roots washed and examined. Three plantings were made, the first May 9th, and later plantings May 16th and May 21st. Of primary interest in these plantings was the stand; how many seedlings failed to emerge when given opportunity for infection.

Table 5 Action of D. zeae on Roots of corn seedlings in field.

<table>
<thead>
<tr>
<th>Plot</th>
<th>No. of seed</th>
<th>Percent stand</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clean seed only</td>
<td>252</td>
<td>98</td>
</tr>
<tr>
<td>Clean seed over Diplodia infected kernels</td>
<td>252</td>
<td>98</td>
</tr>
<tr>
<td>Clean seed over mass of Diplodia inoculum</td>
<td>216</td>
<td>88</td>
</tr>
<tr>
<td>Clean seed over old Diplodia infected stalks</td>
<td>81</td>
<td>65</td>
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</tbody>
</table>

The above figures indicate that in the field Diplodia zeae in proximity to germinating corn kernels may rot them or the young seedling so as to affect stand Fig. (5). In the case of Diplodia rotten seed however, planted with healthy seed, there is no indication of any appreciable spread and injury to the latter.
It must be remembered however, that in the field as in the greenhouse tests the source of infection was in great excess of what would be found naturally and indicates the possibilities rather than what may ordinarily be found.

The main loss from seedling infection by *D. zeae* arises from Diplodia infected seed. The kernels are attacked on the cob in varying degrees by the fungus resulting in dead or weak plants in the field. Field counts in different localities showed a loss in stand often as great as 25% percent. The results of such infection were most striking in test plots in which clean and diseased seed, as determined on the germinator, was planted side by side. Three plots were planted as follows: One plot near Story City on the farm of Clarence Johnson, consisting of 101, 25 hill rows, representing 101 ears of Johnson's corn. A second plot on the Experiment Station farm at Ames, consisting of 201, 10 hill rows representing 201 ears of varying degrees of soundness from different parts of the state. A third plot located near the second consisted of plantings from 28 identical ears, 14 sound and 14 showing 1 to 4 Diplodia infected kernels. These ears were planted with 3 checks every seventh row. Check number 1 being made from two sound ears shelled and mixed. Check number 2 was a composite of all ears used in the plot, and check number 3 was a composite of all the diseased ears in the plot.

The whole of plot No. 3 was divided in 2 parts, planted at different dates. The first planting May 11th consisted of 43, 12 hill rows comprising plantings of the 28 ears and 5 sets of the checks as above described. The row numbers ran from east to west. To check on soil
a duplicate of this planting was made the same date with rows numbering 1 to 43 west to east or just the reverse.

On May 22nd the second part of plot number 3 was planted. It consisted of a replication of the planting on May 11th except there was but one series of 24 hill rows numbering 1 to 43 east to west.

The following data gives the germination of the ears used in the plots above mentioned together with the resulting stand and seedling blight caused by D. zee.

In plot number 3 from the 42 ears showing no Diplodia infection on the germinator, a stand of 90 percent resulted in the field; while on the 42 rows showing 1 to 4 Diplodia infected kernels only 62 percent stand was obtained. The 15 rows of checks from clean seed also gave 90 percent field stand; while the 15 rows of checks representing a composite of all the Diplodia infected ears in the field gave 66 percent.

It would appear from these figures that a germinator test may constitute an index of the Diplodia infection on the resulting seedlings, however, the question arises as to how many kernels from an ear must show Diplodia on the germinator in order to cause material loss of stand.

This relation of Diplodia infected kernels as found on the germinator and the resulting stand as determined with 308 ears planted in plots 1 and 2 is as follows:

<table>
<thead>
<tr>
<th>Percent of Diplodia infected kernels on germinator</th>
<th>Resulting stand</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>98%</td>
</tr>
<tr>
<td>12</td>
<td>86</td>
</tr>
<tr>
<td>25</td>
<td>80</td>
</tr>
<tr>
<td>37</td>
<td>75</td>
</tr>
<tr>
<td>50</td>
<td>45</td>
</tr>
</tbody>
</table>
These averages show a correlation between the number of Diplodia infected kernels on the germinator and the resulting field stand. Individual exceptions to this are often noted however, as ears showing clean on the germinator may not give 100 percent stand in the field.

This is easily possible for dry rot often occurs on small groups of kernels on the ear not evident to the eye and in sampling such kernels may be missed.

Plants past seedling stage.

Second to the consideration of the effect on seedlings and stand is the action on plants after they have established themselves. In the following table is given the percentage of plants having roots rotted in greater or less degree when grown over various types of inoculum.

Table (6) Action of D.zeae on roots maturing plants.

<table>
<thead>
<tr>
<th>Plot</th>
<th>No. plants</th>
<th>No. plants showing rot on roots</th>
</tr>
</thead>
<tbody>
<tr>
<td>Over Diplodia stubble</td>
<td>12</td>
<td>10</td>
</tr>
<tr>
<td>Over Diplodia culture</td>
<td>290</td>
<td>53</td>
</tr>
<tr>
<td>Over Diplodia kernels</td>
<td>60</td>
<td>22</td>
</tr>
</tbody>
</table>

The figures in the above table indicate that Diplodia zaeae may rot the roots of young corn plants if supplied in sufficient quantities, however the experiment is hardly a criterion of what actually happens in the field for the inoculum was of great amount. The point of interest is not, however, how many plants had root lesions,
but how many survived. Once the corn plant is independent of the food from the old kernel its rapidity of growth together with the unfavorable conditions for Diplodia development at that time lessen the chances of root infection. In the above experiment out of the hills planted, one foot apart every second and third was removed for observation leaving a third of each lot, standing 3 feet apart. These plants grew the rest of the season in normal condition. Many of them over 8 feet in height and produced good ears. If the roots sustained any injury from the inoculum it was not manifest. In fact, many plants that were dug later showed clean white roots growing through old masses of dry rot inoculum without discoloration or injury. Isolations made from the crowns of 50 of these stalks, Aug. 3-31, showed D. zaeae in but one instance. The other 48 plants were clean and free of infection though the seed had been planted in a mass of inoculum the size of a walnut. At time of digging some of this material still enveloped the crown of the plant.

In addition to the above observations on plants from seed planted in Diplodia inoculum, an experiment was made on healthy stalks as follows: On July 10th the soil was removed at one side of the base of 22 stalks, care being taken not to injure the roots. A mass of inoculum the size of an egg was tightly packed into the cavity between the roots below the crown of the plant and the soil was covered over it. On August the first, 18 more stalks were similarly treated. The following table gives the results of these tests. Plants grown in a field containing old stubble covered with Diplodia were used as a check.
Table (7) Results of inoculating roots and crowns of plants with D.zeae

<table>
<thead>
<tr>
<th>Date of inoculation</th>
<th>Date of isolation</th>
<th>Number of plants</th>
<th>Percent of D.zeae at crown</th>
</tr>
</thead>
<tbody>
<tr>
<td>July 10</td>
<td>Sept. 11</td>
<td>22</td>
<td>68</td>
</tr>
<tr>
<td>Aug. 1</td>
<td>Sept. 16</td>
<td>18</td>
<td>33</td>
</tr>
<tr>
<td>Natural soil infection</td>
<td>Sept. 1</td>
<td>28</td>
<td>7</td>
</tr>
</tbody>
</table>

It must be said in regard to these figures that they represent isolations from the crown of the plant, which is relatively dead tissue when the plant is larger, and does not represent root infection. On none of the plants were root lesions found. These negative field trials suggest that root infection by D.zeae was not significant under the conditions that prevailed in the field during 1922.

**Effect of D.zeae on maturity and yield.**

In any field of corn toward the period of ripening some stalks will be found that mature sooner than other, considering the prevalence of D.zeae at this time on the lower nodes of the stalks the question arises does this infection play a part in the premature ripening. Counts, in one plot under observation showed that many early maturing plants had no D.zeae at base, conversely many green growing stalks did show D.zeae at the basal nodes.

*Diplodia zea* if it enters the plant may no doubt hasten early ripening, but it is doubtful if it is the cause. In the succeeding
table are figures showing the relation of this maturity to Diplodia infection.

Table 7  Relation of maturity and Diplodia infection at base of stalk

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>19</td>
<td>9</td>
<td>7</td>
<td>1</td>
<td>69</td>
</tr>
<tr>
<td>11</td>
<td>25</td>
<td>6</td>
<td>3</td>
<td>0</td>
<td>96</td>
</tr>
<tr>
<td>21</td>
<td>25</td>
<td>5</td>
<td>8</td>
<td>2</td>
<td>96</td>
</tr>
<tr>
<td>31</td>
<td>13</td>
<td>3</td>
<td>7</td>
<td>3</td>
<td>96</td>
</tr>
<tr>
<td>41</td>
<td>14</td>
<td>13</td>
<td>4</td>
<td>2</td>
<td>96</td>
</tr>
</tbody>
</table>

In the plots on which the above data was gathered rows 1, 11, 21, 31, and 41 were from the same seed and duplicated on different dates 11 days apart. The degree of Diplodia infection recorded does not indicate a relation existing between the number of infected stalks and the number of dying stalks moreover the dying stalks are not necessarily infected ones.

There is some evidence that infection by D. zaeae at the lower nodes of the stalk reduces yield. On 4080 plants observed those rows having an average of 13 stalks infected at base gave a yield of 22.6 lbs. will those rows having an average of 6 diseased stalks yield 25.7 lbs. The stand in all rows was the same. While comparative-
ly few plants are represented in these observations, the 2.6 lb. greater yield on the less infected rows suggests that the invasion of *D. zeae* into the lower nodes may be detrimental to the best functioning of the corn plant.

In addition to the early maturity and dying of individual stalks the symptoms of barrenness and breaking of the stalk are of common occurrence in corn.

The prevalence of dry rot suggests that it may play a part in producing these symptoms. However, on the plants observed in one plot, 19 percent showed dry rot infection at the lower nodes, but only 3 percent of the plants were barren. It would seem that a greater amount of barrenness would occur if dry rot was an agent. Further no relation appeared to exist between disease seed and barrenness for the cleanest seed producing the most perfect stand of vigorous plants may produce more barren plants than badly diseased seed.

In the case of broken stalks a similar situation exists. Broken stalks are generally distributed over the field. The infection by Diplodia locally at the nodes is conducive to breaking. There is no relation between diseased seed and this breaking of the first few nodes above the ground as shown by records in over four thousand hills of corn.

In measuring the effect of dry rot on corn, yield must be the index of the injury.

In the following table are given the results of planting healthy and Diplodia infected kernels, expressed in yield. The figures
represne data on 4564 hills planted 3 kernels to a hill.

Table (9) Yield versus germination and stand.

<table>
<thead>
<tr>
<th>Percent germination</th>
<th>Total yield in lbs.</th>
<th>Percent stand</th>
</tr>
</thead>
<tbody>
<tr>
<td>57 rows of Diplodia free seed 100</td>
<td>1714</td>
<td>90</td>
</tr>
<tr>
<td>57 rows of Diplodia infected seed 75</td>
<td>1272</td>
<td>64</td>
</tr>
</tbody>
</table>

In six localities over the state similar plot comparisons show like results for diplodia infected seed.

The bulk of this loss is no doubt due to lowered stand where infected seed is used, for it is quite obvious that a 64 percent stand would greatly decrease yield.

The effect of D.zeae on seed and seedling while it constitutes the chief destruction of the fungus is not however, the only detrimental effect produced. The results of planting seed from dry rot infected ears indicate that the fungus not only kills a certain percent of the seed as evidenced by lower stand, but also the plants that do grow show weakening. In the following table is given comparative heights of plants from Diplodia infected and Diplodia free seed.

<table>
<thead>
<tr>
<th>Height in feet</th>
<th>Percent stand</th>
</tr>
</thead>
<tbody>
<tr>
<td>45 rows Diplodia free seed 4.0</td>
<td>90</td>
</tr>
<tr>
<td>68 rows Diplodia infected seed 3.2</td>
<td>64</td>
</tr>
</tbody>
</table>
Fig. 17 Resulting height of corn from healthy and Diplodia infected seed. The poll is held horizontal at height of healthy corn on the left.
These figures indicate that the plants that do grow from diseased seed are in some way at a disadvantage and are stunted, differing in height by over one half a foot from the plants from clean seed. Not only do the plants from seed from Diplodia infected ears show decreased height but they are more spindly and yellowish as well. In Fig. (17) is shown the comparative height on July 11th of two of the rows included in the above table of measurements. The difference between the end and center of the pole held horizontally constitutes the difference in height between a row from healthy and infected seed. Diplodia zeae attacks the kernels of an ear in varying degrees, some are killed outright and microscopical examination shows such kernels to be completely overrun with the fungus mycelium. Other kernels are very slightly affected. This is the most probable explanation for the weak plants from dry rot infected ears. The badly infected kernels do not germinate, the slightly affected ones germinate but are weakened by the fungus.
The studies reported in this bulletin seem to warrant the following recommendation for the control of the dry rot disease; namely, a long rotation, early field seed selection and germination before planting in the spring. Since it has been found that the dry rot organism can live for at least three years on old corn stalks in the field, a five year rotation is more desirable than a shorter one. Every effort should be made to hasten the decay of the stalks by thorough covering when the ground is plowed.

The seed corn should be selected as soon as the ears are well filled and dented without regard to the time of frost. Seed ears should only be selected from green standing stalks with sound shanks. After the seed has been gathered it should be cured in a dry, well ventilated place, not exposed to the weather.

Since it has been found that apparently sound ears may be slightly infected when gathered in the fall, and that the fungus may spread in the ear, while it is curing, it is necessary to run germination tests in the spring before planting. The most practical methods of testing seed corn germination have already been described in circular 78 of the Iowa Agricultural Experiment Station.
Part II.

Pathogenicity of Fungal Flora of Seed Corn.

Introduction

In the preceding chapter consideration has been given to Diplodia zeae as one of the most important corn parasites. There are, as is well known numerous other fungi that occur on corn and especially on germinating seed corn. Little is known about the prevalence, destructiveness, or biology of these organisms as they occur on corn. Much confusion exists as to the importance of these organisms found on seed corn and little is known on their relation to the corn plant and the seed. In order to study this problem it became imperative to identify all the fungi associated with D. zeae on germinating seed corn and to record the characteristic symptoms produced by them. In addition to recognizing these on corn, it seemed worth while to determine whether these fungi were on or inside of the seed coat. No attempt will be made to collect all the literature relating to the organism found. Only the papers having a direct bearing on the problem as outlined will be cited. It is significant to note that Oudemans lists the following species as reported on corn kernels:

- Epicoccum purpurascens
- Entoloma demitriacem
- Ustilago grandid
- Rhizopus elegans
- Aspergillus effusus
- Aspergillus sterigmatophorus
- Chromosporium maydis
- Oospora heteromera
- Penicillium glaucum
- Sterigmatocystis italic
- Cladosporium zeae
- Cladosporium herbarum
- Trichosporium maydis
- Fusarium heterosporium
- Fusarium moniliforme
- Sclerotium maydis
Several of these saprophytic molds have been studied in connection with the deterioration of corn and corn meal. As molds alter the acidity of the grain, that factor has been taken into consideration by Van der Bijl (14) and by Besley and Boston (15) in judging the deterioration of corn.

The deterioration of corn due to molds has also been extensively studied in this country and in Italy in connection with Pellagra and stock poisoning. Alsberg and Black (16) made careful investigation of Penicillium puberulum and \textit{stoloniferum} in this connection, in an effort to locate definite toxins formed in maize deteriorated by these molds. Cotton (17) names four Rhynchophora found on stored corn that contribute to its deterioration, and Heald (21) observed \textit{P.purpureum} on corn kernels.

From the standpoint of plant parasitism, relatively few fungi have been cited as occurring on corn. Sheldon in 1903 (18) described \textit{Fusarium moniliforme} on corn while a few years later Burrill and Barrett (1), Smith (7), Heald and Wilcox (2), and Van der Bijl (7), discuss \textit{Diplodia zeae} as a parasite of this corn kernel in field and in storage.

Caffarelli (19) mentions \textit{Aspergillus varians} as parasitic on corn kernels, while Taubenhaus (20) refers to two other \textit{Aspergillae}, as parasitic on the seed, \textit{A. niger} and \textit{A. glauus}. These fungi in addition to \textit{Diplodia zeae} and \textit{Fusarium}, he considered responsible for weak plants and seedling blight.

Chen (21) reports finding a fungus in sweet corn seed which he identified as \textit{Oospora verticilliodes}. He states that Tiroboschi and also Deckenbach found the same fungus in corn kernels. Arzberger (22) in 1916 describes certain symptoms of kernel and cob rot to \textit{Coniosporium geseevi}, classing it as a parasite on the ears.
Bubok (23) to whom he refers for description of this organism considers it saprophytic on the corn ears.

A number of the references to corn molds however, group around various Fusaria and related fungi.

Burrill and Barrett (1) in 1909 give accounts of three Fusaria on corn which, however, they do not specifically name, but designate as I, II, and III. Pammel, King and Seal (24) discuss and figure various Fusaria found on corn. Holbert and Hoffer (25) refer to various organisms on seed corn as responsible for ear and root rots, chief of which are Gibberella acervalis and G. saubinetti. Valleeau (26) found Fusarium moniliforme on the seed and associated with root and stalks rots. Manns and Adams (27, 28) list four fungi as being the most prevalent molds on seed corn as determined from numerous samples from Maryland and also other states. They place them in order of their prevalence as follows: Fusarium moniliforme, Gibberella saubinetti, Cephalosporum sacchari and Diplodia zeae.

Frost (29) in his discussion of the relation of starchy kernels to mold infection recognizes Diplodia zeae specifically, but other molds present on the germinating corn he merely designates as Penicillium spp. and Fusarium spp.

In all of these papers the molds found were either catalogued as present on the corn kernels or were considered from the standpoint of future parasites on the corn plant. No effort is made by the writers to distinguish the superficial, uninjurious molds found on the kernels in the germinator, from the truly parasitic and harmful molds. Adams and Russell (30) are the only exception in that they discuss the confusion arising from the over-running of the germinator with Rhizopus nigricans and the difficulty of determining the degree of germination under such conditions.
Method of Study

Kernels attacked by *D. zeae* can readily be distinguished on the germinator and appear as described in another part of this paper. Out of 1,000 ears of sweet corn tested 1.8% of the kernels were affected with *D. zeae*, representing 62 ears. In the case of 600 ears of field corn tested on the germinator 1.2% of the kernels from 27 ears were affected with *D. zeae*. Aside from these easily recognized numerous others showed molds as herein listed.

It was determined to inquire into the extent of the invasion of these molds, consequently one sample of 100 ears was selected showing molds other than readily recognized as *Diplodia zeae*. These ears had tips that manifest molds on the germinator as figured in Figs. (27-29). A second sample was also selected comprising 26 ears showing vigorous growth and no mold on the germinator. Plantings on agar agar plates were made from the kernel tips and embryos of the kernels. The methods employed were as follows:

Kernels were sampled spirally around the ear from those adjoining the kernels previously showing mold on germinator. These kernels were sterilized 50% aqueous alcohol containing 1 to 1000 HgCl₂, as used by Manns and Adams (27-28). In order to test the effectiveness of this method for killing surface borne organisms, corn kernels heavily dusted with spores of *P. expansum* were subjected to this alcoholic HgCl₂. The following results indicate that dipping or immersion for one-half minute is sufficient to destroy such surface contamination.

<table>
<thead>
<tr>
<th>Time of immersion</th>
<th>Results on plating</th>
</tr>
</thead>
<tbody>
<tr>
<td>dip</td>
<td>all platings clean</td>
</tr>
<tr>
<td>½ minute</td>
<td>&quot;</td>
</tr>
<tr>
<td>1 &quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>2 &quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>3 &quot;</td>
<td>&quot;</td>
</tr>
<tr>
<td>Check untreated</td>
<td>All molded with <em>P. expansum</em></td>
</tr>
</tbody>
</table>
Fig. (18) Layout of instruments preparatory to plating kernel tips and embryos. Glass table top and glass tool rest facilitate sterile conditions. The small Gooch crucible on wire holder enables easy sterilization of kernels, all of which may be immersed and withdrawn at the same time.
Fig 19  Tools adapted to the removal of kernel tips and embryos.
1- light sharp edged tweezers for removing seed coat and gouging out embryo
2- heavy tweezers with toothed tips bent to fit kernel
3-, stout short bladed shears for clipping off tips
The cork on tweezer handles insulates from heat of repeated sterilizing.
Fig. (20) Method of removing tip cap
Stout tweezers are used to grip the kernel while the cap is partially clipped off with a pair of short bladed shears. The tip is then removed to an agar plate with another pair of tweezers.
Method of removing embryo aseptically. The kernel is held firmly with a pair of stout tweezers while with a sharp chisel pointed pair the seed coat is torn back and the embryo lifted out.
Of course such disinfection is not 100 percent effective in all instances, particularly in case of bacteria. However, as the number of plantings was large and time was an element, this method was considered safe for the purpose at hand.

Two sets of plantings were made of the kernels from the 100 ears, one of the tip caps, the other of the excised embryos. In case of the tip cap, the kernels were removed from the ear, as before mentioned, and placed in a gooch crucible as shown in Fig. (28) and immersed in the alcohol-\( \text{HgCl}_2 \) solution. They were withdrawn in one-half minute, and while held with a stout pair of tweezers, filed and bent to fit a kernel, the caps were clipped partly off, with a short bladed shears, Fig. (20). The tip caps were then entirely removed with another pair of tweezers and placed on media. All instruments were dipped in alcohol and flamed before every contact.

The embryos were removed in a similar manner. The kernels were soaked for a few hours to loosen the seed coat, which can then be readily stripped after surface sterilization. A sharp chisel edged pair of tweezers serve well for this purpose and also easily gouged out the embryo, Fig. (21).

Data and Observations

Though mentions are made in the literature of numerous molds found on corn, no attention is given to the relative destructiveness of the various species. It is taken for granted that such molds as Penicillium sp. are only saprophytic and that those belonging to genera containing parasitic species like Fusarium are necessarily parasitic and dangerous parasites.
The following is a list of fungi as found by this writer during the past two years on the ear, and on germinating seed corn.

Helminthosporium turcicum
Rhizopus nigricans
Aspergillus niger
Aspergillus flavus
Aspergillus clavatus
Penicillium expansum
Penicillium citrinum
Penicillium commune
Penicillium divericatum
Penicillium luteum
Penicillium purpurogenum
Gibberella saubinetti
Fusarium moniliforme
Diplodia zeae
Coniosporum geceva
Altenaria sp.
Cephalosporum Acremonium
Fusarium sp.

Some of these molds occur constantly on germinating seed corn, while others are found only occasionally. The flora no doubt varies with the season and locally.

In the following figures (22-30) the symptoms of the most common of these fungi are shown, as they appear on the kernels.

There are doubtless intergradations from saprophytism to parasitism in the fungi found in corn. Three possibilities may arise as follows: First the fungus may be only on the surface of the seed as scant mycelial growth, or as spores; Second it may be present as mycelium just within the seed coat or tip cap; and third, it may penetrate to the embryo. In the case of Diplodia zeae, all three conditions may prevail depending on the time elapsed after exposure to infection and such may possibly by the case with certain of the other fungi.
Fig. (22) Kernel attacked by *P. purpurogenum*. This mold often occurs on germinating corn. The fungus secretes a bright red stain that is soluble in water and permeates the seed coat.
Fig. (23) Kernel on germinator overrun with Rhizopus nigricans. The mycelium of the mold spreads rapidly over large groups of kernels and in ragdolls penetrates numerous layers of the cloth. The spread of this mold and the rotting of the shoots and rootlets by it hampers the determination of the good and bad ears.
Aspergillus niger one of the most common molds found on the germinator. The fungus attacks at any crack or abrasion in the seed coat.
Fig. (25) *Penicillium expansum* growing and fruiting on surface of seed coat. This is a common symptom on kernels on germinator occurring most frequently on those from immature or improperly stored ears.
Fig. (26) Asrostalagmus penicilloides fruiting on surface of seed coat and on abrasion of crown of kernel on germinator
In order to determine the extent of invasion of the fungi, on the seed corn, the tip caps and embryos of the 100 ear samples was tested for the presence of mycelium in the tissues, using the methods described above. In all of these 100 ears the tip cap became moldy on the germinator as shown in Figs. (27-28).

The results of the plantings were as follows: None of the common molds as Penicillium sp. or Aspergillus sp., etc., as above listed were found in the excised tip caps or embryos planted. The inference is therefore that all such molds are surface borne and are eliminated by short surface disinfection. Of 500 kernel tip caps planted, 45 or 9 percent showed the following fungi:

<table>
<thead>
<tr>
<th>No. Kernels</th>
<th>Fungus</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Coniosporium gecevi</td>
</tr>
<tr>
<td>4</td>
<td>Diplodia zeae</td>
</tr>
<tr>
<td>9</td>
<td>Fusarium moniliforme</td>
</tr>
<tr>
<td>22</td>
<td>Cephalosporium acremonium</td>
</tr>
<tr>
<td>5</td>
<td>Fusarium sp.</td>
</tr>
</tbody>
</table>

This represents 34 percent of the ears used. The rest of the ears selected on the basis of showing molds on the tip caps were apparently attacked in a superficial manner.

The 500 embryos planted showed still smaller percent of infection. There were only 11 infected, or 2.2 percent of the kernels, representing 6.6 percent of the ears. The following fungi were found in these embryos:

<table>
<thead>
<tr>
<th>No. Kernels</th>
<th>Fungus</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Diplodia zeae</td>
</tr>
<tr>
<td>1</td>
<td>Coniosporium gecevi</td>
</tr>
<tr>
<td>1</td>
<td>Gibberella saunbinetti</td>
</tr>
<tr>
<td>3</td>
<td>Fusarium moniliforme</td>
</tr>
<tr>
<td>5</td>
<td>Cephalosporium acremonium</td>
</tr>
</tbody>
</table>
A characteristic moldiness often found at the tip of germinating kernels. The mold within the perion of the germination test does not increase much beyond this amount. *Cephalosporium acremonium* manifests such symptoms on germinating kernels.
Fig. (28) Fusarium moniliforme may manifest itself by such slight molding of the kernel tips.
Fig. Purplish discoloration of kernel tip at beginning of germination. *F. moniliforme* has been obtained from such kernels.
Fig (30) Advanced stage of Fusarium development on kernels on germinator.
As a check on the 100 ear sample, the tip caps and embryos of the 26 clean ears above described were also planted on agar plates. Three kernels from two of these ears grew *Fusarium moniliforme* from the excised embryos. This is 2.2 percent of the kernels studied. Nine of the tip caps from these showed mold. One showed *Cephalosporium acremonium* and *F. moniliforme*. This is 6.9 percent of the total kernels.

The results on the 26 clean ears closely coincide with those on the 100 visibly moldy ears. Either in the experiment at hand the amount of fungus infection found to be considered as due to faulty technique or the conclusion to be made that seed corn generally contains a certain amount of fungus mycelium in tip cap or embryo. The later is perhaps the real situation.

It is further suggested by the above data that infection of the corn kernels is one of degree. If given sufficient time for invasion, the fungus may advance as far as the embryo. Where the ear dries or matures shortly after the fungus enters, only the tip cap or seed coat is affected.

Where the former condition prevails the kernels appear weak or dead on the germinator. In the latter case slight molding of tip caps is manifest on germinator and the suberized layer between tip cap and embryo in most instances prevents serious injury.

Such heterogeneous molds as occur on the surface of the kernels cause little injury flourishing only where abrasions of seed coat expose the endosperm.
SUMMARY

The study of the dry rot disease of corn caused by Diplodia zeae shows it to be a prevalent disease in Iowa, resulting in losses, the past two seasons, ranging from 3 to 15 percent of the ears at harvest and a 11 percent damage to the seed corn. The loss in stand from diseased seed in many fields amounted to 15 percent. A still further loss results from nodal infection and weak plants grown from slightly infected seed.

Infected seed either does not germinate at all or produces weak plants. However, the fungus is not systemic and does not grow into the plant from infected seed.

Infected is local not systemic. The fungus may attack the plant through the silk and tips of ears but the nodes and the ear shanks are the chief points of attack. At the time of flowering the leaf sheaths become loosened from the stalk forming a pocket for the collection of pollen, moisture and spores of D. zeae. Within this cavity the fungus attack the sheaths, and nodes. Under conditions of excessive moisture within the sheath, sugar is excreted. The presence of this sugar may also favor dry rot infection. The fungus may spread readily in seed corn while it is curing or in corn cribs in damp, rainy weather.

Heavy rainfall at the end of the growing period very materially favors the development of Diplodia.

The temperature for the growth of the mycelium of Diplodia zeae on media is as follows: minimum about 10°; optimum 30°; and maximum 35°C. It will not grow in the absence of oxygen.

The use of cellulose by the fungus enables it to readily penetrate a modified rag doll germinator, or to weaken the nodes of the stalk. The use of cellulose moreover explains the capacity of the fungus to live in the soil on old stubble.
In experiments on roots of seedlings and older plants, it has been possible to produce only slight infection.

Seedling blight caused by *D. zeae* is the result of infection of the ear the previous year. Planting of badly infected seed results in decreased stand and obviously a lower yield. The viable seed from diseased ears produces weak plants. Diplodia infected seed is difficult or impossible to detect except by germination tests.

A long rotation, the early field selection of seed, and seed germination tests in the spring are probably the most practical means of control.

Seed corn is affected by molds other than *D. zeae*. In the experiments at hand, 17 fungi occurring on germinating seed corn have been identified and listed. Thirteen of these fungi are superficial on the kernel.

Four of the fungi occurring on germinating seed corn are deeper seated. Diplodia zeae, Gibberella sambinetti, Fusarium moniliforme, Cephalosporum acremonium, penetrate the tip cap and to a lesser degree the embryo itself.
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