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SOME RECENT CONTRIBUTIONS TO OUR UNDERSTANDING OF CORN DISEASES

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There is probably no other disease problem of greater significance economically than that of corn. Hence, the purpose of this paper is to sketch briefly the progress that has been made in this study and to point, in a general way, to some phases of the problem that are immediately before us. Because of its complexity and the misconception as to its significance, a beginning in this field was tardy. Our method of attack on the corn problem seems to be passing through the characteristic successive stages of investigation. These stages may be traced as beginning with observation of the significance of the disease, and the causal relationships, followed by intensive biological investigations and field studies. My discussion of the corn-disease situation will follow this order and I hope you will bear with me if I refer frequently to work done in our own laboratory.

SIGNIFICANCE OF CORN DISEASES

Previous to 1910, corn smut was practically the only important corn disease generally recognized, and our knowledge of this disease came largely from the labors of Brefeld (4) in the late seventies of the last century. In other words, the period of years between 1880 and 1910 was not productive so far as increasing our knowledge of corn diseases was concerned. So little was done during that period of three decades that corn came to be looked upon as a crop strikingly free from plant diseases. This was probably because of two conditions operating together: first, the ever-expanding corn belt; and, second, the limited number of workers in the field of plant pathology, most of whom were interested in crops other than corn.

The period between 1879 and 1909 constituted a very unusual period in American agriculture. This span of 30 years brought us an increase of over 36,000,000 in the corn acreage of the United States, or an increase in acreage almost equal to the combined areas planted today in the six leading corn states; namely, Iowa, Illinois, Nebraska, Missouri, Indiana, and Kansas. The expanding corn belt was continually placing corn on new land, and there was little

need for continuous cropping or short rotations. Moreover, during the three decades referred to, our task consisted of cultural problems, such as that of finding suitable varieties, curing the seeds, and determining the rate and distance of planting.

There is some evidence to show that all was not well with the corn crop from the standpoint of disease in the eighties of the last century. This period takes us back to the beginning of plant pathology in this country. Professor Burrill (6), one of the early outstanding plant pathologists, gave us this interesting record on corn diseases in 1889, "From observations now made, it appears that the disease is a very prevalent one and probably has existed during the time that corn has been grown on the continent." In this same article, Burrill records that he found "dwarfed plants, plants with brown nodes, plants with the base of the stalk badly affected, and plants where the leaves as a whole were blighted. Very often the ears infected with the bacterial disease became moldy. The husks are packed full of a close, very white, felt-like fungus. Possibly in some cases the fungus enters alone."

Although we may not choose to accept Burrill's explanation as to the cause of the condition he saw, we cannot question the accuracy of his observations. There were also rumblings concerning corn diseases in another part of the corn belt. In Nebraska, Billings (3), a veterinarian, was busy in 1888 trying to find why the farmers' cattle were dying when allowed to graze in corn fields after harvesting the crop. This lead to his description of the "corn-stalk disease of cattle." Billings' work did much to stimulate plant pathologists to look into the corn disease situation. Heald (11) in 1906 and 1908 records that his work in finding and describing *Diplodia zeae* (Schw.) Lev. was aided by the interest aroused by a case of poisoning of horses pastured in corn fields.

One year later Burrill and Barrett (7) brought forth still further evidence showing that *Diplodia zeae* and certain Fusaria were prevalent in Illinois. They estimated that in 1906 the loss was over $15,000,000 in the State of Illinois. That there were diseases, and that they were taking their annual toll, during this period of marked expansion of the corn acreage of over one million acres per year, is quite certain; yet the opinion prevailed that either diseases in general were not present or were of nominal importance to the corn crop. It is probably safe to assume that many corn diseases followed the westward march of the corn crop, and our lack of knowledge of these diseases was because of the limited number of workers.

However, between 1910 and 1920, there dawned gradually and generally among pathologists, agronomists, and some growers, a realization of the importance of the damage being done to corn. Pammel, King, and Seal (25) in 1915 called attention to a "corn-stalk and ear-rot disease" caused by a Fusarium on the stalks, roots, and ears. The loss in Iowa was estimated in the neighborhood of $15,000,000 in 1914. In 1918, Hoffer and Holbert (14), working in Indiana and Illinois, described a destructive "root-rot disease due to species of Gibberella, Fusarium, Verticillium, Rhizopus, and Pseudomonas as in a great measure responsible for missing hills, slow growing, barren, down stalks, and early blighted stalks. Also that barren stalks and stalks bearing nubbins only seem to be correlated with certain pathologic conditions in the plants and that the rate of seedling development usually referred to as 'vitality' is not a criterion for assuming freedom from infection of the seed by bacteria and Fusaria."

These excellent observations by Burrill (5), Heald (11), Heald, Wilcox and Pool (12), Burrill and Barrett (7), Pammel, King and Seal (25), Hoffer and Holbert (14) also served a useful purpose in arousing interest, although they left much to be desired in the way of a clear understanding of the significance of the corn disease situation. The development of a clearer vision did not take definite form until the beginning of the present decade. In 1920 Holbert (15) ventured the statement that "Those in close touch with the situation feel that these rots are cutting the yields of corn in the state fully 15 per cent." Again, Holbert et al (16) writes that "on the basis of all data reported in this bulletin, as well as on the basis of observations made throughout Illinois for a period of years, the authors feel that where inferior and infected seed is used, losses to the corn crop from diseases, including smut and rust, can very conservatively be placed at 20 per cent." This means that in Illinois alone, the losses amounted to over 76,000,000 bushels, or nearly one-half as many bushels as the total crop produced in the State of Ohio the same year. In Iowa reports of the years 1921-1927 showed that four to ten per cent of the crops were destroyed by Diplodia and Basisporium. The loss caused by these dry rots amounted to an average yearly loss of over 32,000,000 bushels. If all corn diseases were included, doubtlessly, this figure would be doubled. Although the losses in only two states are cited, it should not be assumed that the ravages caused by corn diseases are not general in the middle west and probably wherever corn is grown. The logic of this statement is supported by the

condition in the East. Manns and Adams (22) wrote that "we have been estimating the annual losses from corn diseases in Delaware from three to five per cent. Our recent studies show these estimates are low and that 15 per cent was approximately the losses for the year 1920." If this is true for Delaware, the condition cannot be markedly different in the other eastern states. Less is known about the corn disease situation in the southern states, but there is every reason to believe that definite information from the south is lacking, as well as in the more intensive part of the corn belt. The estimated reduction in yield in the whole United States in 1918 was 158,530,000 bushels and, in 1927, 267,553,000 bushels. The latter is an amount greater than was grown the same year on the total acreage in the states of Indiana, Wisconsin, Pennsylvania, and West Virginia. I do not believe that corn diseases materially increased during the period from 1918 to 1927. I believe the increase of 100,000,000 bushels in the loss estimates, is owing to our increased realization of the significance of the corn disease situation.

To deal in estimated losses, constitutes little contribution to the corn disease problem. The only excuse that I offer for their citation is to make clear the change in conception held earlier, that corn was a crop strikingly free from diseases except for corn smut and some minor troubles, has been thoroughly uprooted.

Causal Relationship

During the past decade splendid progress has been made on the causal relationship of corn diseases. Many contributions have been made in this direction by Burrill (5) and others. The first of these diseases to be unraveled was that caused by *Diplodia zeae*. Although Heald, Wilcox and Pool (12) and Burrill and Barrett (7) proved its pathogenicity, its pathological effect on the corn crop was seriously neglected until Durrell began his study in 1920. Durrell (9), (working in my laboratory), succeeded in showing that infection was local, rather than systemic, and that it did not extend from the roots up through the vascular bundles into the ear as believed by Smith and Hedges (31). Neither Heald, Wilcox, and Pool (12) nor Burrill and Barrett (7) mention any relation or damage of Diplodia to the seed. Durrell (9) found 11 per cent of the seed in two sections of Iowa unfit for seed because of Diplodia infection.

Knowledge of the percentage of infected seed placed new and greater emphasis on the Diplodia dry rot fungus. Hitherto, it had

been considered as damaging chiefly the ears and stalks. Now it became evident that such seed might produce weak plants or seedling blight, but Durrell did not believe the fungus traveled from the kernel up into the seedling. His point of view was that the organism inhibited the development of the seedling by usurping its food supply, thereby causing the seedling to die because of lack of food rather than fungus invasion. Holbert and his co-workers (16) first showed clearly how the Diplodia dry rot fungus migrated to the mesocotyl and roots, causing the characteristic lesions. However, they did not believe that the fungus advanced up the stalk from the crown. In this connection it should be pointed out that Raleigh (27) found Diplodia lesions extending three inches above the crown when the corn was in the milk stage, and that such plants were dead before the crop had passed out of the milk stage. Furthermore, Holbert, et al (16) showed that in field trials extending over a period of four years the use of diseased seed (as compared with healthy) might reduce the yield more than 50 per cent, depending upon the date of planting and the soil temperature and moisture early in the spring. These same investigators have shown that corn grown from Diplodia-infected seed suffers when planted early in cold soil. This would seem to imply that the organism is most active at low temperatures. However, Raleigh (27) has shown, through the use of visible-root sand-cultures held at different constant soil moistures, that Diplodia invasion of the young growing plants increased with the rise in temperature.

The next disease shown to have a detrimental effect on the corn crop was Basisporium dry-rot caused by *Basisporium gallarum* Moll. Its presence on the corn crop in this country was reported first by Artzberger (1) in Ohio. Artzberger was able to culture the fungus, but was unable to obtain infection on living young or old plants. The fungus attacked sterilized green ears or moist crib corn causing decay of the cob. Artzberger felt, however, that Basisporium was a saprophyte and would not in any way injure growing corn.

Durrell (10) first showed that Basisporium might attack the shanks, husks, stalks, and ears, causing a rapid destruction of the parenchymous tissue, and that the fungus might destroy all or only slightly injure the ears. He showed also that Basisporium dry-rot on the ears caused the kernels to be shriveled and loose, the ears light, and the cob brittle. Basisporium was found to be inconspicuous on the kernels and frequently such infected ears found their way into the selected seed. In such cases, the stand

might be reduced the next year and many of the young plants weakened. Moreover, he found that heavy precipitation in August and September, when the crop was maturing, favored the development of the disease. Although Durrell obtained evidence showing that Basisporium dry-rot is a destructive disease on corn, yet at no time was he able to induce artificial infection under controlled conditions, nor was he able to definitely measure the amount of injury caused where slightly infected kernels are used for seed.

Doctor C. S. Reddy, also working in my laboratory, has recently demonstrated the effect of Basisporium growing in infected seed. He has found that when viable seed infected with Basisporium is placed in cold soil, the seed is destroyed by the Basisporium organism. Similar viable seed, treated with several of the well known commercial corn dusts and held as checks, produced comparatively normal plants, thus establishing definitely the pathogenicity of Basisporium in relation to the seed. The symptoms of Basisporium dry-rot on the corn plant are quite unlike those of Diplodia dry-rot and yet the effect on the stalk, ear, and seed is similar.

The splendid work of Reddy and Holbert (30) aided materially in clearing the atmosphere regarding the cause of some of the symptoms ascribed by Burrill (6) to his bacterial disease and by Hoffer and Holbert (14) to their so-called root rot. These writers found that the black bundles occurring in corn was caused by a different organism. Reddy (28) showed that numerous isolations from the blackened bundles gave consistently one organism, and pure culture inoculations proved it to be *Cephalosporium acremonium* Corda.

Reddy and Holbert (30) record that "the most distinguishing symptom of this disease is the presence of blackened vascular bundles in the talks and sometimes in the leaves." Associated with the disease to a notable extent are the following abnormalities: excessive sucker production, prolific stalks, manifestations of which are a tendency for ear development at many nodes or multiple-ear production at one node; a certain type of reddening or purpling of the leaves and stalks; stalks with aborted ears (barren), near Urbana where Burrill made his early studies on corn diseases.

Probably the most conspicous ear-rot disease of corn is caused by *Gibberella saubinetii* (Mont.) Sacc., yet its causal relationship was not established until 1918. Its presence on corn had been noted many times, but it remained for Hoffer, Johnson, and Atanasoff

(13) to report definite experimental evidence as to its causal relationship. The organism may be a virulent parasite on the corn roots. In 1920, Holbert, Dickson, and Biggar (17) reported that in their field inoculations the "germination was lowered, early growth retarded, storm resistance decreased, relative vigor through the season reduced, and average grain production lessened." They recovered Gibberella from the plants showing the above symptoms, especially in the seedling stage. In 1924, Koehler, Dickson and Holbert (21) carried out extensive field trials using seed dipped in spore suspensions and planted on good corn land, that had previously been in blue grass sod. They found that the inoculated seed gave a reduced stand, reduced number of vigorous plants, and a reduced yield in every plot. The discovery of the foregoing diseases is of comparative recent date when we think of corn smut.

Corn smut (*Ustilago zeae* (Beckm.) Unger) has been with us in a destructive form for a long time and bids fair to continue. The control of this disease seems remote, the outlook is not bright. It is true, we know the life history of the organism through the excellent early studies of Brefeld (4), but he left much to be desired regarding the factors influencing infection, such as moisture, temperature, morphology of the host, and relative susceptibility of different varieties and strains of corn. The nature of the problem confronting us in the case of corn smut will receive further consideration in a later chapter. There are several minor diseases of corn that need to be mentioned in passing, not because of their economic importance today, but because they have the capacity to become so under favorable conditions.

The first is *Physoderma zeae-maydis* Shaw which was so thoroughly investigated by Tisdale (32), and shown to be most destructive in the southern part of the corn belt and of little or no importance, at least at the present time, in the more intensive part of the corn belt. The second organism is *Pythium arrhenomanes* Drechs., studied by Johann et al (19). Whether this is the same Pythium reported earlier by Valleau, Karraker and Johnson (33), the literature does not say. This organism is said to manifest itself in any of three different ways: first, as a rot of the embryo, killing the seed; second, as a seedling blight attacking the plant after it is up; and, third, as a root rot causing a reduction in the size, vigor, and yield of the maturing plant. This fungus shows a marked preference for the tips of the small feeding roots and it flourishes at a low temperature. What importance this disease may assume under changed cropping conditions cannot be predicted but it is

safe to say that a Pythium preying on the roots of a plant needs watching. The third organism is *Sclerospora graminicola* (Sacc.) Schraet., first reported on corn by Melhus and Van Haltern (24) in this country. It has been shown, that this organism which occurs normally on *Setaria viridis* (L.) Beauv. may attack corn, also that there is a marked difference in the susceptibility of strains and varieties. The serious condition caused by species of Sclerospora on corn is known from losses in the Orient. Other organisms that we now consider as causing minor diseases include *Pseudomonas holci* Kend., described by Kendrick (20), and species of Helminthosporium, studied by Drechsler (8). Still others might be mentioned. The fact that these diseases do exist in the corn belt, fortunately only to a nominal extent, at least as far as we know now, should serve to keep us on our guard. New varieties that may be introduced may prove extremely susceptible and confront us at any time with a serious problem. This should not be accepted merely as over-emphasis, because we know it has happened repeatedly in the past. Moreover, the destructiveness of the Phycomycetous parasites under favorable conditions cannot be over-emphasized.

INTENSIVE INVESTIGATIONS

It is a natural sequence in the study of a disease or the diseases of any particular crop, first, to survey the whole field and conceive the significance of the problem economically and biologically, before intensive investigation on the significant phases of the problem can begin. However, before discussing the more intensive investigations, it might be asked, what practical relief has come to the corn grower up to this time. The most important contribution is probably the clear definition of the different diseases, their presence in and on the seed, and their seedling blight stages, together with the conditions that foster or retard their development. This work has paved the way and made possible the only directly practical relief extended to the corn grower so far — that of seed treatment. This came as the natural first step after it had been shown that the seed might carry the pathogene, not only on its surface but also inside in the mycelial stage.

Probably the first constructive work on seed treatment in this country was done by Reddy beginning in 1922. This work was significant in that he was using some of the organic mercury compounds. Reddy's (29) preliminary results were published jointly with Holbert in 1924. To be sure, seed treatment of corn was not a new idea. Treatment of seed corn was recommended in this

country as early as 1889 by Bessey (2) of Nebraska. It was recommended for the control of corn smut, but it was soon found that the practice was not well founded. It was only when the organic mercury products began to become available that the significance of corn seed treatment showed real promise. Although seed treatment has rapidly come to find a place in the practice of corn growing in some states, as Iowa, Illinois, and Indiana, yet in others as Nebraska, Kansas and Colorado the practice is doubtful and probably unwarranted, at least in the light of the experimental data that has appeared. So far it seems seed treatment has found greatest favor in the more humid and more intensive part of the corn belt. It is not improbable, however, that when we have determined more accurately the minimum stand, returning the greatest average yield over a period of years that seed treatment may prove worthwhile in the drier part of the corn belt. Its chief value, as far as we know now, seems to lie in the prevention of seed decay and seedling blight. At least experiments by Melhus, Reddy, Raleigh and Burnett (23) and Holbert, Reddy and Koehler (18), and Raleigh (27) in the laboratory and in the field in many places, have clearly shown that diseased seed under somewhat unfavorable growing conditions is benefited most. This suggests that the presence of the disinfectant may serve two purposes: first, to penetrate the seed coat, and, second, to prevent the attack and invasion of the seed by soil-inhabiting microorganisms.

It is fully appreciated that the control of seedling blight, valuable as it may be, leaves much to be achieved in meeting the corn disease problem adequately. Corn that has successfully passed the seedling stage still has a long way to go before it can mature its golden grain. We know little about the organisms that prey on the corn plant during these later stages of its development. It is probably safe to say, that this phase of the corn disease problem has scarcely been scratched. Our work, so far, on the diseases attacking the corn plant after it has passed the seedling stage, merits comparatively little consideration. We have, it is true, studied the biology, at least to some extent, of the organisms concerned. Some investigators have tried to follow the effect on the plant in the field, but always under mixed cultural conditions, which, at best can serve only as experiments pointing the way, rather than actually supplying definite proof. In other words, our understanding of the biology of the organism preying on the corn plant after it passes the seedling stage is shamefully weak. A few

examples should make my point clear. We probably know as much about the biology, pathogenicity, and field response of *Diplodia zeae* as of any other organism attacking corn, yet we know only one stage in the life history of Diplodia. Certainly, the possibility of a perfect stage still looms up before us, and if there is one, then a whole volume about Diplodia may be closed to us today. In the same way, we know that it can and does lead a saprophytic existence, but we do not know whether it feeds on the soil refuse or merely lives in the soil. We do not know how long it may live, nor do we know the conditions that permit it to flourish or die out. Moreover, we know that it attacks the roots, and even moves up on the crown, but there our knowledge stops. We know little about how the organism makes its attack on the underground parts of the host, and nothing about the effect of ecological influences, soil type, and the interaction of the organism and the corn plant.

This lack of a clear understanding of Diplodia stands as a challenge to every pathologist in the corn belt states. An understanding may grow from an extended study of the biology of the parasites, and the development of a technique adequate for testing the relative resistance of our existing varieties and the many additional varieties that are being created through the elaborate corn breeding program under way in several centers. With an increase in our knowledge of these problems it is certain that investigators in other fields will need to take cognizance again of many agronomic problems, such as rate and time of planting, rotation, organic matter and fertilizers, as they may be influenced by the parasitic organism complex existing in our corn soils. The happiest solution is to form a research program broad enough to carry on simultaneusly its several phases. Unfortunately, in spite of much ado about coöperation and coördination, during the past decade little or no progress has been made in this direction on corn studies.

If we understand little of the biology and pathogenicity of *Diplodia zeae*, we understand less of the corn smut fungus — the corn disease that we have probably known longest. Although Brefeld's infection technique was adequate for working out the life history, it has not proven adequate for measuring the relative resistance of different strains of corn. Likewise, the practice consisting of injecting a spore suspension hypodermically has proved inadequate, in that it does not give a true measure of the relative resistance of different strains. Platz (26) has recently called attention to the inter-dependence of infection and the morphology of the young corn plant. His extensive negative evidence bearing on the

infection of the lateral buds by dropping viable spores back of the leaf sheath is also very suggestive. Recent studies in my laboratory by John Trumbower and Glen Davis tend to show that lateral bud infection takes place not by spores dropping in behind the leaf sheath, but by spores entering through the spiral whorl. At least it is certain that delayed lateral bud infection can be induced in this way under controlled conditions. The explanation of the results cannot be definitely given. Possibly the mycelium from such spores either fails to develop before the lateral buds start rapid growth or the mycelium enters the lateral bud primordia and becomes inactive with the temporary cessation of their development. How infection takes place in the axillary buds is not answered, and stands in the way of progress. The finding of mutants and heterothallism has shown to some extent why plant breeders have made little progress along this line and emphasizes the necessity for a large amount of intensive work on the biology and parasitism of the parasite, before substantial progress can be made in hybridizing resistant strains of corn. The same is true with the other organisms attacking corn, such as *Basisporium gallarum, Gibberella saubinetii, Cephalosporium acremonium, Fusarium moniliforme* Sheldon and others. We need intensive biological studies involving each pathogene, studied by itself under controlled conditions in all its variability, pathologically and physiologically, and its response to environmental conditions in soil media varying in texture, organic matter, water-holding capacity, and acid reaction.

In order to be able to do these things, we must have a clearer understanding of the normal functions of the corn plant. What is the response of the corn plant growing in a soil medium free from the various parasites? In other words, we know too little of its normal physiology. Does it stand up, lodge, break over, produce barren stalks, or show decayed roots under such conditions? When we can answer these questions about the response of the plant growing in the absence of corn parasites, and can make use of the uniform plant material such as is available in many pure lines, we should be able to accumulate data of real value. The pure lines that are used in pathological studies should be kept, and be available in large enough quantities so that they can be obtained over a considerable period. Too much of our work has been done utilizing many different pure lines, none or only a few of which are available two years later. The published data in such instances cannot be repeated and frequently is almost lost to progress. Sufficient progress has been made in corn genetics to clearly emphasize its

role. In fact unless genetics is utilized much of the ground will need to be reworked.

Anyone at all familiar with corn diseases knows that following the earing stage the corn plant appears to be most susceptible to parasitic invasion. Before much progress can be made in the direction of a clearer understanding of the disease problem in this stage of the corn plant, we must know more about the translocation phenomena in the corn plant, induced by the filling of the ear. What is the substance or substances lost to the leaves, roots, and stalks that prevents parasitic invasion earlier? On the other hand, it may not be entirely a physiological change in the plant, but possibly a direct effect of the environment, manifesting its effect at this particular stage of the corn plant. By environment, I have in mind not only the physical elements, but also the relation of one plant to another and of different hills to one another. It is conceivable that the rate of planting as measured in terms of yield does not give us the right number and distribution most conducive to the normal development of the plant. Ecologists should be encouraged to take up this problem from the standpoint of root competition, light utilization, moisture requirements, and limitations, and leaf area development. The availability of such data is essential to a clear understanding of the pathological effect of the corn pathogenes working individually and collectively.

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