ECOLOGY OF CORN STALK ROT IN NEBRASKA

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Ecology of Corn Stalk Rot in Nebraska

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INTRODUCTION

Corn stalk rot is often referred to as the most destructive corn disease in Nebraska. Although stalk-rotting fungi can always be found in all corn growing areas, the losses caused by these pathogens vary from field to field and year to year. Stalk rot is usually more of a problem in the Central and Western irrigated areas of the state than in the dryland corn areas in the East. It may cause $10-30 million losses annually.

In Nebraska Fusarium moniliforme and Cephalosporium sp. are thought to be the most frequent causes of corn stalk rot. Early workers felt these two fungi were seed-borne and possibly grew systemically through the plant. However, these assumptions are not accepted by many plant pathologists today. If inoculum is not carried in the seed, it must come from the soil or the air or both. Despite much research it is still controversial just when and how infection occurs. The disease is thought to attack only mature plants, yet the pathogens can be isolated more or less frequently in all stages of plant growth.

Corn stalk rot has been studied in corn-producing areas for over 50 years, yet it is still poorly understood. This research was undertaken to determine why corn stalk rot is more of a problem

in irrigated than in dryland corn in Nebraska, and to ascertain the influence of environmental factors on the incidence and severity of the disease. The likelihood of systemic infection from seed and soil-borne inoculum was investigated in both controlled and natural environments. An attempt was made to determine if the rate of death of pith tissue is a factor in predisposing plants to stalk rot fungi in the field.

LITERATURE REVIEW

Corn stalk rot caused by Fusarium moniliforme Sheld.(Gibberella fujikuroi (Saw.) Wr.) is found in many corn-growing areas of the USA and in many countries throughout the world. The fungus was first described on moldy corn ears in Nebraska in 1904 (33). In the next 30 years it was found in diseased corn stalks in more than 20 states. Pammel, King, and Seal (22) reported the fungus occurred abundantly in the endosperm and embryo of corn kernels. They suggested the pathogen was largely spread by infected seed. Selby (32) suggested that the fungus grew from the roots through the plant to infect the kernels. Valleau (35) found the fungus in over 50% of the surface sterilized seed examined. He isolated it from over 300 ears collected in eight states and noted that red or black discolorations in corn seed coats before or after germination indicated infection with F. moniliforme. However, absence of discoloration did not necessarily mean the seed was not infected with the pathogen. Edgerton and Kidder (6) stated that 44% of the kernels in 52 samples from Louisiana contained the pathogen. Branstetter (5) consistently observed the fungus beneath the pedicels of kernels from infected ears in Missouri.

In 1923 Manns and Adams (17) observed F. moniliforme under the pedicel and suggested that the pathogen grew systemically from the kernel through the plant. Cross and longitudinal sections through the germ end of infected kernels showed the mycelium in the pedicel. Removal of pedicels before surface disinfecting kernels increased germination and decreased infection. Valleau (35) noted that seed infection did not affect germination and only slightly affected plant vigor. Edgerton and Kidder (6) found a slight increase in germination and in stand in field trials with uninfected kernels. Holbert and co-workers (11) transplanted diseased and apparently disease-free seedlings from the germinator to the field. Disease-free seedlings grew more rapidly for the first 25 days, but differences were not noticeable after that. Plants from disease-free seedlings produced more grain than plants from diseased seedlings. Kernels from relatively healthy ears from severely diseased plants were inferior to comparable kernels from healthy plants.

Manns and Phillips (18) reported that greenhouse corn grew better in soil from a corn field with a great deal of stalk rot than from a corn field relatively free of the disease. Plants grown in steam-sterilized soil inoculated with F. moniliforme were not markedly different from control plants. However, Valleau and co-workers (36) showed that more root decay occurred in greenhouse corn grown in soil from a field in continuous corn for 11 years than in a soil from virgin forest. No differences

in growth rates were noted 80 and 93 days after planting. They proposed that this corn root rot was caused entirely by soil-inhabiting organisms. In untreated soil the fungi did not incite seedling blight but caused root rot of plants nearing maturity. Fungus-free kernels and kernels infected with F. moniliforme were planted in sterile sand and watered with nutrient solution. The primary root system was destroyed by the pathogen but the secondary root system remained unblemished. The roots of plants grown from uninfected kernels showed only an occasional lesion. When rotted corn roots were added to the sand, the plant root system was almost completely destroyed.

Voorhees (38) described and photographed the growth of F. moniliforme through the tissues of both naturally and artificially infected corn seedlings. Observations on naturally infected kernels fixed and sectioned through the cotyledonary plate region shortly after germination showed the fungus was well established. He concluded that the fungus entered the seedlings from the soil through wounds or by direct penetration. He did not section and study naturally infected, ungerminated kernels. Young plants grown from infected kernels or attacked by the fungus present in the soil were indistinguishable from uninfected seedlings. However, as the season advanced the effect of seedling infection was reflected in reduced stand, vigor and yield.

Leonian (15) placed germinated corn seedlings in petri dishes of F. moniliforme growing on Pfeffer's nutrient agar. After 50%

of the roots were killed the seedlings were transplanted to the greenhouse or field where they grew as vigorously as the controls. Thus, he and many other workers (5,6,35) concluded that F. moniliforme was only a secondary invader of corn roots and not an important stalk rot pathogen.

Little interest was shown in the seed-borne aspect of corn stalk rot after 1934 until Foley (9) re-opened the controversy in 1962. He found some seed lots in Iowa that did not yield F. moniliforme when kernels were germinated in various media at room temperature. However, nearly 100% of the kernels from those seed lots yielded F. moniliforme when they were germinated on various media at 10-15 C and the seedlings aseptically cut into small sections, crushed, and incubated on mineral agar. He grew seedlings from infected kernels in aerated mineral solution for 28 days before symptoms appeared. However, weekly isolations from roots always yielded F. moniliforme. The fungus was also frequently isolated from roots, nodes, and internodes of apparently healthy field corn. He concluded that the presence of F. moniliforme in kernels and many stalk tissues without symptom expression indicated systemic infection. This theory was recently challenged (14).

Manns and Adams (16) reported Cephalosporium sp. was observed beneath the pedicels of many corn kernels not showing external rot. In inoculation experiments, however, (18) the fungus caused no apparent injury to young corn plants. The fungus was referred to

as C. sacchari Butler and Khan (16), but Reddy and Holbert (30) identified it as C. acremonium Corda, the cause of black bundle disease of corn. The latter workers described purpling, excessive tillering, barrenness and black vascular bundles as characteristic symptoms of the disease. They also observed the fungus under the pedicels and growing from the pedicels in germinating kernels. They theorized that the pathogen developed with the germinating kernel, and caused a systemic infection through the vascular system, eventually invading the ear.

Root inoculations with C. acremonium are reported to cause black bundles in stalks (1). Stalk inoculations at various stages of growth led to black bundles in leaves, leaf sheaths, and stalks. Whorl inoculations were unsuccessful. Results from other research indicate that the black bundles are inherited in certain kinds of corn or caused by environmental conditions (10).

Other organisms found in association with corn stalk rot include Gibberella zeae (Schw.) Petch; Diplodia maydis (Berk.) Sacc. ; Pyrenochaeta terrestris (Hans) Gorenz, J. C. Walker, and Larson; Trichoderma spp.; Nigrospora spp.; and bacteria. Peterson (29) frequently isolated Trichoderma sp. from corn stalks and with toothpick inoculations he demonstrated that it caused tissue decay. Though certain bacteria do cause soft stalk rots (13), the role of bacteria in dry stalk rot is unknown. They are assumed to be of minor importance.

Environmental and cultural conditions seem to affect the incidence of stalk rot. Many workers (21,34) have shown that the N:K fertility balance is critical. Either a nitrogen excess or a potassium deficiency tends to increase stalk rot. High plant population (20,000 plants/acre or more) often leads to more stalk rot (20). Late-maturing corn is not attacked as severely by stalk rot as early-maturing corn (39). In Minnesota more stalk rot was observed in inoculated plants on flooded land than on non-flooded land in a season with ample rainfall (19). However, plants inoculated during periods of insufficient soil moisture were readily killed. In greenhouse experiments more rot occurred at 30 than at 18 C (19). In Ohio stalk rot was reduced and yields increased by planting corn after soybeans in a rotation including oats, wheat and alfalfa as compared to continuous corn after corn (42).

Some inherent qualities of the corn plant determine its susceptibility to stalk rot. In Ontario, Canada it was possible to find root rot without stalk rot, but all rotten stalks had root rot (39). It was suggested that stalk rot resistance resides in the roots. If the disease did not develop on the roots, the stalk remained healthy. Barren stalks and high-yielding stalks were less likely to be damaged by stalk rot than intermediate-yielding stalks (20).

Pappelis discovered that Diplodia maydis and Gibberella zeae invasion in corn stalks is delimited by living tissue (23,25). Hyphae and rot were only observed in dead tissues. He found a highly significant correlation between rate of death of internode tissues and stalk rot with internode inoculations. Pith tissues in upper internodes died prior to pith tissue in the first internode. Simultaneous inoculations of internode one and four in the same plant resulted in much less rot in the lower internode. In all experiments the discolored, rotted area was restricted by borders of living tissue. Root and leaf injury increased the death rate of pith tissue and the amount of stalk rot (24). Soil fertility (27) and planting date (26) also affect the death rate of pith tissue.

Foley (8) reported that rind softening, resulting in loss of stalk strength, was the most frequent effect of natural infection, and thus the best available indication of inherent susceptibility. Inoculation of D. zeae or G. zeae into corn stalks did not cause general stalk disintegration. He noted the primary result of inoculation is discoloration in the immediate vicinity of inoculation loci. Such discoloration did not occur in unwounded diseased plants. Stalk diameter; thickness of rind; ratio of rind thickness to stalk diameter; total number and number/unit area of vascular bundles in the rind, in the pith, and in the stalk; and percentage of sclerenchyma sheath fibers/vascular bundle in the rind and per unit area of the rind are not correlated with stalk strength (2).

MATERIALS AND METHODS

Media and nutrient solutions

The following media were used. Amounts given are per liter of distilled water.

Water agar - 20 g of agar

Potato dextrose agar (PDA) - 20 g dextrose, 15 g agar, and the infusion from 200 g peeled, steamed potatoes

Corn meal agar (CMA) - 20 g corn meal and 15 g dextrose

Czapek - Dox agar (CDA) -

Agar	15 g
$NaNO_3$	2 g
K_2HPO_4	1 g
$MgSO_4 \cdot 7H_2O$	0.5 g
KCl	0.5 g
$FeSO_4 \cdot 7H_2O$	0.01g
Sucrose	30.0g

Mineral agar -

NH_4NO_3	0.004 M
$CaCl_2 \cdot 2H_2O$	0.0038 M
KCl	0.0038 M
$K H_2PO_4$	0.0021 M
$MgSO_4 \cdot 7H_2O$	0.0017 M
Iron chelate	0.05%
$MnCl_2 \cdot 4H_2O$, H_3BO_3, $ZnSO_4$, and MoO_3	trace
Agar	1.7%

Potato dextrose broth (PDB) - 20 g dextrose and the infusion from 200 g peeled, steamed potatoes

Hoagland's nutrient solution (modified)

A. Macronutrients		cc/liter of nutrient solution
M	$NH_4H_2PO_4$	1
M	KNO_3	6
M	$Ca(NO_3)_2 \cdot 4H_2O$	4
M	$MgSO_4 \cdot 7H_2O$	2