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An in-depth look at the Corn-Colletotrichum graminicola (causal organism of anthracnose) pathosystem

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A pathosystem is an ecosystem in which parasitism occurs. Parasitism of corn by *Colletotrichum graminicola* results in anthracnose, an important disease of corn. The disease has three distinct phases:

1. Anthracnose leaf blight that usually occurs between V2 and V12.
2. Anthracnose top dieback that usually occurs from R4 onwards.
3. Anthracnose stalk rot that is usually prevalent around physiological maturity.

Colletotrichum graminicola was not considered an economically important pathogen of corn until the early 1970s. Today it seems like almost every cornfield has plants infected with the pathogen.

Anthracnose stalk rot reduces yields and costs farmers approximately $750 million dollars each year (Frey et al 2010). In Brazil, Cota et al (2012) reported a 17 to 22% reduction in 100 seed weight of seven hybrids that were evaluated over two growing seasons. Perkins and Hooker (1979) reported yield losses of up to 17% in Illinois. Yield losses are primarily due to premature death of the plant before grain fill is complete, however some losses may also occur at harvest when plants are lodged. It is unlikely that anthracnose leaf blight directly contributes to yield loss since symptomatic leaves usually die and defoliate well before grain fill occurs. The leaf lesions, however, do serve as a source of inoculum for infection throughout the growing season.

The anthracnose disease cycle

A thorough understanding of the disease cycle of a pathosystem can enable disease management strategies to be developed or improved.

Survival

C. graminicola does not produce any survival structures such as sclerotia or thick-walled spores. The fungus survived for 18 months in *C. graminicola*-infested crop residue, but spores and mycelia did not survive more than a few days (Vizvary and Warren, 1982).

Inoculum production and dispersal

At the beginning of the growing season, inoculum is produced from *C. graminicola*-infested surface crop residues. Spores are produced in an acervulus, a saucer-shaped fruiting body that bursts through the epidermal tissues. Long dark needle-like structures (setae) protrude from the acervulus, and spores are coated in a salmon-pink gelatinous matrix. The setae and matrix, which look like porcupines can be seen with a hand lens or a microscope, and are diagnostic for the disease. The matrix is ensures survival and successful dispersal of the spores (Bergstrom and Nicholson 1999). The spores are dispersed by splashing and blowing raindrops and thus only travel short distances. Wind currents disperse dry spore masses (Bergstrom and Nicholson 1999).

Spores produced on anthracnose leaf blight lesions serve as a secondary source of inoculum for leaf and stalk infections.

Infection and colonization

Anthracnose is a polycyclic disease meaning that infection can occur multiple times during the season. The fungus can infect any part of the plant, including the roots, crown, leaves and stalk.

C. graminicola is hemibiotrophic (parasitizes living tissue for a period of time and then continues its life cycle on dead tissue). During the first stages of infection, the fungus establishes a biotrophic relationship with its host that is essential to the success of the interaction (Munch el 2008). Spores require 6 to 8 hours to germinate and

temperatures ranging from 55 to 100F once they come into contact with a corn leaf. Upon landing on a corn leaf, the spore immediately starts to produce an adhesive material that binds it to the leaf. Over the next 15-18 hours, an appressorium develops which enables *C. graminicola* to penetrate the host cell wall. Infection appears to only occur between 75 and 85F. An infection vesicle is formed and primary hyphae grow and scavenge nutrients from the host (Bergstrom and Nicholson 1999). During this period, the pathogen changes the surface of its hyphal cell walls to disguise itself so the host defense systems are not triggered. Between 48-72 hours after infection, the necrotrophic phase of the infection begins. Secondary hyphae penetrate the cell membrane and secrete toxins that kill the host cells (Munch el 2008). The fungus rapidly establishes itself as a necrotroph and colonizes localized host tissues until it invades the xylem vessels. *C. graminicola* lives off of sucrose present in the xylem vessels systemically colonizes the plant (Bergstron and Nicholoson 1999).

Stalk boring insects facilitate infection of corn by providing entry points for the *C. graminicola*, however, Venard and Vaillancourt (2007) recently showed the fungus can also penetrate unwounded stalks, although this route was not as efficient as infection via wounds. In their research, fungal penetration appeared to be influenced by environment and/or genetic variables, but once penetration of the epidermis had occurred, colonization of stalk tissues occurred at a similar rate to colonization that resulted from an infection via a wound.

It has been suggested that spores produced from the leaf blight phase of anthracnose are washed behind the maize sheaths and may directly penetrate stalks. No relationship was found between the incidence of anthracnose leaf blight at V4 to V5 and mean stalk rot severity in a long term rotation study near Ames, IA (Robertson, unpublished). Similarly, Jirak-Peterson and Esker (2011) reported no association between the two phases of the disease. A relationship between the onset of top dieback and stalk rot severity was recorded in an observational trial in Iowa (Robertson, unpublished).

Infection may also occur via the roots of developing seedlings. Sukno et al (2008) used an isolate of *C. graminicola* labeled with the green fluorescent protein to visualize infection of corn roots of an anthracnose-susceptible and resistant hybrid by spores of the pathogen. The fungus successfully infected and colonized the root tissue of both hybrids. Furthermore, the pathogen was able to colonize the vascular system and spread to above ground parts of the plant without causing symptoms. Lipps (1985) however, reported that although root infection of seedlings was detected on roots that had grown through inoculum, the pathogen could not be recovered from the roots of mature plants.

Symptoms

Anthracnose leaf blight symptoms include round to oval, dark brown to purple lesions that occur on the lower 3 to 5 leaves of young plants early on in the growing season. Once canopy closure occurs, it is unusual to the leaf blight phase of anthracnose in Iowa.

Anthracnose top die back can be recognized as yellowing and death of the tops of scattered plants from R4 onwards. If the leaf sheath is pealed back at the top of the affected plant, characteristic back discoloration of the talk should be evident. If conditions are humid or damp, a pink jellylike substance (spore masses) may be present on the stalk. Care should be taken not to misdiagnose top dieback. Death of top leaves may be due to one or more of several factors that include hybrid characteristics, environmental stress, and corn borer damage.

Dark discoloration of the stalk rind and/or dark discoloration and disintegration of pith tissues are characteristic symptoms of anthracnose stalk rot. It a stalk sample is taken and placed on a damp paper towel in a Ziploc bag over night, a pink jellylike substance should form on the discolored tissues.

Management

As with all diseases, resistance should be the cornerstone of any management plan. Resistance to the leaf blight and stalk rot phases of anthracnose are inherited independently and controlled by one to a few major genes and several genes with minor effects. Hybrids differ in their susceptibility to anthracnose stalk rot. The incidence of anthracnose stalk rot and reduction in grain weight varied between hybrids (Cota et al. 2012) suggesting that some hybrids may have resistance to infection, but when infection occurs colonization occurs quickly and impacts yield. Recently the *Rcg1* locus was incorporated into corn hybrids. This locus reduced anthracnose stalk rot development on near-isogenic hybrids containing the *Rcg1* locus compared with those that did not contain the locus (Frey et al 2011). Furthermore, yields of the hybrids were similar under inoculated conditions indicating no fitness cost was associated

with the *Rcg1*. Interestingly, light intensity may affect resistance. Low light intensity associated with cloudy, overcast conditions, increased the relative susceptibility of corn (Bergstrom and Nicholson 1999).

Since infested surface corn residue provides the primary inoculum for disease development, continuous cornfields are at greater risk for anthracnose. Anthracnose leaf blight severity was higher in a corn-corn rotation compared to a corn-soybean rotation in Wisconsin and Iowa (Jirak-Peterson and Esker 2011, Robertson, unpublished). Lipps (1985) reported a 1-year rotation away from corn significantly decreased anthracnose.

Data on the role of tillage in reducing anthracnose stalk rot differs. Jirak-Peterson and Esker (2011) reported a higher incidence of anthracnose stalk rot in chisel-plowed fields that suggests putting infested corn residue into the root zone may increase the risk of stalk rot as a result of root infection by the pathogen (Sukno et al 2008). Lipps (1985), however, reported buried residues were not a considerable source of inoculum for stalk rot. In Lipps (1985) field studies in OH, the incidence of leaf blight and stalk rot were greater ($P=0.05$) when surface residue was present and correlated with distance from the residue area ($P=0.05$).

Although foliar fungicides do not directly affect the anthracnose pathogen, theer may be some indirect effects. Foliar fungicide applications during grainfill reduced the incidence of top dieback (Robertson et al 2008) and stalk rot incidence (Shriver and Robertson 2009), although there was no evidence of an effect on yield ($P<0.1$). There are many more reports, however, in which foliar fungicide applications had no effect on either top dieback or stalk rot. Foliar applications made at V5 to V6 are too late to affect anthracnose leaf blight development, and the jury is still out on their effect on anthracnose stalk rot development.

References

Bergstrom, G.C. and Nicholson, R.L. 1999. The biology of corn anthracnose. Plant Dis. 83:596-608.

Cota, L.V., da Costa, R.V., Silva, D.D., Casela, C.R. and Parreira, D.F. 2012. Quantification of yield losses due to anthracnose stalk rot on corn in Brazilian conditions. J. Phytopathol. 160: 680-684.

Frey, T.J., Weldekidan, T., Colbert, T. Wolters, P.J.C.C. and Hawk, J.A. 2011. Fitness evaluation of *Rcg1*, a locus that confers resistance to *Colletotrichum graminicola* (Ces.) G.W.Wils. using near isogenic lines. Crop Sci 51:1551-1563.

Jirak-Peterson, J.C. and Esker, P.D. 2011. Tillage, crop rotation and hybrid effects on residue and corn anthracnose occurrence in Wisconsin. Plant Dis. 95:601-610.

Lipps, P. 1985. Influence of inoculum from buried and surface corn residues on the incidence of corn anthracnose. Phytopathology 75: 1212-1216.

Munch, S., Lingner, U., Floss, D.S., Ludwig, N. Sauer, N. and Deising, H.B. 2007. The hemibiotrophic lifestyle of *Colletotrichum* species. J. Plant Physiol. 165:41-51.

Perkins J.M. and Hooker, A.L. 1979. Effects of anthracnose stalk rot on corn yields in Illinois. Plant Dis. Rep. 63:26-30.

Robertson, A.E., Pecinovsky, K. and Liu, L. 2010. Effect of fungicides on anthracnose top dieback, frost severity and yield of hybrid corn at Nashua, Iowa in 2009. Plant Disease Management Reports 4:FC087.

Shriver, J. M. and Robertson, A.E. 2009. Comparison of fungicide products for disease control and yield in corn at Crawfordsville, Iowa 2008. Plant Disease Management Reports 3:FC015.

Sukno, S.A., Garcia, V.M., Shaw, B.D. and Thon, M.R. 2008. Root infection and systemic colonization of maize by *Colletotrichum graminicola*. Applied Environ. Microbiol. 73:823-832.

Vernard, C. and Vaillancourt, L. 2007. Penetration and colonization of unwounded maize tissues by the maize anthracnose pathogen *Colletotrichum graminicola* and the related nonpathogen *C. sublineolum*.

Vizvary M.A. and Warren, H.L. 1982. Survival of *Colletotrichum graminicola* in soil. Phytopathology 72:522-525.