GOSS'S WILT: A 2012 RECAP AND LOOKING AHEAD TO 2013[1]

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Introduction

Goss's wilt is a disease of corn caused by the Gram positive bacterium *Clavibacter michiganensis* subsp. *nebraskensis* (Cmn). The disease was first identified in Nebraska in Dawson County in 1969 (Clafin, 1999). Over the next decade, the disease was reported in 53 Nebraska counties and five of the six bordering states where it resulted in substantial (40 to 60 %) yield loss. Corn breeders successfully identified genetic resistance in field corn, and thereafter the disease occurred sporadically and rarely caused yield loss.

Within the past six years, Goss's wilt has re-emerged as a threat to corn production throughout the western Corn Belt. Since 2006, the disease has been confirmed in more than 60 Nebraska counties (Jackson et al, 2010). In Iowa, Goss's wilt was confirmed for the first time in 25 years in 8 counties in 2008, and by 2011 it had been confirmed in over 80 counties (Robertson, *unpublished data*). Yield losses of 50 percent or more due to Goss's wilt have already been documented during this more recent epidemic. Furthermore, fields with a high severity of Goss's wilt have a higher prevalence of stalk rot and consequently lodging and harvest complications.

Besides increasing in prevalence in NE and IA, Goss's wilt also is spreading east and north across the Corn Belt. The disease was reported for the first time in Indiana in 2008 (Ruhl et al., 2009), where it caused yield losses of up to 60 bushels/acre in the north-western part of the state (Wise et al, 2010), in Minnesota in 2009 (Malvick et al., 2010) and in Texas in 2010 (Korus et al., 2010). Sporadic outbreaks of the disease during the past three years have been reported in Illinois and Wisconsin.

The exact cause for resurgence of the disease is unknown, but is likely due to a combination of factors including continuous corn combined with conservation tillage practices that favor survival of the pathogen, widespread use of Goss's wilt susceptible hybrids and weather conditions during the growing season that favor infection and disease development (Jackson et al., 2007).

Symptoms

Goss's wilt symptoms may be easily mistaken for other diseases including northern leaf blight, Stewart's wilt and drought or heat stress. In Iowa, Goss's wilt usually appears soon after silking as leaf blight symptoms in the top canopy of the plant. Lesions are large, grey to reddish and start at the tips of the leaves and extend downwards, often along the edge of the leaf. Cigar-shaped lesions may also occur away from the edge of the leaf. Rather than a distinct delimitation between diseased and healthy tissue (like with northern leaf blight), the border of Goss's wilt lesions is usually indistinct and may be grey-green. Within this "border", the characteristic freckles associated with Goss's wilt are seen. The freckles are one of the characteristic symptoms

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that MUST be used to correctly diagnose this disease. The bacterium often oozes out of lesions and dries on the surface of the leaf as shiny exudates. This exudate is often more visible on the underside of the leaf. The pathogen can also infect the vascular system of the plant, and then wilting can occur. This symptom is more common on young plants. Discoloration of a few bundles in the vascular system may occur together with a wet, slimy stalk rot.

Disease Cycle

The primary source of inoculum for Goss's wilt is Cmn-infested corn residue. The bacterium can survive at least 10 months in surface residue. Dissemination of the bacterium from the residue to corn has not been studied but it is hypothesized the bacterium is splashed dispersed onto the leaves of young corn seedlings. Smidt and Vidaver (1986) isolated Cmn from the surfaces of apparently healthy corn plants in early June, and these epiphytic populations of the bacterium increased on the leaves throughout the growing season. Physical damage to the plant by hail, wind or sand is necessary for infection by the bacterium, and all plant parts can be infected. Unlike Stewart's wilt, insects are not known to be involved with spread of the disease or infection and disease development. The optimum temperature for disease development is approximately 80F. Another source of inoculum may be seed, since the bacterium is seedborne and can be seed transmitted at very low rates (0.1-0.4% in inoculated seed).

Ongoing Research

In 2011, Drs Tamra Jackson-Ziems and Greg Kruger at the University of Nebraska, and I received funding from USDA-NIFA to conduct various research projects on the epidemiology of Goss's wilt. The first objective was a survey of Goss's wilt throughout the Corn Belt. Corn leaf samples with and without symptoms of Goss's wilt were collected from 486 fields in eight states in 2011, and tested for the presence of Cmn. Data on the history of the field, hybrid, and agronomic practices were also collected and subjected to Classification and Regression Tree (CART; Breiman and Friedman, 1984) and Random Forest (Breiman, 2001) analyses to identify environmental and agronomic risk factors for Goss's wilt. The top five risk factors for Goss wilt disease development that were identified were: Goss's wilt hybrid rating, planting population, crop rotation, planting date and percent surface crop residue.

The second objective of this research project was to assess the genetic diversity of Cmn. Dr Jackson's lab is currently assessing the genetic diversity of the Cmn isolates collected from the survey and comparing it with the genetic diversity of isolates collected during the first epidemic during the 1970s to 1980s.

In Iowa, we have been studying the role of epiphytic Cmn in disease development in different agronomic environments. Preliminary work suggests that for disease to occur, a threshold population of Cmn needs to occur on the surface of the leaves. Furthermore populations of Cmn are greater on susceptible hybrids compared to tolerant hybrids, and when Cmn-infested surface residue is present versus no surface residue.

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