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## Research insights in Goss's wilt and leaf blight

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**Presenter Information**

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## Research insights in Goss's wilt and leaf blight

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Goss's wilt and leaf blight is caused by the bacterium *Clavibacter michiganensis* subsp. *nebraskensis* (*Cmn*). The disease was first reported in Nebraska in 1969 and soon after in the surrounding states including Iowa. Corn breeders identified resistance to the bacterium and by the 1980s the disease was no longer a threat to corn production except in eastern Nebraska. In 2008, Goss's leaf blight was reported in eight counties in Iowa. In 2011, the disease was widespread throughout the state and up to 50 percent yield losses occurred in some fields.

### Disease cycle

Sources of inoculum for *Cmn* are infested corn residue and seed. The bacterium is dispersed by rain, and infects wounds caused by hail or wind (Jackson et al., 2007). Infection of corn soon after planting usually results in the wilt phase of the disease, while the leaf blight phase is more characteristic of infection that occurs later in the crop development.

Our understanding of the biology of interaction of *Cmn* with corn and the environment however is rudimentary. For the past three years, we have been researching various aspects of the Goss's disease cycle.

### Goss's leaf blight severity, *Cmn* seed infection, and transmission in corn

Biddle et al. (1990) demonstrated that *Cmn* was transmitted to seedlings at 0.1-0.4% from seeds inoculated with the pathogen. They were, however, unable to demonstrate seed transmission from seeds harvested from plants that were inoculated in the field. They concluded that while the pathogen might be introduced into new geographic areas by *Cmn*-infected seeds, the risk of significant economic impact was low. Despite this research, there have been concerns that the widespread occurrence of Goss's wilt in the US Corn Belt maybe due to seed transmission.

In collaboration with Monsanto we established field trials in 2012 in which hybrids that varied in their susceptibility of Goss's were either inoculated or non-inoculated to attain different levels of Goss's leaf blight severity. We harvested the seed from these trials and determined the percent of seed infected with *Cmn*. We found that plants with more severe leaf blight had a higher incidence of *Cmn*-infected kernels (Table 1). Similarly, Block et al. (1999) showed a relationship between disease severity levels on leaves associated with Stewart's bacterial wilt and seed infection and transmission. In 2013, we planted three of the most severely infected seed lots in a field with no history of Goss's wilt. The trial was scouted every few days for wilted plants, and percent seed transmission determined. Percent seed transmission, although correlated with percent seed infection (Table 1), was similar to that reported by Biddle et al (1990).

**Table 1.** Goss's wilt and leaf blight severity, incidence of *Cmn*-seedborne infection, and number of seed transmission events observed on four corn hybrids in Iowa.

Hybrid	Inoc with <i>Cmn</i>	Final leaf blight severity (%)	Incidence of seedborne infection (%)	No. seed transmission events/ no. seed planted	Rate of seed transmission (%)	
					All plants	Infected seed only
Susceptible	Yes	88.0	27.0	9 / 51,051	0.02	0.06
	No	27.0	7.5	0 / 44,332	0.00	0.00
Moderately resistant	Yes	31.0	6.25	2 / 40,672	0.005	0.08
	No	5.0	1.0	Not done	-	-
Moderately resistant	Yes	18.0	3.0	Not done	-	-
	No	6.0	1.5	Not done	-	-
Resistant	Yes	2.0	1.5	Not done	-	-
	No	1.0	1.5	Not done	-	-

### Dissemination of *Cmn* and Goss's disease from a point source of inoculum

In an effort to understand the spatial and temporal spread of *Cmn* and Goss's disease from a point source of inoculum simulating a seed transmission event or *Cmn*-infested residue, we conducted field trials in 2012 and 2013 at the ISU Horticultural Farm, that had no previous history of Goss's wilt and leaf blight. We planted a susceptible hybrid in plots that were 8 rows wide (spaced 30-in apart) and 30-ft long (approx. 50 plants per row). A 10-ft border of a Goss's resistant hybrid was used to border each plot. The center four plants of each plot were either inoculated with a suspension of *Cmn* at the V2 growth stage, or *Cmn*-infested corn residue was placed at their base. Non-inoculated plots served as controls. Every 7 to 10 days, plots were scouted for symptoms of Goss's wilt or leaf blight, and the exact positions (row, plant number) of symptomatic plants were recorded and mapped.

Goss's leaf blight symptoms were detected first on the plants inoculated with a suspension of *Cmn*. Disease incidence (percentage of symptomatic plants in the plot) recorded at the end of the growing season was 16% and 14% in 2012 and 2013, respectively. Goss's leaf blight symptoms also developed in plots inoculated with *Cmn*-infested residue, but at a slower rate, and disease incidence at the end of the season (8% and 3% in 2012 and 2013, respectively) was lower than that in wound-inoculated plots. Symptomatic plants were found in one of the four control plots in 2011, and in none of the plots in 2012.

These data show point sources of inoculum such as an infected plant or *Cmn*-infested crop residue can serve as infection foci from which Goss's wilt may develop, but it is unlikely that whole fields could become infected within a single growing season. It is more likely that the disease goes unnoticed in the first year, and then becomes epidemic in subsequent years if the environment is favorable for disease development. Reduced tillage and continually planting *Cmn*-susceptible hybrids in a field allow the pathogen inoculum to build up and as disease foci enlarge and coalesce large areas of a field can be infected.

### Role of epiphytic *Cmn* in Goss's wilt and leaf blight development

Smidt and Vidaver (1986) monitored the population of *Cmn* on healthy corn leaves in the field over two growing seasons and suggested this bacterium also has an epiphytic relationship with its host. *Cmn*-infested residue is considered the primary source of inoculum for Goss's wilt in a field (Schuster, 1975). We hypothesize that the bacterium is splash-dispersed from *Cmn*-infested residue onto young seedlings, where it survives and multiplies as an epiphyte. During favorable weather, injury to the plants caused by hail and/or high winds enables the bacterium to enter and infect the plant and cause disease.

Observations from field trials that we conducted in Iowa and Nebraska suggested that *Cmn* was able to survive equally well on both Goss's resistant and susceptible hybrids. We established two field trials in 2013 to monitor

*Cmn* populations on asymptomatic corn leaves sampled from a resistant and a susceptible hybrid in plots with and without *Cmn*-infested residue. We found that Goss's leaf blight developed only in the susceptible hybrid, even though epiphytic populations did not differ between the hybrids. Epiphytic populations were higher in plots that had *Cmn*-infested residue, and the incidence of disease was greater in the plots with residue ( $P=0.0356$ ).

### How does corn resist infection by *Cmn*?

In the greenhouse we inoculated *Cmn* at the midpoint of leaves of a susceptible and a resistant hybrid. We monitored *Cmn* populations and colonization within the leaf tissue from the site of infection for two weeks. We also used microscopy to observe colonization of the host tissue and morphological changes that may occur. Pataky (1983) found the reaction of sweet corn hybrids to Goss's wilt and Stewart's wilt were highly correlated, and consequently, the mechanisms of resistance to each bacterial pathogen may be similar. He reported no difference in the growth rate of *Pantoea stewartii* in mesophyll tissues of resistant and susceptible seedlings. In our studies, we observed leaf tissue colonization prior to the development of leaf symptoms. Lesion expansion towards the tips of the leaves developed at the same rate on the susceptible and resistant hybrids, but lesions expanded more slowly towards the proximal (stalk) end of the leaf on the resistant hybrid. At 14 days after inoculation, a dense matrix was visible in the infected leaf tissues; this resistance response restricted the movement of *Cmn*. Braun (1982) also observed host-derived vessel-plugging materials in corn seedlings that were resistant to Stewart's wilt, and suggested that this is a host response that localizes the pathogen.

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