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Reemergence of Goss's Wilt and Blight of Corn to the Central High Plains

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Goss's bacterial wilt and blight, also known as leaf freckles and wilt, is caused by *Clavibacter michiganensis* subsp. *nebraskensis* (Vidaver & Mandel) Davis et al. and was first identified in Dawson Co. in south central Nebraska in 1969 (2). Over the next 10 years, the disease was identified in 53 additional counties in the state and at least one county in five of the six states bordering Nebraska (4). Corn breeders were successful in identifying genetic resistance in field corn. Since then the disease has occurred only sporadically, rarely causing severe damage and yield loss with the exception of fields planted with susceptible popcorn, sweet, and dent corn hybrids that sustained physical injury. There is no known vector and the pathogen requires wounds for entry into the plant. Disease is most common and severe following hail storms. Bacteria are splashed from crop residue where they overwinter to wounded leaves.

Fig. 1. Small, dark discontinuous lesions, or 'freckles,' near the edges of expanding lesions are characteristic of Goss's wilt and blight.

Symptoms of infection can be manifested as both a leaf blight phase and a systemic vascular wilt. Foliar lesions are dark and water-soaked, similar in appearance to those associated with Stewart's bacterial wilt disease. Characteristic small, dark, discontinuous, water-soaked areas, often referred to as 'freckles,' can develop at the margins of expanding lesions (Fig. 1). Orange bacterial exudate (Fig. 2, bottom) may also be observed on the surface of mature lesions giving leaves a glistening appearance when dried (Fig. 2, top). The less common wilt phase can occur when the pathogen infects the vascular system and moves systemically within the xylem, resulting in discoloration of vascular bundles (Fig. 3) and can progress to a stalk rot and early plant death (Fig. 4). Systemic infection can kill plants at any time during the season, including young seedlings, and can cause losses of up to 50% during severe epidemics (2).

Fig. 2. Orange bacterial exudate can be secreted on the surface of leaves (lower photo) and can appear shiny when dried (upper photo).

Fig. 3. Discoloration of vascular bundles in a systemically infected plant.

Fig. 4. Systemically infected plants can die prematurely.

Widespread development of these symptoms in western Nebraska, southeastern Wyoming, and eastern Colorado early in the 2006 growing season led to submission of more than 50 samples to the UNL Panhandle Research and Extension Center's Plant Disease Diagnostic Clinic in Scottsbluff, NE for analysis. Samples were submitted from numerous counties across the tri-state region with no obvious disease epicenter. In contrast, samples received by the UNL Plant and Pest Diagnostic Clinic in Lincoln reflected sparse development of Goss's wilt and blight in eastern Nebraska. Only two of six samples (out of the 147 corn samples) with Goss's wilt and blight were submitted from counties in eastern Nebraska. Prior to the 2006 season, a total of only 40 samples out of more than 1,200 submitted from corn fields to both clinics since 1998 were diagnosed with Goss's wilt and blight. Disease severity since 2004 ranged from slight to severe and was accompanied by estimated yield losses of more than 50% in some fields with early symptom development and plant mortality. The impact on yield was most apparent in two adjacent fields in northeast Colorado with a history of comparable yields. Despite the lack of hail in 2004, disease severity and yield in the two fields differed by 63% between resistant and susceptible hybrids producing 195 and 72 bu/acre, respectively. However, in 2006 some seed company representatives in the central High Plains observed that early season infection through wounds created by sandblasting during high winds often led to severe disease and yield loss, regardless of reported hybrid disease ratings.

Pathogen identification was made based on a combination of test results including positive Gram stain and KOH reactions, catalase-positive and oxidase-negative tests, cell morphology consisting of coryneform-shaped, motile rods, and the development of fluidal orange-yellow colonies on nutrient-broth yeast extract medium (NBY) and the semi-selective medium CNS (*Corynebacterium nebraskense* selective) (3). Confirmation also included reproduction of the disease in the greenhouse on 'Golden Cross Bantam' sweet corn plants (Koch's

postulates). Plants were inoculated at the 3 to 5 leaf stage by needle inoculation of the hypocotyl with approximately 1×10^7 CFU/ml in 5 µl of bacterial suspension prepared in 12.5 mM of phosphate buffer, pH 7.1. Bacteria were reisolated from symptomatic plants onto NBY or CNS for identity verification.

The exact cause for the resurgence of the disease in this region is unknown but is likely due to a combination of factors that have favored both pathogen survival and disease development. A major factor is the use of continuous corn cropping practices, often for more than 50 years in some fields, which provides a consistent source of pathogen-infested residue. In addition, the increased popularity of reduced tillage practices decreases the potential for the breakdown of infested residue. Due to the lack of severe and persistent disease pressure, less than 25% of seed corn companies in Nebraska currently evaluate hybrids for their reaction to the disease and this may also have contributed to the widespread distribution of susceptible commercial hybrids. Finally, early season hail storms and strong winds in many of the affected fields provided the means for early infection and development of systemic disease in young plants, which are known to be more susceptible than mature plants regardless of their levels of resistance (1). At this time, seed are not considered a source of inoculum.

The 2006 epidemic of Goss's wilt and blight in the central high plains was an important reminder that this pathogen will likely persist, especially considering the increase in minimum tillage and planting of continuous corn. More emphasis should be placed on the selection of resistant hybrids and crop rotation in areas with a history of the disease. When practical, producers could consider a tillage practice to bury debris and encourage decomposition at least 60 to 90 days before planting. Lightly discing the residue in the spring prior to planting may also reduce the inoculum and risk of disease development.

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