Genetic Analysis of Goss's Bacterial Wilt and Leaf Blight in North American Maize

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Goss's wilt and leaf blight is a bacterial disease of maize (*Zea mays* L.) caused by gram positive bacterium *Clavibacter michiganensis* subsp. *nebraskensis* (*Cmn*) (Schuster et al. 1972). Goss's wilt was first discovered in south central Nebraska in 1969 and quickly spread to different counties within Nebraska as well as Iowa, Kansas, Colorado, and South Dakota. In disease evaluation trials, varying levels of resistance were found in maize inbred lines (Calub et al., 1974; Schuster et al., 1972). Partially resistant hybrids were developed and due to deployment of resistant hybrids over a period of ten years, the disease became sporadic, caused only minor damage, and occurred only in fields planted with susceptible maize hybrids (Jackson et al., 2007). However, around 2006, Goss's wilt re-emerged as a serious disease in the North American Corn Belt (Jackson et al., 2007). Since then, Goss's wilt has been observed in 60 counties in Nebraska (Jackson and Rees, 2010) and 80 counties in Iowa (Robertson, 2012). Goss's wilt has also been reported in Colorado, Illinois, Iowa, Kansas, Michigan, Minnesota, South Dakota, Wisconsin, Texas, and Louisiana. Goss's wilt infection can occur at any developmental stage with the bacteria entering into the plant through leaves, stems, and roots (Schuster, 1975). There are two possible phases of Goss's wilt: 1) leaf blight and 2) systemic wilt. Infection starts through wounds on the plant surface leading to water soaked spots (or freckles), which progress to grey lesions and eventually lead to leaf blight. Less common systemic wilt occurs when the pathogen infects the vascular system and moves systemically through the xylem leading to blockage of vascular bundles. The systemic wilt phase is more common if infection occurs during early growth stages (Jackson et al., 2007). Loss in yield can be severe if Goss's wilt occurs on susceptible hybrids. Yield loss caused by Goss's wilt under artificial inoculation was estimated to be 44% in susceptible maize hybrids (Claflin et al., 1978). Inheritance of resistance to Goss's wilt has been studied previously using classical techniques including diallel mating designs and generation mean analysis. Early studies indicated that additive variation accounts for most genotypic variation for Goss's wilt (Ngong-Nassah et al., 1992; Treat and Tracy, 1990).

Using the maize NAM population, numerous QTL with small additive effects have been identified for diseases of maize such as southern corn leaf blight and northern corn leaf blight (Kump et al., 2011; Poland et al., 2011). Such studies are lacking for Goss's wilt because of its sporadic occurrence over several years. The recent reemergence of Goss's wilt, however, has increased interest in understanding the genetic basis of resistance to Goss's wilt. The objectives of this study are: to map quantitative trait loci (QTL) underlying the resistance to Goss's wilt using linkage and association mapping approaches, and to assess the prospects of genomic selection for Goss's wilt. Joint linkage mapping as well as linkage mapping were used to map the QTL in linkage mapping families. The three bi-parental linkage mapping families B73 x Oh43,

B73 x HP301, and B73 x P39 were phenotyped for Goss's wilt in Nebraska. The bi-parental families were previously genotyped with 1106 single nucleotide polymorphism (SNP) markers. Eleven QTL were detected on chromosomes 1, 2, 3, 4, 5, and 10 through joint linkage mapping. The detected QTL together explained 45% of the phenotypic variation for Goss's wilt. Linkage mapping in three families separately, identified nine, six, and four QTL in families B73 x Oh43, B73 x HP301, and B73 x P39 respectively. For association mapping, a diverse panel of maize inbred lines was selected and evaluated for reaction to Goss's wilt in 2014 and 2015 in Nebraska. A genotyping by sequencing (GBS) dataset consisting of 955,690 SNPs was used for association analysis. To assess the genomic prediction accuracy for Goss's wilt, a ten-fold cross validation approach within bi-parental families and association mapping panel was used. Results from this study will improve our knowledge about the genetic basis of underlying variation for resistance to Goss's wilt, and may assist the plant breeders to incorporate resistance to Goss's wilt into maize.

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