GOSS'S WILT AND LEAF BLIGHT OF CORN: CHEMICAL CONTROL, RESIDUE MANAGEMENT AND RESISTANCE

BY

KELSEY M. MEHL

THESIS

Submitted in partial fulfillment of the requirements
for the degree of Master of Science in Crop Sciences
in the Graduate College of the
University of Illinois Urbana-Champaign, 2015

Urbana, Illinois

Master's Committee:

Associate Professor Carl Bradley, Chair
Associate Professor Martin Bohn
Associate Professor Youfu Zhao
Professor Emeritus Jerald Pataky

ABSTRACT

Goss's wilt and leaf blight of corn has been a re-emerging disease in the United States. Caused by the bacterium *Clavibacter michiganensis* subsp. *nebraskensis* (Cmn), Goss's wilt has been increasing in Illinois due to factors that include continuous maize rotations, reduced tillage which increases debris harboring inoculum and the use of susceptible hybrids. There are several ways that could help control the spread of Goss's wilt, including chemical control, residue management and the use of resistant hybrids. These three areas of control were the subject of this research.

While there are resistant hybrids for some regions, the availability of resistant hybrids may be limited for other regions. In areas where Goss's wilt was more severe, recommendations were provided to farmers to apply different products as "rescue treatments" even though there was no data to support or oppose them. This study was developed in order to understand the effect of various chemicals on Cmn and to see if it was a viable method of control while more resistant hybrids were being developed. A field study was done to evaluate the effect of copper hydroxide and citric acid on the severity of Goss's wilt and on the yield of corn. Products were applied relative to inoculation with Cmn and were compared to inoculated, non-treated control. Non-inoculated treatments were also included. While treatments that were not inoculated with Cmn had significantly ($P \leq 0.05$) lower Goss's wilt severity compared with those that were inoculated, there was no significant differences among treatments within Cmn inoculated plots or within non-inoculated plots. Yield was reduced in Cmn-inoculated plots compared to the non-inoculated plots. Our research found that copper hydroxide and citric acid were not a viable management technique for Goss's wilt.

There is a current shift in farming practices in Illinois towards growing continuous maize without rotational crops. Cmn overwinters in corn debris, and increasing the debris left in the field could lead to epidemics. In order to understand the role of corn residue on the incidence and severity of Goss's wilt, a field study was conducted in the summers of 2012 and 2013. Three different tillage methods were used on a field that historically has severe Goss's wilt in order to vary the amount of residue left on the surface. These tillage methods included no-till, chisel plow and moldboard plow. Ten susceptible hybrids were evaluated for the incidence and severity of Goss's wilt from natural infection. The incidence (%) and severity (0-9 scale) data were used to calculate a disease severity index (DSI), where DSI – (% incidence) X (severity) / 9. The different tillage treatments had a significant effect on both DSI (P = 0.0004) and yield (P = 0.0005). The no-till treatment resulted in the highest amount of residue and a significantly higher DSI than the other two treatments. The moldboard plow treatment had the lowest amount of residue left on the surface and had significantly lower DSI than no-till and chisel plow. These results have shown that by limiting the amount of infected corn residue left on the surface, Goss's wilt may be managed.

Resistant hybrids are limited in some regions of the United States. With the re-emergence of Goss's wilt, more resistant hybrids are being developed for more areas. The University of Illinois plant pathology inbred collection has over 2,000 inbred lines collected from all over the world. This collection has been evaluated for other potential sources of resistance to other maize diseases such as Aspergillus ear rot (*Aspergillus flavus*) and grey leaf spot (*Cercospora zeae-maydis*). An initial screen was conducted near Urbana, IL in 2011. Over 1,500 inbred lines were screened, inoculated with a Cmn cell suspension and had the Goss's wilt severity rated with a 1-

9 severity scale, with a 1 = resistant and 9 = susceptible. Over 150 lines were identified to have potentially high levels of resistance to Goss's wilt. These lines were moved to a second stage of screening in 2012. During the second stage, not all the lines preformed as well as the previous year. Nine lines were chosen to move to the final stage of screening. These nine lines had a mean severity score of 1.9, while the susceptible check had a mean score of 6.4. From our research, we found that there are potential sources of resistance to Goss's wilt in the inbred collection that could be used for future breeding efforts.

Acknowledgments

I would like to thank Dr. Carl Bradley for giving me this amazing opportunity and for his help and direction throughout this study. It has been a long road, but I have learned many things and it is with my sincere gratitude that I thank Carl for allowing me to be a part of his lab.

Special thanks to my fellow lab members and friends, Jafe Weems, Toyo Ajayi-Oyetunde, and Kaitlyn Bissonnette for all their help, support and guidance throughout this process. They helped me so much with anything I ever needed and were there for me when times were tough. I will be forever thankful that I met such wonderful friends during my time in the Bradley lab. I am also very appreciative of Keith Ames and Ursula Reuter-Carlson for all the extensive help and support in the field and lab as I fumbled and learned my way around. Their patience and guidance for me will always be remembered. There have also been many lab members, undergraduate and hourly workers that have helped me in many different ways and my research would not have been possible without them.

Thank you to Jerald Pataky who provided me with an opportunity to work in his lab as an undergraduate. His enthusiasm for his work, support and advice helped me find my way when I was still wondering what I wanted to do with the rest of my life.

I would also like to thank my committee members, Youfu Zhao and Martin Bohn for their advice and input in my studies. Thank you also to Mark Mikel for his help and advice.

I am so grateful for my parents and my sister as they supported me through this degree. Their constant support, encouragement and love helped me through the last few years, and I would not have been able to get through it without them

TABLE OF CONTENTS

CHAPTER 1: EVALUATION OF FOLIAR-APPLIED COPPER HYDROXIDE AND CITRIC ACID FOR THE CONTROL OF GOSS'S WILT AND LEAF BLIGHT OF CORN

ABSTRACT

Goss's wilt and leaf blight, caused by *Clavibacter michiganensis* subsp. *nebraskensis* (Cmn), is a re-emerging disease of corn (*Zea mays*) in portions of the Midwestern USA. Although resistant corn hybrids have been developed for some regions, the availability of adapted resistant hybrids may be limited in other areas; therefore, chemical control could potentially serve as an interim control measure while resistant hybrids are being developed. A field experiment was initiated to evaluate the effect of copper hydroxide and citric acid on Goss's wilt severity and yield of corn. Chemicals were applied relative to inoculation with Cmn, and non-inoculated plots also were included. Treatments not inoculated with Cmn had significantly ($P \leq 0.05$) lower Goss's wilt severity values compared with those that were inoculated, but no significant differences among treatments within Cmn-inoculated or within non-inoculated plots were observed. Neither Cmn-inoculation nor chemicals applied had a significant effect on corn yield. Overall, corn yields were reduced by approximately 9% in Cmn-inoculated plots compared with non-inoculated plots. From our research, application of copper hydroxide or citric acid was not a viable Goss's wilt management practice. Therefore, corn growers should continue to rely on cultural practices such as crop rotation and tillage to limit Cmn inoculum in fields, and grow resistant hybrids if they are available.

INTRODUCTION

Goss's wilt of corn (*Zea mays*), caused by the bacterial pathogen *Clavibacter michiganensis* subspecies *nebraskensis* (Cmn), was first observed in Dawson County, NE in 1969 (Wysong et al., 1973). Soon after, it was found in the surrounding states Iowa, Kansas, and South Dakota. Eventually, corn breeders were successful in developing resistant hybrids, and only sporadic observations of this disease were reported in the central high plains. Most of these reports were from fields planted to susceptible hybrids with physical damage (caused by hail or high winds) (Jackson et al., 2007). Goss's wilt can be observed as either a leaf blight or systemic wilt on susceptible hosts.

Cells of Cmn are splashed onto corn leaves from debris on the soil and enter the plant tissue through wounds caused by hail or damaging winds (Wysong et al., 1981). The symptoms that are associated with this disease include water-soaked, irregular-shaped lesions with wavy margins on the foliage, as well as dark green to black spots (known as "freckles") within these lesions. These lesions can coalesce into larger lesions, resulting in typical blight symptoms. As the disease progresses, bacterial exudates may be seen on the leaf surface. When these exudates dry, they leave behind a shiny sheen that can be seen on the leaf surface. Severe infections can lead to wilting and premature death when the vascular tissue is infected. When affected corn stalks are split open, orange to brown discolored vascular tissue may be observed. If the disease leads to a vascular wilt, the leaf symptoms may resemble drought stress. Symptoms can develop at any stage of the plant. Younger plants may die early, while older plants may or may not produces tassels or ears.

Although Goss's wilt had been first observed in Illinois in 1980 (Wysong et al., 1981), the disease did not cause much damage within the state for several years. However, starting in the late 2000s, reports of corn fields affected by Goss's wilt came from some Midwestern states such as Indiana, Illinois and Minnesota (Bradley, 2012; Malvick et al., 2010; Ruhl et al., 2009). In 2011, Goss's wilt was confirmed in 31 Illinois counties (Bradley, 2012).

Management practices for Goss's wilt include planting resistant hybrids, rotating to non-host crops, and tilling to speed up corn residue decomposition. Since Cmn overwinters in corn debris, fields using conservation or no-tillage practices are at a higher risk for Goss's wilt (Wysong et al., 1981) Rotating to a non-host crop and planting resistant corn hybrids will also help manage Goss's wilt (Claflin, 1999; Treat and Tracy, 1990). Unfortunately, adapted resistant hybrids may not be available in areas in which Goss's wilt has been recently observed for the first time or in areas where the disease is resurging. In these areas, chemical applications would be a helpful interim management tool that could be used until resistant hybrids are available. Unfortunately, only a few Goss's wilt chemical efficacy trials have been reported (Korus et al., 2009; Oser et al., 2013; Wise et al., 2014). Therefore, the objective of this research study was to evaluate potential chemical treatments for their effect on Goss's wilt and corn yield.

MATERIALS AND METHODS

Treatments and experimental design

In 2012 and 2013, corn hybrids susceptible to Goss's wilt were planted at the University of Illinois Crop Sciences Research and Education Center (CSREC) near Champaign and Urbana, IL. In 2012, the research field was located on the Cruse Tract of the CSREC, and in 2013, two

different fields were utilized at the CFAR Tract of the CSREC. All research fields were previously planted to corn. Plots were planted on 11 May 2012, 30 May 2013 and 13 June 2013 using an Almaco 360 research plot planter (Almaco Co., Nevada, IA). The planting population was approximately 84,000 seeds/ha. Plots were planted 4 rows wide on 76 cm centers and 7.6 m long, but only the two middle rows were treated and utilized for data collection.

Treatments consisted of a non-treated control and different chemicals applied at different timings relative to inoculation with Cmn. In total, ten treatments were evaluated (Table 1.1). The chemical treatments consisted of copper hydroxide (Kocide 3000; DuPont, Wilmington, DE) and citric acid (Procidic; Greenspire Global, Inc., Des Moines, IA). Plants were inoculated with Cmn once at approximately the 10^{th} leaf stage. Plots were arranged in a randomized complete block design (RCBD) with 4 replications at the Cruse Tract in 2012 and one of the CFAR Tracts in 2013 and 3 replications at the CFAR Tract in 2013.

Chemical treatments were applied with a CO_2-pressurized backpack sprayer calibrated to deliver 187 liters/ha at 276 kPa with TwinJet 8002 spray nozzles (Spraying Systems Co., Wheaton, IL). The sprayer boom was set 46 cm above the canopy on 51 cm centers.

Inoculum production, inoculation technique.

Cmn inoculum production and plant inoculation procedures followed those reported by Pataky (1985). An isolate of Cmn obtained from infected leaves from a corn field in Champaign County, IL in 2011 was used for inoculations in this study. To initiate inoculum production, Cmn was cultured using nutrient broth (Becton, Dickinson and Company, Franklin Lakes, NJ) in Erlenmeyer flasks on a shaker table for 2 days at room temperature (between 23-25°C) under

constant light (15 watt fluorescent bulbs). A spectrophotometer (SmartSpec 3000; Bio-Rad Lab., Hercules, CA) was used to determine bacterial concentration (OD = 600). Bacterial concentrations were adjusted to 10^7 colony forming units/ml using a 0.1 M NaCl solution at inoculation. This inoculum was used to inoculate early-planted 'Jubilee' sweet corn in the field to increase inocula. Plants were inoculated with Cmn using the pinprick technique previously described (Blanco et al, 1977; Pataky, 1985) at approximately the V4 growth stage (Ritchie et al., 1997). To inoculate the field trial, symptomatic leaves were collected from the 'Jubilee' sweet corn disease nursery. Ten symptomatic leaves collected from the 'Jubilee' disease nursery were cut into smaller pieces (approximately 5 cm^2), placed into a commercial blender (Model 38BL52; Waring Products, New Hartford, CT) containing 3.8 liters of a 0.1 M NaCl solution, and blended for 30 seconds. The blended leaf suspension was then strained through a kitchen strainer to remove large pieces of leaf tissue. The suspension was then used to inoculate plants with the pinprick method at the V10 growth stage.

Disease and yield assessments

Goss's wilt severity was rated 3-4 weeks after inoculation using a 1 to 9 scale previously reported by Pataky and Suparyono (1989), where 1 = no symptoms present; 2 = lesions with water soaking, freckles, chlorosis, and necrosis spread <6 cm from inoculation site; 3 = lesions spread appreciably toward the tip of the leaf from the inoculation site; 4 = lesions spread appreciably toward the base of the leaf from the inoculation site; 5 = limited systemic infection, with lesions appearing on non-inoculated leaves; 6 = 25-50% of total leaf area affected with severe leaf lesions, water soaking, chlorosis, necrosis, and stunting of the plant; 7 = 50-75% of

total leaf area affected, and severe stunting of the plant; 8 = 75-90% of the total leaf area affected, with severe stunting of the plant; and 9 = plant death. Three ratings were collected from three different areas of each plot. The mean of these three ratings were calculated for each plot prior to data analysis. Plots were harvested on 29 October 2012 and 11 November 2013 with a Kincaid 8-XP small plot combine (Kincaid Equipment Manufacturing, Haven, KS) equipped with a HarvestMaster grain gauge (Juniper Systems, Inc., Logan, UT) to calculate grain moisture and weight for each plot. Individual plot weights were adjusted to 15% moisture and yields were calculated to kg/ha.

Data were analyzed using the mixed linear model procedure (PROC MIXED) in SAS (Version 9.3; SAS Institute Inc., Cary, NC). Treatment was considered a fixed effect, while environment and replications were considered random effects. Least square means were compared using the PDMIX800 macro (Saxton, 1998) where $\alpha = 0.05$.

RESULTS

The main effect of treatment was significant ($P = 0.0001$) for Goss's wilt severity, but not for yield ($P = 0.3526$). The treatment × environment interaction was not significant for yield ($P = 0.1328$), but was significant for Goss's wilt severity ($P = 0.0406$). Since environment was considered a random effect, only the main effect of treatment is presented.

Mean disease severities ranged from 0 to 3.5 (Table 1.1). Treatments not inoculated with Cmn had significantly ($P \leq 0.05$) lower disease severity values compared to those that were inoculated. No significant differences among inoculated treatments were observed, and no significant differences among non-inoculated treatments were observed. Yields ranged from

7,205 to 8,576 kg/ha, but means were not statistically compared, because the main effect of treatment was not significant for yield.

DISCUSSION

In this experiment, the only significant difference among treatments for Goss's wilt severity was between Cmn-inoculated and non-inoculated treatments. Among Cmn-inoculated treatments, none of the chemical treatments reduced Goss's wilt severity compared to the non-treated control. This is similar to what was reported by Oser et al. (2013) and Wise et al. (2014), where copper products applied to corn did not have any significant effect on Goss's wilt in Nebraska and Indiana, respectively. Korus et al. (2010) did report that copper hydroxide applied post-inoculation with Cmn did significantly reduce Goss's wilt compared to a Cmn-inoculated, no pesticide control in Nebraska on a susceptible corn hybrid, but no effect was observed on a resistant hybrid.

Chemical treatments have been evaluated for their effect on the related bacterium, *Clavibacter michiganensis* subsp. *michiganensis* (Cmm), which causes bacterial canker of tomato (*Solanum lycopersicum*). Farley and Miller (1973) reported that tribasic copper sulfate, tetracycline HCl, or streptomycin sulfate were not effective in controlling bacterial canker. Hausbeck et al. (2000) reported that copper hydroxide reduced Cmm population size, but not the incidence of tomato fruit spotting in the field. Similarly, Werner et al. (2002) reported that copper hydroxide reduced Cmm population size on seedling tomatoes, but not fruit spotting in the greenhouse. Based on the research by Hausbeck et al. (2000) and Werner et al. (2002), it is possible that Cmn population size could have been affected in my research, but it was not

measured. Regardless of any potential effects on Cmn population size, Goss's wilt severity was not impacted by copper hydroxide in my study. In future chemical evaluations for Goss's wilt control, the treatments should be expanded to include treatments that previously have been shown to be effective against the related bacterium Cmm. An in vitro study by Thompson (1986) showed that several compounds inhibited Cmm, of which 2,2-methylenebis (4-chlorophenol), benzalkonium chloride, and cetrimide caused the most inhibition. Hausbeck et al. (2000) and Werner et al. (2002) also reported that mixing chemicals, such as mancozeb, streptomycin, or acibenzolar-*S*-methyl, with copper hydroxide may provide additional inhibition of Cmm.

Although not statistically significant, yields were lower in Cmn-inoculated treatments compared to non-inoculated treatments in my trial. Conducting research on sweet corn, Pataky et al. (1988) reported that yield (primary ear weight) reduction caused by Goss's wilt varied by year, with up to nearly 100% yield reduction occurring when Goss's wilt severity was over 50%. Also working with sweet corn, Suparyono and Pataky (1989) reported a damage threshold of approximately 40% severity for susceptible and moderately-susceptible hybrids, where ear weight and ear number was decreased by 17% and 19%, respectively, for each 10% increase in severity with losses up to 100% being reported. In my dent corn study, grain yield was reduced by approximately 9% when Cmn-inoculated treatments were compared to non-inoculated treatments. Jackson et al. (2007) reported observational data from two adjacent fields in Colorado, in which the yield of a susceptible hybrid was reduced by 63% compared to the yield of a resistant hybrid. The highest mean severity value in my trial was less than 4.0, which would be equivalent to approximately 3%-5% of the leaf area affected with non-systemic leaf blight (Suparyono and Pataky, 1989). This level of Goss's wilt is below the damage threshold of 40%

severity reported by Suparyono and Pataky (1989), and was likely too low to cause statistically significant differences in yields between Cmn-inoculated and non-inoculated treatments. However, the approximate 9% yield reduction observed in my study is high enough to cause economic losses for corn growers in the U.S.

In this study, neither copper hydroxide nor citric acid were observed to reduce Goss's wilt severity regardless of the application timing, but future research should focus on additional chemicals that could potentially inhibit Cmn. Until chemical products are identified that have efficacy against Goss's wilt, growers should rely on planting resistant hybrids, rotating to non-host crops, and tilling soil to increase degradation of Cmn-infested debris.

REFERENCES

Bradley, C. A. 2012. Wilts, spots, blights and streaks: Old and emerging diseases of corn. Pages 18-21 in Proc. Univ. Illinois Corn Soybean Classics. http://extension.cropsci.illinois.edu/fieldcrops/classics/pdfs/2012.pdf.

Blanco, M. H., Johnson, M. G., Colbert, T. R., and Zuber, M. S. 1977. An inoculation technique for Stewart's wilt disease of corn. Plant Dis. Rep. 61:413-416

Calub, A. G., Schuster, M. L., Compton, W. A., and Gardner, C. O. 1974. Improved technique for evaluating resistance of corn to *Corynebacterium nebraskense*. Crop Sci. 14:716-718.

Claflin, L. E. 1999. Goss's bacterial wilt and blight. Pages 4-5 in: Compendium of Corn Diseases, 3rd Edition. D. G. White, ed. American Phytopathological Society, St. Paul, MN.

Farley, J. D., and Miller, T. D. 1973. Spread and control of *Corynebacterium michiganense* in tomato transplants during clipping. Plant Dis. Rep. 57:767-769

Hausbeck, M. K., Bell, J., Medina-Mora, C., Podolsky, R., and Fullbright, D. W. 2000. Effect of bactericides on population sizes and spread of by *Clavibacter michiganensis* subsp.

michiganensis on tomatoes in the greenhouse and on disease development and crop yield in the field. Phytopathology 90:38-44.

Jackson, T. A., Harveson, R. M., and Vidaver, A. K. 2007. Reemergence of Goss's wilt and blight of corn to the central High Plains. Online. Plant Health Progress doi:10.1094/PHP-2007-0919-01-BR.

Korus, K. A., Jackson, T.A., Behn, J. L., and Schleicher. C. 2010. Evaluation of foliar treatments and application timings for management of Goss's bacterial wilt and blight of field corn in Nebraska, 2009. Plant Dis. Manag. Rep. 4:FC083. Online publication. doi:10.1094/PDMR04.

Malvick, D., Syverson, R., Mollov, D., and Ishimaru, C. A. 2010. Goss's bacterial blight and wilt of corn caused by *Clavibacter michiganensis* subsp. *nebraskensis* occurs in Minnesota. Plant Dis. 94:1064

Oser, H. H., Jackson-Ziems, T.A., and Brungardt, J. L. 2013. Foliar treatment timing trials for management of Goss's bacterial wilt and blight of field corn in Nebraska, 2012. Plant Dis. Manag. Rep. 7:FC045. Online publication. doi:10.1094/PDMR07.

Pataky, J. K. 1985. Relationships among reactions of sweet corn hybrids to Goss' wilt, Stewart's bacterial wilt, and northern corn leaf blight. Plant Dis. 69:845-848.

Pataky, J. K., Headrick, J. M., and Suparyono. 1988. Classification of sweet corn hybrid reactions to common rust, northern corn leaf blight, Stewart's wilt and Goss's wilt and associated yield reductions. Phytopathology 78:172-178.

Ritchie, S.W. and Hanway, J. J. 1997. How a corn plant develops. Special Report No. 48, Iowa State University, CES, Ames. IA.

Ruhl, G., Wise, K., Creswell, T., Leonberger, A., and Speers, C. 2009. First report of Goss's bacterial wilt and leaf blight on corn caused by *Clavibacter michiganensis* subsp. *nebraskensis* in Indiana. Plant Dis. 93:841.

Saxton, A. M. 1998. A macro for converting mean separation output to letter groupings in Proc Mixed. Pages 1243-1246 in: Proc. 23rd SAS Users Group International.

Suparyono and Pataky, J. K. 1989. Influence of host resistance and growth stage at the time of inoculation on Stewart's wilt and Goss's wilt development and sweet corn hybrid yield. Plant Dis. 73:339-345.

Thompson, E. T. 1986. The toxicity of a number of different bactericides to *Clavibacter michiganense* subsp. *michiganense* (Smith 1910) Jensen 1934 comb. nov. [basonym *Corynebacterium michiganense* pv. *michiganense* (AL)] and to the tomato plant, *Lycopersicon esculentum. J. Appl. Bacteriol. 61:427-436.*

Treat, C. L., and Tracy, W. F. 1990. Inheritance of resistance to Goss's wilt in sweet corn. J. Amer. Soc. Hort. Sci. 115:672-674.

Werner, N. A., Fullbright, D. W., Podolsky, R., Bell, J., and Hausbeck, M. K. 2002. Limiting populations and spread of *Clavibacter michiganensis* subsp. *michiganensis* on seedling tomatoes in the greenhouse. Plant Dis. 86:535-542.

Wise, K., Ravellette. J., Anderson, N., and Buechley, G. 2014. Evaluation of foliar products for Goss's wilt management in central Indiana, 2013. Plant Dis. Manag. Rep. 8:FC253. Online publication. doi:10.1094/PDMR08.

Wysong, D. S., Doupnik Jr, B., and Lane, L. 1981. Goss's wilt and corn lethal necrosis—can they become a major problem? Pages 104-130 in: Proc. Annu. Corn Sorghum Res. Conf., 36th. Am. Seed Trade Assoc. Washington, DC.

Wysong, D. S., Vidaver, A. K., Stevens, H. and Stenberg, D. 1973. Occurrence and spread of an undescribed species of *Corynebacterium* pathogenic on corn in the western corn belt. Plant Dis. Rep. 57:291-294

Table 1.1. Effect of chemical treatments on Goss's wilt severity and yield of corn. Means are averaged across a total of three environments from Champaign and Urbana, IL in 2012 and 2013.

Treatment (active ingredient %)	Product rate	Application time[a]	Cmn inoculation[b]	Goss's wilt severity (1-9)[c]	Yield (kg/ha)
Non-treated	n/a	n/a	No	0.3 A[d]	8,576 A
Copper hydroxide (46.1%)	1.68 kg/ha	24 hours before	No	0 A	8,293 A
Citric acid (3.5%)	980 ml/ha	24 hours before	No	0 A	8,089 A
Non-treated	n/a	n/a	Yes	3.5 B	7,853 A
Copper hydroxide (46.1%)	1.68 kg/ha	24 hours before	Yes	3.4 B	7,495 A
Citric acid (3.5%)	980 ml/ha	24 hours before	Yes	3.5 B	7,591 A
Copper hydroxide (46.1%)	1.68 kg/ha	24 hours after	Yes	3.2 B	7,706 A
Citric acid (3.5%)	980 ml/ha	24 hours after	Yes	3.4 B	7,585 A
Copper hydroxide (46.1%)	1.68 kg/ha	5-7 days after	Yes	3.5 B	7,205 A
Citric acid (3.5%)	980 ml/ha	5-7 days after	Yes	3.4 B	7,566 A

[a]Application time was relative to when plants were inoculated with *Clavibacter michiganensis* subsp. *nebraskensis*.

[b]Plots were either inoculated or not inoculated with *C. michiganensis* subsp. *nebraskensis* at approximately the V10 growth stage.

[c]Plots were rated for Goss's wilt severity approximately 3-4 weeks after inoculation using a 1-9 scale where 1 = no symptoms present and 9 = plant death.

[d]Means followed by the same letter within a column are not significantly different ($\alpha = 0.0$

CHAPTER 2: EFFECT OF CORN RESIDUE MANAGEMENT PRACTICES ON GOSS'S WILT AND LEAF BLIGHT OF CORN

ABSTRACT

Goss's wilt of corn which is caused by *Clavibacter michiganensis* subsp. *nebraskensis* (Cmn) is a reemerging bacterial disease of corn in Illinois. Since Cmn overwinters in corn debris, fields that have corn residue are more at risk for severe Goss's wilt outbreaks. With the current shift in farming practices towards continuing corn rotations in Illinois, farmers in Illinois face a higher risk of an epidemic occurring if proper corn residue management techniques are not developed. In order to understand the role of different types of residue management on Goss's wilt, a field study was conducted in the summers of 2012 and 2013 in a field near Urbana, Il. Three different tillage regimes consisted of moldboard plow, chisel plow and no- till were used on a field that historically has severe Goss's wilt. Ten different hybrids that were susceptible to Goss's wilt were planted. The severity and incidence were evaluated and the yields were determined. The incidence (%) and severity (0-9 scale) data were used to calculate a disease severity index (DSI), where DSI – (% incidence) X (severity) / 9. Plots that were no tilled had the greatest amount of residue in 2012 and 2013 (33% and 55% respectively), while plots that were moldboard plowed has the least amount of residue in both years (0 % and 2.5% respectively). Tillage had a significant effect on both DSI ($P = 0.0004$) and yield ($P = 0.0005$). The no-till treatment resulted in the greatest amount of residue in both years, and had a significantly higher DSI than either the chisel plow or moldboard plow treatments. The moldboard plow treatment resulted in the least amount of residue and a significantly lower DSI

than either the no-till or chisel plow treatments. These results have shown that limiting corn residue by using different tillage methods can be an effective way to manage Goss's wilt.

INTRODUCTION

Goss's wilt of corn (*Zea mays*) is caused by the bacterial pathogen *Clavibacter michiganensis* subspecies *nebraskensis* (Cmn). It was first observed in Nebraska in 1969 and was soon found in the surrounding states (Wysong et al., 1973). Eventually, corn breeders were able to develop resistant hybrids, and establishment of Cmn and Goss's wilt in the Midwestern Corn Belt appeared to be deterred. However, in the late 2000s, fields that were affected by Goss's wilt in the Midwest started to appear (Bradley, 2012; Jackson et al., 2007; Malvick et al., 2010; Ruhl et al., 2009).

Goss's wilt occurs as a leaf blight or a systemic wilt on susceptible hosts (Wysong et al., 1981). Symptoms can develop at any stage of corn development. Typical leaf blight symptoms include water-soaked, irregular lesions that can coalesce into larger lesions. Within these lesions, dark green to black spots that appear similar to "freckles" can be found. Bacterial exudates may also be observed on the leaf surface. As these exudates dry, they leave behind a shiny sheen on the leaf surface. Severe infections can lead to a systemic wilt. If the vascular tissue gets infected by Cmn, wilting and premature death can be observed. Stalks of severely affected plants can be split open to reveal pockets of orange to brown discolored vascular tissue. Cmn is spread by rain splashing of bacterial cells from corn residue on the soil up onto the plants. The bacteria can then enter the plant through wounds or natural openings.

Goss's wilt management practices include planting resistant hybrids, rotating to non-host crops, and tilling to reduce the amount of corn debris left on the soil surface. Tillage has long been a practice to help reduce risk of disease by removing pathogen-infested debris, which reduces primary inocula. Nevertheless, there has been an increase in conservation tillage practices due to many factors, including concerns over soil erosion and the rising cost of fuel (Sumner et al., 1981). Fields that use conservation or no-till practices are at higher risk for Goss's wilt, because Cmn overwinters in debris (Wysong et al., 1981). In fields that have severe Goss's wilt, rotating to a non-host crop and planting hybrids that are resistant to Goss's wilt will help manage the disease in the following years (Claflin, 1999). With recent economics favoring corn over other crops, the practice of planting continuous corn has become more prominent. Considering this, it is even more vital to manage residue to prevent the spread of disease. The objective of this study was to evaluate the effect of different amounts of Cmn-infested corn residue on the development of Goss's wilt in the field.

MATERIALS AND METHODS

Treatments and experimental design

Field research trials were conducted in the summers of 2012 and 2013 at the University of Illinois Crop Sciences Research and Education Center (CSREC) near Champaign, IL. The fields were located on the Cruse Tract of the CSREC in both years, but different fields were used each year. In the fall of 2011 and 2012, replicated blocks of tillage treatments, (no-till, chisel plow, or moldboard plow) were set up. Border rows with no residue on the soil surface were placed between different tillage blocks to minimize plot to plot contamination. Plots were

planted on 10 May 2012 and 6 June 2013 using an Almaco 360 research plot planter (Almaco Co., Nevada, IA). Seeds were planted at approximately 84,000 seeds/ha. Plots were 4 rows wide on 76 cm centers and 7.6 m long. Only the two middle rows were utilized for data collection.

Plots were arranged in a split plot design with 4 replications. The main plots consisted of no-till, chisel plow, or moldboard plow tillage treatments, and subplots consisted of 10 different hybrids susceptible to Goss's wilt. Hybrids were provided by different seed companies with an agreement to keep hybrid names confidential.

Inoculum production, inoculation technique

To set the trials up for the following year, the entire field was planted to a Goss's wilt susceptible corn hybrid and inoculated the preceding year for the study each year, so that the resulting residue after harvest would be colonized by Cmn. A Cmn isolate originally collected from a corn field in Illinois was used to inoculate plants for this study. Cmn was cultured in nutrient broth (Becton, Dickinson and Company, Franklin Lakes, NJ) in 1 liter Erlenmeyer flasks. The flasks were placed on a shaker table for 2 days at room temperature (between 23-25°C) under constant light (15 watt fluorescent bulbs). Plants were inoculated at approximately the V6 growth stage using the pinprick method (Blanco et al, 1977; Pataky, 1985).

Disease, residue, and yield assessments

Goss's wilt incidence and severity data were collected, and then used to calculate a disease severity index (DSI). The DSI was calculated by: (severity value × % incidence) / 9, which resulted in a value between 0 and 100. Each plant in the two middle rows of each plot

was scored for Goss's wilt severity. Since there were no border rows between tillage blocks in 2012, the first 5 and last 5 plants within each row were not counted toward the incidence and severity of a plot. Goss's wilt severity was rated when the corn reached the R1-R3 stage. A 1 to 9 scale was used to rate Goss's wilt severity (Suparyono and Pataky, 1989), in which 1 = no symptoms present; 2 = lesions with water soaking, freckles, chlorosis, and necrosis spread <6 cm from inoculation site; 3 = lesions spread appreciably toward the tip of the leaf from the inoculation site; 4 = lesions spread appreciably toward the base of the leaf from the inoculation site; 5 = limited systemic infection, with lesions appearing on non-inoculated leaves; 6 = 25-50% of total leaf area affected with severe leaf lesions, water soaking, chlorosis, necrosis, and stunting of the plant; 7 = 50-75% of total leaf area affected, and severe stunting of the plant; 8 = 75-90% of the total leaf area affected, with severe stunting of the plant; and 9 = plant death.

The amount of residue was estimated visually on a plot basis. The ratings were done using the photo-comparison method as described by Eck and Brown (2004). Ratings took place on 13 August 2012 and 30 August 2013.

Plots were harvested on 7 July 2012 and 30 August 2013 with a Kincaid 8-XP small plot combine (Kincaid Equipment Manufacturing, Haven, KS) equipped with a HarvestMaster grain gauge (Juniper Systems, Inc., Logan, UT) to calculate grain moisture and weight for each plot. Individual plot weights were adjusted to 15% moisture and yields were calculated to kg/ha.

Data were analyzed using both the mixed linear model procedure (PROC MIXED) and a Tobit model procedure in SAS (Version 9.3; SAS Institute Inc., Cary, NC). Tillage type was considered a fixed effect while year, replication, and hybrid were considered random effects. Differences of least square means were adjusted using the Tukey-Kramer method.

RESULTS

The main effect of tillage treatment was significant for DSI (P = 0.0004) and yield (P = 0.0005). The main effect of hybrid was not significant (P = 0.0914) for DSI, but was significant (P = 0.0003) for yield. The main effect of year was not significant (P = 0.8829) for DSI, but was significant (P = 0.0122) for yield. The tillage treatment × hybrid interaction was significant for both DSI (P = 0.01) and yield (P = 0.0001). The tillage treatment × year interaction was not significant for DSI (P = 0.09), but was significant for yield (P = 0.0001). The year × hybrid interaction was not significant (P = 0.4268) for DSI, but was significant (P = 0.0001) for yield. The three way interaction of tillage treatment × hybrid × year was not significant for DSI (P = 0.9876) or yield (P = 0.7189). Since hybrid and year were considered random effects, only the main effect of tillage treatment is presented. Pairwise comparisons were used in order to determine the significance of treatments.

Tillage treatments affected the amount of residue covering the soil. The no-till treatment resulted into the significantly greatest amount of residue (44.1%) (Table 2.1). The chisel plow treatment resulted into significantly less residue than the no-till treatment (10.4%), but greater residue than the moldboard plow treatment (1.3%). Tillage treatments also affected Goss's wilt DSI values, with the significantly greatest DSI observed in the no-till treatment (DSI = 1.5). The chisel plow treatment resulted into a significantly less DSI value than the no-till treatment (DSI = 0.6), but a significantly greater DSI value than the moldboard plow (DSI = 0.2). The greatest yield was observed with the moldboard plow treatment (7,407 kg/ha) (Table 2.1), which was not significantly different than the chisel plow treatment (7,079 kg/ha), but was significantly greater than the no-till treatment (6,336 kg/ha).

DISCUSSION

Among other recommended practices, deep plowing of infested debris is a practice recommended for management of Goss's wilt (Claflin, 1999). However, no previous research evaluating the effect of tillage of debris on Goss's wilt has been published to our knowledge. In our research, reducing infested debris with tillage resulted into lower levels of Goss's wilt. Since Cmn is known to overwinter in corn residues on the soil surface (Schuster and Coyne, 1974), our results were not surprising.

Although significantly less than other tillage treatments, Goss's wilt was still detected in the moldboard plow treatment. The moldboard plow treatment provided very low levels of residue coverage of the soil, with a mean value of 1.3% coverage. This indicates that Goss's wilt can still occur in a field with very low levels of Cmn-infested residue. In light of this finding, cultural management practices that impact initial inoculum levels, such as tillage and crop rotation, may not provide complete control of Goss's wilt if low levels of Cmn-infested residue are still present in a field. Therefore, in fields where Goss's wilt has occurred in the past, the management practice of planting hybrids with the highest levels of Goss's wilt resistance should be utilized along with crop rotation and possibly tillage.

REFERENCES

Bradley, C. A. 2012. Wilts, spots, blights and streaks: Old and emerging diseases of corn. Pages 18-21 in Proc. Univ. Illinois Corn Soybean Classics. http://extension.cropsci.illinois.edu/fieldcrops/classics/pdfs/2012.pdf.

Blanco, M. H., Johnson, M. G., Colbert, T. R., and Zuber, M. S. 1977. An inoculation technique for Stewart's wilt disease of corn. Plant Dis. Rep. 61:413-416

Claflin, L. E. 1999. Goss's bacterial wilt and blight. Pages 4-5 in: Compendium of Corn Diseases, 3rd Edition. D. G. White, ed. American Phytopathological Society, St. Paul, MN.

Eck, K, J., and Brown, D.E. 2004. Estimating corn and soybean residue cover. Agronomy Guide, West Lafayette, IN. AY-269-W.

Jackson, T. A., Harveson, R. M., and Vidaver, A. K. 2007. Reemergence of Goss's wilt and blight of corn to the central High Plains. Online. Plant Health Progress doi:10.1094/PHP-2007-0919-01-BR.

Malvick, D., Syverson, R., Mollov, D., and Ishimaru, C. A. 2010. Goss's bacterial blight and wilt of corn caused by *Clavibacter michiganensis* subsp. *nebraskensis* occurs in Minnesota. Plant Dis. 94:1064

Pataky, J. K. 1985. Relationships among reactions of sweet corn hybrids to Goss' wilt, Stewart's bacterial wilt, and northern corn leaf blight. Plant Dis. 69:845-848.

Ruhl, G., Wise, K., Creswell, T., Leonberger, A., and Speers, C. 2009. First report of Goss's bacterial wilt and leaf blight on corn caused by *Clavibacter michiganensis* subsp. *nebraskensis* in Indiana. Plant Dis. 93:841.

Schuster, M. L., and Coyne, D. P. 1974. Survival mechanisms of phytopathogenic bacteria. Ann. Rev. Phytopathol. 12:199-221.

Sumner, D. R., Doupnik Jr, B. and Boosalis, M. G. 1981. Effects of reduced tillage and multiple cropping on plant diseases. Ann. Rev. Phytopathol. 19:167-187.

Suparyono and Pataky, J. K. 1989. Influence of host resistance and growth stage at the time of inoculation on Stewart's wilt and Goss's wilt development and sweet corn hybrid yield. Plant Dis. 73:339-345.

Treat, C. L., and Tracy, W. F. 1990. Inheritance of resistance to Goss's wilt in sweet corn. J. Amer. Soc. Hort. Sci. 115:672-674.

Wysong, D. S., Doupnik Jr, B., and Lane, L. 1981. Goss's wilt and corn lethal necrosis—can they become a major problem? Pages 104-130 in: Proc. Annu. Corn Sorghum Res. Conf., 36th. Am. Seed Trade Assoc. Washington, DC.

Wysong, D. S., Vidaver, A. K., Stevens, H., and Stenberg, D. 1973. Occurrence and spread of an undescribed species of *Corynebacterium* pathogenic on corn in the western corn belt. Plant Dis. Rep. 57:291-294.

Table 2.1. Effect of tillage on corn residue coverage of soil, disease severity index of Goss's wilt, and corn yield in Champaign, IL. Means are averaged over 10 corn hybrids susceptible to Goss's wilt and over two environments (2012 and 2013).

Tillage treatment	**Residue coverage (%)[a]**	**Disease severity index (0-100)**	**Yield (kg/ha)**
No-till	44.1 A	1.5 A	6,336 B
Chisel plow	10.4 B	0.6 B	7,079 AB
Moldboard plow	1.3 C	0.2 C	7,407 A

[a]Means within a column followed by the same letter are not significantly different ($P \leq 0.05$) from each other.

CHAPTER 3: EVALUATION OF THE UNIVERSITY OF ILLINOIS PLANT PATHOLOGY INBRED MAIZE COLLECTION FOR RESISTANCE AGAINST GOSS'S WILT AND LEAF BLIGHT OF CORN

ABSTRACT

Goss's wilt of corn, caused by the bacterium *Clavibacter michiganensis* subsp. *nebraskensis* (Cmn), is a reemerging disease in the Midwestern United States. This reemergence is possibly due to an increasing amount of inoculum which comes from growing continuous maize with limited crop rotations, decreased amounts of tillage which leaves corn debris on the soil surface where the bacterium overwinters and the use of susceptible hybrids. The University of Illinois plant pathology inbred collection consists of over 2,000 inbred lines that have been collected from all over the world and maintained. An initial screen was conducted near Urbana, Il in 2011. These lines were inoculated with a Cmn cell suspension and rated for Goss's wilt severity using a 1-9 severity scale. The initial screen identified over 150 lines that had high levels of resistance. These lines were then used in a second stage of screening. From this second stage, 9 lines moved to the third stage. The mean Goss's wilt severity rating of the resistant lines in the last stage was 1.9, while the susceptible check had a mean score of 6.4. The most resistant lines identified from this research are potential sources of resistance to Goss's wilt, and could be used in maize breeding programs to develop Goss's wilt resistant hybrids.

INTRODUCTION

In 1969, Goss's wilt of corn (*Zea mays*), caused by the bacterial pathogen *Clavibacter michiganensis* subsp. *nebraskensis* (Cmn), was first observed in Dawson County, Nebraska (Wysong et al., 1973). In the following years, the spread of Goss's wilt was observed in the states surrounding Nebraska, such as Iowa, Kansas and South Dakota. By 1981, Goss's wilt had been found in 58 counties in Nebraska, and in Colorado, Illinois, Iowa, Kansas and South Dakota, and unconfirmed observations of Goss's wilt had been reported in Minnesota and Wisconsin (Wysong et al., 1981).

Goss's wilt is observed as a leaf blight and/or a systemic wilt on corn. Tan leaf lesions and, distinct, water-soaked spots will be observed. These spots, known as freckles, are dark green to black in appearance. As the lesions become larger and coalesce, they form blight symptoms on leaves (Wysong et al., 1981). Droplets of Cmn exudate may be seen on the surface of the leaf. As these droplets dry on the surface of the leaf, they leave behind a crystalline substance. This substance glistens in the presence of light. A plant systemically infected by Cmn may have pockets of orange bacterial exudates within the stalk of the plant (Wysong et al., 1981). The vascular bundles of the corn stalk will be discolored, and severely affected plants may have wet-rotted tissue present at the intermodal pith and nodal plates. This will result in a soft rot that is odiferous. Systemically-infected plants may show signs of drought stress rather than the typical blight symptoms because of the nature of the pathogen. Severe disease can cause up to 50% yield losses in fields (Wysong et al., 1981). Plants may be infected at any stage. Seedlings may die.

There are a variety of ways to manage Goss's wilt in the field, including planting resistant hybrids, utilizing rotations with non-host crops, and managing Cmn-infested debris in the field. Since Cmn overwinters in debris, managing debris through tillage or other methods may be a viable way to limit the initial inoculum in a field, but this is not practical in areas where conservation tillage is practiced. Rotating corn with non-host crops and using resistant hybrids have been shown to manage Goss's wilt (Claflin, 1999; Wysong et al., 1981). The most effective way to manage Goss's wilt is to use resistant hybrids. Corn breeders were successful in developing resistant hybrids of field corn shortly after the discovery of Goss's wilt (Jackson et al., 2007). Since the use of resistant hybrids, there have only been sporadic reports of Goss's wilt in the high plains of the United States. However, Goss's wilt can still be observed in fields that have been physically damaged by hail or high winds.

Although corn hybrids have been developed that have high levels of resistance to Goss's wilt, all sources of resistance may not yet be known. Some researchers have identified a few sources of resistance (Calub et al., 1974; Gardner and Schuster, 1973; Martin et al., 1975; Ngong-Nassah et al., 1992; Schuster et al., 1972; Treat and Tracy, 1990; Treat et al., 1990), but a comprehensive evaluation of a large germplasm collection has not been conducted. The objective of this study was to evaluate the University of Illinois Plant Pathology maize inbred collection for sources of resistance to Goss's wilt. This inbred collection has been utilized in the past to identify new sources of resistance to other diseases, such as, Aspergillus ear rot, Fusarium ear and kernel rot, and gray leaf spot (Campbell and White, 1995; Clements et al., 2004; Coates and White, 1994).

MATERIALS AND METHODS

Field Trials

Inbred lines from the University of Illinois maize inbred collection were screened for resistance to Goss's wilt in field trials located at the University of Illinois Crop Sciences Research and Education Center (CSREC) near Champaign and Urbana, IL. In 2011 and 2012, the research field was located on the Cruse Tract of the CSREC. In 2013, one field was utilized at the CFAR Tract of the CSREC. Corn was the previous crop in all fields.

The screening process was conducted in three stages over a period of three years. The first stage of screening was conducted in 2011. The purpose of this stage was to identify potential sources of resistance and to remove the most susceptible lines. In total, 1,316 inbred lines were selected to be screened from the inbred maize collection for the first stage. This was almost the entire collection, but excluded lines with a very limited seed supply. Plots were planted on 24 May 2011 with approximately 20 seeds per row. Each plot was 1 row, and plots were arranged in a randomized complete block design (RCBD) with two replications. Plots were 6.1 m long, and spaced 76 cm apart. Plants were inoculated with a Cmn isolate that was collected from a field in Champaign County, IL in 2010. Cmn inoculum production and plant inoculation procedures followed those reported by Pataky (1985). To initiate inoculum production, Cmn was cultured zin 1 liter Erlenmeyer flasks containing nutrient yeast broth at room temperature (between 23-25°C) under constant light (15 watt fluorescent bulbs). After 2 days, a spectrophotometer (SmartSpec 3000; Bio-Rad Lab., Hercules, CA) was used to determine Cmn cell concentration (OD_{600}), and were adjusted to 1×10^7 cfu/ml for inoculations. Plants were inoculated the first time at approximately the V4 growth stage (Ritchie et al., 1997) on 22 June,

and again 6 days later, using the pinprick method previously described by Blanco et al (1977), Calub et al. (1974) and Pataky (1985).

On 11 July, plots were rated for Goss's wilt severity using a 1 – 9 scale previously reported by Suparyono and Pataky (1989), where: 1 = no symptoms present at the inoculation site; 2 = <6 cm lesions present at the inoculation site, with water soaking, freckles, chlorotic and necrotic spread; 3 = >6 cm lesions spreading appreciably toward the tip of the leaf; 4 = lesions from the inoculation site spreading appreciably toward the base of the leaf; 5 = limited systemic infection, with lesions appearing on non-inoculated leaves; 6 = 25-50% of total leaf area is affected with leaf lesions, water soaking, chlorosis, necrosis and minimal stunting of the plant; 7 = 50-75% of the total leaf area is affected with water soaked lesions and freckles, chlorosis, necrosis and stunting of the plant; 8 = 75-90% of the total leaf area is affected with Goss's wilt with severe stunting; and 9 = plant death. Three ratings were obtained from three separate locations in each plot. The mean of these three ratings was calculated for each plot. Disease severity means for each inbred were used to sort the lines. Lines with a mean disease rating score of ≤2.5 were advanced to stage 2.

Of the 1,316 lines tested in the first stage, 177 lines were advanced to stage 2 for re-evaluation in the field in 2012. Stage 2 screening was conducted in the same manner as stage 1, except that 3 replications were utilized in stage 2. Plots were planted 10 May and inoculated on 12 and 18 June. Plots were rated for disease severity on 17 July using the methods previously described.

A third stage of screening was conducted in 2013, in which a subset of 10 inbred lines was selected to be screened against four different isolates of Cmn. This subset included 9 lines

considered to be resistant to Cmn based on the results from stages 1 and 2 (Fr36, Fr481, Fr4326, H101, 191-0477, 191-7323, 191-7387, 191-7388, and 191-7393), and 1 susceptible line (CQ 183). Cmn isolates consisted of the isolate from Illinois mentioned previously and 3 isolates from Nebraska (CN18-1, G2 and 13424). The isolates from Nebraska were chosen based on their genetic diversity (Agarkova et al., 2011), and were obtained from Dr. Anne Vidaver, University of Nebraska (Lincoln, NE). All methods were identical to those used in previous years except that 4 replications were utilized in stage 3. Plots were planted on 6 June, and plants were inoculated on 11 and 22 July. Unfortunately, the Cmn isolates from Nebraska did not cause disease symptoms on any of the resistant lines or the susceptible line. Therefore, only data from plots inoculated with the Illinois Cmn isolate were analyzed. Plots were rated on 20 August 2013 using the methods described previously.

Greenhouse trial

A greenhouse study was conducted to evaluate the effect of different inoculation methods on the resistance of the inbred lines. The 10 inbred lines used in the stage 3 field trial were used (9 resistant lines and 1 susceptible line). One seed of each line was planted in 3.8 liter, high density polyethylene pots containing a steam pasteurized potting mixture that consisted of 1:1:1 ratio of soil, peat and perlite. Growing conditions in the greenhouse included a 16-h photoperiod and day and night temps of 28°C, and pots were watered daily for the duration of the study. Pots were arranged in a RCBD with three replications, and the study was repeated once over time.

Inoculum production followed the same procedure previously described, and the Illinois Cmn isolate was used. Plants were inoculated approximately 20 days after planting (DAP). The

inoculation methods consisted of inoculating plants through the leaves, stems, or roots with 3 ml of a Cmn solution. When inoculating through the roots, 10 spots were chosen along the main root and injected with three ml of a Cmn cell suspension total. When inoculating through the stem, one spot was chosen approximately one inch above the soil line and injected with three ml of the Cmn solution. When inoculating through the leaves, the pinprick method previously described by Blanco et al (1977), and Pataky (1985) was used. Disease severity was rated approximately 35 DAP using the same 1-9 scale used for the field trials.

Data analysis

Data from stages 2 and 3 of the field trials were analyzed with the mixed model procedure (PROC MIXED) of SAS (Version 9.3; SAS Institute Inc., Cary, NC). Least square means were compared using PDMIX800 macro (Saxton, 1998) where $\alpha = 0.05$. Inbred line was considered a fixed effect, while replication was considered random.

Data for the greenhouse trial were analyzed using the mixed model procedure (PROC MIXED) in SAS. Least square means were compared using the PDMIX800 macro (Saxton, 1998) where $\alpha = 0.05$. Inoculation type and inbred line were considered fixed effects, while trial and replication were considered random.

Pedigree history analysis

The pedigree history of lines with known pedigrees was found in order to understand where resistance may have originated from. The pedigree history of 32 lines with 7 check lines was analyzed using KIN (Tinker and Mather, 1993). A dendrogram of pedigree distance was

constructed from the KIN output by using genetic dissimilarity (1 – CP) as a measure of genetic (pedigree) distance among all possible pairings of the 39 inbreds. The dendrogram tree was constructed using the software NTSYSpc (Rolf, 2007) using unweighted pair group method analysis (UPGMA) in the SAHN program module for clustering by pedigree distance followed by the construction of the dendrogram using NTSYXpc Tree Plot program module.

In order to gain understanding of the putative sources of Goss's wilt resistance, the same 39 lines were selected, and their genetic contribution (percent of genes by descent) from progenitor lines associated with Goss wilt resistance was estimated. KIN was used again on the same pedigree database mentioned earlier, but to facilitate genetic contribution from each respective progenitor, the database was analyzed separately for each progenitor with its respective parentage (ancestor contribution) set to zero. This enabled determination of each specific progenitor's genetic contribution to its progeny without comingling the contribution of its ancestors.

RESULTS

Field trials

The disease severity means ranged from 1 to 8.5 for stage 1 (Appendix A.1.), and overall mean and median disease severity values were 3.6 and 3.8, respectively. Although 266 lines had a score of 2.5 or lower (Fig. 1), only 177 lines were advanced to stage 2 because of low seed amounts.

For stage 2, the mean disease severity for the 174 lines tested ranged from 1.1 to 7.4 (Fig. 2), and the overall mean and median disease severity values were 2.9 and 2.8, respectively

(Table 3.1). Inbred line FR4326 has the lowest mean severity score (1.1), but it was not significantly different from the next 22 lines. Even though all these lines performed well in 2011, not all of them performed well in 2012. All the lines chosen for 2012 had a mean average score of 2.5 or lower. In 2012, 114 lines had an average mean score above 2.5, while only 60 lines had a mean average score of 2.5 or less. There were six lines (A654, KY209, CH74161, A188, N31 and CQ184A) that had a mean average score of about 4.5. Lines A188, N31 and CQ184A did significantly worse than any other lines, with mean severity scores of 6.6, 6.6 and 7.4, respectively.

For stage 3, the range of the disease severity ratings was 1.1 to 2.9 (Table 3.2). The overall mean disease severity ratings of the 9 resistant lines and the susceptible line was 1.9 and 6.4, respectively. These 9 lines had significantly ($P = 0.05$) lower disease rating scores than the susceptible line (Table 3.2). Inbred line FR4326 had the lowest mean severity score (1.2), but it was not significantly different than FR36, 191-7393, FR481 and 191-0477. The susceptible check CQ 183 had the highest mean severity rating (6.4), and was significantly different than all of the other inbred lines.

Greenhouse trial

The main effects of inbred line and inoculation type for the greenhouse trial were significant at $P < 0.0001$ and $P = 0.0010$, respectively for Goss's wilt severity. The interactive effect of inbred line × inoculation method was not significant ($P = 0.5041$).

Mean severities for inoculation types ranged from 1.6 to 3.2 (Table 3.3). Plants inoculated through the leaves or stems had the highest severity ratings, which were not

significantly different from each other, but were significantly greater than the disease severity of root-inoculated plants. Mean severities for inbred lines ranged from 1.3 to 7.0 (Table 3.4). The most resistant inbred was FR481, but it was not significantly different ($P = 0.05$) from the next seven inbred lines. H101 had significantly more disease than the best 8 lines, but it was also significantly better than the last line, CQ183, which also served as the susceptible check.

Pedigree history analysis

Thirty four inbred lines were analyzed for the pedigree distance, along with 5 lines that were inserted as pedigree check lines. Of the 39 lines that were analyzed for the pedigree distance (Fig. 3), 22 are from the same heterotic group, Lancaster Sure Crop, while only 5 are from a separate heterotic group, Iowa stiff Stalk Synthetic (BSSS). There were also 8 lines that came from miscellaneous heterotic groups not related to Lancaster or BSSS, including an Iodent group.

The same 34 inbred lines were analyzed for their genetic contribution of genes from a few putative sources associated with disease resistance. Of these 34 lines, pedigree line of K148, which had a mean disease score of 2.2, is 100% from Pride of Saline (Table 3.5) while Va15 and R802A both had Illinois High Oil genetic contributions. Eleven lines, including FR20A, FR36, FR812, LH51, LH55 and LH58, descended from MO17. Inbred FR4326, which had a mean disease score of 1.1, descended from both MO17 and OH40B.

DISCUSSION

In this research, several inbred lines that consistently showed resistance to Goss's wilt were observed. There have been few studies conducted on the reactions of inbred genotypes to Goss's wilt. Schuster et al. (1972) evaluated 100 corn inbreds and found that most of the inbreds were susceptible to Cmn. However, they did find some inbreds that they classified as tolerant and moderately susceptible. Of the 100 inbreds that Schuster et al. tested, 54 of them were evaluated in our initial evaluation in 2011 and only 9 of those inbreds moved to the 2^{nd} stage. There were several inbreds evaluated by Schuster et al. that we also identified as showing resistance and moved to the 2^{nd} stage of our inbred screen. These included C123, OH507, OH510, H88, B57, B73 and MO17. Of these lines, Schuster et al. only identified H88 as having resistance. Gardner and Schuster (1973) performed a series of experiments in the greenhouse and the field to test the tolerance and susceptibility of various lines and their resulting hybrids. Twenty three lines were tested in 2 diallel crosses in the greenhouse. They did not find any resistant lines, but found that some lines were more resistant than others. In the second greenhouse experiment, F_1, F_2 and backcrossed generations were tested. The plants were intermediate in their reactions and the ratings that were obtained were close to what was expected based on the additive model of genetic inheritance. The last experiment was a field study using the same lines used in the second greenhouse study. In general, it was found that the ratings in the greenhouse matched the field study, but the correlation coefficient between the greenhouse and the field study was only 0.55, indicating that the predictive values of the greenhouse study did not always match the field study. Calub et al. (1974) evaluated 113 corn genotypes in 3 experiments. Of these 113 genotypes, we evaluated 55 in our initial screen and 12

of the 55 moved onto the 2nd stage of testing. None of the lines they identified as resistant were included in our experiments. However, Calub et al. identified that 3 of their 4 most susceptible genotypes (B59, N124 and A632) were closely related to B14. We also identified that most of our resistant lines, in which pedigrees were available, were not related to B14 or BSSS. Wysong et al. (1981) tested 69 inbred corn lines. We tested 51 out of those 69 lines, and 8 of those lines moved onto the 2nd stage of our evaluation. The lines that had a low disease rating during our 2nd stage of testing and the testing done by Wysong et al. included MO13. However, the other 7 lines (A654, B73, C123, N31, NC230, SC213 and VA26) did not perform as well in 2012 as Wysong et al. reported. Treat et al. (1990) tested 39 different inbred genotypes. Of the 39 genotypes, we evaluated 21 in 2011 and 3 were chosen to be evaluated again in 2012. The top 2 lines (SDp310 and Mo17) that performed the best in Treat et al.'s experiment were 2 of the lines that moved onto the 2nd stage of testing. Treat et al. also conducted a study in which known susceptible and resistant lines were crossed. It was found that when MO17 was used as a parent, the resulting offspring were generally more resistant than when using other resistant parents. It was also found that no resistant inbreds were related to B14, which is a BSSS type. In addition, no inbreds in the C103 family, which is a Lancaster line, were found to be susceptible to Cmn. In our pedigree analysis, we found few lines that were related to the BSSS family, while there were many more lines related to the Lancaster family. Ngong-Nassah et al (1992) tested 10 inbred lines in a diallel cross. They found that, in general, resistant lines contributed resistance to the F_1 progeny, while susceptible lines contributed susceptibility to the F_1 progeny. However, in crosses that involved intermediate parents, the F_1 generation was less predictable in their behavior. The conclusions

drawn from this study indicated that resistance of the parental inbred lines for Goss's wilt is a good indicator of how the resulting hybrids will behave.

The exact mechanism of Goss's wilt resistance is not known. Gardner and Schuster (1973) found that there was probably more than one major gene locus that controlled the disease resistance in maize. Martin et al. (1975) hypothesized that the susceptibility and resistance to Cmn was quantitative and additive in nature. They also hypothesized that partial dominance was a factor in susceptibility. Treat et al. (1990) did a study crossing known susceptible and resistant lines. They found in their diallel crosses of various genotypes that the general combining ability (GCA) was much greater than the specific combining ability (SCA), indicating that the additive gene action was more important than dominance for resistance to Cmn. However, the importance of dominance depended on the cross and the environments. It was also found that when MO17 was used as a parent, the resulting offspring were generally more resistant than when using other resistant parents. Ngong-Nassah et al. (1992) found that the additive gene action is primary in importance to the inheritance of resistance to Goss's wilt. There were significant positive SCA effects in one of their field trials, indicating that nonadditive genetic effects interacted more with the environment than the additive genetic effects. This shows that evaluation for disease resistance in hybrids should take place over several environments to get a more precise estimate in how hybrids will react.

In recent times, there has been a shift in the genetic composition of commercial hybrids that are available to the public. Mikel and Dudley (2008) reported that much of the North American dent corn germplasm that is commercially available today comes from just 7 progenitor lines. This germplasm is then sorted into various heterotic groups, including Lancaster

Sure Crop, Iodent, and BSSS. A large portion of the inbred lines that were advanced in the screening field trials come from the Lancaster background. This Lancaster background appears to be a source of resistance to Goss's wilt, while there are less sources of resistance in lines that come from a BSSS or Iodent background.

A pedigree study with 1,132 U.S. plant variety protection (PVP) lines and/or utility patent registered corn inbred lines from 1984 to 2008 done by Mikel (2011) indicated that the genetic contribution of the public line Mo17, which has a Lancaster background, has decreased from 8.6% to 1.7%. There is some circumstantial evidence that resistance to Goss's wilt is in smaller progenitor groups, as opposed to the major groups that are used now. However, from the dendrogram that was produced, we see that K148 is more closely related to the Lancaster branch than the BSSS derived lines.

There have not been many studies done on the relation of the location of injury on the plant to infection. Most studies inoculate maize plants by puncturing either the stem or the leaves. In our greenhouse study, we found that Cmn infection will occur if the roots, stem or leaves of the maize plants are exposed to Cmn. Disease severity was actually significantly higher when inoculated through the roots, while the disease severity was not significantly different if inoculated through the stem or leaves. In either case, the resistant plants still showed resistance, while the susceptible check was still susceptible, indicating that resistance will still be effective, regardless of where the plant is infected. One study done by Schuster (1972) tested various means of infection, including through the roots. Schuster found that seeds that were germinated on petri dishes and exposed to Cmn as the root grew did not display any symptoms of Goss's wilt on the leaves, even though there was evidence of Cmn bacterial growth between the pericarp

and the ruptured coleorhiza and between the coleorhiza and the primary root. In Schuster's greenhouse trial, the roots of maize plants were wounded and exposed to Cmn. These wounded plants did show symptoms of Goss's wilt on the leaves of the wounded plants. This study agrees with our findings that the roots of the maize plants can be infected with Cmn if the roots are wounded. However, there needs to be more studies done on infection occurring though natural openings or wounds in the growing roots.

The goal of this project was to identify inbred lines that displayed potential sources of resistance to Cmn. While we did identify sources of resistance in the inbred collection, unfortunately many of the inbred lines used in this study come from unknown pedigrees. Future work using these lines could include genotyping these lines and finding the relationship between lines.

The University of Illinois inbred maize collection has sources of genetic resistance to Goss's wilt. Throughout our evaluation, we were able to screen out lines that were susceptible to Cmn, and were able to identify potential sources of resistance for future breeding programs. Further studies should also be done to investigate the infection process of Cmn and Goss's wilt development. We have shown that plants can be infected through other parts of the plant besides the leaves. Although host resistance was shown to work regardless of how the plant was inoculated, Goss's wilt severity was significantly less when plants were inoculated through the roots than either the stem or leaves, showing that there may be some unknown defense that the corn has when infected through the roots. Finally, we were able to construct a dendrogram showing the genetic relationship of resistant lines with known pedigrees. This will be able to help future breeding programs decide where sources of resistance should be found according to

maize pedigree. Future programs should focus on looking at either PVP line progenitors, commercial lines or other inbred collections for potential sources of resistance. A study done on current corn hybrids could give an idea of the vulnerability of today's germplasm.

REFERENCES

Agarkova, I. V., Lambrecht, P. A., and Vidaver, A. K. 2001. Genetic diversity and population structure of *Clavibacter michiganensis* subsp. *nebraskensis*. Can. J. Microbiol. 57:366-374

Blanco, M. H., Johnson, M. G., Colbert, T. R., and Zuber, M. S. 1977. An inoculation technique for Stewart's wilt disease of corn. Plant Dis. Rep. 61:413-416

Calub, A. G., Schuster, M. L., Compton, W. A., and Gardner, C. O. 1974. Improved technique for evaluating resistance of corn to *Corynebacterium nebraskense*. Crop Sci. 14:716-718.

Campbell, K. W., and White, D. G. 1995. Evaluation of corn genotypes for resistance to Aspergillus ear rot, kernel infection and aflatoxin production. Plant Dis. 79:1039-1045.

Claflin, L. E. 1999. Goss's bacterial wilt and blight. Pages 4-5 in: Compendium of Corn Diseases, 3rd Edition. D. G. White, ed. American Phytopathological Society, St. Paul, MN.

Clements, M. J., Maragos, C. M., Pataky, J. K., and White, D. G. 2004. Sources of resistance to fumonisin accumulation in grain and Fusarium ear and kernel rot of corn. Phytopathology 94:251-260.

Coates, S. T., and White, D. G. 1994. Sources of resistance for grey leaf spot of corn. Plant Dis. 78:1153-1155.

Gardner, C. O., and Schuster, M. L. 1973. Genetic studies of susceptibility to bacterial leaf freckles and wilt, *Corynebacterium nebraskense*. Maize Genet. Coop. Newsl. 47:155-157.

Jackson, T. A., Harveson, R. M., and Vidaver, A. K. 2007. Reemergence of Goss's wilt and blight of corn to the central High Plains. Online. Plant Health Progress doi:10.1094/PHP-2007-0919-01-BR.

Martin, P. R., Gardner, C. O., Calub, A. G., and Schuster, M. L. 1975. Inheritance of susceptibility and tolerance to leaf freckles and wilt (*Corynebacterium nebraskense*) of corn. Maize Genet. Coop. Newsl. 49:173-138.

Mikel, M. A. 2011. Genetic composition of contemporary U.S. commercial dent corn germplasm. Crop Sci. 51:592-599.

Mikel, M. A., and Dudley, J. D. 2006. Evolution of North American dent corn from public to proprietary germplasm. Crop Sci. 46:1193-1205.

Ngong-Nassah, E. N., Carson, M. L., Wicks III, Z. W. 1992. Inheritance of resistance to leaf freckles and wilt caused by *Clavibacter michiganense* subsp. *nebraskense* in early maturing maize inbred lines. Phytopathology. 82:142-146.

Pataky, J. K. 1985. Relationships among reactions of sweet corn hybrids to Goss' wilt, Stewart's bacterial wilt, and northern corn leaf blight. Plant Dis. 69:845-848.

Ritchie, S.W. and Hanway, J. J. 1997. How a corn plant develops. Special Report No. 48, Iowa State University, CES, Ames. IA.

Rolf, F. J. 2007. NTSYSpc: numerical taxonomy system. Version 2.20n. Exeter Publishing, Setauket.

Saxton, A. M. 1998. A macro for converting mean separation output to letter groupings in Proc Mixed. Pages 1243-1246 in: Proc. 23rd SAS Users Group International.

Schuster. M., L. 1972. Leaf freckles and wilt, a new corn disease. Pages 176-191 in: Proc. Annu. Corn Sorghum Res. Conf. 27th.

Suparyono and Pataky, J. K. 1989. Influence of host resistance and growth stage at the time of inoculation on Stewart's wilt and Goss's wilt development and sweet corn hybrid yield. Plant Dis. 73:339-345.

Tinker, N.A., and D.E. Mather. 1993. KIN: Software for computing kinship coefficients. J. Hered. 84:238.

Treat, C. L., Tracy, W. F., Drolsom, P. N., Coors, J. G. 1990. Inheritance of resistance to Goss's wilt in maize. Crop Sci. 30:893-896.

Wysong, D. S., Doupnik Jr., B., and Lane, L. 1981. Goss's wilt and corn lethal necrosis—can they become a major problem? Pages 104-430 in Proc. Annu. Corn Sorghum Res. Conf., 36th. Am. Seed Trade Assoc

Wysong, D.S., A.K. Vidaver, H. Stevens, and D. Stenberg. 1973. Occurrence and spread of an undescribed specie of Corynebacterium pathogenic on corn in the western corn belt. Plant Dis. Rep. 57:291-294.

Table 3.1. Mean Goss's wilt severity ratings for 177 corn inbred lines screened in a field trial near Champaign, IL in 2012.

Inbred line	Mean severity rating[a]	Inbred line	Mean severity rating	Inbred line	Mean severity rating	Inbred line	Mean severity rating
FR4326	1.1	191-7323	2.4	WCI141	2.8	B46	3.3
191-7393	1.6	FR2142	2.4	FR1128	2.9	B73	3.3
R802A	1.6	K4	2.4	FR303	2.9	FR802	3.3
MO13	1.7	OH40B	2.4	FR6	2.9	K809	3.3
O61	1.7	R805	2.4	H10	2.9	LH18	3.3
Y4Y1	1.7	485.0	2.4	SDP311	2.9	SC301E	3.3
OH561	1.8	H101	2.4	FR22	2.9	VR1	3.3
K720	1.8	LH118	2.4	M14N160	2.9	A165	3.4
H116	1.9	LH22	2.4	N8A	2.9	VA7	3.4
191-0477	2.0	LH55	2.4	N8B	2.9	ChHaK14	3.4
191-7330	2.0	OH510	2.4	OH563	2.9	PA32	3.4
191-7331	2.0	PA7317	2.4	OH91613	2.9	A652	3.6
191-7387	2.0	Y4	2.4	PI430478	2.9	CK25	3.6
FR36	2.0	B1498	2.5	C114	3.0	FR554	3.6
J826	2.0	FR825	2.5	C123	3.0	SC213	3.6
LH58	2.0	LH53	2.5	FR21Sel	3.0	75R004ReRu1	3.7
MO17	2.0	NC258	2.6	FR824	3.0	A662	3.7
N613	2.0	NC264	2.6	FRB73	3.0	CH592132	3.7
SP332	2.0	SDP060	2.6	J848	3.0	CH59317	3.7
VA20	2.0	W406A	2.6	LH48	3.0	CQ207	3.7
Y5	2.0	FR20	2.6	LH63	3.0	B1138Tsel	3.8
191-7388	2.1	H110	2.6	MO16W	3.0	FR460	3.8
FR812	2.1	M14N225	2.6	R75	3.0	MBS1032	3.8
25744.0	2.2	B57	2.7	SDP310	3.0	R216A	3.8
191-7339	2.2	BC577	2.7	SP264	3.0	CQ174	3.9
Cl30	2.2	ChHa485	2.7	VA59	3.0	H56	3.9
FR20A	2.2	CK54	2.7	B164	3.1	FR809	4.0
FR33	2.2	Cl27	2.7	CQ191	3.1	CH593132	4.1
OH507	2.2	Cl38	2.7	KY27	3.1	Cl42A	4.1
Va21	2.2	CQ190	2.7	LH130	3.1	CH20	4.2
FR18Sel	2.2	EX466	2.7	MBS18	3.1	LNAB	4.2
FR481	2.2	FR810	2.7	R222	3.1	CH5939	4.3
FR992	2.2	ILA	2.7	AR42	3.1	N7A	4.3
LH51	2.2	LH120	2.7	B40	3.1	VA26	4.3
W406	2.2	LH47	2.7	D714	3.1	CQ171	4.4
FR515	2.2	M14NR	2.7	E2558W	3.1	VA10	4.4
K148	2.2	MASGTgk	2.7	FR564	3.1	A654	4.6
NC250	2.2	MOG1253	2.7	Troyer106	3.1	Ky209	4.6
B149W	2.3	PB78	2.7	SDP309	3.2	CH74161	5.7
Cl82B	2.3	SD25	2.7	Cl38B	3.2	A188	6.6
EE5647	2.3	CQ181	2.8	NC230	3.2	N31	6.6
FR103D	2.3	K44	2.8	R146	3.2	CQ184A	7.4

Table 3.1. (Cont.)

Inbred line	Mean severity rating[a]	Inbred line	Mean severity rating	Inbred line	Mean severity rating	Inbred line	Mean severity rating
H88	2.3	N611	2.8	W703	3.2		
VA15	2.3	SDP312	2.8	A158	3.3	LSD 0.05	1.0

[a]Goss's wilt severity was rated using a 1 to 9 scale, where 1 = no symptoms and 9 = plant death.

Table 3.2. Mean Goss's wilt severity ratings for 10 corn inbred lines screened in a field trial near Urbana, IL in 2013.

Inbred line	Mean severity rating[a]
Fr4326	1.2 A
Fr36	1.3 A
191-7393	1.3 A
Fr481	1.7 AB
191-0477	1.8 AB
191-7388	2.2 BC
191-7387	2.3 BC
H101	2.8 BC
191-7323	2.9 C
CQ183	6.4 D

[a]Goss's wilt severity was rated using a 1 to 9 scale, where 1 = no symptoms and 9 = plant death.

Table 3.3. Effect of different *Clavibacter michiganensis* subsp. *nebraskensis* inoculation methods used in the greenhouse on Goss's wilt severity of corn inbred lines. Means are averaged over two trials and 10 corn inbred lines.

Inoculation method[a]	Mean severity rating[b]
Root	1.6 A
Leaf	2.8 B
Stem	3.2 B

[a]Corn inbred lines were inoculated with a *C. michiganensis* subsp. *nebraskensis* cell suspension through roots, stems, or leaves.
[b]Goss's wilt severity was rated using a 1 to 9 scale, where 1 = no symptoms and 9 = plant death.

Table 3.4. Goss's wilt severity ratings of 10 corn inbred lines inoculated in the greenhouse. Means are averaged over two trials and three different inoculation methods.

Inbred line	Mean severity rating[a]
Fr481	1.33 A
191-7393	1.56 A
191-7387	1.61 A
191-7388	1.83 A
191-0477	2.00 A
Fr4326	2.11 A
191-7323	2.12 A
Fr36	2.28 A
H101	3.72 B
CQ183	7.00 C

[a]Goss's wilt severity was rated using a 1 to 9 scale, where 1 = no symptoms and 9 = plant death.

Table 3.5. Percent genetic contribution of genes from each putative source associated with disease resistance

Inbred	Pride Of Saline	IL high oil	OH40B	C103[a]	CI187-2[a]	Krug[a]	MO17[a]	CI90A	FR4326
FR4326	0	0	13	22	22	22	44	0	100
R802A	0	13	0	0	5	5	0	0	0
MO13	0	0	0	0	0	0	0	0	0
OH561	0	0	0	0	3	3	0	0	0
K720	0	0	0	0	0	0	0	0	0
H116	0	0	0	44	0	0	0	0	0
LH58	0	0	0	38	50	50	75	0	0
FR36	0	0	13	22	22	22	44	0	100
MO17	0	0	0	50	50	50	100	0	0
VA20	0	0	75	0	0	0	0	0	0
FR812	9	0	0	75	0	0	0	0	0
LH51	0	0	0	50	50	50	100	0	0
K148	100	0	0	0	0	0	0	0	0
NC250	0	0	0	0	5	5	0	0	0
CI30	0	0	0	22	22	22	44	0	0
FR20A	0	0	0	44	44	44	88	0	0
FR33	0	0	0	50	0	0	0	0	0
VA21	0	0	0	0	0	0	0	0	0
FR18	0	0	13	0	0	0	0	0	0
FR481	0	0	0	25	25	25	50	0	0
FR992	0	0	0	0	4	4	0	0	0
W406	0	0	38	25	0	0	0	0	0
C103[b]	0	0	0	100	0	0	0	0	0
H88	0	0	25	0	0	0	0	0	0
CI82B	0	0	0	0	0	0	0	0	0
VA15	0	50	0	0	0	0	0	0	0
OH40B	0	0	100	0	0	0	0	0	0
K4	0	0	0	0	0	0	0	0	0
LH55	0	0	0	38	50	50	75	0	0
H101	0	0	25	0	0	0	0	25	0
R805	0	0	0	0	0	0	0	0	0
LH22	25	0	0	38	0	0	0	0	0
LH53	0	0	0	50	50	50	100	0	0
FR825	19	0	13	38	13	13	25	0	0

[a]Pedigree redundancy in that MO17 is from the biparental cross C103 / CI187-2, and CI187-2 is derived from Krug.
[b]FR1103D in screen set is an *Ht* isoline conversion of C103.

Figure 3.1. Frequency distribution of Goss's wilt severity ratings for 1,316 corn inbred lines screened in a field trial located near Champaign, IL in 2011. Disease severity was rated using a 1 to 9 scale, where 1 = no symptoms and 9 = plant death.

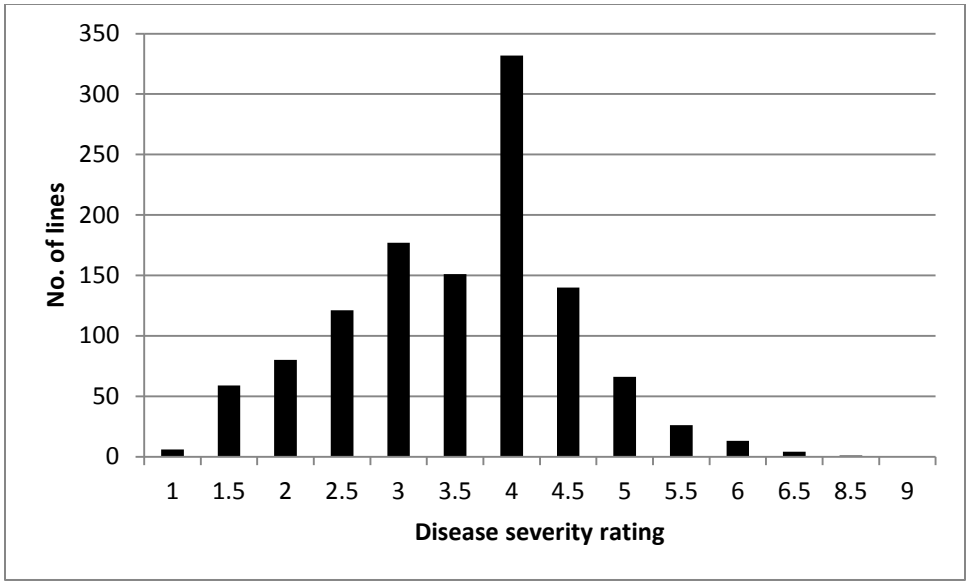


Figure 3.2. Frequency distribution of Goss's wilt severity ratings for 177 corn inbred lines screened in a field trial located near Champaign, IL in 2012. Disease severity was rated using a 1 to 9 scale, where 1 = no symptoms and 9 = plant death.

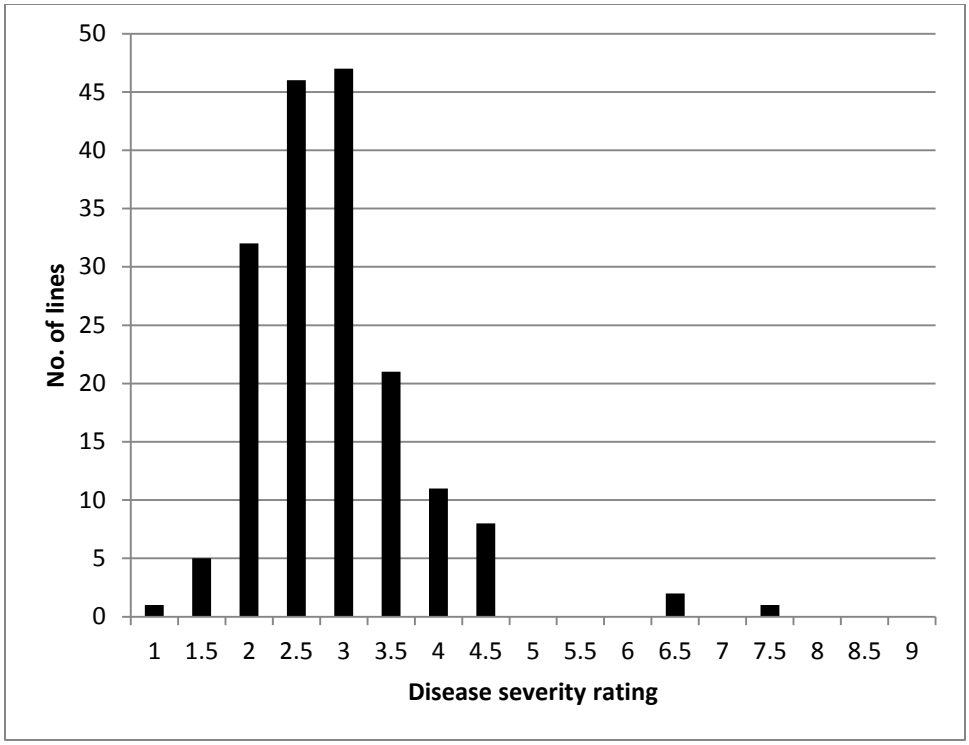
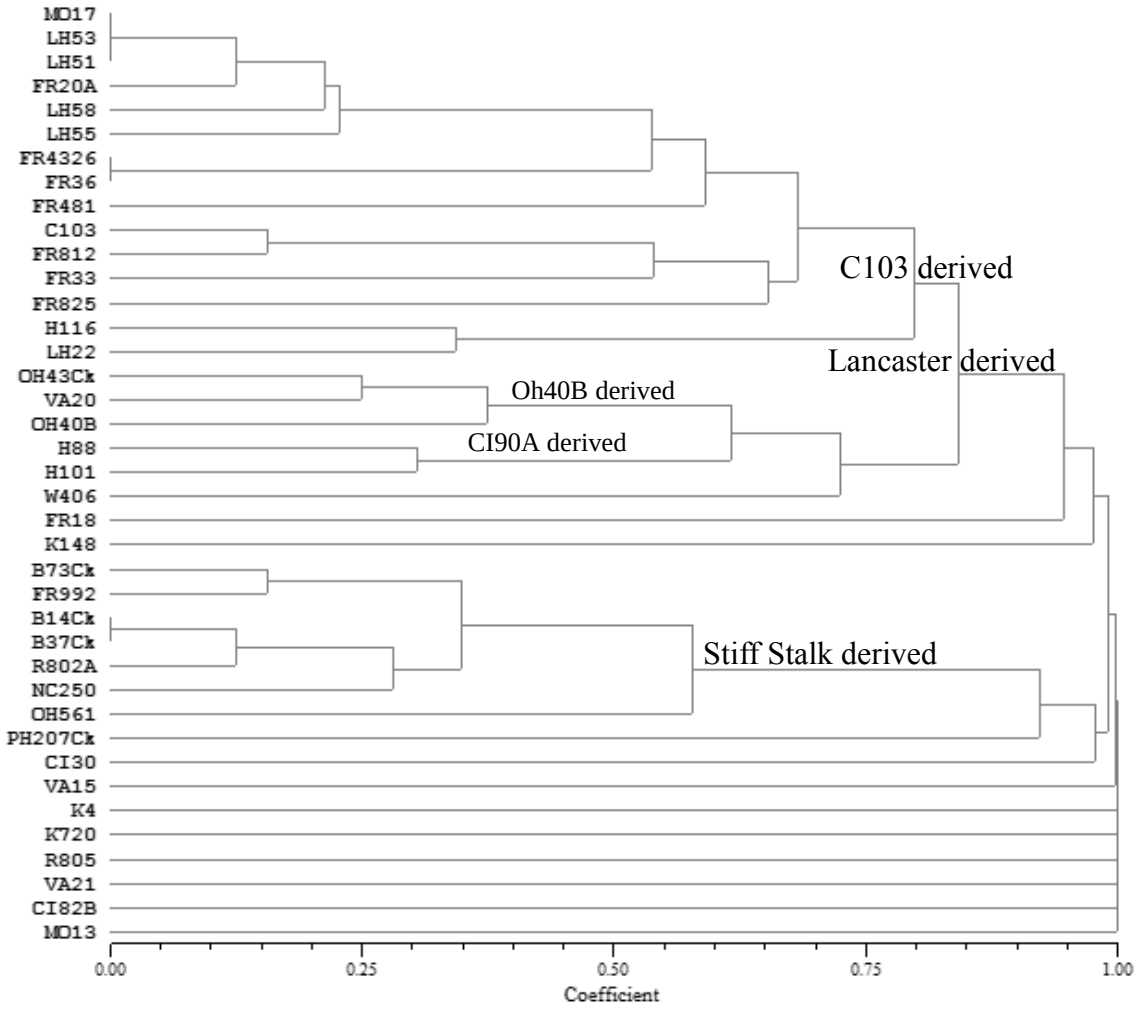


Figure 3.3. Pedigree distance of Goss's wilt resistant lines with known pedigrees[a]



[a]Includes five inserted check lines with known pedigrees, indicated by Ck.

APPENDIX A

Table A.1. Mean severity ratings of Goss's wilt for 1316 inbred lines in 2011.

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
191-0477	1	Pa33	3	J769	3.75	Ay307	4.25
Ch-Ha-K14	1	Pa36	3	J770	3.75	Ay308	4.25
FR824	1	Pa74-14	3	J803	3.75	B52	4.25
FR36	1.25	Pa74-2	3	J849	3.75	CG14	4.25
FR812	1.25	Pa884p	3	J856	3.75	CG4	4.25
Ky209	1.25	PI430473	3	K14	3.75	CK24	4.25
191-7339	1.5	PI430475	3	K55	3.75	Cl116	4.25
191-7387	1.5	Q705	3	K61	3.75	CM234	4.25
191-7388	1.5	R177	3	K9390	3.75	COMSTOCK FLINT	4.25
191-7393	1.5	R220	3	KTM-2-1-1	3.75	CQ203	4.25
A188	1.5	S48	3	Ky39	3.75	E43-26	4.25
A503NR	1.5	SP256	3	Ky55-562	3.75	EX196	4.25
B149W	1.5	SP304	3	L304Af	3.75	F481	4.25
C103D	1.5	Va31	3	LH72	3.75	F542	4.25
CH593-9	1.5	Va32	3	LH74	3.75	FR151	4.25
Cl30	1.5	Va46	3	M179	3.75	FR153R	4.25
E2558W	1.5	Va58	3	M182	3.75	FR153Sel	4.25
FR303	1.5	Va71	3	M189	3.75	FR19	4.25

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
FR481	1.5	VR4	3	M848W	3.75	FR2128	4.25
FR803W	1.5	W22R	3	MBS501	3.75	FR41	4.25
FR809	1.5	W59E	3	MEB164	3.75	FRO88	4.25
FR4326	1.5	W61	3	MO6	3.75	H5	4.25
H10	1.5	W9	3	MO9	3.75	H52	4.25
H110	1.5	WGI 1-41	3	MOL3	3.75	H55	4.25
H116	1.5	WV7	3	MS72	3.75	HY	4.25
K148	1.5	Y3	3	MS73	3.75	J740	4.25
LH48	1.5	Y8	3	MS80	3.75	K64	4.25
LH53	1.5	ZPL-22	3	N28	3.75	Laguna	4.25
LH55	1.5	1434	3.25	NC234	3.75	LB31	4.25
LH58	1.5	A1012W	3.25	NC350	3.75	LH132	4.25
MO17	1.5	A25	3.25	ND283	3.75	M162W	4.25
O61	1.5	A308	3.25	NYH1	3.75	MI904	4.25
OH26	1.5	A334	3.25	NYN40	3.75	MI915	4.25
OH507	1.5	A34	3.25	O7	3.75	MI925	4.25
OH562	1.5	A357	3.25	OH513	3.75	MICH-1	4.25
OH563	1.5	A629	3.25	OH56	3.75	MO10	4.25
Pa86	1.5	A649	3.25	OMVO89C	3.75	MO3	4.25
PI430478	1.5	A650	3.25	Pa76-1	3.75	MS128	4.25
SP264	1.5	A657	3.25	Pioneer2	3.75	MS142	4.25
Va15	1.5	Ay305	3.25	R175	3.75	MS92	4.25
Va21	1.5	B80	3.25	R182	3.75	N38	4.25
W406A	1.5	B85	3.25	R188	3.75	ND203	4.25

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
Y4	1.5	CH576-3	3.25	R217	3.75	ND230	4.25
Y4Y1	1.5	CH732-12	3.25	S61	3.75	ND481	4.25
485	1.75	Cl2	3.25	SC301D	3.75	ND486	4.25
C114	1.75	Cl90A	3.25	SD15	3.75	ND77	4.25
CH20	1.75	CM139	3.25	SD26	3.75	NY2	4.25
Ch-Ha-485	1.75	CM64	3.25	SD9	3.75	OH43B	4.25
Cl82B	1.75	Dan705	3.25	SDP031	3.75	OH67-1044	4.25
FR20A	1.75	DS:74:1032	3.25	SDP034	3.75	Pa349	4.25
FR33	1.75	DS:74:1071	3.25	SDP084	3.75	Pa351	4.25
FR515	1.75	DS:74:1074	3.25	SDP211	3.75	PI430508	4.25
H88	1.75	F534	3.25	SDP254	3.75	Pride of Saline	4.25
K4	1.75	FR1141	3.25	SDP264	3.75	Q188	4.25
LH51	1.75	FR13	3.25	SP223	3.75	R101	4.25
M14	1.75	FR25	3.25	SP288	3.75	R157	4.25
MAS(GT)gk	1.75	FR27rhm	3.25	SP293	3.75	R181	4.25
MBS1032	1.75	FR3	3.25	SP306	3.75	R193	4.25
N8B	1.75	FR495	3.25	SP312	3.75	R197	4.25
N613	1.75	FR6019	3.25	SPA321	3.75	R87	4.25
R216A	1.75	H21	3.25	SSDL12	3.75	R902	4.25
R222	1.75	H23	3.25	SZV66	3.75	R95	4.25
R805	1.75	H84	3.25	T82	3.75	R98	4.25
Va6	1.75	HMz850	3.25	TEX2	3.75	S304A	4.25
Va59	1.75	I69	3.25	TR07	3.75	SC212M	4.25
191-7331	2	IA1263	3.25	TR227	3.75	SC375	4.25

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
A165	2	IF192	3.25	U350Y	3.75	SD29	4.25
B73	2	IL Hullless	3.25	V7	3.75	SD48	4.25
BC577	2	Jinhuan 795-75	3.25	Va43	3.75	SP226	4.25
C123	2	K9214	3.25	Va50	3.75	SP233	4.25
CH593-17	2	KP58K	3.25	Va91	3.75	SP249	4.25
CQ174	2	KTM1	3.25	VR5	3.75	SP297	4.25
CQ181	2	KTX3101	3.25	VR6	3.75	SP300	4.25
CQ207	2	Ky216	3.25	VR7	3.75	T244	4.25
FR18Sel	2	Ky225	3.25	W16	3.75	T498	4.25
FR20	2	L289	3.25	W59M	3.75	TBA76085	4.25
FR103D	2	LH82	3.25	90	4	TBA76124	4.25
FR564	2	Long Kan II	3.25	187	4	Va85	4.25
FR802	2	MI901	3.25	407	4	Zongxi31	4.25
FR810	2	MICH-3	3.25	4-8	4	191	4.5
FR825	2	MICH-75-6	3.25	67242	4	902	4.5
FR1128	2	MO11	3.25	33-16	4	5003	4.5
FR2142	2	MS211	3.25	38-11	4	187-2	4.5
H100	2	MS214	3.25	4Co82	4	A272	4.5
H103	2	MS4	3.25	61M	4	A295	4.5
IL'A'	2	N103	3.25	75-R002(Ru10)	4	A340	4.5
J826	2	N6	3.25	75-R003(Ru11)	4	A385	4.5
J848	2	NC283	3.25	75-R006(Ru9)	4	A427	4.5
LH63	2	ND302	3.25	82-R2597	4	A628	4.5
LH120	2	OH4C	3.25	83-R2324	4	A635	4.5

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
MO13	2	OH512	3.25	A	4	A636	4.5
MOG-125-3	2	OH551	3.25	A131	4	A640	4.5
NC258	2	OH565	3.25	A148	4	B14A	4.5
OH45B	2	OH84	3.25	A17	4	BC488	4.5
OH91613	2	OS426	3.25	A171	4	C105	4.5
Pa73-17	2	Pa70	3.25	A21	4	C14-8	4.5
PB78	2	Pa73-3	3.25	A223	4	C153	4.5
R153	2	Pa75-4	3.25	A258	4	CG13	4.5
R802A	2	Pa76-7	3.25	A293	4	CH581-13	4.5
SC301E	2	PI194387	3.25	A310	4	CH701-36	4.5
SD25	2	PI427109	3.25	A311	4	CH732	4.5
SDP060	2	PI430474	3.25	A312	4	CK22	4.5
SDP310	2	R2040	3.25	A344	4	CM169	4.5
SP332	2	SDP035	3.25	A392	4	CO158	4.5
Va5	2	SP316	3.25	A441-5	4	CQ202	4.5
Va7	2	SQ18	3.25	A632	4	CQ708	4.5
W406	2	T14	3.25	A638	4	F234	4.5
Y5	2	TR206	3.25	A639	4	F39	4.5
150	2.25	Va79-419-1-9	3.25	A641	4	F502	4.5
191-7330	2.25	VR3	3.25	A648	4	F546	4.5
A158	2.25	VR9	3.25	A665	4	F575	4.5
AR42	2.25	W22	3.25	A667	4	F59B	4.5
B46	2.25	W51A	3.25	A668	4	FR1454	4.5
B164	2.25	ZPL-11	3.25	A669	4	FR43	4.5

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
B1138 Tsel	2.25	1596	3.5	A68-6	4	FR619	4.5
CH593-13-2	2.25	4722	3.5	A77-8	4	FR634	4.5
Cl38B	2.25	67232	3.5	A90	4	FR635	4.5
Cl42A	2.25	67236	3.5	A96	4	GT112	4.5
CK54	2.25	191-7340	3.5	Ay499	4	H46	4.5
CQ184A	2.25	5120B	3.5	Ay560	4	H91	4.5
CQ191	2.25	75-R007 (chin1*11)	3.5	B14	4	K150	4.5
FR460	2.25	75-R011	3.5	B16	4	LH38Block	4.5
FR554	2.25	A286	3.5	B54	4	M16	4.5
FR992	2.25	A375	3.5	B58	4	MO14W	4.5
FRB73	2.25	A395	3.5	B63	4	MO75:736	4.5
H101	2.25	A655	3.5	B65	4	MP313E	4.5
LH118	2.25	A659	3.5	B70	4	N101	4.5
LH130	2.25	A663	3.5	B82y	4	N139	4.5
LNAB	2.25	A682	3.5	B83	4	N159	4.5
M14N(160)	2.25	A71	3.5	B9	4	N162	4.5
M14N(225)	2.25	B2	3.5	C14	4	N24	4.5
MBS18	2.25	B41	3.5	C17	4	N8	4.5
MI923	2.25	B56	3.5	CB59L	4	N9	4.5
N7A	2.25	B69	3.5	CG10	4	ND468	4.5
N8A	2.25	B78	3.5	Ch Q31	4	ND483	4.5
NC250	2.25	B79	3.5	CH591-36	4	ND77-1	4.5
OH40B	2.25	C106	3.5	CH606-11	4	NY327	4.5

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
OH510	2.25	C107	3.5	CH661-17	4	OH514	4.5
Pa32	2.25	CG1	3.5	CH671-28	4	OH91653	4.5
R75	2.25	Ch-Ha-Long Kang 11	3.5	Ch-Ha-K10	4	Pa73-13	4.5
SDP309	2.25	Ch-Ha-Lou Ji	3.5	Cl187-2	4	Pa77-34	4.5
SDP311	2.25	Ch-Ha-Ql Ji 947	3.5	Cl5	4	PI430487	4.5
Troyer106	2.25	CHIMERA	3.5	Cl61	4	PR1	4.5
Va24	2.25	Cl1	3.5	Cl64	4	PRC517	4.5
VR1	2.25	Cl28A	3.5	CM182	4	Q83W	4.5
75-R004(ReRu1)	2.5	Cl4-8	3.5	CM42	4	R129	4.5
191-7323	2.5	Cl540	3.5	CM51	4	R218	4.5
340	2.5	CM7	3.5	CM74	4	R218A	4.5
25744	2.5	CQ176	3.5	CMV3	4	R30	4.5
A251	2.5	CQ209	3.5	CO235	4	R43	4.5
A652	2.5	D26	3.5	CQ169	4	R74	4.5
A654	2.5	De811	3.5	CQ179	4	R804	4.5
A662	2.5	De819	3.5	CQ186A	4	R84	4.5
B40	2.5	De833	3.5	CQ188	4	R909	4.5
B57	2.5	Dong 237	3.5	CQ197	4	S444	4.5
B66	2.5	DRS-1	3.5	CQ199	4	SA2	4.5
B1498	2.5	DS:74:1054	3.5	CQ201	4	SD102	4.5
C103	2.5	E1053	3.5	CQ205	4	SD30	4.5
CH592-13-2	2.5	EX556	3.5	CQ212	4	SDP001	4.5
CH741-6-1	2.5	F574FIANCE	3.5	CQ709	4	SDP016	4.5

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
Cl27	2.5	FF1	3.5	CV3	4	SDP2	4.5
Cl38	2.5	FR49	3.5	De815	4	SDP2A	4.5
CK25	2.5	FR4B	3.5	De848	4	SEY76138	4.5
CQ171	2.5	FR814	3.5	Duohuang8	4	Shen 5003	4.5
CQ190	2.5	H111	3.5	E2891	4	SP222	4.5
D71-4	2.5	H98	3.5	F2	4	SP228	4.5
EE5647	2.5	Huang Zao 4	3.5	F2834T	4	SP232	4.5
EX466	2.5	HY-2	3.5	F522	4	SP334	4.5
FR6	2.5	IB Fertile	3.5	F7	4	SU80-1	4.5
FR21Sel	2.5	IFHY-2	3.5	FR1420	4	T111	4.5
FR22	2.5	ILL535A	3.5	FR14A	4	T220	4.5
FRO7	2.5	J818	3.5	FR15	4	TBA76125	4.5
Ga209	2.5	J823	3.5	FR15A	4	V8	4.5
H42	2.5	KDX2903	3.5	FR23	4	Va38	4.5
H56	2.5	Ky21	3.5	FR239	4	Va3B	4.5
K44	2.5	Ky217	3.5	FR29	4	W52	4.5
K720	2.5	Ky222	3.5	FR618	4	W739A	4.5
K809	2.5	Ky55-537	3.5	FR815	4	WH	4.5
KP39-2	2.5	Ky58K	3.5	FR8P	4	67244	4.75
Ky27	2.5	LH117	3.5	FR902	4	75-R008(Wu105)	4.75
LH18	2.5	LH32	3.5	FRP8	4	A116	4.75
LH22	2.5	LH39	3.5	Ga199	4	A23R	4.75
LH47	2.5	LH42	3.5	GE440	4	A305	4.75
M14NR	2.5	LH73A	3.5	GI4-8	4	A554	4.75

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
MBS5	2.5	LH87	3.5	H22	4	ACR male	4.75
MO16W	2.5	LH92	3.5	H45	4	CG15	4.75
MO21R	2.5	LH99	3.5	H50	4	CH663-8	4.75
[(MS713xMcMair11S1) xMS71]S6	2.5	Liaolon814	3.5	H53	4	CH74-6-1	4.75
N31	2.5	MBS61	3.5	H54	4	CK31	4.75
N611	2.5	MEX72:1586-1	3.5	H59	4	CM105	4.75
NC230	2.5	MI909	3.5	H89	4	E739	4.75
NC264	2.5	MI912	3.5	H92	4	FR2114	4.75
NC344	2.5	MI920	3.5	J855	4	FR2156	4.75
OH561	2.5	MO20W	3.5	Jellicorse MSY	4	FR632	4.75
Pa75-14	2.5	MO34	3.5	K11	4	FR818	4.75
R146	2.5	MP305	3.5	K155	4	Gaspe Flint	4.75
R806	2.5	MP77:115	3.5	K222	4	J847	4.75
R853	2.5	MS141	3.5	K6	4	Laguna69	4.75
SC213	2.5	MS153	3.5	Ky108	4	LH20	4.75
SDP312	2.5	MS57	3.5	Ky49	4	MICH-80-3	4.75
T337	2.5	MS62	3.5	Ky56-180	4	MO18W	4.75
Va10	2.5	N104	3.5	L	4	MO4	4.75
Va20	2.5	N132	3.5	LH112	4	MO940	4.75
Va26	2.5	N138	3.5	LH119	4	NC348	4.75
W703	2.5	N152	3.5	LH123	4	ND255	4.75
WCI 1-41	2.5	N156	3.5	LH24	4	OH07B	4.75
337	2.75	N158	3.5	LH7	4	OH45	4.75

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
890	2.75	N16	3.5	LH77	4	OH5	4.75
81-R2191	2.75	N20	3.5	LH97	4	Pa405	4.75
A374	2.75	NC352	3.5	M185	4	Pa54	4.75
B42	2.75	ND364	3.5	M37W	4	Purdue1031	4.75
B6	2.75	ND385	3.5	MBS1334	4	Q199	4.75
B68	2.75	ND407	3.5	MEX MIX-3	4	R203	4.75
B7	2.75	NY401	3.5	MI905	4	R917	4.75
B75	2.75	O72	3.5	MI916	4	SEG75118	4.75
B76	2.75	OH517	3.5	MI919	4	SP280	4.75
B77	2.75	OH91624	3.5	MICH-4	4	SP289	4.75
Ch-Ha-Long Kang 24m	2.75	Pa419p	3.5	MICH-78-1	4	Tie84	4.75
Cl40	2.75	Pa74-7	3.5	MO12	4	YUBC3	4.75
Cl44	2.75	Pa762	3.5	MO2	4	67238	5
Cl45	2.75	Pa77-58	3.5	MO39	4	67253	5
Cl88A	2.75	PI222480	3.5	MP311	4	A239	5
CQ208	2.75	PR	3.5	MP420	4	A622	5
CQ585	2.75	R151	3.5	MP78:62	4	A631	5
EX505	2.75	R158	3.5	MS132	4	A73	5
FR1064	2.75	R168	3.5	MS67	4	BS2	5
FR2352	2.75	R189	3.5	N142	4	BwKKTM2-1-1	5
FR302	2.75	R4	3.5	N157	4	C14-IMP	5
FR4C	2.75	R74A	3.5	N22	4	CG11	5
FR811	2.75	R906	3.5	N22A	4	CG12	5
FR819	2.75	R907	3.5	NC34	4	Cl43	5

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
FR9	2.75	SD105	3.5	NC83	4	CM109	5
H74	2.75	SD12	3.5	ND309	4	CM174	5
H94	2.75	SDP068	3.5	ND363	4	CM53	5
HP62-52	2.75	SDP157	3.5	ND478	4	CM99	5
Jellicorse	2.75	SDP260	3.5	OH07	4	CQ189	5
KTX2602	2.75	SP214	3.5	OH07A	4	EP1	5
LA48	2.75	SP215	3.5	OH07K	4	EX370	5
LH116	2.75	SP274	3.5	OH29	4	F431	5
LH71	2.75	SP292	3.5	OH516	4	F486	5
LV4	2.75	SP298	3.5	OH51A	4	FR2164	5
M14Wx	2.75	SP299	3.5	OH578	4	FR2200	5
MBS335	2.75	SP320	3.5	OH7N	4	H41	5
MBS50	2.75	SP333	3.5	OH91678	4	I205	5
MICH-78-3	2.75	T13	3.5	OS420	4	K441	5
MO44	2.75	T206	3.5	P8	4	MI917	5
MS116	2.75	T434	3.5	Pa11	4	MI918	5
MS71	2.75	T474	3.5	Pa409	4	MO42	5
N15	2.75	TEX1	3.5	Pa83	4	New Mex. Blue Flour	5
N160	2.75	TEX4	3.5	Pa84	4	OH41	5
N88	2.75	TEX5	3.5	Pa91	4	OH43E	5
OH2	2.75	TR207	3.5	PI427111	4	Pa73-11	5
OH545	2.75	TR210	3.5	R134	4	PH9-DMR	5
OH91610	2.75	TR293	3.5	R138	4	PI430482	5

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
OH91618	2.75	Troyer107	3.5	R174	4	Q189	5
Pa33A	2.75	TX403	3.5	R223	4	R104	5
Pa75-20	2.75	Va17	3.5	R53	4	R61	5
R199	2.75	Va22	3.5	SA3	4	R76	5
R221	2.75	Va25	3.5	SAF76242	4	R803	5
R78	2.75	Va36	3.5	SD10	4	SA1	5
R904A	2.75	Va39	3.5	SD107	4	T133	5
SD22	2.75	Va3A	3.5	SD5	4	T163	5
SP255	2.75	Va84	3.5	SDP014	4	T220A	5
Va29	2.75	Va94	3.5	SDP022	4	T246	5
Va92	2.75	VR2	3.5	SDP212	4	T490	5
W22G	2.75	VR8	3.5	SDP214	4	TBA76123	5
126	3	W10	3.5	SDP275	4	25801	5.25
198	3	W17	3.5	SDP282	4	A238	5.25
4226	3	W23	3.5	SG1533	4	FR24	5.25
9385	3	W32	3.5	SG18	4	FR600	5.25
67266	3	W438	3.5	SP244	4	MO36	5.25
A204	3	W454	3.5	SP252	4	MO5	5.25
A556	3	YUC103	3.5	SP258	4	MP307	5.25
A637	3	107	3.75	SP279	4	MS107	5.25
A653	3	25800	3.75	SP281	4	MS1334	5.25
A660	3	67235	3.75	SP291	4	N376	5.25
A664	3	191-7324	3.75	SP326	4	NY16	5.25
B49	3	75-4165	3.75	SUWAN3	4	NY302	5.25

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
		(CHI63)					
B55	3	75-R012	3.75	SXR	4	OH91634	5.25
		(CHIN69)					
BC555	3	80-2	3.75	SZV149	4	Pa347	5.25
BC9	3	A15	3.75	T101	4	PI430479	5.25
C127	3	A257	3.75	T115	4	SP253	5.25
Chun 7	3	A347	3.75	T162	4	SP276	5.25
Cl3	3	A408	3.75	T202	4	SP283	5.25
Cl317B	3	A413R	3.75	T218	4	TEX9	5.25
Cl31A	3	A502	3.75	T222	4	A634	5.5
Cl82	3	A545	3.75	T224	4	B64	5.5
CM37	3	A619	3.75	T226	4	B81	5.5
CQ170	3	A624	3.75	T232	4	CB59G	5.5
CQ710	3	A646	3.75	T236	4	CM39	5.5
D71-7	3	A656	3.75	T416	4	F517	5.5
DS:74:1004	3	Ay562	3.75	T8	4	MEX72:1556-1	5.5
DS:74:1113	3	B45	3.75	TBA76086	4	MI903	5.5
EX535	3	B67	3.75	TBA76126	4	MI922	5.5
EX555	3	B8	3.75	TEX3	4	N141	5.5
FR1087	3	CH24	3.75	TEX7	4	NN14	5.5
FR37	3	CH66-17	3.75	TR107	4	NY378	5.5
FR981	3	Cl41	3.75	TR110	4	PI430511	5.5
H105	3	Cl7	3.75	TRTR213	4	Q177	5.5
H73	3	CM75	3.75	TX29A	4	R219	5.5

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
H95	3	CO237	3.75	TX533	4	R80	5.5
H96	3	CQ182	3.75	U280Y	4	R914	5.5
I137TN	3	CQ187	3.75	Va27	4	W729C	5.5
I153	3	CQ192	3.75	Va28	4	E184	5.75
IF153	3	CQ196	3.75	Va35	4	F21	5.75
IF195	3	CQ206	3.75	Va4	4	F477white	5.75
K41	3	CQ213	3.75	Va41	4	PI430481	5.75
K804	3	CQ214	3.75	Va45	4	Purdue1026	5.75
KSX2301	3	CQ215	3.75	Va52	4	SD28	5.75
Ky211	3	D21	3.75	Va55	4	SKC02	5.75
KYS	3	EX371	3.75	Va72	4	TEX8	5.75
L578	3	F570FIANCE	3.75	Va93	4	A322	6
LH44	3	F6	3.75	W25	4	Ay561	6
LH5	3	FR16	3.75	W485	4	Cl66	6
LH98	3	FR2A	3.75	W513	4	E184R	6
LK	3	FR2B	3.75	W629A	4	F235	6
LS78	3	FR64A	3.75	W64A	4	FRK55	6
McNair11	3	FR816	3.75	W729D	4	MI913	6
MI910	3	GE54	3.75	WD Blacks	4	OH91672	6
MO22	3	GE62	3.75	WF9 (29)	4	R71	6
MO7	3	H117	3.75	WF9 C	4	TEX6	6
MS106	3	H85	3.75	Y6	4	TR	6
MS213	3	H97	3.75	67243	4.25	W633	6
N610	3	H99	3.75	67245(Etoxcbc)	4.25	CQ183	6.25

Table A.1. (Cont.)

Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating	Inbred Lines	Mean severity rating
N6D	3	Hangzhou4	3.75	75-R001(YING 04)	4.25	A77-3	6.5
N7B	3	HP62-49	3.75	A498	4.25	CM5	6.5
NC354	3	I234	3.75	A509	4.25	Laguna70	6.5
OH509A	3	J2705TV	3.75	A651	4.25	R105	6.5
OH51	3	J-63	3.75	A661	4.25	MI902	8.5