STUDIES ON SWEET CORN: STEWART'S WILT FORECASTING, THE EFFECT OF MAIZE DWARF MOSAIC ON FOLIAR DISEASES, AND HERBICIDE SENSITIVITY

BY

MICHAEL DEVIN MEYER

THESIS

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Adviser:

Professor Jerald K. Pataky

ABSTRACT

Diseases and sensitivity to P450-metabolized herbicides can limit the production of high quality sweet corn. Separate studies were done to determine the probability of exceeding 1% and 5% incidence of Stewart's wilt on sweet corn with different reactions to *Pantoea stewartii*, the effect of maize dwarf mosaic (MDM) on eight foliar diseases, and the effect of hybrid CYP genotype on sweet corn yield following postemergence applications of mesotrione or nicosulfuron. Stewart's disease can be forecasted using the Stevens, Stevens-Boewe, or Iowa State forecasts. These forecasts assume a high degree of host susceptibility. Host resistance affects the incidence and severity of disease, and therefore can affect the accuracy forecasts of Stewart's disease. Levels of host resistance affected the incidence of systemic seedling wilt within ranges of winter temperatures used by each of the forecasts. Frequency distributions of Stewart's wilt incidence on moderate and resistant hybrids did not differ among the three temperature ranges above -2.8ºC (27ºF). Conversely, distributions of Stewart's wilt incidence on susceptible hybrids differed among each of the four ranges of winter temperature from the Stevens-Boewe forecast (i.e., >0.6ºC, -1.1º to 0.6ºC, -2.8º to -1.1ºC, and <-2.8ºC). The probability of exceeding the 1% and 5% incidence thresholds that warrant the use of seed treatment insecticides also differed among hybrids with susceptible, moderate, or resistant reactions to Stewart's wilt. The probability of exceeding 1% incidence of systemic Stewart's wilt was greater than 0.5 on moderately susceptible to susceptible sweet corn hybrids when mean winter temperature was above -2.8ºC (27ºF). When mean winter temperature was below -2.8ºC (27ºF), using a seed treatment insecticide on moderate and resistant hybrids for Stewart's wilt control was not economical because

the probability of exceeding 1% incidence of systemic Stewart's wilt was about 0.04. The probability of exceeding 5% incidence was less than 0.1 except when the mean winter temperature was above -2.8ºC (27ºF) and a moderately susceptible to susceptible hybrid was grown. A separate study also was done to evaluate the effect of MDM on eight foliar diseases of sweet corn. MDM-infection substantially increased the severity of five diseases including: southern corn leaf blight (SCLB), northern corn leaf spot (NCLS), gray leaf spot (GLS), Diplodia leaf streak (DLS), and eyespot. Among MDM-susceptible hybrids, mean severity ratings of SCLB, NCLS, GLS, DLS, and eyespot on MDM-infected plants typically were double those of virus-free plants. Three diseases not substantially increased by MDM-infection included: common rust, northern corn leaf blight, and Stewart's wilt. MDM-infection appears to affect the severity of diseases caused by necrotrophic foliar fungi that colonize mesophyll tissue. MDM-infection did not appear to substantially affect diseases caused by pathogens which form haustoria or invade the vascular system. The extent to which SCLB severity is increased by MDM in terms of changes in level of host resistance also was determined. For MDM-susceptible hybrids, reactions to SCLB ranged from resistant to moderately-susceptible in virus-free treatments, but each of these hybrids was classified as moderately susceptible to susceptible when infected with MDM. Reactions to SCLB did not differ appreciably among viral treatments for MDM-resistant hybrids, ranging from moderately-resistant to moderately-susceptible. An additional study was done to determine if hybrid genotype at a major cytochrome P450 (CYP) locus on chromosome 5S affected yield following postemergence applications of mesotrione (HPPD-inhibiting herbicide) or nicosulfuron (ALS-inhibiting herbicide) at two growth stages. Yield of

hybrids with two mutant cyp alleles (i.e., *cypcyp*) was reduced by applications of mesotrione. These hybrids were not evaluated for nicosulfuron because ALS-inhibiting herbicides kill these hybrids. Yield of hybrids heterozygous for mutant and functional CYP alleles (i.e., *CYPcyp*) were reduced by nicosulfuron but unaffected by mesotrione. Yield of hybrids with two functional CYP alleles (i.e., *CYPCYP*) were unaffected by mesotrione or nicosulfuron. Results of this experiment demonstrate that the genetic condition of hybrids at the CYP locus on chromosome 5S affects the yield of sweet corn following application of mesotrione or nicosulfuron at the V3 to V5 or the V5 to V7 growth stages.

To my fiancée and family

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CHAPTER 1

INFLUENCE OF HOST RESISTANCE ON STEWART'S WILT FORECASTS AND THE PROBABILITY OF EXCEEDING THRESHOLDS THAT WARRANT THE USE OF SEED TREATMENT INSECTICIDES ON SWEET CORN

ABSTRACT

Many sweet corn (*Zea mays* L.) hybrids commercially available today have greater levels of resistance to Stewart's disease (caused by *Pantoea stewartii* subsp. *stewartii* (Smith) Mergaert et al.) than the cultivars from which Stevens developed the first forecast of this disease in the 1930s. Incorporating levels of host resistance into forecasts of the seedling wilt phase of Stewart's disease (i.e., Stewart's wilt) could improve pre-plant control decisions for sweet corn. Incidence of systemic infection of seedlings was assessed on 27 sweet corn hybrids with a range of reactions to *P. stewartii*. A total of 741 observations were collected from 1998 to 2009 in 79 field trials at 15 locations throughout Illinois and one each in Kentucky and Delaware. Relative frequency distributions of the incidence of systemic Stewart's wilt were developed and compared among hybrids with different levels of resistance in combination with ranges of winter temperature from the Stevens, Stevens-Boewe, and Iowa State forecasts. The probability of exceeding the 1% and 5% incidence thresholds that warrant the use of seed treatment insecticides was determined from these frequency distributions. Levels of host resistance affected the incidence of systemic seedling wilt within ranges of winter temperatures used by each of the forecasts. Frequency distributions of Stewart's wilt incidence and mean incidence ranging from 0.7 to 1.8%

on moderate and resistant hybrids did not differ among three temperature ranges above -2.8ºC (27ºF). Conversely, distributions of Stewart's wilt incidence on susceptible hybrids differed among each of the four ranges of winter temperature from the Stevens-Boewe forecast (i.e., >0.6ºC, -1.1º to 0.6ºC, -2.8º to -1.1ºC, and <-2.8ºC) with mean incidence ranging from 0.5 to 8.5%. Occurrence of Stewart's wilt also differed among trials varying in number of winter months above -4.4ºC (24ºF) which is used by the Iowa State forecast to predict this disease. Levels of resistance to *P. stewartii* also affected the occurrence of Stewart's wilt as predicted by the Iowa State method. The probability of exceeding economic thresholds of 1% or 5% incidence of systemic Stewart's wilt depended on levels of host resistance and winter temperature. Stewart's wilt is unlikely to exceed economical thresholds when the mean winter temperature is below -4.4ºC (24ºF). The probability of exceeding 1% incidence of systemic Stewart's wilt was 0.59 on susceptible sweet corn hybrids, and 0.28 on moderate and resistant hybrids when mean winter temperature was above -2.8ºC (27ºF). When mean winter temperature was below -2.8ºC (27ºF) the probability of exceeding 1% incidence of systemic Stewart's wilt was about 0.22 and 0.04 on susceptible and moderate or resistant sweet corn hybrids, respectively. The probability of exceeding 5% incidence was less than 0.1 except when the mean winter temperature was above -2.8ºC (27ºF) and susceptible hybrids were grown.

INTRODUCTION

Plant disease forecasts can be used to guide disease management decisions. Forecasts have been developed for several economically important vegetable diseases such as: Stewart's disease (caused by *Pantoea stewartii* subsp. *stewartii* (Smith)

Mergaert et al. syn. *Erwinia stewartii*) of sweet corn (2, 6, 18, 28), late blight (caused by *Phytophthora infestans*) of potato (1), root rot (caused by *Aphanomyces euteiches* and *Fusarium solani* f.sp. *pisi*) of pea (27), and early blight (caused by *Alternaria solani*) of tomato (15). Most forecasts are based on a single element of the disease triangle (14). Weather is the most commonly used predictor of disease occurrence because environmental factors affect the production or effectiveness of primary or secondary inocula (5, 10). Most forecasts are developed with the assumption of uniform host susceptibility (9, 14).

Host resistance affects the incidence and severity of disease, and therefore can affect the accuracy of plant disease forecasts. For example, unwarranted fungicide applications were eliminated by accounting for levels of host resistance in two forecast models for late blight of potato (9). Similarly, separate models were developed to forecast stripe rust (caused by *Puccinia striiformis*) on different wheat cultivars (3). Host resistance also was incorporated into a model to predict gray leaf spot (caused by *Cercospora zeae-maydis*) of maize in Iowa (26).

The seedling wilt phase of Stewart's disease (i.e., Stewart's wilt) is an economically important disease of sweet corn. *Pantoea stewartii* is vectored by the corn flea beetle, *Chaetocnema pulicaria* Melsh. (7). Sweet corn hybrids experience significant yield losses when Stewart's wilt is prevalent, particularly when susceptible hybrids are infected at early stages of growth (29). Yield reduction is linearly associated with the incidence of systemic infection which primarily results from the seedling wilt phase of the disease (8). Incidence of systemic infection is affected by levels of host resistance (16, 21). In spite of potential yield losses, susceptible sweet

corn hybrids may be grown because they possess other desirable traits, such as early maturity, proper ear shape, high yields, and/or kernel quality (8).

In the 1930s, Stevens (28) associated various levels of the seedling wilt phase of Stewart's disease with a temperature index based on mean daily temperatures in December, January, and February preceding planting (Table 1.1). An additional temperature category was added when the forecast was expanded later to predict the leaf blight phase of Stewart's disease (2). Prediction of the seedling wilt phase is based on the abundance of primary inocula as affected by the presence and size of overwintering populations of corn flea beetles. *Chaetocnema pulicaria* does not survive prolonged periods of cold weather (4). The Stevens forecast assumes that in-season weather conditions are conducive for corn flea beetles and disease development, although factors other than winter temperature likely affect the prevalence of Stewart's disease (2). After planting, corn flea beetle populations are affected by weather conditions such as wind, rain, and temperature (4). Also, the incidence of Stewart's wilt usually decreases with later plantings from April to early June, associated with a decline in the size of populations of flea beetles that overwintered from the previous season (4, 11, 23). The Stevens forecast also assumes host susceptibility. Stevens' original associations were based primarily on the occurrence of disease on open pollinated sweet corn cultivars (e.g., 'Golden Bantam') known to be relatively susceptible to Stewart's wilt (12, 20, 22).

Recently, a model was developed to predict the occurrence of Stewart's disease at the time of phytosanitary inspections in seed corn fields (6, 18). This model, often referred to as the Iowa State method, predicts Stewart's disease from the number of

winter months with mean monthly air temperatures above -4.4ºC (24ºF) (Table 1.2). The presence or absence of Stewart's disease in seed production fields, which is predicted by the Iowa State method, is crucial to international marketing of corn seed because seed-borne *P. stewartii* is regulated by quarantines in many countries. Seed-production fields must be free of the disease to pass phytosanitary inspections late in the season.

Stewart's wilt in sweet corn is controlled primarily through pre-plant decisions to grow a resistant or moderately resistant hybrid or to use a seed treatment insecticide. Resistance decreased the incidence of systemic infection by fourfold or more compared to susceptible hybrids when plants were infected naturally under mild to severe occurrences of Stewart's wilt (16). Neonicotinoid insecticide seed treatments (e.g., imidacloprid, thiamethoxam, and chlothianidin) reduced the incidence of Stewart's wilt on sweet corn by 50 to 90% (13, 17, 24, 25). The decision to use a seed treatment insecticide depends on crop value and the expected level of Stewart's wilt in the absence of control. Neonicotinoid seed treatment of sweet corn is economical (i.e., the value of control equals or exceeds the cost of treatment) when the incidence of systemic Stewart's wilt is about 5% on crops grown for processing and about 1% on crops grown for fresh market (25).

Today, many commercially available sweet corn hybrids have much greater levels of Stewart's wilt resistance than the cultivars from which Stevens developed his forecast in the 1930s. Incorporating levels of host resistance into forecasts of Stewart's wilt could improve pre-plant control decisions for sweet corn. The objective of this study was to develop and compare relative frequency distributions of the incidence of

systemic Stewart's wilt on sweet corn hybrids with different levels of resistance to *P. stewartii* within ranges of winter temperature from the Stevens forecast, and to use those distributions to determine the probability of exceeding the 1% and 5% incidence thresholds that warrant the use of seed treatment insecticides.

MATERIALS AND METHODS

Observation of Stewart's wilt in field trials. Incidence of systemic Stewart's wilt infection was evaluated on sweet corn hybrids having different reactions to *P. stewartii* in 79 field trials from 1998 to 2009 at 15 locations throughout Illinois and one each in Kentucky and Delaware (Fig. 1.1). Planting dates varied among locations and years (Appendix A.1 and A.2). Ten to twelve sweet corn hybrids were planted at each trial location. The experimental design was a randomized complete block with 2 or 3 replicates. An experimental unit consisted of four rows of a sweet corn hybrid 7 m long spaced 76 cm apart, with about 40 plants per row. Plants between the 3- and 7-leaf stage were rated for incidence of systemic Stewart's wilt infection following early season feeding from naturally occurring populations of the corn flea beetle. Randomly selected, systemically-infected plants from each trial were examined microscopically for bacterial ooze.

Twenty-seven different hybrids were grown throughout the experiment. Seed were produced in Idaho or Chile where *P. stewartii* does not occur. Hybrid reactions to Stewart's wilt ranged from susceptible to resistant based on multiple years of evaluation in disease nurseries (Table 1.3) (19). Reactions from individual disease nurseries were averaged over all years of evaluation. For this study hybrids were grouped as resistant 1-3, moderate 4-6, or susceptible 7-9 based on mean reactions.

Winter weather data. Daily air temperature (i.e., mean of the daily high and low) during December, January, and February were obtained from the Midwestern Regional Climate Center (MRCC) (http://mrcc.isws.illinois.edu/) database. If temperature data were not available from a trial location, data from at least two nearby MRCC locations were averaged to estimate the daily air temperature at the trial location. Mean winter temperature preceding each growing season was then calculated as the mean of the daily air temperatures for December, January, and February. Trials were categorized by four ranges of winter temperature based on indices from the Stevens-Boewe forecast (Table 1.1) (2).

Relative frequency distributions and probability. Relative frequency distributions of Stewart's wilt incidence (0%; >0% to <1%; 1% to <3%; 3% to <5%; and ≥5.0%) on susceptible, moderate, and resistant hybrids were developed for each range of winter temperature. Contingency tables generated using the Proc Freq procedure of SAS version 9.2 (SAS Institute Inc., Cary, NC) were used in chi-square analyses to test the homogeneity of relative frequency distributions for all possible pairwise combinations of distributions within each range of winter temperatures and each hybrid reaction. The probability of exceeding 1% and 5% Stewart's wilt incidence was determined from relative frequency distributions for all combinations of susceptible, moderate, or resistant hybrids and the four ranges of winter temperature. Binomial tests of proportions ($P = 0.05$) were used to compare the probabilities of exceeding 1% or 5% Stewart's wilt incidence on hybrids with different levels of resistance within each temperature range.

Iowa State method. Mean winter temperatures preceding planting also were grouped according to the Iowa State method for predicting the occurrence of Stewart's disease. Mean daily air temperatures during December, January, and February were calculated and used to group trials by the number of winter months preceding planting with mean daily air temperatures above -4.4ºC (24ºF). Occurrence of Stewart's wilt at each location (i.e., the number of trials where Stewart's wilt was observed divided by total number of trials) was determined within each category of the Iowa State method. The occurrence of Stewart's wilt (i.e., the presence or absence) on hybrids with susceptible, moderate, and resistant reactions also was compared using the Iowa State method.

Weather at planting. Within each temperature category, standard z-scores were calculated to compare the mean incidence of Stewart's wilt on individual susceptible, moderate, and resistant hybrids within each trial to the mean incidence of Stewart's wilt for all hybrids with those reactions. For trials in which at least one susceptible, moderate, and resistant hybrid had appreciably positive or negative (±0.35) z-scores, rainfall and temperature data were collected for the period immediately before and after planting (April 15 to May 31). These data were used to determine if an association existed between rainfall and temperature at the time of planting and a higher or lower than expected incidence of Stewart's wilt.

RESULTS

Summary statistics. Mean winter temperature preceding planting ranged from -5.8°C (21.6°F) to 5.7°C (42.2°F), and averaged -1.1°C (30°F). There were 17 to 25 trials with 153 to 216 hybrid observations for each of the four ranges of winter temperature (Table 1.4). Stewart's wilt occurred in 59 of 79 trials, and on 330 of 741 hybrids observed in

those trials (Table 1.4). Among the 20 trials without Stewart's wilt, 11 occurred in 2008 and 2009, when mean winter temperature for all trial locations was -2.6°C (27.3°F) and -2.8°C (27.0°F), respectively. Five occurred in 2005, when daily low temperatures from mid-April to mid-May were below 4.4°C (27.0°F) for at least 11 and 7 days at central and southern Illinois locations, respectively, and daily low temperatures were below 0°C at least once. Mean incidence was 5.7, 1, and 0.8% for susceptible, moderate, and resistant hybrids, respectively. Mean incidence was 0.3% when the winter temperature was below -2.8°C (27.0°F), and ranged from 2.2 to 4.3% for the other three ranges of winter temperature.

Stewart's wilt was more prevalent at trial locations in southern Illinois and Kentucky than in northern or central Illinois. Stewart's wilt did not occur in 11, 3, and 1 trial each in northern, central, and southern Illinois, respectively. When winter temperature was below -2.8°C (27°F), Stewart's wilt was not observed on 80% of the hybrids evaluated (154 of 192 observations). When winter temperature was above -2.8°C (27°F), Stewart's wilt was not observed on 47% of the hybrids evaluated (257 of 549 observations).

Comparisons of relative frequency distributions. Relative frequency distributions of incidence of systemic Stewart's wilt differed among hybrid reactions and ranges of winter temperature (Tables 1.5 and 1.6; Fig. 1.2). Frequency distributions did not differ between moderate and resistant hybrids within each of the four ranges of winter temperature, whereas frequency distributions for susceptible hybrids differed from those of moderate and resistant hybrids within each winter temperature range (Table 1.5; Fig. 1.2). For susceptible hybrids, frequency distributions were different for each of the four

ranges of winter temperature (Table 1.6; Fig. 1.2). Frequency distributions did not differ among the three ranges of winter temperature above -2.8ºC (27ºF) for moderate and resistant hybrids, but distributions of incidence on moderate and resistant hybrids for each of the three temperature ranges above -2.8ºC were significantly different from the distributions when the winter temperature was below -2.8ºC (Table 1.6; Fig. 1.2).

Probability of 1% and 5% Stewart's wilt incidence. The probability of exceeding 1% or 5% incidence of systemic Stewart's wilt differed among hybrids with different reactions to *P. stewartii* and among ranges of winter temperature (Figs. 1.3 and 1.4). The probability of exceeding 1% or 5% Stewart's wilt incidence was greatest on susceptible hybrids, and did not differ significantly between moderate and resistant hybrids. For susceptible hybrids, the probability of exceeding 1% incidence was 0.22 when the winter temperature was below -2.8ºC (27ºF), and ranged from 0.5 to 0.66 when mean winter temperature was above -2.8ºC (Fig. 1.3). For moderate and resistant hybrids, the probability of exceeding 1% incidence was 0.06 or 0 when mean winter temperature was below -2.8ºC (27ºF), and ranged from 0.15 to 0.39 when mean winter temperature was above -2.8ºC (Fig 1.3). When data were combined for frequency distributions that did not differ among moderate and resistant hybrids or among ranges of winter temperatures above -2.8ºC (27ºF), the probability of exceeding 1% incidence of systemic Stewart's wilt was 0.59 and 0.22 for susceptible hybrids when winter temperature was above or below -2.8ºC, respectively; and 0.28 and 0.04 for moderate and resistant hybrids when winter temperature was above or below -2.8ºC, respectively (Table 1.7).

Probabilities of exceeding 5% incidence of systemic Stewart's wilt were similar to but lower than those for 1% incidence. For all hybrids, incidence did not exceed 5% when the mean winter temperature was below -2.8ºC (27ºF). For susceptible hybrids, the probability of exceeding 5% incidence the probability ranged from 0.24 to 0.48 when winter temperature was above -2.8ºC (Fig. 1.4). For moderate and resistant hybrids, the probability of exceeding 5% incidence ranged from 0.02 to 0.1 when winter temperature was above -2.8ºC (Fig. 1.4). When data were combined for frequency distributions with ranges of winter temperature above -2.8ºC, the probability of exceeding 5% incidence of systemic Stewart's wilt was 0.34 for susceptible hybrids and 0.07 for moderate and resistant hybrids (Table 1.7).

Iowa State method. Mean temperatures were above -4.4ºC (24ºF) for one, two, or three months in 8, 28, and 43 trials, respectively. Mean temperatures were not below -4.4ºC for all three months in any of the 79 trials. Occurrence of Stewart's wilt varied among trials having different number of months with mean temperatures above -4.4ºC and among hybrids with different reactions to *P. stewartii* (Table 1.8). Stewart's wilt occurred in 38%, 71%, and 84% of the trials with one, two, or three winter months above -4.4ºC, respectively (Table 1.8). Stewart's wilt was present only half as often on resistant hybrids as on susceptible hybrids for each of the three categories from the Iowa State method (Table 1.8). Among hybrids with susceptible reactions to *P. stewartii*, Stewart's wilt occurrence was 33%, 60%, and 71% when mean temperature was above -4.4ºC for one, two and three winter months, respectively. Stewart's wilt occurred on 9%, 31%, and 35% of the hybrids with resistant reactions to *P. stewartii* when mean

winter temperature was above -4.4ºC for one, two and three months, respectively (Table 1.8).

Assessment of weather effects around planting. Z-scores of at least one susceptible, moderate, and resistant hybrid were ≤-0.35 or ≥0.35 in 27 trials. Twenty-one trials had negative z-scores, and six had positive z-scores. Rainfall events greater than 3.8 cm (1.5 in.) over a 24 h period were observed in 13 of the 21 trials with negative z-scores and 2 of the 6 trials with positive z-scores (Appendix A.3). Total rainfall from April 15 to May 31 did not differ appreciably for trials with negative or positive z-scores. Nightly low temperatures differed among trials with positive and negative z-scores. Among 21 trials with negative z-scores, 10 had at least three consecutive nights with air temperature below 4.4ºC (40ºF). None of the six trials with positive z-scores had three consecutive nights with temperatures this low.

DISCUSSION

For nearly 75 years, the Stevens or Stevens-Boewe forecast has been used as a rule of thumb to predict the prevalence of Stewart's wilt based on the effects of winter temperatures on overwintering populations of corn flea beetles, the vector of *P. stewartii* (2, 28). Based on 12 years of field observations from which our dataset was generated, levels of host resistance appear to affect the incidence of systemic seedling wilt within ranges of winter temperatures used by these forecasts. Frequency distributions of Stewart's wilt incidence and mean incidence ranging from 0.7 to 1.8% on moderate and resistant hybrids did not differ among three temperature ranges above -2.8ºC (27ºF). Conversely, distributions of Stewart's wilt incidence on susceptible hybrids differed among each of the four ranges of winter temperature from the Stevens-

Boewe forecast with mean incidence of 8%, 8.5%, 4.8%, and 0.5% when mean winter temperature was above 0.6ºC (>33ºF), -1.1 to 0.6ºC (30-33ºF), -2.8 to -1.1ºC (27-30ºF), and below -2.8ºC (<27ºF), respectively.

Stevens proposed his forecast based on observed associations between winter temperature and the occurrence of Stewart's disease on open pollinated sweet corn cultivars in the early 1930s (28). Since many of those cultivars were relatively susceptible to *P. stewartii* (12, 20, 22), it is not unexpected that the ranges of winter temperature which define Stevens' indices have a similar association with Stewart's wilt incidence on modern, commercial sweet corn hybrids that are classified as moderately susceptible to susceptible. Similarly, it is not unexpected that incidence of Stewart's wilt on hybrids with greater levels of resistance cannot be differentiated as distinctly by these temperatures. Host resistance has been shown to influence disease forecasts of other crops (3, 9, 26). In this and a previous study (16), incidence of naturally-occurring, systemic Stewart's wilt at vegetative growth stages was lower on resistant and moderately resistant hybrids than on susceptible hybrids.

Host resistance also appears to affect the Iowa State method which predicts the risk of the occurrence of Stewart's disease assessed later in the growing season. In this study, the occurrence of the seedling wilt phase of Stewart's disease was more frequent as the number of winter months above -4.4ºC (24ºF) increased and as levels of resistance decreased. Stewart's wilt was present only half as often on resistant hybrids compared to susceptible hybrids for each of the three categories of the Iowa State method. Thus, similar to the Stevens-Boewe forecast, accounting for the Stewart's wilt

reaction of a hybrid or inbred could improve the ability of the Iowa State method to predict the occurrence of Stewart's disease at the time of phytosanitary inspection.

The economic value of neonicotinoid seed treatments (e.g., imidacloprid, thiamethoxam, and chlothianidin) relative to their control of Stewart's wilt has previously been shown to be equivalent to yield reductions resulting from about 1% systemic Stewart's wilt infection of sweet corn grown for fresh market and about 5% systemic Stewart's wilt infection of sweet corn grown for processing (8, 24, 25). Based on data from this study, the probability of exceeding 1% or 5% incidence of systemic Stewart's wilt depends on the Stewart's wilt reaction of the hybrid being grown and winter temperatures. For fresh market growers, the decision to treat or not to treat seed with these insecticides seems clear cut in three cases. Neonicotinoid seed treatments would not be warranted when the mean winter temperature is below -4.4ºC (24ºF) based on a recent summary of data on the occurrence of Stewart's wilt from seed production fields in Iowa (6, 18). Similarly, using a seed treatment insecticide on moderate and resistant hybrids would not be economical when the mean winter temperature is below -2.8ºC because the probability of exceeding 1% incidence of systemic Stewart's wilt is only about 4%. Conversely, moderately susceptible to susceptible sweet corn hybrids should be treated with a seed treatment insecticide when mean winter temperature is above -2.8ºC (27ºF) in order to avoid yield losses from Stewart's wilt because the probability of exceeding 1% incidence of systemic Stewart's wilt is greater than 50%. In two other cases, the decision to treat or not to treat is less obvious and probably depends on the risk aversion of the grower. Use of a seed treatment insecticide on moderate and resistant hybrids would not be economical in the long run when the mean

winter temperature is above -2.8ºC or on a susceptible hybrid when the mean winter temperature is below -2.8ºC because the probability of exceeding 1% incidence is below 0.5. However, in both of these situations there may be utility in using a seed treatment insecticide as an insurance against substantial fluctuations in production when the disease exceeds 1% incidence, which has about a 25% chance of occurring.

For sweet corn grown for processing, the use of seed treatment insecticides to control Stewart's wilt would not be economical on moderate and resistant hybrids regardless of mean winter temperature, since the probability of exceeding 5% incidence of systemic Stewart's wilt is very low. Similarly, if mean winter temperature is below -2.8ºC (27ºF), the use of a seed treatment insecticide would not be economical on susceptible sweet corn hybrids. However, if mean winter temperature is above -2.8ºC (27ºF) and a susceptible sweet corn hybrid is grown, the use of a seed treatment insecticide may have utility for a risk-averse grower as an insurance against more severe occurrence and yield losses due to Stewart's wilt.

Other factors besides winter temperature undoubtedly affect the prevalence of Stewart's wilt (2). As proposed previously (4, 6) the occurrence of Stewart's wilt the previous growing season and/or the size of the flea beetle population at the end of the previous season may be an important indicators of the amount of local inoculum for the following season. Planting date and in-season weather also may affect the development of Stewart's wilt (11, 25). Stevens (28) did not identify relationships between Stewart's wilt incidence and total rainfall during the growing season or temperature during the growing season. However, Cook et al. (4) observed that weather affects corn flea beetle populations. In this study, trials with less Stewart's wilt than expected based on trial

means often had consecutive nights with cool temperatures. Cool temperatures and excessive rainfall likely result in flea beetle mortality. Although a strong relationship was not observed from this data, we previously observed low levels of Stewart's wilt associated with cold, wet weather. Further examination of the effects of in-season weather conditions may explain some of the variation in Stewart's wilt forecasts that is not a result of host resistance. However, since Stewart's wilt control decisions are made prior to planting, in-season prediction has limited value with regard to disease management.

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Table 1.1. The Stevens forecast for predicting the seedling wilt phase of Stewart's disease

Index[a]	Mean daily temperature		Prevalence of Stewart's wilt
	Celsius	Fahrenheit	
>100	>0.6º	>33.3º	Destructive
90-100	-1.1º to 0.6º	30º to 33.3º	Intermediate
<90	<-1.1º	<30º	Usually absent
<80[b]	<-2.8º	<27.3º	Absent

[a] Sum of the mean daily air temperature (F) in December, January, and February preceding planting (28).

[b] An additional temperature index was added when the Stevens forecast was expanded to include the leaf blight phase of Stewart's disease (2).

Table 1.2. The Iowa State method for predicting Stewart's disease of corn

Number of months[a] ≥-4.4ºC (24ºF)	Predicted risk of occurrence
0	Negligible
1	Low to moderate
2	Moderate to high
3	High

[a] Based on the mean daily air temperature in the December, January, and February preceding planting (18).

Table 1.3. Sweet corn hybrids classified from resistant to susceptible to Stewart's wilt based on reactions in disease nurseries

		Stewart's wilt reaction[b]		
Reaction and hybrid	**Seed source[a]**	**Mean**	**Standard deviation**	**Number of years evaluated**
Resistant (1-3)				
Bonus	Rog	1.2	0.4	13
Terminator	Cr	1.7	1.0	11
Ambrosia	Cr	1.8	0.9	18
Summer Sweet 7630	AC	2.0	1.3	12
182A	IFS	2.2	0.6	7
GH 2628	Rog	2.3	1.0	4
Moderate (4-6)				
Crisp and Sweet 710	Cr	3.5	1.5	16
Incredible	Cr	3.5	1.6	19
Crisp and Sweet 710A	Cr	4.0	1.1	8
Shimmer	Sem	4.3	1.4	14
GH 1829	Rog	4.4	1.0	9
Bodacious	Cr	4.6	1.5	20
Even Sweeter	Sem	5.3	1.6	12
273A	IFS	5.3	1.1	10
Honey Select	Rog	5.5	1.1	8
GH 2690	Rog	5.6	2.2	5
Rival	Sem	5.7	1.6	11
Marvel	Cr	6.0	1.1	11
Double Gem	MM	6.1	1.4	8
Temptation	Sem	6.3	0.7	8
Susceptible (7-9)				
Royal Sweet	Rog	6.5	1.9	4
Snow White	HM	6.9	1.7	19
Coho	HM	7.3	1.5	9
Lumina	HM	7.6	1.1	11
Supersweet Jubilee Plus	Rog	8.1	1.2	8
Jubilee	Rog	8.7	0.6	24
Supersweet Jubilee	Rog	8.9	0.3	12

[a] Cr = Crookham Co.; HM = Harris Moran Seed Co.; IFS = Illinois Foundation Seeds Inc.; MM = Mesa Maize; Rog = Syngenta Seeds Inc., Rogers Brands; Sem = Seminis Vegetable Seeds Inc.

[b] Reaction to infection by *Pantoea stewartii* in University of Illinois sweet corn disease nurseries (19), where 1 = resistant, 3 = moderately resistant, 5 = moderate, 7 = moderately susceptible, and 9 = susceptible.

Table 1.4. Occurrence and incidence of systemic Stewart's wilt in 79 trials from 1998 to 2009 grouped by four ranges of winter temperature

Mean winter temperature[a]		Trials		Hybrid observations[b]		Stewart's wilt incidence (%)	
Celsius	Fahrenheit	Total	With Stewart's wilt	Total	With Stewart's wilt	Mean	Range
>0.6º	>33º	25	24	216	112	3.9	0 – 77
-1.1º to 0.6º	30º – 33º	19	15	153	91	4.3	0 – 62
-2.8º to -1.1º	27º – 30º	17	12	180	89	2.2	0 – 73
<-2.8º	<27º	18	8	192	38	0.3	0 – 16

[a] Mean daily air temperature for December, January, and February.
[b] Hybrid observations - 741 hybrid observations in 79 trials.

Table 1.5. Chi-square test of homogeneity of distributions of Stewart's wilt incidence on sweet corn hybrids with susceptible, moderate, and resistant reactions[a] to *Pantoea stewartii* within four ranges of mean winter temperature[b]

Comparison of hybrid reactions within temperature ranges	Chi-square value	*P*[c]
>0.6ºC (>33ºF)		
susceptible vs. moderate	26.28	<.01
susceptible vs. resistant	39.34	<.01
moderate vs. resistant	4.15	0.39
-1.1 to 0.6ºC (30-33ºF)		
susceptible vs. moderate	27.16	<.01
susceptible vs. resistant	21.47	<.01
moderate vs. resistant	1.42	0.84
-2.8 to -1.1ºC (27-30ºF)		
susceptible vs. moderate	13.57	<.01
susceptible vs. resistant	16.85	<.01
moderate vs. resistant	3.37	0.50
<-2.8ºC (<27ºF)		
susceptible vs. moderate	10.04	0.02
susceptible vs. resistant	12.53	0.01
moderate vs. resistant	3.12	0.21

[a] Reactions to infection by *Pantoea stewartii* (19).
[b] Mean daily air temperature in the December, January, and February preceding planting.
[c] Probability of exceeding the chi-square value when the hypothesis of homogeneity is true.

Table 1.6. Chi-square test of homogeneity of distributions of Stewart's wilt incidence at four ranges of mean winter temperature[a] within groups of sweet corn hybrids with susceptible, moderate, and resistant reactions[b] to *Pantoea stewartii*

Comparison of hybrid reactions within temperature ranges	**Chi-square value**	***P*[c]**
Susceptible		
>0.6ºC vs. -1.1 to 0.6ºC	21.2	<.01
>0.6ºC vs. -2.8 to -1.1ºC	11.2	0.03
>0.6ºC vs. <-2.8ºC	40.6	<.01
-1.1 to 0.6ºC vs. -2.8 to -1.1ºC	10.6	0.03
-1.1 to 0.6ºC vs. <-2.8ºC	41.9	<.01
-2.8 to -1.1ºC vs. <-2.8ºC	20.4	<.01
Moderate		
>0.6ºC vs. -1.1 to 0.6ºC	4.6	0.33
>0.6ºC vs. -2.8 to -1.1ºC	3.2	0.53
>0.6ºC vs. <-2.8ºC	17.5	<.01
-1.1 to 0.6ºC vs. -2.8 to -1.1ºC	1.4	0.84
-1.1 to 0.6ºC vs. <-2.8ºC	28.2	<.01
-2.8 to -1.1ºC vs. <-2.8ºC	20.7	<.01
Resistant		
>0.6ºC vs. -1.1 to 0.6ºC	4.7	0.31
>0.6ºC vs. -2.8 to -1.1ºC	8.7	0.07
>0.6ºC vs. <-2.8ºC	14.7	0.01
-1.1 to 0.6ºC vs. -2.8 to -1.1ºC	5.9	0.21
-1.1 to 0.6ºC vs. <-2.8ºC	22.4	<.01
-2.8 to -1.1ºC vs. <-2.8ºC	11.5	0.02

[a] Mean daily air temperature in the December, January, and February preceding planting.
[b] Reactions to infection by *Pantoea stewartii* (19).
[c] Probability of exceeding the chi-square value when the hypothesis of homogeneity is true.

Table 1.7. Probability of exceeding 1% or 5% incidence of systemic Stewart's wilt on sweet corn hybrids with susceptible or moderate to resistant reactions to *Pantoea stewartii* when mean winter temperature was above or below -2.8°C based on 741 observations of hybrids in 79 field trials from 1998 to 2009

		Systemic Stewart's wilt incidence			
Mean winter temperature[a]		**≥1%**		**≥5%**	
Celsius	**Fahrenheit**	**S[b]**	**M and R**	**S**	**M and R**
>-2.8°C	>27°F	0.59	0.28	0.34	0.07
<-2.8°C	<27°F	0.22	0.04	0	0

[a] Mean daily air temperature in the December, January, and February preceding planting.

[b] Reaction to infection by *Pantoea stewartii*, where: S = susceptible, M = moderate, and R = resistant.

Table 1.8. Occurrence[a] of systemic Stewart's wilt in 79 field trials from 1998 to 2009 when mean winter temperatures are grouped by the Iowa State Method of forecasting the occurrence of Stewart's disease on sweet corn hybrids with susceptible, moderate, or resistant reactions to *Pantoea stewartii*

			Hybrid reaction to Stewart's wilt					
Months[b] above -4.4ºC	**Trials**		**Susceptible**		**Moderate**		**Resistant**	
	n	**Occurrence[c] (%)**	**n**	**Occurrence (%)**	**n**	**Occurrence (%)**	**n**	**Occurrence (%)**
1	8	38	30	33	32	16	22	9
2	28	71	98	60	105	40	72	31
3	43	84	139	71	133	41	110	35

[a] Occurrence = presence or absence.

[b] The number of winter months (December, January or February) where mean daily air temperature was above 4.4ºC.

[c] Number of trials with Stewart's wilt divided by the total number of trials within a temperature range.

Figure 1.1. Locations of field trials in which incidence of systemic Stewart's wilt was assessed on sweet corn hybrids from 1998 to 2009.

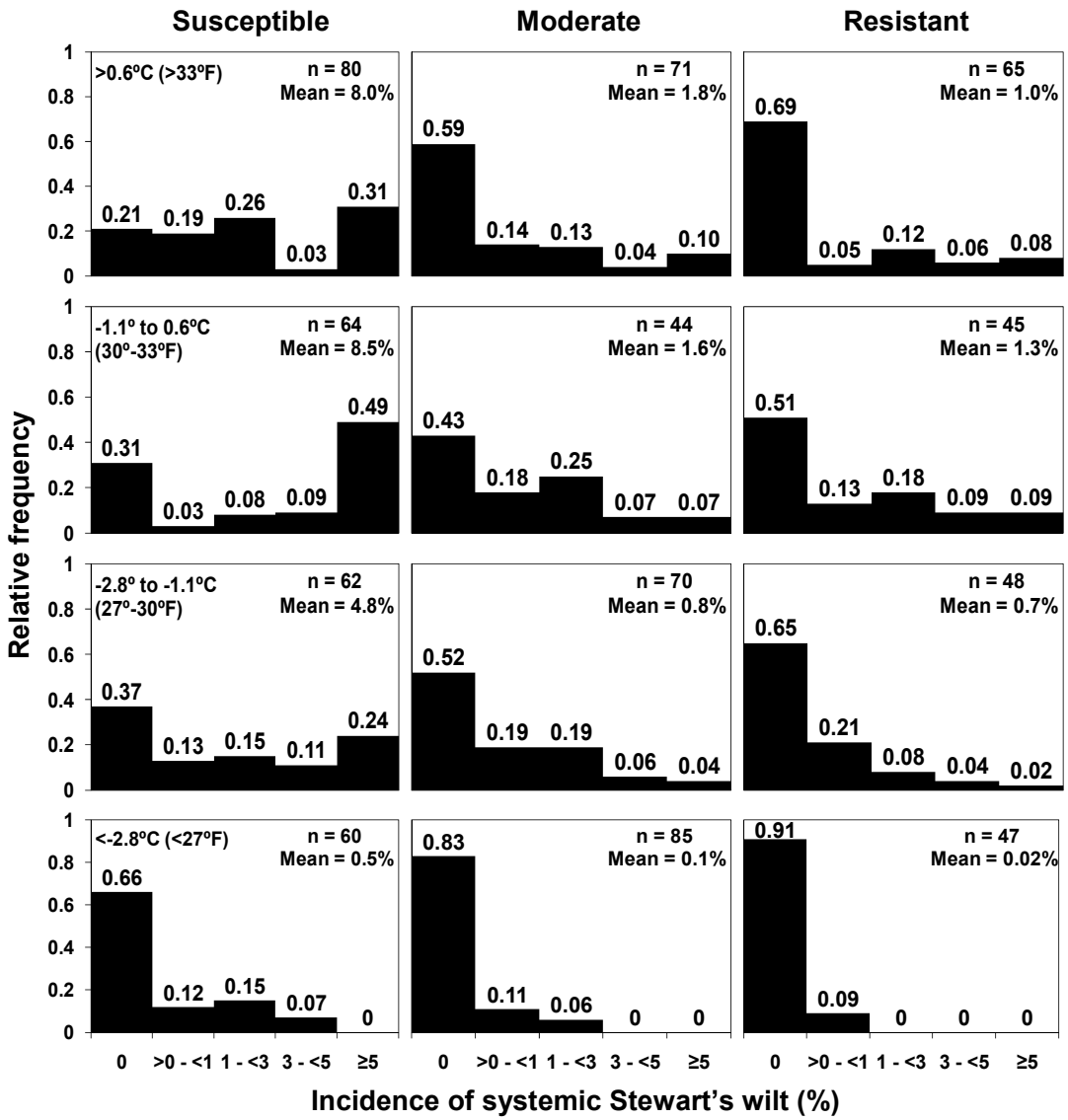

Figure 1.2. Frequency distributions of incidence of systemic Stewart's wilt on sweet corn hybrids with susceptible, moderate, and resistant reactions to *Pantoea stewartii* grouped by four ranges of mean winter temperature.

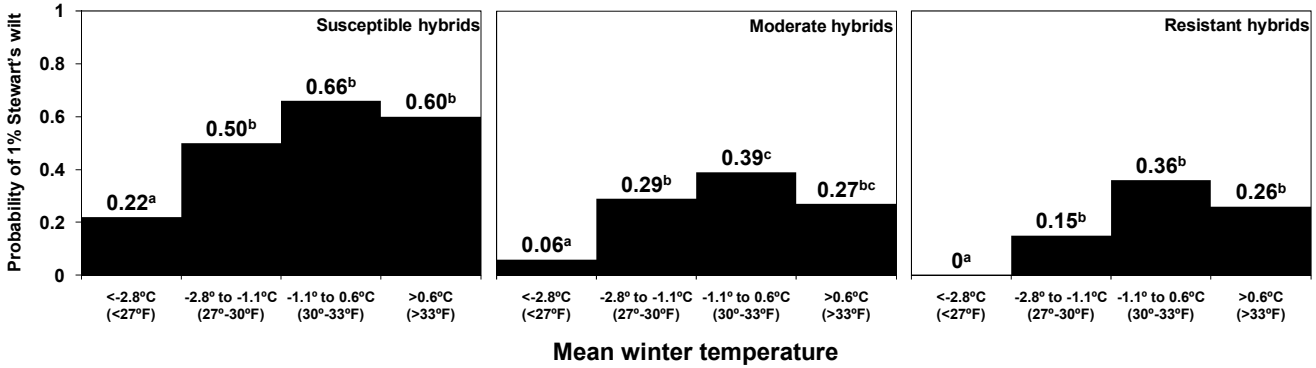

Figure 1.3. Probability of exceeding 1% incidence of Stewart's wilt on hybrids with susceptible, moderate, and resistant reactions to *Pantoea stewartii* following one of four ranges of mean winter temperatures. Within levels of resistance, probabilities followed by the same letter are not significantly different at $P = 0.05$.

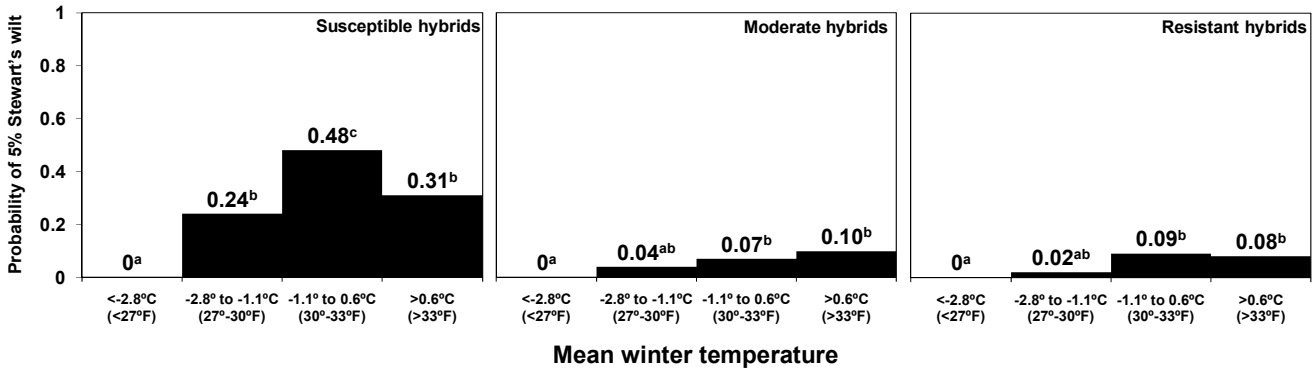

Figure 1.4. Probability of exceeding 5% incidence of Stewart's wilt on hybrids with susceptible, moderate, and resistant reactions to *Pantoea stewartii* following one of four ranges of mean winter temperatures. Within levels of resistance, probabilities followed by the same letter are not significantly different at $P = 0.05$.

CHAPTER 2

INCREASED SEVERITY OF FOLIAR DISEASES OF SWEET CORN INFECTED WITH MAIZE DWARF MOSAIC

ABSTRACT

Maize dwarf mosaic (MDM), caused by *Maize dwarf mosaic virus* (MDMV) and/or *Sugarcane mosaic virus* (SCMV), is an economically important viral disease of sweet corn (*Zea mays* L.), and is known to increase the severity of fungal root rots and southern corn leaf blight. The effect of infection with MDMV-A and SCMV on eight foliar diseases was evaluated on 32 sweet corn hybrids (27 MDM-susceptible hybrids and five MDM-resistant hybrids) in 2007, 2008, and 2009. MDM infection substantially increased the severity of five diseases including: southern corn leaf blight (SCLB), northern corn leaf spot (NCLS), Diplodia leaf streak (DLS), eyespot, and gray leaf spot (GLS). Among MDM-susceptible hybrids, mean ratings of SCLB, NCLS, GLS, DLS, and eyespot severity on MDM-infected plants were typically double those of virus-free plants. Three diseases were not substantially increased by MDM including: common rust, northern corn leaf blight, and Stewart's wilt. MDM infection appears to affect the severity of diseases caused by necrotrophic foliar fungi that colonize mesophyll tissue. Whereas, MDM infection did not appear to substantially affect the severity of diseases caused by pathogens which form haustoria or invade the vascular system. The extent to which SCLB severity is increased by MDM in terms of changes in level of host resistance also was determined. For MDM-susceptible hybrids, reactions to SCLB ranged from resistant to moderately susceptible in virus-free

treatments, but each of these hybrids was classified as moderately susceptible to susceptible when infected with MDM. The results of this experiment demonstrate the importance of breeding for MDM resistance not only to control this important viral disease of sweet corn, but also to lower the potential for detrimental effects from several other foliar diseases that are of minor importance on sweet corn.

INTRODUCTION

Interactions between viral and fungal pathogens occur in many crops. In rice, infection by *Hoja blanca virus* increases susceptibility to brown spot caused by *Cochliobolus miyabeanus* (anamorph: *Bipolaris oryzae*) (16). *Beet yellows virus* infection increases the severity of Cercospora leaf spot in sugar beet (8). Increased susceptibility to Curvularia leaf spot in gladiolus is associated with viral infection (4, 5). When infected with *Bean yellow mosaic virus* and *Bean leaf roll virus*, faba bean is more susceptible to infection by *Botrytis fabae* and *B. cinerea* (26). Virus infection also increases susceptibility to root rot in many crops including: cucumber (2, 25), pea (6, 11), and red clover (38).

Virus infection does not always increase susceptibility of plants to fungal diseases. Corn infected with *Maize streak virus* was not more susceptible to downy mildew fungi compared to virus-free plants (9). Severity of foliar blight caused by *Septoria apiicola* was reduced on celery infected with *Celery mosaic virus* and *Parsnip yellow fleck virus* (1).

Maize dwarf mosaic (MDM), caused by *Maize dwarf mosaic virus* (MDMV) and by *Sugarcane mosaic virus* (SCMV), is the most prevalent viral disease of corn in the continental United States. Soon after MDM first was described on corn (14),

increased occurrence of fungal root rots were associated with infection by MDMV (12, 23, 37, 39). Root rots caused by *Pythium graminicola* and *Diplodia zeae*, and seedling blights caused by *Fusarium moniliforme, F. roseum, Penicillium funiculosum, P. oxalicum,* and *P. purpurognum* were more severe in MDM-infected plants than in virus-free plants (23). Incidence of *F. moniliforme* infection in sweet corn seedlings was 55% greater in MDM-infected plants than in virus-free plants (12). MDM-infected corn seedlings also were more susceptible to root infections caused by *Gibberella zeae* and *Helminthosporium pedicellatum* (37).

Southern corn leaf blight, caused by *Biploris maydis* (Nisikado & Miyake) Shoemaker, is the only foliar disease of corn known to be increased by MDM infection. Severity of SCLB caused by *B. maydis* race T and race O was greater on MDM-infected corn plants than on healthy plants (3, 34). Water soaking prior to lesion development, lesion size, lesion number, and sporulation were greater on leaf sections from MDM-infected plants than non-infected plants (3).

Altered permeability of plant tissues and increased exudates may cause increased susceptibility to *B. maydis* in MDM-infected corn plants (34). Several physiological and cytological changes occur in MDM-infected cells of corn and sorghum including a reduction in chloroplasts, an increase in nuclear material, and degradation of the mesophyll cells (3, 24). Reduced rates of photosynthesis and higher rates of respiration also result from MDM infection (36).

Although increased susceptibility to fungal root rots and SCLB in MDM-infected corn plants is well documented, the effects of MDM on other foliar diseases of sweet corn is unknown. Several foliar diseases are economically important on sweet

corn including: common rust caused by *Puccinia sorghi* Schwein, northern corn leaf blight (NCLB) caused by *Exserohilum turcicum* (Pass.) K.J. Leonard & E.G. Suggs, Stewart's wilt caused by *Pantoea stewartii* subsp. *stewartii* (Smith) Mergaert et al., and southern corn leaf blight (SCLB). About 300 to 400 commercial and pre-commercial sweet corn hybrids and inbreds are screened annually in the University of Illinois disease nursery for their reaction to these and other prevalent diseases (28). In the 2006 disease nursery, symptoms of SCLB were substantially more severe on sweet corn plants infected serendipitously with MDM than on non-infected plants (19).

The objectives of this study were to quantify the extent to which SCLB severity is increased by MDM in terms of changes in level of host resistance and to determine if MDM increases the severity of other foliar diseases of sweet corn including common rust, northern corn leaf blight, Stewart's wilt, northern corn leaf spot (NCLS) caused by *Bipolaris zeicola* (G.L. Stout) Shoemaker, Diplodia leaf streak (DLS) caused by *Stenocarpella macrospora* (Earle) Sutton, eyespot caused by *Kabatiella zeae* Narita & Hiratsuka, and gray leaf spot (GLS) caused by *Cercospora zeae-maydis* Tehon & E.Y. Daniels.

MATERIALS AND METHODS

Field trials were done in 2007, 2008, and 2009 at the University of Illinois Vegetable Crops Research Farm, Champaign, IL. Each trial included 32 commercial sweet corn hybrids with known reactions to MDM. Twenty-seven hybrids were susceptible to MDM; and five MDM-resistant hybrids were included as controls (Appendix B.1). Six foliar diseases (common rust, GLS, NCLB, NCLS, SCLB, and Stewart's wilt) were evaluated in separate experiments. Each experimental design was

a split-plot arrangement of a randomized complete block with four replicates. Hybrids were grown in main plots, and virus treatments were applied to subplots in 2007. In 2008 and 2009, virus treatments were applied to main plots, and hybrids were grown in subplots. An experimental unit was one 3.5 m-long row with approximately 16 plants. Rows were spaced 76 cm apart. Trials were planted on 14 May 2007, 29 May 2008, and 29 May 2009.

DLS and eyespot trials. Trials assessing the effects of MDM on DLS and eyespot were done in 2009. Each disease was evaluated in two separate trials planted on 29 May and 9 June. Four MDM-susceptible hybrids were grown. The experimental design was a split-plot arrangement of a randomized complete block design with four replicates. Viral treatments were applied to main plots and hybrids were grown in subplots. An experimental unit was one 3.5 m-long row with approximately 16 plants. Rows were spaced 76 cm apart.

Viral treatments. All trials included two viral treatments: a noninoculated control and plants inoculated with a combination of MDMV-A and SCMV. In three additional SCLB trials, plants were inoculated with either MDMV-A (2008 and 2009) or SCMV (2008). Non-inoculated plots were maintained virus-free by rouging all MDM-symptomatic plants.

MDMV-A-infected Johnsongrass (*Sorghum halepense*) and SCMV-infected sweet corn plants were maintained in a greenhouse to provide a source of inoculum. To increase inoculum, MDM-susceptible sweet corn hybrids were inoculated in isolated field plots in early May. Fields of plants inoculated with MDMV-A or SCMV for the

purpose of increasing inoculum were separated by at least 400 m. Inoculum from these fields was prepared prior to the arrival of aphid vectors of the viruses.

Leaves of MDMV-A- and SCMV-infected plants were collected from inoculum increase fields. Infectious sap was extracted by blending equal quantities of MDMV-A- and SCMV-infected tissue in 0.1 M potassium phosphate buffer at pH 7 (approximately 1 g leaf tissue per 10 ml of buffer) for approximately 30 s. The homogenate was filtered through a 3.8 L paint strainer (Trimaco LLC, Durham, NC). Inoculum was prepared by mixing approximately 3.8 L of infectious sap extracted from MDMV-A- and SCMV-infected tissue, 7.6 L of 0.1 M potassium phosphate buffer, and 13 g of 320-grit carborundum powder. Plants at the 3- to 4-leaf stage were mechanically inoculated twice using a motorized mist blower (Model 444; Solo, Newport News, VA) on 8 and 11 June 2007, 17 and 18 June 2008, 15 and 19 June 2009, and 30 June and 1 July 2009.

Inoculation procedures and maintenance of pathogen cultures. Plants were inoculated with fungal or bacterial pathogens seven to ten days after MDM inoculations. Dates of inoculation varied among years and diseases (Appendix B.2). Suspensions of about 10^3 conidia per ml plus mycelial fragments of *B. maydis*, *B. zeicola*, *E. turcicum*, *C. zeae-maydis*, *S. macrospora*, or *K. zeae* were sprayed directly into leaf whorls between the 4- to 7-leaf stages to initiate primary infection. Inoculum was prepared by blending 10- to 14-day-old cultures in water. Cultures were grown at room temperature (24 to 26ºC) under diurnal fluorescent light. *Bipolaris maydis*, *B. zeicola*, and *E. turcicum* were cultured on lactose-casein hydrolysate agar (LCA) and sterile sorghum seeds. *Cercospora zeae-maydis* (isolate obtained from D. G. White, University of Illinois, Urbana) was cultured on V-8 agar. *Stenocarpella macrospora*

(isolate obtained from C. A. Bradley, University of Illinois, Urbana) was cultured on PDA amended with autoclaved corn leaves (18) and a synthetic media described by Morant et al. (22). *Kabatiella zeae* (isolate obtained from J. G. Kinsey, Monsanto Co., Waterloo, IL) was cultured on potato dextrose agar (PDA).

Suspensions of *P. sorghi* urediniospores also were sprayed directly into leaf whorls to initiate primary infection. Urediniospores of *P. sorghi* were collected from infected plants in plots used to increase inoculum similar to those used to increase MDMV-A and SCMV. Urediniospores were suspended in water with a few drops of Tween 20. Urediniospores collected the previous summer were maintained at -80ºC until inoculum was increased in early planted fields.

Pantoea stewartii was isolated from infected plants collected throughout Illinois as described previously (35). Plants between the 4- to 6-leaf stage were inoculated with *P. stewartii* using the pinprick inoculation method (7).

Disease assessments. Incidence of MDM-infected plants was measured in each experimental unit 7 to 10 days after inoculation. Severity of the eight foliar diseases was rated visually on a row basis one to three weeks after anthesis. GLS was rated on a 1 to 9 scale of severity, where 1 = chlorotic flecks with little secondary spread and 9 = dense bands of necrotic rectangular lesions with abundant secondary spread (29). NCLS was rated on a 1 to 9 scale of severity, where 1 = chlorotic flecks and 9 = dense necrotic bands of the "strand of pearls" symptom with spread to upper leaves (Appendix B.3). SCLB was rated on a 1 to 9 scale, where 1 = chlorotic flecks and 9 = large necrotic spots and more than 75% of tissue is blighted (Appendix B.4). Eyespot was rated on a 1 to 5 scale, where 1 = few necrotic spots less than 1 mm with chlorotic halos

and 5 = necrotic spots greater than 2-3 mm with substantial spread to upper leaves (Appendix B.5). The percentage of total symptomatic leaf area was rated from 0 to 100% for common rust, DLS, and NCLB. Severity of Stewart's wilt was rated about 14 to 21 days after inoculation on a 1 to 9 scale (35). Each experimental unit was rated separately by two people. Ratings were averaged for each experimental unit. Disease ratings generally reflected secondary infection rather than primary infection resulting from inoculation.

Three ear leaves were sampled from plants in the middle of each row in two replicates of the common rust, eyespot, DLS, GLS, NCLB, NCLS, and SCLB trials. Leaves were pressed and dried using a forced air drier. Leaves were digitally scanned with an Epson scanner (Model Expression 1640 XL, Seiko Epson Corp, Suwa-shi, NGN, Japan). The percentage of total leaf area diseased was determined for each sample using Assess Image Analysis Software for Disease Quantification v. 2.0 (American Phytopathological Society, St. Paul, MN). The percentage leaf area diseased on each of three leaves subsampled from an experimental unit was averaged.

Statistical analysis. Since the arrangement of treatments differed among years, analyses were done separately for each year. Field ratings and Assess data for each foliar disease were analyzed by analysis of variance (ANOVA) using the Proc Mixed procedure of SAS v. 9.2 (The SAS Institute, Cary, NC). Data were analyzed twice with and without MDM-resistant hybrid controls in the analyses. Blocks and hybrids were considered random variables. Viral treatments were considered fixed variables. Effects were declared significant at $P \leq 0.05$ unless otherwise stated. Sums of squares were examined to identify the portion of total variation accounted for by treatment main

effects and the interaction term. Residuals were tested for normality using the Proc Univariate procedure of SAS. Residuals were plotted against predicted values using the Proc Gplot procedure of SAS to assess homogeneity of error variance.

Effect of MDM on hybrid reactions to SCLB. Hybrid reactions to SCLB were used to determine the effect of MDM infection on host resistance to SCLB. Since severity of SCLB was affected similarly by MDMV-A, SCMV, or the combination of MDMV-A and SCMV, ratings were averaged for all five SCLB trials. For each combination of hybrid and viral treatment, z-scores of SCLB severity were calculated using the mean and standard deviation of the virus-free treatment. Criteria used to classify hybrid reactions to SCLB were based on the mean of z-scores used as boundaries for SCLB reaction categories in nine previous University of Illinois sweet corn disease nurseries (Table 2.1; Appendix B.6). Although this procedure produces statistically "overlapping" groups without clear-cut differences among hybrids in adjacent categories (e.g., the hybrids with the least severe symptoms in the MR class may not differ significantly from the hybrids with the most severe symptoms in the R class) a consistent response of a hybrid over several trials produces a reasonable estimate of the disease reaction of that hybrid relative to the response of other hybrids.

RESULTS

Disease ratings. The effect of MDM infection on disease severity differed among the eight foliar diseases evaluated. Severity of five diseases (SCLB, NCLS, GLS, DLS, and eyespot) was increased substantially by MDM infection. Three diseases (common rust, NCLB, and Stewart's wilt) were not affected substantially. When analyses of disease ratings for SCLB, NCLS, and GLS included MDM-resistant and -susceptible hybrids,

viral treatments accounted for 44 to 71% of the total variation and the hybrid by virus interaction term accounted for 6 to 18% of the total variation in the ANOVAs (Table 2.2). When disease severity ratings for SCLB, NCLS, GLS, DLS, and eyespot were analyzed excluding MDM-resistant hybrids, viral treatments accounted for 56 to 87% of the total variation and the hybrid by virus interaction term accounted for only 2 to 8% of the total variation in the ANOVAs (Table 2.2). The significance of the hybrid by viral treatment interaction term resulted primarily from responses of MDM-resistant hybrids and differences in susceptibility to foliar diseases among the other 27 hybrids evaluated. For example, ratings of SCLB severity on hybrids susceptible to SCLB were affected less by MDM-infection than SCLB severity on hybrids with resistance to SCLB because non-MDM-infected, susceptible hybrids were already severely infected by SCLB (Fig. 2.1).

Among MDM-susceptible hybrids, mean ratings of SCLB, NCLS, GLS, DLS, and eyespot severity on MDM-infected plants were typically double those of virus-free plants (Fig. 2.2A and 2.3). SCLB ratings and differences among MDM-infected and virus-free treatments were similar among trials where MDMV-A and SCMV were inoculated separately or in combination; indicating that infection by either virus or the mixture of viruses had a similar effect on host susceptibility to SCLB. Except for the SCLB-B trial in 2008, disease severity ratings for MDM-resistant hybrids did not differ significantly among MDM-infected and virus-free treatments (Fig. 2.2B).

MDM-infection did not substantially increase severity of common rust, NCLB, or Stewart's wilt. Although the main effect of viral treatments and the hybrid by viral treatment interaction were significant in some of the ANOVAs of common rust, NCLB,

and Stewart's wilt ratings, viral treatments only accounted for 0 to 19% for these diseases compared to 44 to 87% for the other five foliar disease (Tables 2.2 and 2.3). Mean ratings of common rust, NCLB, and Stewart's wilt severity did not differ substantially on MDM-infected plants and virus-free plants of MDM-susceptible hybrids or MDM-resistant hybrids (Fig. 2.4A and B). On MDM-susceptible hybrids, mean severity ratings for common rust in 2007, NCLB in 2007, and Stewart's wilt in 2009 were significantly, but not substantially greater on MDM-infected plants than virus-free plants (Fig. 2.4A). In 2008, mean NCLB severity was 52 and 39% on MDM-infected and virus-free plants, respectively. Although severity of Stewart's wilt infection was not appreciably different among viral treatments, chlorotic, water-soaked tissues symptomatic of Stewart's wilt senesced more rapidly on MDM-infected plants than virus-free plants. A greater amount of necrotic tissue may have caused the difference in Stewart's wilt ratings in 2009. On MDM-resistant plants, severity of common rust, NCLB, and Stewart's wilt did not differ between the two viral treatments (Fig. 2.4B).

Percentage leaf area diseased data. Analyses of percentage leaf area diseased data were generally similar to those of visual ratings. When MDM-resistant hybrids were excluded from the analysis, viral treatments accounted for 10 to 84% of the total variation in the ANOVAs of percentage leaf area diseased for common rust, DLS, eyespot, GLS, NCLB, NCLS, and SCLB (Appendix B.7). The hybrid by viral treatment interaction accounted for 0 to 13% of the total variation, but was not significant. For MDM-susceptible hybrids, the percentage leaf area infected with common rust, DLS, eyespot, GLS, NCLB, NCLS and SCLB was substantially greater

in MDM-infected leaf samples than in virus-free leaf samples (Appendix B.8). For MDM-resistant hybrids, the percentage leaf area diseased was similar among viral treatments.

Effects of MDM on SCLB resistance. Among the five SCLB trials, SCLB ratings for virus-free hybrids ranged from 1.8 to 4.7, with a mean of 3.3 and standard deviation of 0.8 (Table 2.4). For MDM-infected hybrids, SCLB ratings ranged from 4.4 to 7.3, with a mean of 5.6 and standard deviation of 0.9. Z-scores of SCLB ratings calculated from grand means and standard deviations from the virus-free treatments ranged from -1.9 to 1.5 among virus-free hybrids and from 1.4 to 7.0 among MDM-infected hybrids. For MDM-susceptible hybrids, reactions to SCLB ranged from resistant to moderately susceptible in virus-free treatments, but each of these hybrids was classified as moderately susceptible to susceptible when infected with MDM (Table 2.4). Reactions to SCLB did not differ appreciably among viral treatments for three MDM-resistant hybrids (i.e., El Toro, Eliminator, and Bonus) (Table 2.4). SCLB reactions were increased for two MDM-resistant hybrids (i.e., Bold and Snow White) with lower levels of MDM resistance. Possibly, this was due to MDM-infected plants of Bold and Snow White from inoculation with SCMV-B. Hybrid SCLB reactions based on 2 to 15 years of evaluation in University of Illinois disease nurseries were highly correlated with SCLB reactions and mean SCLB severity ratings from virus-free treatments with correlation coefficients of 0.81 and 0.85, respectively.

DISCUSSION

Maize dwarf mosaic infection increases severity of at least five foliar diseases of sweet corn, including SCLB, NCLS, GLS, DLS, and eyespot. Increased severity of

MDM-infected plants to SCLB was known previously (3, 34). Severity of common rust, NCLB, and Stewart's wilt were not affected substantially by MDM infection. Thus, MDM infection appears to affect the severity of diseases caused by necrotrophic foliar fungi that colonize mesophyll tissue. Increased susceptibility likely results from the degradation of mesophyll cells by MDM (24), cellular leakage, and altered permeability of host tissues (34). MDM infection did not appear to substantially affect *P. sorghi* which forms haustoria or *E. turcicum* and *P. stewartii* which invade the vascular system.

Pazur et al. (31) reported previously that MDM did not affect severity of common rust on sweet corn. Others have noted that virus infection did not increase the severity of rust diseases caused by *Puccina recondita* f.sp. *tritici*, *P. hordei*, or *P. coronata* f.sp. *avenae* in small grains (17, 32, 33). Some viruses inhibit rust development (10, 40). Severity of NCLB was significantly increased by MDM infection, but to a lesser extent than other fungal foliar diseases that primarily colonize mesophyll tissue. Perhaps NCLB severity is increased slightly because the size of lesions is affected by the ability of *E. turcicum* to grow in mesophyll tissue as well as in vascular bundles. Nevertheless, NCLB severity was not increased as substantially by MDM infection as was the other necrotrophic foliar fungal diseases. Stewart's wilt ratings possibly were higher in MDM-infected plants in 2009 because tissues senesced more rapidly on MDM-infected plants than virus-free plants. Total spread of Stewart's wilt symptoms did not differ appreciably between MDM-infected plants and virus-free plants.

MDM infection substantially affected hybrid resistance reactions to SCLB. Disease reactions are used to assess the potential for diseases to become severe on a particular hybrid. Although, the hybrids selected for this experiment were known to have a wide range of reactions to SCLB, all of the MDM-susceptible hybrids were classified as moderately susceptible to susceptible to SCLB when infected with MDM. MDM infection likely has a similar effect on hybrid reactions to other foliar diseases based on the substantial increases in severity ratings we observed in this study. In the presence of MDM, these foliar diseases, which are usually of minor importance on sweet corn, could be severe due to reduced levels of resistance.

Growing MDM-resistant hybrids is the most practical method of controlling MDM and preventing increased susceptibility to foliar diseases associated with MDM infection. Although MDM resistance is generally successful in preventing MDM development, no MDM-resistant hybrid has 100% incidence of asymptomatic plants when inoculated at the 2- to 3-leaf stage (15). Maize dwarf mosaic is endemic on sweet corn in areas like the lower Ohio River bottomlands and the Central Valley of California. It also occurs sporadically in many other areas where sweet corn is produced. In addition to the adverse affects of MDM on sweet corn yield (13, 15, 20, 21, 30), MDM-infected plants may experience increased susceptibility to several other foliar diseases. While these diseases typically are of minor importance on sweet corn, their increased severity on MDM-infected plants may contribute to yield losses that typically are attributed to MDM. Thus, breeding for MDM resistance not only improves control of this important viral disease of sweet corn, but it also lowers the potential for detrimental effects from several other foliar diseases of sweet corn.

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Table 2.1. Standard z-scores used to classify sweet corn hybrids into nine categories of disease reaction to southern corn leaf blight (SCLB)

Disease reaction		z-score[a]
Resistant (R)	1	≤-1.2
R to MR	2	>-1.2 and ≤-0.7
Moderately resistant (MR)	3	>-0.7 and ≤-0.3
MR to M	4	>-0.3 and ≤0.2
Moderate (M)	5	>0.2 and ≤0.6
M to MS	6	>0.6 and ≤1.3
Moderately susceptible (MS)	7	>1.3 and ≤2.0
MS to S	8	>2.0 and ≤2.9
Susceptible (S)	9	>2.9

[a] Z-scores used as boundaries between resistance categories are the means of z-scores used to classify reactions in nine previous University of Illinois sweet corn disease nurseries.

Table 2.2. Sum of squares for treatment sources of variation, expressed as percentages of the total variation, from ANOVAs of disease severity based on factorial experiments with 32 sweet corn hybrids and two maize dwarf mosaic treatments

			Percentage (%) of total sums of squares[b]					
			ANOVA with MDM-R hybrids[c]			ANOVA without MDM-R hybrids[d]		
Foliar disease	MDM[a]	Year	Hybrid	Virus	Hybrid x virus	Hybrid	Virus	Hybrid x virus
Diplodia leaf streak	AB	2009	…[e]	…	…	3	87***[f]	2
		2009	…	…	…	7	82***	7***
Eyespot	AB	2009	…	…	…	15	80**	2**
		2009	…	…	…	8	77**	8**
Gray leaf spot	AB	2007	21	44***	11***	16*	56***	5**
		2009	9	67***	7***	4	81***	4**
Northern corn leaf spot	AB	2007	24*	54***	10***	18***	66***	4***
		2009	21	57***	18***	11	77***	8***
Southern corn leaf blight	AB	2007	24	52***	13***	17**	67***	5***
		2008	20**	68***	7***	17***	75***	3***
Southern corn leaf blight	A	2008	22**	62***	9***	18***	71***	4***
		2009	24	47***	14***	18*	62***	6***
Southern corn leaf blight	B	2008	16***	71**	6***	13**	77***	4***

[a] MDM treatment, where AB = inoculation with MDMV-A and SCMV, A = inoculation with MDMV-A only, B = inoculation with SCMV only.

[b] Treatment sum of squares divided by total sums of squares multiplied by 100.

[c] Data analyzed with 27 MDM-susceptible and five MDM-resistant hybrid controls.

[d] Data analyzed without five MDM-resistant hybrid controls.

[e] MDM-resistant hybrids not included.

[f] Asterisks indicate corresponding mean squares statistically significant, where * = $P<0.05$,** = $P<0.01$, and *** = $P<0.001$.

Table 2.3. Sum of squares for treatment sources of variation, expressed as percentages of the total variation, from ANOVAs of disease severity based on factorial experiments with 32 sweet corn hybrids and two maize dwarf mosaic treatments

		Percentage (%) of total sums of squares[a]					
		ANOVA with MDM-R hybrids[b]			ANOVA without MDM-R hybrids[c]		
Foliar disease	Year	Hybrid	Virus	Hybrid x virus	Hybrid	Virus	Hybrid x virus
Common rust	2007	62***[d]	8***	8***	48***	14***	12***
	2008	83***	0	4**	52***	0	14***
Northern corn leaf blight	2007	64***	6***	5***	63***	8***	4**
	2008	54***	16*	7***	54***	19**	5***
Stewart's wilt	2008	62***	3	6	52***	5	7
	2009	69***	7*	5*	61***	10*	4

[a] Treatment sum of squares divided by total sums of squares multiplied by 100.
[b] Data analyzed with 27 MDM-susceptible hybrids and five MDM-resistant hybrid controls.
[c] Data analyzed without five MDM-resistant hybrid controls.
[d] Asterisks indicate corresponding mean squares statistically significant, where * = $P<0.05$, ** = $P<0.01$, and *** = $P<0.001$.

Table 2.4. Hybrid reactions to southern corn leaf blight (SCLB) and maize dwarf mosaic (MDM), mean severity of SCLB on virus-free and MDM-infected plants in five trials, and resistance reactions of hybrids based on mean SCLB severity from those trials

	Hybrid reaction[a]		Virus-free		MDM-infected	
MDM-reaction and hybrid	MDM	SCLB	Mean SCLB severity[b]	SCLB reaction[c]	Mean SCLB severity	SCLB reaction
Susceptible						
Tuxedo	9 [7]	2 [9]	1.8	1	4.4	7
Sensor	9 [12]	3 [13]	2.1	1	4.8	7
Precious Gem	9 [11]	2 [8]	2.3	1	5.4	8
Lancelot	9 [7]	4 [8]	2.3	1	5.4	8
Merlin	9 [11]	2 [11]	1.9	1	5.5	8
1280	8 [6]	1 [5]	2.6	2	5.2	8
Delectable	9 [11]	3 [12]	2.5	2	5.8	9
2178	9 [4]	3 [2]	2.9	3	5.2	8
Brocade TSW	9 [10]	3 [8]	2.9	3	5.7	9
1179	8 [3]	2 [3]	2.8	3	5.9	9
Passion	9 [5]	3 [6]	2.9	3	6.0	9
Devotion	9 [8]	4 [6]	3.3	4	5.7	9
282A	9 [9]	3 [7]	3.0	4	5.8	9
Argent	9 [12]	4 [13]	3.1	4	5.8	9
Vision	9 [7]	3 [5]	3.3	4	6.0	9
Absolute	9 [10]	4 [8]	3.2	4	6.2	9
277A	9 [11]	3 [9]	3.3	4	6.4	9
Miracle	9 [13]	4 [15]	3.4	5	6.1	9
Jubilee	9 [13]	4 [14]	3.4	5	6.2	9
Synergy	8 [5]	4 [5]	3.7	5	6.6	9
Native Gem	9 [5]	6 [5]	4.1	6	5.3	8
Harvest Gold	8 [8]	6 [7]	4.1	6	5.9	9
Ambrosia	9 [13]	6 [13]	4.3	6	6.2	9
Augusta	9 [5]	6 [5]	4.0	6	6.4	9
White Saturn	9 [4]	6 [3]	4.3	6	7.1	9
179A	8 [7]	5 [7]	4.7	7	6.7	9
Bojangles	9 [7]	5 [6]	4.6	7	7.3	9
Resistant						
El Toro	3 [13]	3 [12]	2.5	2	2.8	2
Bold	4 [9]	4 [8]	3.0	3	4.2	6
Snow White	4 [12]	3 [14]	3.6	5	5.4	8
Eliminator	2 [13]	6 [14]	3.9	6	4.2	6
Bonus	2 [13]	6 [12]	4.5	7	4.8	7

[a] Hybrid reactions and years of evaluation in University of Illinois disease nurseries.
[b] Severity of SCLB rated on a 1 to 9 scale (Appendix B.4).
[c] Reaction to infection by *Bipolaris maydis*, where 1 = resistant, 2 - 4 = moderately resistant, 5 = moderate, 6 - 8 = moderately susceptible, and 9 = susceptible.

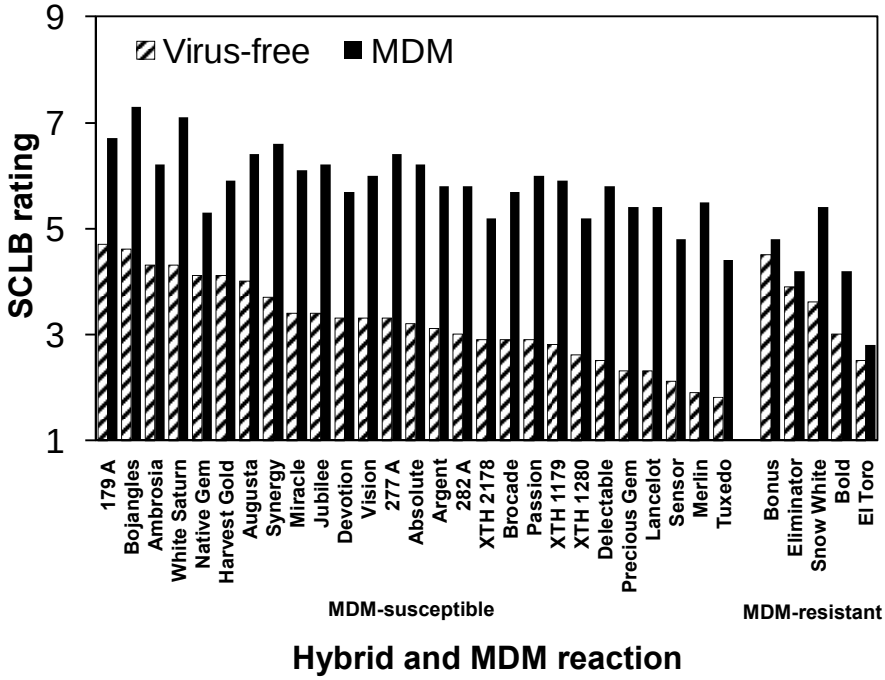

Figure 2.1. Mean ratings of southern corn leaf blight (SCLB) on virus-free and MDMV-A-infected sweet corn hybrids in field trials assessing the effect of maize dwarf mosaic (MDM) on SCLB severity.

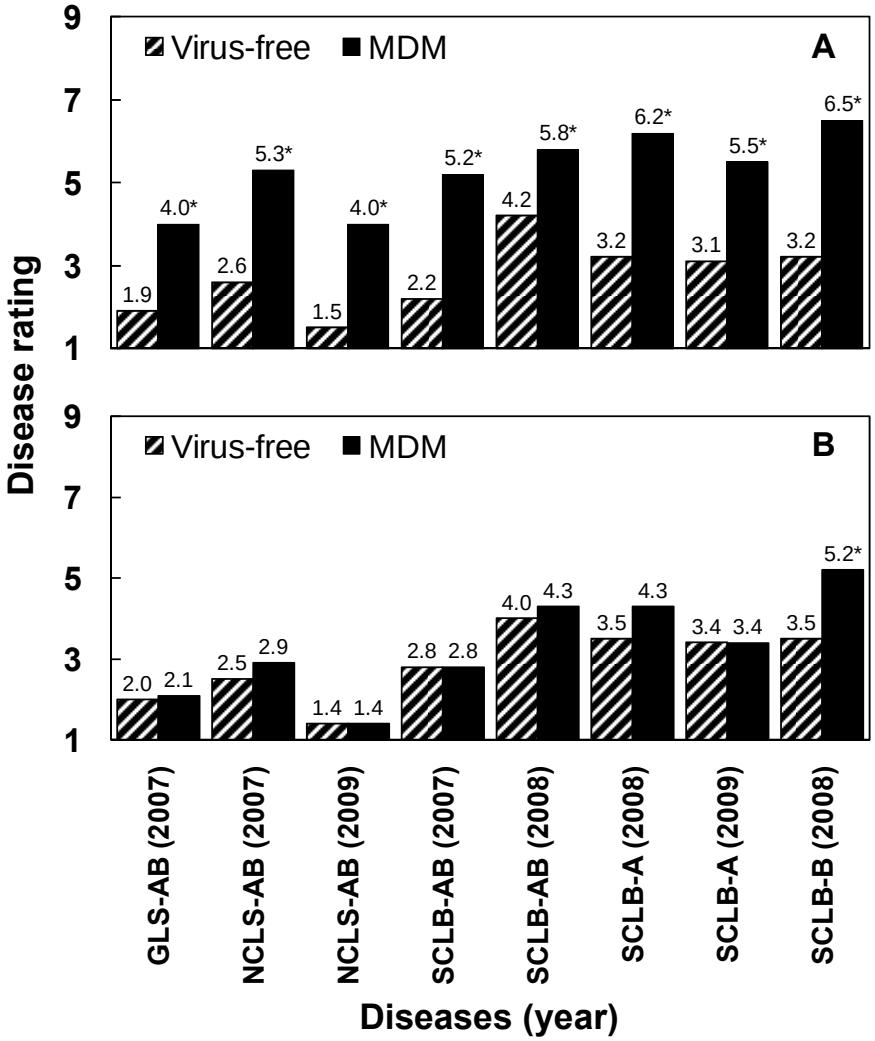

Figure 2.2. Mean ratings of gray leaf spot (GLS), northern corn leaf spot (NCLS), and southern corn leaf blight (SCLB) on virus-free and MDM-infected sweet corn hybrids that were: **A,** MDM-susceptible and **B,** MDM-resistant. Trials were inoculated with MDMV-A and SCMV (AB), MDMV-A only (A), or SCMV only (B). Asterisk indicates the MDM-infected treatment was significantly different from the virus-free treatment at $P \leq 0.05$.

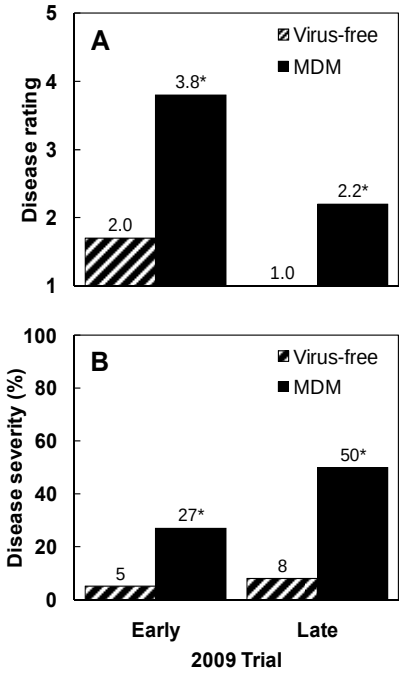

Figure 2.3. Mean disease severity ratings for **A,** eyespot and **B,** Diplodia leaf streak on virus-free and MDM-infected sweet corn hybrids that were MDM-susceptible. Asterisk indicates the MDM-infected treatment was significantly different from the virus-free treatment at $P \leq 0.05$.

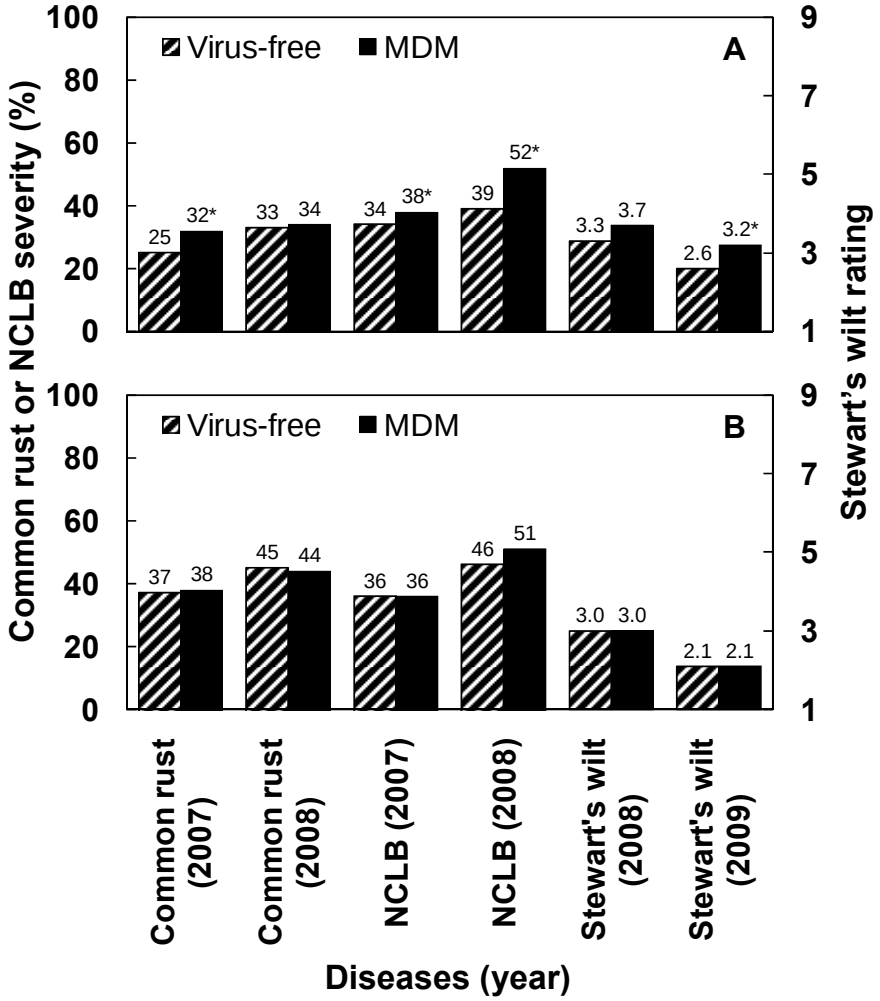

Figure 2.4. Mean ratings of common rust, northern corn leaf blight (NCLB), and Stewart's wilt on virus-free and MDM-infected sweet corn hybrids that were: **A,** MDM-susceptible and **B,** MDM-resistant. Asterisk indicates the MDM-infected treatment was significantly different from the virus-free treatment at $P \leq 0.05$.

CHAPTER 3

GENETIC FACTORS INFLUENCING ADVERSE EFFECTS OF MESOTRIONE AND NICOSULFURON ON SWEET CORN YIELD

ABSTRACT

Sensitivity to certain P450-metabolized herbicides in corn (*Zea mays* L.) is largely conditioned by a single cytochrome P450 (CYP) gene. Little to no research has been done to evaluate the effect of CYP genotype on sweet corn yield. Yield of 23 sweet corn hybrids of known CYP genotype was evaluated in 2007, 2008, and 2009 following postemergence applications of mesotrione, an hydroxyphenylpyruvate dioxygenase-inhibitor, or nicosulfuron, an acetolactate synthase-inhibitor, at two growth stages. Mesotrione and nicosulfuron were evaluated in separate experiments. Treatments included herbicide application during the V3 to V5 or V5 to V7 growth stages and a nontreated control. Crop injury, measured 7 days after treatment (DAT), ranged from 0 to 87% for mesotrione and 0 to 54% for nicosulfuron among CYP genotypes. Injury from both mesotrione and nicosulfuron was most severe following application during V3 to V5 growth stages on hybrids with mutant (nonfunctional) CYP allele(s). Stunting from mesotrione occurred primarily on *cypcyp* hybrids. For nicosulfuron, stunting was more frequent and severe on *CYPcyp* hybrids, although *CYPCYP* hybrids occasionally had mild stunting. Only cypcyp hybrids suffered yield losses from mesotione, ranging from 9 to 40%. Hybrids homozygous for mutant cyp alleles (i.e., *cypcyp*) were not evaluated for nicosulfuron because applications of ALS-inhibiting herbicides kill these hybrids. Nicosulfuron significantly reduced the yield of *CYPcyp* hybrids only. Yield

losses from nicosulfuron ranged from 9 to 35% among *CYPcyp* hybrids and were associated with moderate to severe loss of kernel rows (i.e., ear pinching). Yield of *CYPCYP* hybrids was not affected by mesotrione or nicosulfuron.

INTRODUCTION

Sensitivity of certain sweet corn hybrids to postemergence applications of cytochrome P450-metabolized herbicides, including mesotrione, a hydroxyphenylpyruvate dioxygenase- (HPPD) inhibitor, and nicosulfuron, an acetolactate synthase- (ALS) inhibitor, has been well documented in the past two decades (8, 9, 15, 18, 19, 20, 24, 28, 29, 35, 37). However, not all sweet corn hybrids are sensitive to P450-metabolized herbicides. Variation among hybrid responses to sulfonylurea herbicides, such as nicosulfuron, can be attributed to differential rates of herbicide metabolism. Tolerant plants detoxify herbicides more rapidly than sensitive plants (14). Cytochrome P450 enzymes are an important class of enzymes that are responsible for the primary detoxification of some corn herbicides (3, 17). Although corn has many cytochrome P450 genes (CYP genes), the number of P450 enzymes involved in metabolic inactivation of herbicides, their expression levels, and their levels of herbicide metabolism are not clearly understood (3, 4, 12, 13, 34). The possibility of a "super P450" that metabolizes multiple corn herbicides was proposed nearly 15 years ago (5).

A series of studies over the past two decades has documented that sensitivity of corn to multiple P450-metabolized herbicides is regulated primarily by a single CYP gene or a group of closely-linked CYP genes on the short arm of chromosome 5. A recessive gene from the corn inbred W703a conditioned sensitive responses to

nicosulfuron and was designated as *nsf1* (16), while a recessive gene from the corn inbred GA209 conditioned sensitive responses to bentazon and was designated as *ben* (11). The bentazon-sensitive inbred GA209 also displayed greater sensitivity to dicamba, imazethapyr, nicosulfuron, and primisulfuron than a bentazon-tolerant inbred B73 (6). Using a mapped-based cloning approach, the *nsf1* gene was located on the short arm of chromosome 5 and the dominant, functional allele, *Nsf1*, was sequenced from a nicosulfuron-tolerant inbred, B73 (42). The *Nsf1* gene was one of four closely-linked genes with significant sequence similarity to CYP genes. This gene also was very similar in sequence and function to a CYP gene in rice that conditions tolerant responses to multiple herbicides (30). Nicosulfuron- and bentazon-sensitive inbreds W703a and GA209 contained a 392 bp insertion in the *Nsf1* gene sequence relative to B73. Thus, it appears that the *nsf1* and *ben* alleles identified from W703a and GA209, respectively, are the same 392 bp insertion mutation of a CYP allele, very likely inhibiting the function of the "super P450" proposed by Barrett et al. (5).

The sweet corn inbred Cr1 is sensitive to nicosulfuron, mesotrione, and at least seven other P450-metabolized herbicides with five different modes of action (23, 31, 40, 38). A QTL that conditions cross-sensitivity to these P450-metabolized herbicides was detected in a segregating population of Cr1 x Cr2 (a herbicide tolerant inbred) on the short arm of chromosome 5 in tight linkage disequilibrium with the CYP locus previously identified as the *nsf1* and *ben* genes (23). Based on segregation of progeny from F_2 generations and from testcrosses with Cr1 and Cr2, 45 sweet corn hybrids and 29 sweet corn inbreds were identified as possessing a gene that is the same as or very closely linked to the gene in Cr1 that conditions sensitivity to multiple postemergence

herbicides (33). This group of hybrids and inbreds consists of lines developed by 12 independent, commercial breeding programs and includes sugary, sugary enhancer, and shrunken-2 endosperm types that are grown for processing and fresh consumption in world-wide markets. Thus, a common genetic basis for herbicide sensitivity occurs throughout the sweet corn industry.

Although this mutant CYP allele appeared to be recessive based on initial inheritance studies of nicosulfuron and bentazon sensitivity (11, 16), the rate of herbicide metabolism of *CYPcyp* hybrids (i.e., hybrids heterozygous for a functional and mutant CYP allele) appears to be intermediate to that of *cypcyp* and *CYPCYP* hybrids (i.e., hybrids homozygous for mutant or functional CYP alleles, respectively). The intermediate rate of metabolism of *CYPcyp* hybrids results in varied phenotypic responses under different conditions (32, 39). For example, when low rates of HPPD-inhibiting herbicides (e.g., mesotrione, tembotrione, and topramezone) were applied, *CYPcyp* hybrids generally were not injured and had a phenotype similar to *CYPCYP* hybrids as compared to *cypcyp* hybrids, which were injured. Thus, the CYP allele appeared to have dominant gene action. Conversely, at higher application rates, phenotypes of *CYPcyp* hybrids were intermediate to those of uninjured *CYPCYP* and severely injured *cypcyp* hybrids, which would be interpreted as partially-dominant or co-dominant gene action (39). Similarly, injury following postemergence applications of mesotione, nicosulfuron, or foramsulfuron in twelve herbicide trials in six states was 1.5 to 2.3 times greater on *CYPcyp* hybrids than on *CYPCYP* hybrids, but substantially less than on *cypcyp* hybrids, which were severely injured or killed in many trials (32).

Although applications of certain P450-metabolized herbicides can reduce yields of sweet corn, effects on yield are varied similar to variable phenotypic responses based on visual injury. Mesotrione applied postemergence at 100 g/ha^{-1} reduced the yield of 'Del Monte 20-38' by 7% (29). Increasing the application rate to 200 g/ ha^{-1} resulted in a 28% yield reduction. Despite the presence of injury 7 and 14 days after treatment, yield of eight sweet corn hybrids was unaffected by postemergence applications of nicosulfuron in one study (20), whereas in a similar study, nicosulfuron significantly reduced the yield of 'Silver Xtra Sweet' by 74 to 100% (35). Nicosulfuron plus rimsulfuron applied postermergence reduced the yield of six sweet corn hybrids including 'Del Monte 20-38', 'GH 1698', 'Cabara', 'Supersweet Jubilee', 'GH 1685', and 'Even Sweeter' by 30 to 100% (24). Yield reductions were more common when the application rate was increased from 25 g/ha^{-1} to 50 g/ha^{-1}. Foramsulfuron, an ALS-inhibiting herbicide, applied postemergence resulted in plant death or yield reductions of more than 92% to 'Del Monte 20-38'; whereas yield of the hybrids 'Calico Belle' and 'GH 2684' were reduced 34 to 54% (8). Bentazon, a photosystem II-inhibiting herbicide, applied postemergence reduced the yield of 'Del Monte 20-38' by 94% (9).

In field corn, postemergence applications of foramsulfuron resulted in yield losses of 6 and 15% in two trials (7). Applications made at later growth stages (i.e., V8 to V12) resulted in greater levels of injury and yield reduction than those made at earlier growth stages (i.e., V2 to V6) (7). ALS-inhibiting herbicides applied at early growth stages appear to be metabolized before the initiation of ear development, whereas symptoms of ear malformation and loss of kernel rows (ear pinching) result from applications of ALS-inhibiting herbicides at later growth stages. At the V12 growth

stage, applications of foramsulfuron caused pinching in 25 to 40% of ears harvested from two different hybrids (7).

Although differential yield responses of sweet corn hybrids to postemergence herbicides occur, little or no research relates sweet corn yield to hybrid genotype for the CYP alleles affecting herbicide metabolism. The objective of this research was to determine if yield is affected by the genetic condition of sweet corn hybrids at the CYP locus controlling P450 metabolism of herbicides when mesotrione and nicosulfuron are applied during the V3 to V5 and V5 to V7 growth stages.

MATERIALS AND METHODS

Twenty-three hybrids developed by seven seed companies and the University of Illinois were evaluated in 2007, 2008, and 2009 at the University of Illinois Vegetable Crops Farm, Champaign, IL (Table 3.1). Mesotrione and nicosulfuron were evaluated in separate experiments. Each trial included 16 hybrids. Eleven hybrids were common to both trials. The genotype of each hybrid at the CYP locus affecting herbicide metabolism was identified in a previous experiment based on segregation of F_2 and testcross progeny (32). Mesotrione trials included five hybrids homozygous for functional CYP alleles, five hybrids homozygous for mutant (i.e., nonfunctional) CYP alleles, and six hybrids heterozygous for functional and mutant CYP alleles. Nicosulfuron trials included eight hybrids homozygous for functional CYP alleles and eight heterozygous hybrids. Hybrids homozygous for mutant alleles were not included in nicosulfuron trials, because ALS-inhibiting herbicides kill these hybrids. Each trial included 13 or 14 commercially-available hybrids and a set of near isogenic hybrids differing primarily for alleles at the CYP locus on chromosome 5S (Table 3.1). Near

isogenic hybrids (46 x 52 or 95 x 69) were produced from crosses of near isogenic inbred lines selected in the F_6 generation for tolerant (T) and sensitive (S) reactions to mesotrione.

Experimental approach. The experimental design was a split-block arrangement of a randomized complete block, with four replicates. Each CYP genotype was replicated within a block. Herbicide treatments were applied to main plots. Hybrids were grown in subplots. An experimental unit was four 3.5 m-long rows, spaced 76 cm apart with approximately 16 plants, representing a commercial field population of about 67,700 plants ha^{-1} (≈ 27,400 plants/A). The soil type was a Flanagan silt loam (fine, smectitic, mesic Aquic Argiudoll). Fields were fertilized with 202 kg N ha^{-1}. Preplant tillage included two passes each of a field cultivator and spring-tine harrow. Early-season weeds were controlled with a preemergence application of 2.2 kg atrazine ha^{-1} plus 1.8 kg S-metolachlor ha^{-1}. Hand weeding was done as necessary.

Three treatments included a non-treated control and herbicide applications between the growth stages of V3 and V5 or V5 and V7. Mesotrione was applied at 210 $g \cdot ha^{-1}$ a.i. plus 1% v/v petroleum oil concentrate. Nicosulfuron was applied at 70 $g \cdot ha^{-1}$ a.i. plus 1% v/v petroleum oil concentrate and 2.2 $kg \cdot ha^{-1}$ ammonium sulfate. Herbicides were applied at 190 $L \cdot ha^{-1}$ spray volume between May 29 and June 26 (Table 3.2). Plants were assessed visually 7 days after treatment (DAT) for the percentage leaf area chlorotic or necrotic and for stunting. Height of three representative plants in each plot was measured 7 and 14 DAT after application of mesotrione or nicosulfuron. Height of plants in herbicide treated plots was calculated as a percentage of height of plants in the nontreated control for each hybrid.

The date when silks had emerged 1 to 2 cm from 50% of plants in an experimental unit (i.e., the mid-silk stage) was recorded for each hybrid. Primary ears of ten consecutive plants in the middle two rows of each experimental unit were harvested and weighed 21 days after the mid-silk stage. Yield, measured as green ear weight, was converted to tons per ha^{-1}. After weighing, ears harvested from nicosulfuron trials were husked to assess loss of kernel rows.

Statistical analysis. Yield, crop injury, plant height, and ear pinching were analyzed separately by analysis of variance (ANOVA) using the Proc Mixed procedure of SAS, version 9.2 (SAS Institute, 2007). Years and blocks were considered random variables. Genotypes and herbicide treatments were considered fixed variables. Effects were declared significant at $P \leq 0.05$. Residuals were tested for normality using the Proc Univariate procedure of SAS. Residuals were plotted against predicted values using the Proc Gplot procedure of SAS to assess homogeneity of error variance. Assumptions of ANOVA were met. Slice statements were used to partition the genotype by herbicide treatment interaction by genotype. Comparisons of herbicide treatments within genotypic classes were done with single degree of freedom contrast statements. Hybrid means were separated by Fisher's least significant difference (LSD) using the pdmix800 SAS macro (36).

RESULTS

Years were a significant source of variation in the statistical analysis of injury, plant height, yield, and ear pinching. Therefore, analyses were done separately for each year.

Mesotrione trials. In 2007 and 2009, injury from mesotrione applications during the V3 to V5 or V5 to V7 growth stages differed significantly among hybrids with different CYP genotypes. Crop injury 7 DAT was greater on hybrids with fewer functional CYP alleles (Fig 3.1). Injury also was greater following application of mesotrione during the V3 to V5 growth stages than during the V5 toV7 growth stages (Fig. 3.1). Injury 7 DAT with mesotrione at the V3 to V5 stage varied among years, ranging from 4 to 18% on *CYPCYP* hybrids, 16 to 44% on *CYPcyp* hybrids, and 39 to 87% on *cypcyp* hybrids. Injury 7 DAT with mesotrione at the V5 to V7 stage also varied among years and ranged from 0 to 1%, 1 to 8%, and 21 to 33% on *CYPCYP* hybrids, *CYPcyp* hybrids, and *cypcyp* hybrids, respectively. Injury from mesotrione was appreciably lower on all genotypes in 2008 than in 2007 or 2009. Severity of injury was nearly equivalent in 2007 and 2009. No injury occurred in the nontreated control plots.

Mesotrione treatments also resulted in mild to substantial stunting (Appendix C.1). Severity of stunting varied among years, treatments, and CYP genotypes. Stunting was most frequent and severe following mesotrione application during the V3 to V5 growth stages. Heights of *CYPCYP* hybrids treated with mesotrione did not differ significantly from the nontreated control. In general, *CYPcyp* hybrids were unaffected or stunted mildly (i.e., ≤11%) following mesotrione application. In four of five comparisons at 14 DAT, *cypcyp* hybrids were stunted more than 10%, including V3-V5 applications in 2007 and 2009 that caused greater than 30% stunting 14 DAT (Appendix C.1).

The effect of mesotrione on sweet corn yield differed significantly among hybrids with different CYP genotypes. The CYP genotype by herbicide treatment

interaction term also was significant in the ANOVA of yield in 2007 and 2009. Slice statements partitioning the interaction by CYP genotype indicated the effect of mesotrione treatments were significant only for *cypcyp* hybrids (Appendix C.2). Yield of *CYPCYP* and *CYPcyp* hybrids was unaffected by mesotrione (Fig. 3.1; Appendix C.2). Yield of the group of *cypcyp* hybrids was reduced 27 and 24% by mesotrione applications during the V3 to V5 growth stages in 2007 and 2009, respectively; whereas yield of this group of hybrids was significantly reduced 13 and 7% by mesotrione applications during the V5 to V7 growth stages in 2007 and 2009, respectively (Fig. 3.1). Mesotrione treatments did not affect yield in 2008.

Among the five *cypcyp* hybrids, yield reductions resulting from mesotrione applications during the V3 to V5 growth stages ranged from 12 to 36% in 2007 and from 14 to 40% in 2009 (Table 3.3). Applications of mesotrione during the V5 to V7 growth stages reduced yields of individual *cypcyp* hybrids from 9 to 22% in 2007 and from 10 to 12% in 2009 (Table 3.3). Yields were not reduced on *cypcyp* hybrids in 2008 (Table 3.3). Among these five hybrids, yield of '177 A' was affected most and yield of 'EX 08705770' was affected least by mesotrione.

Nicosulfuron trials. In 2007 and 2009, injury from nicosulfuron applications during the V3 to V5 or V5 to V7 growth stages differed significantly between *CYPCYP* and *CYPcyp* hybrids. Crop injury 7 DAT was greatest on *CYPcyp* hybrids following application during the V3 to V5 growth stages (Fig. 3.2). Injury 7 DAT with nicosulfuron during the V3 to V5 growth stages was 5 and 21% on *CYPCYP* hybrids and 28 and 54% on *CYPcyp* hybrids in 2007 and 2009, respectively (Fig. 3.2). Injury 7 DAT with nicosulfuron during the V5 to V7 growth stages was 4 and 7% on *CYPCYP*

hybrids and 16% on *CYPcyp* hybrids in 2007 and 2009, respectively (Fig. 3.2). No visible injury resulted from nicosulfuron applications in 2008.

Nicosulfuron treatments caused mild plant stunting (Appendix C.1). Severity of stunting varied among years, treatments, and CYP genotypes. Stunting 7 or 14 DAT with nicosulfuron during the V3 to V5 growth stages was greatest on *CYPcyp* hybrids, ranging from 10 to 25% (Appendix C.1). Plants of *CYPCYP* hybrids were stunted up to 12% 7 or 14 DAT with nicosulfuron during the V3 to V5 growth stages (Appendix C.1). Stunting caused by nicosulfuron application during the V5 to V7 growth stages ranged from 5 to 8% on *CYPcyp* hybrids and from 4 to 6% on *CYPCYP* hybrids (Appendix C.1).

The effect of nicosulfuron on sweet corn yield differed significantly among hybrids with different CYP genotypes, as indicated by a significant CYP genotype by herbicide treatment interaction in the ANOVA of yield in 2007 and 2009. Applications of nicosulfuron did not affect the yield of *CYPCYP* hybrids (Fig. 3.2; Appendix C.3). Yield of *CYPcyp* hybrids was significantly reduced 8 to 22% by nicosulfuron applications during the V3 to V5 or V5 to V7 growth stages (Fig. 3.2; Appendix C.3).

Among eight *CYPcyp* hybrids, significant yield reductions due to nicosulfuron applications during the V3 to V5 growth stages ranged from 9 to 57% in 2007, 12 to 28% in 2008, and 11 to 57% in 2009 (Table 3.4). When nicosulfuron was applied during the V5 to V7 growth stage, significant yield reductions to individual *CYPcyp* hybrids ranged from 9 to 18% in 2007, 12 to 18% in 2008, and 15 to 50% in 2009 (Table 3.4). Although the effect of nicosulfuron on yield of these hybrids varied among

hybrids, years, and time of application, 'Double Gem' was consistently affected more severely than the other seven hybrids.

Nicosulfuron treatments caused a reduction in kernel rows of harvested ears. The percentage of affected ears differed significantly among hybrids with different CYP genotypes in 2007 and 2009 (Appendix C.4). Loss of kernel rows was more severe on *CYPcyp* hybrids, ranging from 9 to 12% and 13 to 66% at the V3 to V5 and V5 to V7 growth stages, respectively (Appendix C.4). On *CYPCYP* hybrids, less than 10% of ears lost kernel rows following applications of nicosulfuron except for the V5 to V7 applications in 2008 and 2009, which resulted in 14 and 25% affected ears, respectively (Appendix C.4).

DISCUSSION

A single CYP gene or group of closely linked CYP genes on the short arm of chromosome 5 affect the response of sweet corn hybrids to several postemergence corn herbicides with multiple modes of action, including nicosulfuron and mesotrione (33). Levels of injury differ among sweet corn hybrids with different CYP genotypes probably as a result of differential rates of herbicide metabolism (32). Hybrids homozygous for functional CYP alleles (i.e., *CYPCYP*) are not substantially or consistently injured by P450-metabolized herbicides. Hybrids homozygous for mutant alleles (i.e., *cypcyp*) are frequently killed or severely injured by P450-metabolized herbicides. Injury to hybrids heterozygous for a functional and a mutant allele (i.e., *CYPcyp*) is variable but typically more similar to injury on *CYPCYP* hybrids than *cypcyp* hybrids. Environment and other factors also affect the degree to which sweet corn is injured by these herbicides. When trials were conducted in arid regions, such as

Idaho and Colorado, *CYPcyp* and *cypcyp* hybrids were uninjured by P450-metabolized herbicides (32).

Results of this experiment demonstrate that the genetic condition of hybrids at the CYP locus on chromosome 5S affects the yield of sweet corn following application of mesotrione or nicosulfuron at the V3 to V5 or the V5 to V7 growth stages. However, yields are not affected entirely the same as injury responses. Injury to hybrids with different CYP genotypes was not always associated with significant yield losses. Following mesotrione application, *cypcyp* hybrids were severely injured and yield was reduced significantly except in 2008 when weather was dry prior to application. Yields of *CYPcyp* and *CYPCYP* hybrids were not reduced by mesotrione even though *CYPcyp* hybrids had levels of injury as high as 44%. Injury from nicosulfuron was more severe on *CYPcyp* hybrids than on *CYPCYP* hybrids, and yields of *CYPcyp* hybrids were reduced by nicosulfuron. Yields of *CYPCYP* hybrids were unaffected. Thus, while the CYP genotype may determine whether or not a hybrid is likely to be injured by a P450-metabolized herbicide, adverse effects on yield may depend on the type and severity of injury caused by herbicides with different modes of action.

Some of the sweet corn hybrids evaluated for yield responses in previous herbicide trials have subsequently been identified as homozygous or heterozygous for CYP alleles on the short arm of chromosome 5 (33). As in this study, yields of *cypcyp* hybrids were significantly reduced by mesotrione or ALS-inhibiting herbicides. Effects of ALS-inhibiting herbicides on yield of *CYPcyp* hybrids were variable. Yield of *CYPcyp* hybrids was unaffected by mesotrione. Yields of *CYPCYP* hybrids usually were not affected by these herbicides. Yields of two *cypcyp* hybrids (‘Merit’ and ‘Del

Monte 20-38') were reduced significantly in each of 30 evaluations of ALS-inhibiting herbicides in 16 previous trials (8, 19, 24, 25, 27). Yields of both hybrids were reduced 92 to 100% because application of ALS-inhibiting herbicides usually killed these hybrids. Also, yield of 'Del Monte 20-38' was reduced 7 and 28% in two previous evaluations of mesotrione (29). Yields of six *CYPcyp* hybrids ('Delectable', 'Eliminator', 'Even Sweeter', 'How Sweet It Is', 'Jubilee', and 'Supersweet Jubilee') were reduced significantly in 8 of 52 evaluations of ALS-inhibiting herbicides in 18 previous trials. Among these hybrids, yield losses ranged from 14 to 48% (15, 24, 26, 35). Yield of 'Supersweet Jubilee' was unaffected by mesotrione in one trial (29). Yields of three *CYPCYP* hybrids ('Challenger', 'Incredible', and 'Miracle') were not affected in 22 of 24 evaluations of ALS-inhibiting herbicides in nine previous trials (8, 19, 24, 25, 26, 27). Yield of 'Challenger' was reduced 15 to 23% by applications of nicosulfuron plus rimsulfuron in one trial (26). Yield losses on *CYPCYP* hybrids following application of mesotrione have not yet been reported.

The potential for crop injury and yield loss following applications of P450-metabolized herbicides is primarily affected by CYP genotype. Environmental conditions and other inherent genetic differences among hybrids also affect the potential for yield loss. In this experiment, yield losses from mesotrione were generally consistent among *cypcyp* hybrids, but varied among years. In 2008, abnormally dry weather conditions preceded herbicide applications (Table 3.2), and plants exhibited symptoms of drought stress. In 18 site-years, the lowest levels of mesotrione injury to sweet corn occurred in arid climates or when abnormally dry conditions preceded

herbicide application (32, 41). In this experiment, mesotrione caused less crop injury and yields were not reduced significantly among *cypcyp* hybrids in 2008.

In addition to CYP genotype and environmental conditions, applications of P450-metabolized insecticides or other herbicides could affect the potential for injury and yield loss following applications of P450-metabolized herbicides. Organophosphate insecticides are known to interfere with P450-metabolism in corn (2). Terbufos, an organophosphate insecticide, reduces metabolism of nicosulfuron in corn while increasing its uptake (10). Reduced rates of nicosulfuron metabolism due to organophosphate application in *CYPcyp* and *cypcyp* hybrids could result in increased injury and yield loss. Injury from nicosulfuron on 'Jubilee', a *CYPcyp* hybrid, was greater when plants also were treated with terbufos (22, 21). Nicosulfuron reduced the yield of 'Jubilee' plants treated with terburos, while yield of plants not treated with terbufos was unaffected by nicosulfuron (21). Similarly, applications of herbicides with different detoxification pathways could enhance injury from P450-metabolized herbicides on hybrids with mutant cyp allele(s). Mesotrione and atrazine, a photosystem II inhibitor, are known to have synergistic activity (1). Recently, postemergence applications of topramezone (another HPPD-inhibiting herbicide) and atrazine killed *cypcyp* sweet corn inbreds and hybrids in Idaho nurseries (J. K. Pataky, personal communication, 2009). Additional research is necessary to determine the extent to which P450-metabolized insecticides and other herbicides interact with P450-metabolized herbicides to affect crop injury and yield of *CYPcyp* and *cypcyp* hybrids.

Loss of kernel rows caused by nicosulfuron occurred on both *CYPCYP* and *CYPcyp* hybrids. Previously, loss of kernel rows following application of

foramsulfuron, an ALS-inhibiting herbicide, was reported on dent corn hybrids differing in foramsulfuron tolerance (7). These malformed ears are unmarketable in sweet corn sold for fresh consumption. Thus, yield losses from ALS-inhibiting herbicides may be even greater to fresh market sweet corn where yields are based on the number of marketable ears.

The results of this experiment demonstrate the importance of eliminating mutant (i.e., nonfunctional) cyp alleles from commercial sweet corn hybrids, inbreds, and breeding germplasm. Nevertheless, *CYPcyp* or *cypcyp* hybrids may still be grown because of their superior agronomic or horticultural traits. Herbicides and application timing should be selected carefully to avoid injury when these hybrids are grown. For example, certain HPPD-inhibiting herbicides applied alone, such as topramezone, appear safe for all CYP genotypes at labeled rates (39). Crop safeners, such as isoxadifen-ethyl, can enhance P450 metabolism of herbicides and reduce injury to hybrids with at least one functional CYP allele (38, 39). However, crop safeners do not appear to enhance P450 metabolism in hybrids that lack a functional CYP allele (39).

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Table 3.1. Sweet corn hybrids differing in number of functional CYP alleles grown to evaluate yield responses to postemergence applications of mesotrione or nicosulfuron in field experiments in 2007, 2008, and 2009

Injury phenotype, CYP genotype[a], and hybrid cultivar	Seed source[b]	Experiment[c]
Tolerant (*CYPCYP*)		
46 x 52 TT	UI	nico
95 x 69 TT	UI	meso
Ambrosia	Cr	meso, nico
GH 6462	Rog	meso, nico
GSS 1477	Rog	meso, nico
HM 2390	HM	meso, nico
Luscious TSW	MM	nico
Obsession	Sem	nico
Rustler	HM	nico
Intermediate (*CYPcyp*)		
46 x 52 TS	UI	nico
95 x 69 TS	UI	meso
277A	IFSI	meso, nico
Argent	Cr	meso, nico
Coho	HM	meso, nico
Double Gem	MM	nico
GH 2669	Rog	meso, nico
Max	HM	nico
Supersweet Jubilee Plus	Rog	meso, nico
Sensitive (*cypcyp*)		
95 x 69 SS	UI	meso
177A	IFSI	meso
Del Monte 20-38	DMC	meso
EX 08705770	Sem	meso
Merit	Sem	meso

[a] Hybrid genotype at a single CYP locus identified in a previous experiment based on segregation of F_2 and testcross progeny (32).

[b] Cr = Crookham Seed Co.; DMC = Del Monte Corp.; HM = Harris Moran Seed Co.; IFSI = Illinois Foundation Seed, Inc.; MM = Mesa Maize; Rog = Syngenta Seeds, Inc., Rogers Brands; Sem = Seminis, Inc.; UI = Dr. Jerald Pataky, University of Illinois.

[c] Indicates experiment, where meso = mesotrione and nico = nicosulfuron.

Table 3.2. Environmental conditions during, and total precipitation seven days prior to, application of mesotrione or nicosulfuron to sweet corn hybrids at the V3-V5 or V5-V7 growth stages in 2007, 2008, and 2009

	2007		2008		2009	
Conditions	**V3-V5[a]**	**V5-V7**	**V3-V5**	**V5-V7**	**V3-V5**	**V5-V7**
Application date	May 29	June 6	June 18	June 24	June 17	June 26
Temperature at application (C)	33	24	26	28	27	30
Relative humidity at application (%)	35	35	27	39	65	57
Total precipitation seven days prior (cm)[b]	1.3	0.8	0.2	0.3	7.1	4.5

[a] Number of visible leaf collars when herbicides were applied.
[b] Total rainfall in the seven days prior to application.

Table 3.3. Relative yield (ear weight) of sweet corn hybrids homozygous for mutant cyp alleles, expressed as a percentage yield of the nontreated control, following applications of mesotrione during the V3-V5 or V5-V7 growth stages in 2007, 2008, and 2009

	2007		2008		2009		
Hybrid	**V3-V5[a]**	**V5-V7**	**V3-V5**	**V5-V7**	**V3-V5**	**V5-V7**	**Mean**
	----------Yield as % of control----------						
177A	66*	81*	85	93	60*	90*	79
95 x 69 SS	64*	90*	116	102	75*	90*	90
Del Monte 20-38	76*	78*	92	101	77*	88*	85
EX 08705770	88*	94	103	103	86*	106	97
Merit	69*	91*	95	101	82*	90*	88

[a] Growth stage determined by the number of visible leaf collars.
[b] Within a given year and hybrid combination, values with an asterisk differed significantly from the nontreated control at $P \leq 0.05$.

Table 3.4. Relative yield (ear weight) of sweet corn hybrids heterozygous for functional and mutant CYP alleles, expressed as a percentage yield of the nontreated control, following applications of nicosulfuron during V3-V5 or V5-V7 growth stages in 2007, 2008, and 2009

	2007		2008		2009		
Hybrid	**V3-V5[a]**	**V5-V7**	**V3-V5**	**V5-V7**	**V3-V5**	**V5-V7**	**Mean**
	----------Yield as % of control----------						
277A	88*[b]	86*	94	98	92	94	92
46 x 52 TS	94	94	90	90	90	83*	90
Argent	84*	99	95	95	89*	92	92
Coho	90*	93	93	82*	92	84*	89
Double Gem	43*	82*	72*	88*	43*	50*	63
GH 2669	91*	98	103	93	92	85*	94
Max	87*	91*	99	102	87*	79*	91
Supersweet Jubilee Plus	90*	89*	88*	87*	82*	65*	84

[a] Growth stage determined by the number of visible leaf collars.

[b] Within a given year and hybrid combination, values with an asterisk differed significantly from the nontreated control at $P \leq 0.05$.

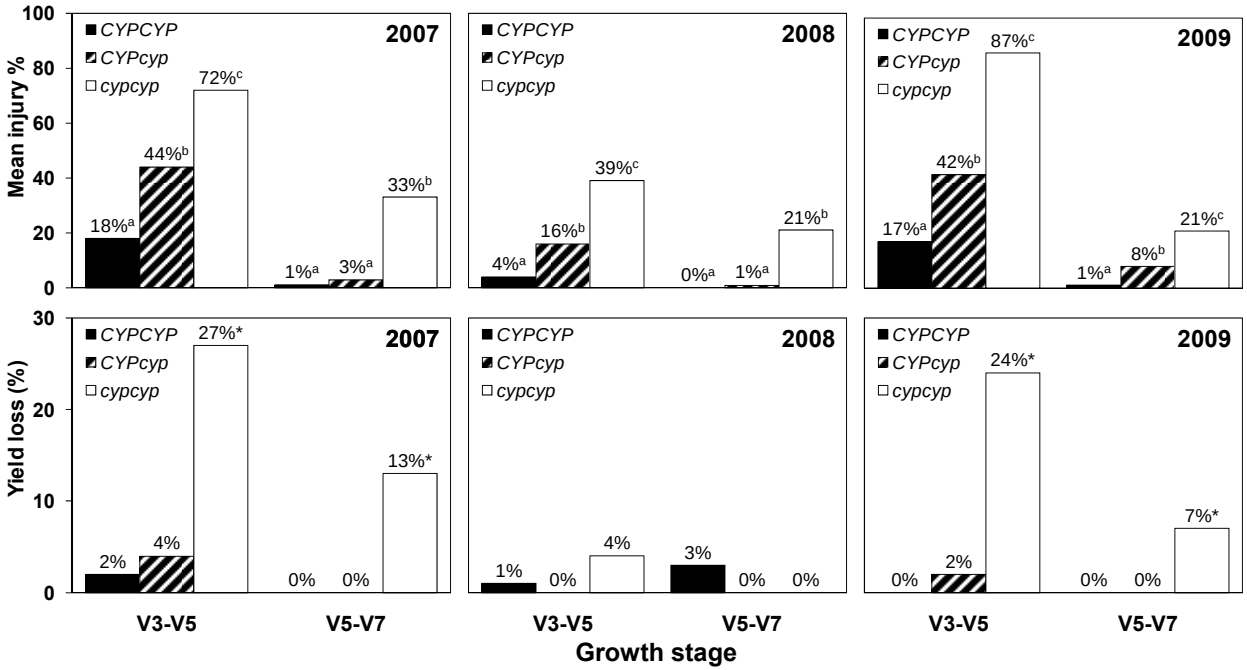


Figure 3.1. Injury 7 DAT and yield loss in sweet corn hybrids differing in number of functional *CYP* alleles following mesotrione application during the V3-V5 or V5-V7 growth stages. Within a given year and growth stage, injury values followed by different letters differ significantly at $P \leq 0.05$. Mean yield loss followed by an asterisk differs significantly from the nontreated control at $P \leq 0.05$.

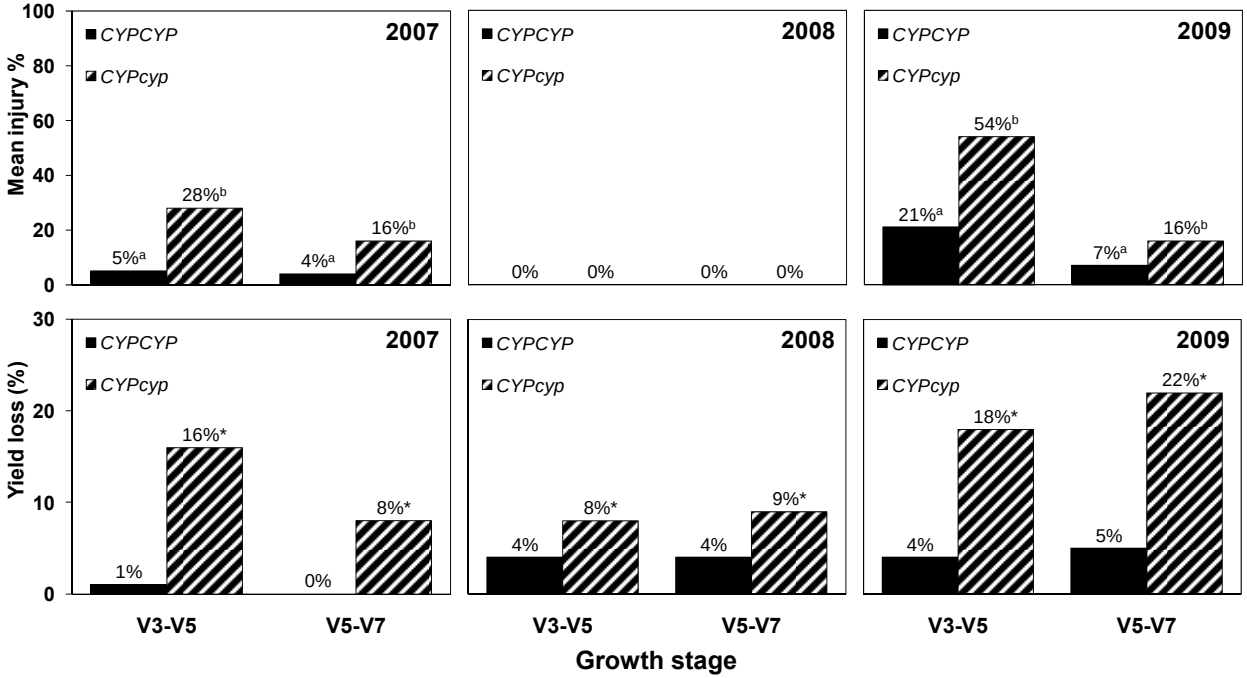


Figure 3.2. Injury 7 DAT and yield loss in sweet corn hybrids differing in number of functional *CYP* alleles following nicosulfuron application during the V3-V5 or V5-V7 growth stages. Within a given year and growth stage, injury values followed by different letters differ significantly at $P \leq 0.05$. Mean yield loss followed by an asterisk differs significantly from the nontreated control at $P \leq 0.05$.

APPENDIX A

ADDITIONAL TABLES FROM CHAPTER 1

Appendix A.1. Locations and planting dates of field trials in which the incidence of systemic Stewart's wilt was assessed from 1998 to 2003

Year and location	Planting date
1998	
Mendota, IL	June 20
Urbana, IL	April 24
Urbana, IL	June 30
1999	
Arcola, IL	May 17
Brownstown, IL	May 20
Collinsville, IL	May 2
Dixon Springs, IL	May 21
Henderson, KY	April 23
Manito, IL	May 1
Manito, IL	July 5
Mendota, IL	July 9
Shelbyville, IL	May 4
Urbana, IL	April 30
Urbana, IL	July 6
2000	
Manito, IL	April 26
Urbana, IL	April 27
2001	
Newark, DE	April 25
2002	
Urbana, IL	April 16
2003	
Urbana, IL	July 22

Appendix A.2. Locations and planting dates of field trials in which incidence of systemic Stewart's wilt was assessed from 2003 to 2009

	Planting date						
Location	**2003**	**2004**	**2005**	**2006**	**2007**	**2008**	**2009**
Belleville	May 23	April 19	May 4	May 8	April 19	nt[a]	May 22
Brownstown	May 28	April 19	April 25	May 8	April 20	nt	nt
Carbondale	May 24	May 12	April 25	May 8	April 20	April 23	May 21
Dwight	April 25	April 16	April 29	April 28	April 23	May 10	May 23
Goodfield	April 27	April 16	April 20	April 28	April 24	May 6	May 24
Monmouth	April 15	April 15	April 19	April 17	April 21	May 6	May 6
New Berlin	April 14	April 14	April 18	April 27	April 21	April 24	April 24
Perry	April 14	April 15	April 15	April 17	April 22	May 8	April 23
Urbana	April 24	April 17	April 16	April 17	April 23	April 30	May 23

[a] No trial due to flooding.

Appendix A.3. A summary of weather data from April 15 to May 31 for trials in which hybrid z-scores for mean Stewart's wilt incidence were ≥0.35 or ≤-0.35 relative to mean incidence within a temperature index

			Number of trials with	
Z-scores	**n**	**Mean precipitation total (cm)**	**Precipitation events above 3.8 cm[a]**	**Temperature below 4.4ºC for 3 or more consecutive nights**
Positive	6	18.2	2	0
Negative	21	15.2	13	10

[a] Rainfall events greater than 3.8 cm in a 24 h period.

APPENDIX B

ADDITIONAL TABLES FROM CHAPTER 2

Appendix B.1. Sweet corn hybrids with susceptible or resistant reactions to maize dwarf mosaic grown in field trials assessing the effect of MDM infection on the severity of several fungal foliar diseases and one bacterial wilt disease in 2007, 2008, and 2009

Hybrid	Seed source[a]	MDM reaction[b]	Years evaluated
1179	IFS	7.7	3
1280	IFS	8.3	6
2178	IFS	8.3	3
179 A	IFS	8.4	7
277 A	IFS	8.5	11
282 A	IFS	8.9	9
Absolute	Sem	8.6	10
Ambrosia	Cr	9.0	13
Argent	Cr	8.8	12
Augusta	MM	8.6	5
Bojangles	Cr	8.9	7
Bold	Rog	3.8	9
Bonus	Rog	2.0	13
Brocade TSW	MM	8.9	10
Delectable	Cr	8.7	11
Devotion	Sem	9.0	8
El Toro	Sem	2.8	13
Eliminator	Cr	2.0	13
Harvest Gold	Sem	8.3	8
Jubilee	Rog	9.0	13
Lancelot	MM	8.6	7
Merlin	MM	8.6	11
Miracle	Cr	9.0	13
Native Gem	MM	8.6	5
Passion	Sem	8.6	5
Precious Gem	MM	9.0	11
Sensor	Sem	9.0	12
Snow White	HM	4.0	12
Synergy	Sem	8.4	5
Tuxedo	MM	8.7	7
Vision	IFS	8.6	7
White Saturn	Sdw	8.8	4

[a] Seed source: Cr = Crookham Co.; HM = Harris Moran Seed Co.; IFS = Illinois Foundation Seeds Inc.; MM = Mesa Maize; Rog = Syngenta Seeds Inc., Rogers Brands; Sem = Seminis Vegetable Seeds Inc.; Sdw = Seedway, LLC.

[b] Reaction to infection by MDMV and SCMV in University of Illinois sweet corn disease nurseries (Pataky et al., 2009), where 1 = resistant, 3 = moderately resistant, 5 = moderate, 7 = moderately susceptible, and 9 = susceptible.

Appendix B.2. Dates of inoculation in field trials in which the effect of maize dwarf mosaic infection on the severity of several fungal foliar diseases and one bacterial wilt disease of sweet corn was assessed in 2007, 2008, and 2009

	Date of inoculation					
Year and Disease[a]	**1**	**2**	**3**	**4**	**5**	**6**
2007						
MDM	8 June	11 June				
Common rust	18 June	21 June	29 June	3 July		
GLS	22 June	27 June				
NCLB	12 June	19 June	22 June			
NCLS	14 June	22 June	27 June			
SCLB	14 June	20 June	22 June	3 July		
2008						
MDM	17 June	18 June				
Common rust	23 June	25 June	1 July	3 July	8 July	
NCLB	24 June	30 June	3 July	9 July	10 July	
SCLB-A[b]	27 June	30 June	8 July			
SCLB	23 June	27 June	8 July	11 July		
SCLB-B[c]	27 June	30 June	8 July			
Stewart's wilt	24 June	26 June				
2009						
MDM	15 June	19 June				
MDM late[d]	30 June	1 July				
DLS trial 1	26 June	1 July	9 July			
DLS trial 2[e]	9 July	15 July	20 July	22 July		
Eyespot trial 1	25 June	30 June	1 July	8 July		
Eyespot trial 2[e]	20 July	22 July				
GLS	25 June	30 June	1 July	8 July	9 July	15 July
NCLS	25 June	30 June	2 July	8 July	14 July	
SCLB-A	26 June	30 June	2 July	8 July	23 July	
Stewart's wilt	25 June	29 June				

[a] Disease abbreviations: DLS = diplodia leaf streak, GLS = gray leaf spot, NCLB = northern corn leaf blight, NCLS = northern corn leaf spot, and SCLB = southern corn leaf blight.
[b] Trial inoculated with MDMV-A only.
[c] Trial inoculated with SCMV only.
[d] Inoculation of MDM in late-planted DLS and eyespot trials in 2009.
[e] Repeated trial of experiment in 2009.

Appendix B.3. Rating scale used to assess the severity of northern corn leaf spot caused by *Bipolaris zeicola* in field experiments to evaluate the effect of maize dwarf mosaic infection on the severity of foliar disease in 2007, 2008, and 2009

Rating	Description
1	pinpoint-sized chlorotic flecks
2	chlorotic 'strands of pearls' symptom with some necrotic spots 1-2 mm in width
3	scattered narrow, necrotic strands 10-20 mm in length and 1 mm in width
4	abundant narrow, necrotic strands 20-30 mm in length and 1 mm in width
5	sparse bands of necrotic strands 30-50 mm in length and 1 mm in width
6	sparse bands of necrotic strands 50-60 mm in length and 2-3 mm in width
7	dense bands of necrotic strands 60 mm in length and 2-3 mm in width
8	dense bands of necrotic strands ≥60 mm long and 2-3 mm in width, some spread to upper leaves
9	dense bands of necrotic strands >60 mm long and 2-3 mm in width, spread to upper leaves

Appendix B.4. Rating scale used to assess the severity of southern corn leaf blight caused by *Bipolaris maydis* in field experiments to evaluate the effect of maize dwarf mosaic infection on the severity of foliar disease in 2007, 2008, and 2009

Rating	Description
1	chlorotic spots
2	chlorotic spots with scattered, small necrotic spots 1-2 mm in diameter
3	small necrotic spots 1-2 mm in diameter
4	medium necrotic spots 3-7 mm in diameter
5	medium necrotic spots 7-15 mm in diameter forming a sparse band
6	large necrotic spots ≥15 mm in diameter forming a dense band
7	large necrotic spots ≥15 mm with some spread approx. 50% of leaf tissue blighted
8	large necrotic spots ≥15 mm with approx. 75% of leaf tissue blighted
9	large necrotic spots ≥15 mm with approx. >75% of leaf tissue blighted

Appendix B.5. Rating scale used to assess the severity of eyespot caused by *Kabatiella zeae* in field experiments to evaluate the effect of maize dwarf mosaic infection on the severity of foliar disease in 2007, 2008, and 2009

Rating	Description
1	disperse necrotic spots <1 mm with large chlorotic halos
2	necrotic spots ≥1 mm lacking chlorotic halos
3	abundant necrotic spots ≥1 mm, little secondary spread
4	abundant necrotic spots >1 mm, moderate secondary spread
5	necrotic spots ≥2-3 mm in diameter with abundant secondary spread

Appendix B.6. Standardized z-scores used to classify sweet corn hybrids into nine categories of disease reaction to southern corn leaf blight (SCLB) in sweet corn hybrid disease nurseries at the University of Illinois from 1999 to 2009

	SCLB reaction category[a]								
	R		MR		M		MS		S
Year	1	2	3	4	5	6	7	8	9
1999	-1.4[b]	.	-0.8	-0.1	0.5	1.1	1.8	3.0	3.0
2000	-0.9	-0.5	0	0.9	.	1.8	2.8	3.7	3.7
2001	-1.4	-0.7	-0.1	0.3	0.6	1.3	1.9	.	1.9
2004	-0.9	-0.7	-0.6	0	0.4	1.2	2.1	3.0	3.0
2005	-1.1	-0.7	-0.02	0.3	0.6	1.3	2.1	2.8	2.8
2006	-1.0	-0.6	-0.1	0.4	0.8	1.3	2.2	3.1	3.1
2007	-0.9	-0.6	-0.2	0.1	0.5	0.9	1.2	1.9	1.9
2008	-1.8	-1.3	-0.7	-0.1	0.5	1.0	1.6	2.2	2.2
2009	-1.4	-0.8	-0.3	0.2	0.7	1.5	2.1	3.4	3.4
Mean	-1.2	-0.7	-0.3	0.2	0.6	1.3	2.0	2.9	2.9

[a] Reaction to SCLB, where 1 = resistant (R), 3 = moderately resistant (MR), 5 = moderate (M), 7 = moderately susceptible (MS), and 9 = susceptible (S).

[b] Standardized z-scores of the SCLB severity rating used as a boundary for reaction categories based on the mean severity and standard deviation of SCLB severity in each trial.

Appendix B.7. Sum of squares for treatment sources of variation, expressed as percentages of the total variation, from ANOVAs of the percentage of leaf area diseased based on factorial experiments with 32 sweet corn hybrids and two maize dwarf mosaic treatments

			Percentage (%) of total sums of squares[b]					
			ANOVA with MDM-R hybrids[c]			ANOVA without MDM-R hybrids[d]		
Foliar disease	MDM[a]	Year	Hybrid	Virus	Hybrid x virus	Hybrid	Virus	Hybrid x virus
Common rust	AB	2007	47	10***[e]	8	46	12***	7
Diplodia leaf streak	AB	2009	…[f]	…	…	0	22	0
		2009	…	…	…	4	10	3
Eyespot	AB	2009	…	…	…	0	84*	1
		2009	…	…	…	3	49	3
Gray leaf spot	AB	2007	26	41***	15**	18	51***	11
Northern corn leaf blight	AB	2007	37	30***	18*	31	40***	13
Northern corn leaf spot	AB	2007	18	54***	12	9	67***	6
		2009	20	38*	16*	13	48*	12
Southern corn leaf blight	AB	2007	20	54***	16**	12**	68***	9
Southern corn leaf blight	A	2009	19	42*	14	15	48*	10

[a] MDM treatment, where AB = inoculation with MDMV-A and SCMV and A = inoculation with MDMV-A only.
[b] Treatment sum of squares divided by total sums of squares multiplied by 100.
[c] Data analyzed with 27 MDM-susceptible and five MDM-resistant hybrid controls.
[d] Data analyzed without five MDM-resistant hybrid controls.
[e] Asterisks indicate corresponding mean squares statistically significant, where * = $P<0.05$,** = $P<0.01$, and *** = $P<0.001$.
[f] MDM-resistant hybrids not included.

Appendix B.8. The percentage leaf area diseased by common rust, Diplodia leaf streak, eyespot, gray leaf spot, northern corn leaf spot, southern corn leaf blight or northern corn leaf blight on leaves sampled from virus-free and maize dwarf mosaic (MDM) infected sweet corn plants in separate field trials assessing the effect of MDM infection on the severity of foliar diseases in 2007 and 2009

		Percentage (%) leaf area diseased[b]			
		MDM-susceptible[c]		MDM-resistant	
Disease[a]	Year	Virus-free	MDM	Virus-free	MDM
Common rust	2007	19	32	26	32
DLS	2009	32	64	…[d]	…
	2009	41	56	…	…
Eyespot	2009	3	33	…	…
	2009	1	8	…	…
GLS	2007	6	23	7	5
NCLB	2007	28	49	37	35
NCLS	2007	10	39	14	9
	2009	6	29	3	7
SCLB-AB[d]	2007	5	28	8	5
SCLB-A	2009	6	35	5	9

[a] Foliar fungal diseases, where DLS = Diplodia leaf streak, GLS = gray leaf spot, NCLS = northern corn leaf spot, NCLB = northern corn leaf blight and SCLB = southern corn leaf blight.

[b] Percentage of leaf area diseased determined using Assess.

[c] Hybrids susceptible or resistant to maize dwarf mosaic infection (Pataky et al., 2004).

[d] … = MDM resistant hybrids not included in trial.

[e] MDM treatment, where AB = MDMV-A/SCMV and A = MDMV-A only.

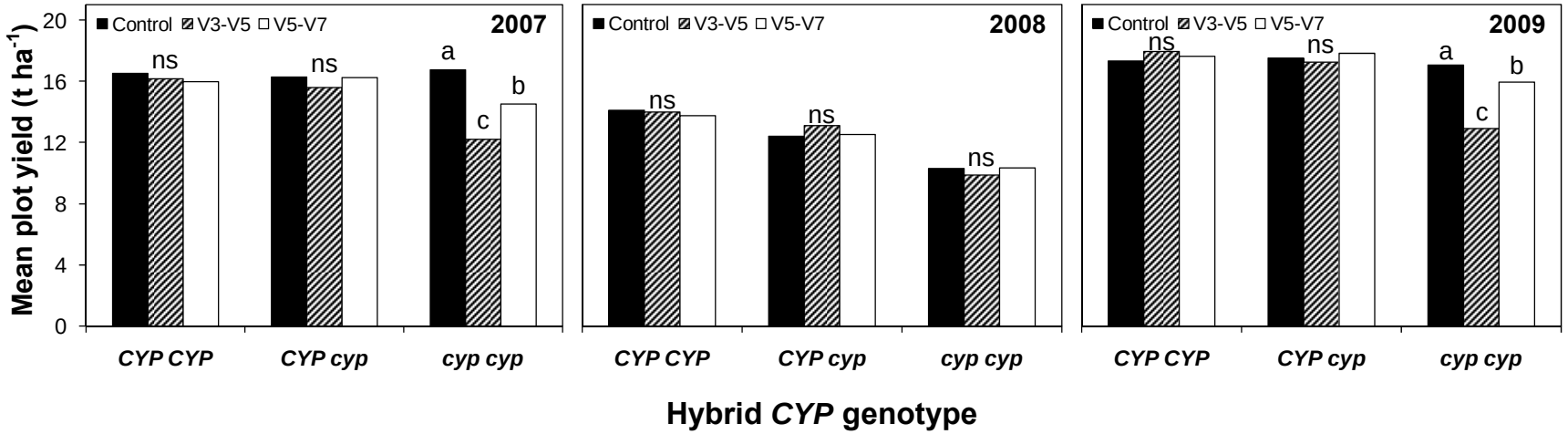
APPENDIX C

ADDITIONAL TABLES AND FIGURES FROM CHAPTER 3

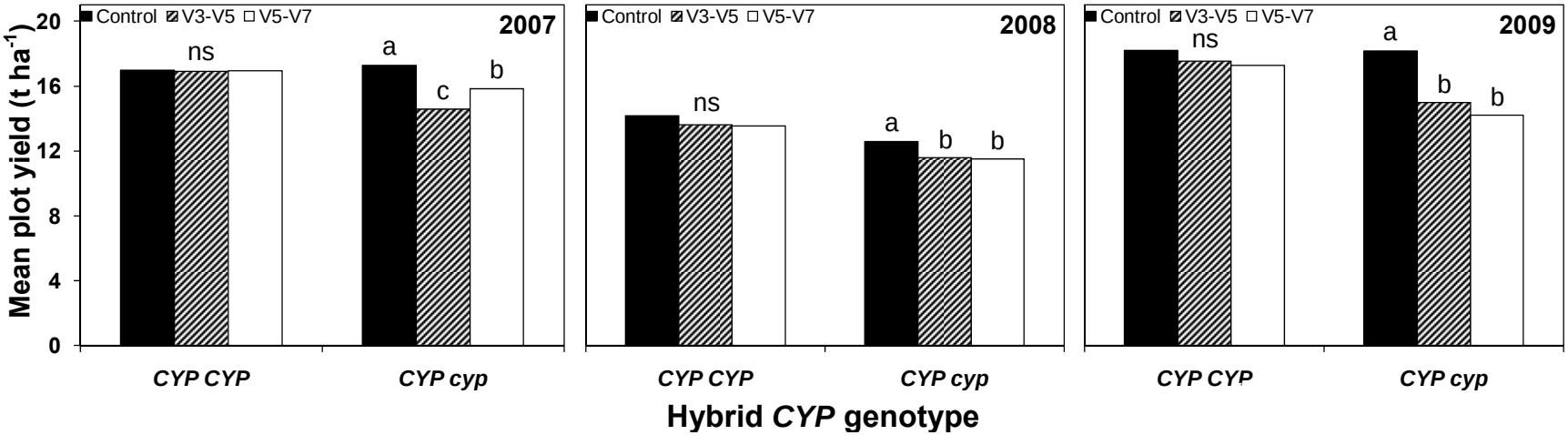
Appendix C.1. Height of sweet corn hybrids differing in number of functional CYP alleles expressed as a percentage of the nontreated control height 7 and 14 days after applications of mesotrione or nicosulfuron at V3-V5 or V5-V7 growth stages in 2007, 2008, and 2009

		2007				2008				2009			
Herbicide and	**Injury**	**V3-V5[b]**		**V5-V7**		**V3-V5**		**V5-V7**		**V3-V5**		**V5-V7**	
CYP genotype[a]	**phenotype**	**7[c]**	**14**	**7**	**14**	**7**	**14**	**7**	**14**	**7**	**14**	**7**	**14**
mesotrione		----------Height as % of control----------											
CYPCYP	tolerant	101	99	101	102	105	98	95	103	98	98	103	.[d]
CYPcyp	intermediate	92*[e]	89*	97*	95*	100	96	100	102	94	97	100	.
cypcyp	sensitive	77*	64*	98*	87*	94	84*	102	100	71*	65*	102	.
nicosulfuron													
CYPCYP	tolerant	92*	97	99	98	.	93*	.	96*	88*	99	94*	.
CYPcyp	intermediate	84*	86*	95*	95*	.	90*	.	97	75*	81*	92*	.

[a] Hybrid CYP genotype at a single CYP locus identified in a previous experiment based on segregation of F_2 and testcross progeny (Pataky et al., 2008).
[b] Growth stage determined by the number of visible leaf collars.
[c] Stunting measured 7 and 14 DAT.
[d] Height measurements unavailable.
[e] Within a year and genotype combination, means followed by an asterisk differ significantly from the nontreated control at P=0.05.



Appendix C.2. Yield (ear weight) of sweet corn hybrids differing in number of functional CYP alleles following mesotrione application during the V3-V5 or V5-V7 growth stages. Means followed by ns or the same letter do not differ significantly at P=0.05 for comparison of application treatments within CYP genotype.



Appendix C.3. Yield (ear weight) of sweet corn hybrids differing in number of functional CYP alleles following nicosulfuron application during the V3-V5 or V5-V7 growth stages. Means followed by ns or the same letter do not differ significantly at P=0.05 for comparison of application treatments within CYP genotype.

Appendix C.4. Percentage of ears with loss of kernel rows (i.e., ear pinching) on *CYPCYP* and *CYPcyp* sweet corn hybrids following applications of nicosulfuron during the V3-V5 or V5-V7 growth stages in 2007-2009

		Percentage of ears pinched[z]					
		2007		2008		2009	
CYP genotype[y]	Injury phenotype	V3-V5[x]	V5-V7	V3-V5	V5-V7	V3-V5	V5-V7
CYPCYP	tolerant	5 [a w]	7 [a]	7 [a]	14 [a]	4 [a]	24*[v a]
CYPcyp	intermediate	12*[a]	18*[b]	9 [a]	13 [a]	11* [a]	66* [b]

[z] Mean percentage of ears harvested from an experimental unit with visible loss of kernel rows (i.e., ear pinching).
[y] Hybrid genotype at a single CYP locus identified in a previous experiment based on segregation of F_2 and testcross progeny (Pataky et al., 2008).
[x] Number of visible leaf collars when herbicides were applied.
[w] Within a given year by growth stage combination, values followed by a different letter differed significantly at P=0.05.
[v] Within a given year by treatment combination, values with an asterisk differed significantly from the nontreated control at P=0.05.