Sugarcane Mosaic Virus Resistance in the Wisconsin Sweet Corn Diversity Panel

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ABSTRACT. Sugarcane mosaic virus [SCMV (*Potyvirus sugarcane mosaic virus*)] is an ssRNA virus that negatively affects yield in maize (*Zea mays*) worldwide. Resistance to SCMV is controlled primarily by a single dominant gene (*Scm1*). The goal of this study was to identify sweet corn (*Z. mays*) inbreds that demonstrate resistance to SCMV, confirm the presence of genomic regions previously identified in maize associated with resistance, and identify other resistant loci in sweet corn. Eight plants from each of 563 primarily sweet corn inbred lines were tested for SCMV resistance. Plants were inoculated 14 d after planting and observed for signs of infection 24 d after planting. A subset of 420 inbred lines were genotyped using 7504 high-quality genotyping-by-sequencing single-nucleotide polymorphism markers. Population structure of the panel was observed, and a genome-wide association study was conducted to identify loci associated with SCMV resistance. Forty-six of the inbreds were found to be resistant to SCMV 10 d after inoculation. The *Scm1* locus was confirmed with the presence of two significant loci on chromosome 6 ($P = 2.5 \times 10^{-8}$ and 1.6×10^{-8}), 5 Mb downstream of the *Scm1* gene previously located at Chr6: 14194429,14198587 and the surrounding 2.7-Mb presence–absence variation. We did not identify other loci associated with resistance. This research has increased information on publicly available SCMV-resistant germplasm useful to future breeding projects and demonstrated that SCMV resistance in this sweet corn panel is driven by the *Scm1* gene.

Sugarcane mosaic virus [SCMV (*Potyvirus sugarcane mosaic virus*)] has a worldwide geographic range and causes significant yield losses in maize [*Zea mays* (Kuntze et al., 1995; Shukla et al., 1989)]. SCMV causes an average of 10% to 15% yield loss in maize grown in China and is said to be the most important viral maize disease in Europe (Kuntze et al., 1997; Zhang et al., 2003). If plants are infected when young, SCMV can reduce ear weight, plant height, and total plant weight (Johnson et al., 1972; Kuntze et al., 1995). When SCMV coinfects with *Machlomovirus maize chlorotic mottle virus*, it can result in maize lethal necrosis disease (MLN), causing total yield loss (Wangai et al., 2012). SCMV is one of the positive-sense, single-strand RNA viruses found within the Potyviridae family, previously denoted as *Maize dwarf mosaic virus-B* (Shukla et al., 1989). Other viruses within the Potyviridae include *Potyvirus johnsongrass mosaic virus*, and *Potyvirus maize dwarf mosaic virus* (Shukla et al., 1989, 1992). In addition to maize, SCMV also infects sugarcane (*Saccharum* sp.) and sorghum (*Sorghum* sp.), causing stunting and chlorosis (Kuntze et al., 1995; Shukla et al., 1989). The virus is transmitted by corn

leaf aphids (*Rhopalosiphum maidis*) in a nonpersistent manner, and it is diagnosed with symptoms such as chlorosis, stunting, and reduced plant weight (Knocke et al., 1974). Chemical or agronomic control of aphids are not effective at eliminating transmission of SCMV (Charpentier, 1956). However, genetic resistance has proven to be an effective means of controlling SCMV (Liu et al., 2017; Xing et al., 2006). Genetic resistance is primarily linked with the *Scm1* gene within a large presence–absence variation (PAV) on chromosome 6 (Gustafson et al., 2018). Mosaic virus genetic resistance was transferred intentionally into sweet corn (*Z. mays*) as early as 1983 (Mikel et al., 1984).

The reaction to SCMV infection in field maize has been studied extensively. Kuntze et al. (1995) tested 124 European flint and dent maize inbreds for lasting resistance to SCMV under multiple conditions. Twenty-four inbreds showed resistance in both greenhouse and field conditions 4 weeks after inoculation. They also found only three inbreds completely resistant to SCMV 7 weeks after inoculation. Nineteen inbreds displayed delayed symptom expression (Kuntze et al., 1995). Subsequently, Kuntze et al. (1997) screened 122 European maize inbreds in greenhouse conditions and showed 34 inbreds had no symptoms 10 to 14 d after inoculation (DAI). A second experiment in field conditions found only three inbreds (D21, D32, and FAP1360A) that were completely resistant to SCMV and four inbreds (D06, D09, R2306, and FAP1396A) with partial resistance when evaluated 28 and 49 DAI (Kuntze et al., 1997).

Gustafson et al. (2018) evaluated SCMV resistance in field maize within the Wisconsin Diversity Panel under inoculated

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field conditions and found that 31 of the 578 inbreds tested were resistant to SCMV at 35 DAI. Gustafson et al. (2018) found that when inoculation was delayed from the three- to the six-leaf growth stage, symptom development was reduced, but inbreds that did not have the *Scm1* gene, due to the absence variant, were susceptible regardless of inoculation time points. In agreement, an earlier study found that younger plants are most susceptible to infection through inoculation, and that incidence of disease was halved when susceptible maize hybrids were inoculated at 11-leaf stage compared with five-leaf stage (Rosenkranz and Scott, 1978). Previous studies have determined that evaluation of SCMV resistance is not influenced significantly by testing in the field compared with greenhouse or by methods of manual inoculation, such as the rub method or the air-brush method (Kuntze et al., 1995).

Two loci conferring resistance to SCMV are known in maize, *Scm1* and *Scm2* (or *Scmv1* and *Scmv2*), on the short arm of chromosome 6 and the centromeric region of chromosome 3, respectively (Gustafson et al., 2018; McMullen and Louie, 1989; Melchinger et al., 1998; Simcox et al., 1995). *Scm1* and *Scm2* were first mapped in association with SCMV in 1989, and both were identified as dominant genes (McMullen and Louie, 1989). *Scm1* is a dominant gene, conferring resistance to infection at all developmental stages, especially resistance in early stages of development (DuBle et al., 2000; Gustafson et al., 2018; Jones et al., 2007; Tao et al., 2013). *Scm2* is associated with higher degree of resistance to SCMV infection in populations, especially at later stages of development and behaves epistatically with *Scm1* (DuBle et al., 2000; Xing et al., 2006). *Scm1* is located at Chr6: 14194429.14198587 in B73 v3 and is within a 2.7-Mb PAV spanning from 12.9 to 15.4 Mb. Gustafson et al. (2018), showed that all susceptible lines with the absence variant were found to be susceptible to SCMV and had an allele associated with susceptibility downstream at 19.4 Mb, due to the inability of making associations with missing single-nucleotide polymorphisms (SNPs). Liu et al. (2017) determined the causal gene at *Scm1* to be *Zmtrxh* which encodes an atypical h-type thioredoxin and suppresses viral RNA accumulation in the cytoplasm. *Scm2* is partially dominant for resistance to SCMV but often does not appear in association with resistance at early time points (Xia et al., 1999). Minor quantitative trait loci (QTL) have also been identified on chromosomes 1, 3, 5, and 10 that explain 6% to 10% of phenotypic variance in total (Gustafson et al., 2018; Melchinger et al., 1998; Xia et al., 1999).

Genome-wide association studies (GWAS) are frequently used in maize research to detect associations of genetic markers with traits of interest (Xiao et al., 2017). GWAS can identify associated loci with high resolution by using diverse populations that have undergone many generations of recombination, leading to a more rapid decay of linkage disequilibrium (LD; Flint-Garcia et al., 2003). Association mapping panels combined with GWAS have been used to gain insights on the genetic architecture of MLN and have found 24 SNPs associated with MLN resistance linked to 20 putative candidate genes that can potentially be used in MLN resistance breeding programs (Gowda et al., 2015). Although GWAS identifies associated loci and candidate genes, follow-up is often required to directly connect those loci with causal genes, making it useful predictively in many cases but not always immediately applicable (Broekema et al., 2020; Pierce et al., 2020). Additionally, GWAS has trouble identifying rare alleles or alleles that are highly fixed in the

population (Hamazaki et al., 2020). Three GWAS have been done previously on the Wisconsin Sweet Corn Diversity Panel (WSCDP), investigating loci associated with tocochromanols, carotenoid, and elemental variation in fresh sweet corn kernels. These studies suggest that GWAS are effective in the WSCDP (Baseggio et al., 2019, 2020, 2021).

The objectives of this study were to identify sweet corn inbreds with genetic resistance to SCMV, confirm the presence of genomic regions previously identified in field maize associated with resistance (the *Scm1* and *Scm2* loci), and determine if there were other putative resistance loci unique to the sweet corn panel.

Materials and Methods

GERMPLASM. In 2019, 563 sweet corn inbreds from the WSCDP were evaluated for resistance to SCMV under greenhouse conditions (Supplemental Table 1). These inbreds were part of the WSCDP, a diversity panel designed to evaluate the genetic architecture and breadth of molecular and phenotypic diversity within public, temperate, sweet corn germplasm. The WSCDP has 581 inbreds in the full panel, through the size and composition of the inbreds used in WSCDP experiments depending on the experiment's focus, germplasm availability, and resources (Baseggio et al., 2019, 2020, 2021). The pedigrees of many of the inbreds within the panel are known through internal records. Inbreds found to be SCMV-resistant had their pedigree traced for obvious inherited sources of genetic resistance. A large portion of the lines within the WSCDP are sourced from the University of Illinois–Urbana and University of Wisconsin–Madison sweet corn breeding programs (330 of the 563 in this study), due to availability and the extent of modern sweet corn germplasm derived from those programs.

PHENOTYPIC EVALUATION. The experiment was conducted at the University of Wisconsin–Madison Walnut Street Greenhouses (Madison). The greenhouse was kept at 24 to 28 °C during the day and 18 °C at night. Lighting was supplied by 400-W high-pressure sodium lights, four lights over each bench of 60 pots. Lights remained on for 16 h·d⁻¹ or until 400 μmol of natural light was reached. One hundred twenty inbreds were planted at a time in sets of 60 pots per bench and with the inbred's planting order determined at random. Eight kernels of each inbred were planted in 19.7-cm-diameter pots about 2 cm deep in media composed of 65% to 75% sphagnum peatmoss and 8% to 35% perlite (ProMix HP, Premier Tech Horticulture, Quakertown, PA). Plants were fertilized three times per week with 20N–4.4P–16.6K fertilizer (Peters Professional 20–10–20 Peat Lite Special Fertilizer; ICL Specialty Fertilizers, Summerville, SC).

Seedlings were inoculated 14 d after planting (DAP), when all inbreds were around the three-leaf stage to maximize susceptibility (Rosenkranz and Scott, 1978). The SCMV isolate used for inoculation was collected in 1996 from a Wisconsin sweet corn field. The amplified isolate was confirmed to be a SCMV isolate via RNA sequencing analysis and enzyme-linked immunoassay (Agdia, Elkhart, IN) and tested negative for *Maize dwarf mosaic virus*, *Wheat mosaic virus*, and *Wheat streak mosaic virus* (Gustafson et al., 2018). SCMV inoculum was maintained on a susceptible sweet corn hybrid, GH4927 (Syngenta, Basel, Switzerland), under isolated greenhouse conditions. A cycle of available inoculum was maintained by inoculating uninfected GH4927 14 DAP and planting new GH4927 every 14 d. Inoculum was prepared by grinding six to 10 leaves of the 21-d-old infected hybrid, with ≈250 mL of

municipal water and 5 g of carborundum using a mortar and pestle. Inoculation was done mechanically with the carborundum creating injury points by dipping a gloved hand in the inoculum and running the seedlings' leaves through pinched fingers, known as the rub-inoculation method (Knoke et al., 1974). Stand count was recorded for each pot on the same day as inoculation. Inbreds with zero germination were dropped from the study.

Symptoms were visually scored 10 DAI, and the number of plants per pot displaying any symptoms were recorded. Diagnostic symptoms were mosaic chlorosis on leaves with contrasting green on a background of paler green or yellow chlorotic areas, occasionally with streaks or stripes (Fuchs and Grüntzig, 1995). Uninfected plants did not show signs of chlorosis or mosaic patterning. To eliminate the possibility of false resistance ratings due to mistakenly uninoculated pots, inbreds that displayed no symptoms on any seedlings were tested again with three additional pots of eight kernels each planted all within the same round. Four control pots of GH4927 were included at random on each bench of 60 pots. The control pots were inoculated and served as a check for inoculation effectiveness. Each seedling was either rated as symptomatic or asymptomatic regardless of intensity of symptoms. Each pot of eight kernels was considered an experimental unit. The percentage of symptomatic plants for each inbred was calculated by dividing the total number of symptomatic seedlings by the total number of seedlings grown across all replicates. Inbreds with no symptomatic plants 10 DAI within their first planting and no symptomatic plants 10 DAI in their three follow-up replicates were labeled as resistant to SCMV. The rates of germination and symptomatic plants were evaluated for the susceptible control and for the panel at large. Correlation between rates of symptomatic plants and planting order or bench position were assessed, and no correlation was found.

GENOTYPING. In 2013, samples from each inbred in the WSCDP were taken and sequenced at the Cornell Biotechnology Resource Center (Ithaca, NY) using genotyping-by-sequencing (GBS) as described previously (Baseggio et al., 2019; Elshire et al., 2011). The sequence data used in this study has been archived in the National Center of Biotechnology Information (Bethesda, MD) BioProject under accession number PRJNA482446 and in the Sequence Read Archives under accession SRP154923. The raw GBS sequence data consisted of 955,690 SNPs called using the default parameters in the TASSEL 5 GBSv1 production pipeline (Bradbury et al., 2007) with the ZeaGBSv2.7 Production TagsOn-PhysicalMap file in B73 RefGen_v2 coordinates (Panzea, Ithaca, NY). The SNP genotype calls from this study were combined with the raw sequencing data of inbreds included in this screening within the genotyping study of the U.S. Department of Agriculture, Agriculture Research Service, North Central Regional Plant Introduction Station collection in Ames, IA (Romay et al., 2013). The raw SNP genotype calls from the combined set were filtered to retain only biallelic SNPs with greater than 10% call rate. This raw combined sequencing dataset has been used previously in several studies when analyzing the WSCDP for other traits (Baseggio et al., 2019, 2020, 2021). Markers were further filtered by removing SNPs with minor allele frequency of less than 5%, and SNPs with call rates of less than 90%—percentage of SNPs successfully genotyped for each inbred—within TASSEL and were pruned for LD at a rate of 0.98 or higher within SNPRelate (Bradbury et al., 2007; Zheng et al., 2012). The result was a set of 7504 non-imputed high-quality marker sites for 420 of the inbreds tested. Previous studies involving the WSCDP used the same GBS data, filtered differently

and imputed (Baseggio et al., 2019, 2020, 2021). We avoided imputation and worked with the raw sequencing data to ensure PAVs were maintained correctly.

ASSOCIATION MAPPING. This study's GWAS was conducted on these markers using the general P + K mixed model through the genetic analysis R package GWASpoly (Rosyara et al., 2016) with three principal components and the kinship matrix calculated by the realized relationship matrix based on VanRaden's method (VanRaden, 2008). The GWAS was run using a general model, meaning all heterozygotes have the same effect and there is no weighting based on allelic dosage. GWAS was run on the 420 inbreds with GBS data. One hundred forty-three inbred lines did not have associated genetic information because sequencing was conducted before the SCMV screening. These lines were screened despite the lack of sequencing data for the purpose of identifying their SCMV resistance status for the benefit of sweet corn breeding programs.

We estimated the presence of *Scm1* in the inbreds by comparing their haplotype near the *Scm1* locus to IL793a, an inbred with known *Scm1* presence in its pedigree and 0% symptomatic plants in this screening. We will refer to this haplotype as the *Scm1*-IL793a haplotype. The *Scm1*-IL793a haplotype was classified by three loci: the locus at 19.9 Mb which was associated with resistance during the initial GWAS that was closest to the *Scm1* gene and two SNPs nearest that 19.9-Mb locus. The locus at 19.9 Mb was chosen as the basis for the haplotype due to a lack of SNP coverage in the PAV where *Scm1* is located. A closer haplotype could not be used to infer *Scm1* presence because there was only one SNP within the PAV sequenced, and it was not enough of a high-quality locus to be included within our filtering parameters. Due to the commonality of the absence variation within the diversity panel, the PAV was not able to be captured by our GBS technique. Previous studies used a similar associated locus at 19.4 Mb to infer *Scm1* presence (Gustafson et al., 2018). A simple *t* test of the percentage of symptomatic plants was conducted between the inbreds with and without the *Scm1*-IL793a haplotype. We also conducted GWAS on the subset of 360 inbreds that did not have the *Scm1*-IL793a haplotype. Population structure analysis and visualization in the form of principal component analysis (PCA) was conducted with TASSEL (Bradbury et al., 2007), and R packages SNPRelate (Zheng et al., 2012) on the 420 inbred group. Population structure analysis was done specifically for this study due to our unique filtering parameters.

ASSOCIATED GENE DISCOVERY. Linkage disequilibrium decay was calculated using a full matrix comparison in TASSEL and visualized in R (Bradbury et al., 2007). We used the same filtering as described earlier, without LD pruning for a total of 12,012 marker sites to calculate the genome-wide LD within the 420 inbreds genetically analyzed. The LD decay was used to inform our candidate gene window of 250 kb. The loci of interest that scored above the 5% Bonferroni threshold were deemed significant, and gene models within a 250-kb window of the loci's flanking region were recorded as associated genes. The Maize B73 RefGen_v3 genome assembly was consulted for associated genes through MaizeGDB (Portwood et al., 2019).

Results

VARIATION IN RESISTANCE. Germination among the screened inbreds was strong with 96% of inbreds germinating three or more seedlings, the median stand count being seven seedlings

Fig. 1. Number of percent symptomatic inbred sweet corn plants among (A) the susceptible controls and (B) all screened inbreds.

from eight planted kernels, and the mean being 6.6 seedlings. Ninety-six percent of the control plants (Syngenta GH4927), which has the *scm1* allele, were symptomatic and 21% of the control pots had one or more asymptomatic plants (Fig. 1A). Given that *Scm1* confers a dominant form of resistance, asymptomatic plants among the controls are likely due to inoculation escapes or minor seed lot impurity.

Among the 563 inbreds tested, 559 germinated adequately for screening. From those 559 inbreds, at 10 DAI, 46 inbreds had 0% symptomatic plants. Forty-eight inbreds had fewer than 10% symptomatic plants at 10 DAI. Notably, C.I.540 was included and screened twice, and was rated as 0% and 6.7% symptomatic in the two screenings. Sixty-two inbreds had between 10% and $\leq 50\%$ plants showing symptoms (Supplemental Table 1). Two

hundred and thirty-six inbreds had between 50% and 90% plants with symptoms and 211 inbreds were $>90\%$ symptomatic (Fig. 1B).

Of the 46 inbreds that were classified as resistant to SCMV, 33 inbreds had associated GBS data. Within our sequenced panel, 60 inbreds were found with the *Scm1*- IL793a haplotype and 360 without. Our *t* test between the inbreds with and without the *Scm1*- IL793a haplotype found that the groups had significantly different percent symptoms [$t = 5.0$, $P = 4.6 \times 10^{-6}$ (Fig. 2)]. The group with the *Scm1*- IL793a haplotype had a mean symptomatic plant percentage of 50% and the group without had a mean percentage of 77%. Twenty-one of the 60 inbreds with the *Scm1*- IL793a haplotype had no plants that showed symptoms and were therefore classified as resistant.

Fig. 2. Number of percent symptomatic inbred sweet corn plants among the screened inbreds (A) with the *Scm1*- IL793a associated haplotype and (B) without the *Scm1*- IL793a associated haplotype.

Fig. 3. Principal component (PC) analysis of 420 sweet corn inbreds within the Wisconsin Sweet Corn Diversity Panel, labeled with their program of origin: CT = University of Connecticut–Storrs; IA = Iowa State University–Ames; IL = University of Illinois–Urbana/Champaign (Champaign, IL); IN = Purdue University (West Lafayette, IN); MN = University of Minnesota–Twin Cities (Minneapolis, MN); SD = South Dakota State University (Brookings, SD); WI = University of Wisconsin–Madison; Other = all other programs within the panel.

POPULATION STRUCTURE. Population structure was observed among the 420 sequenced inbreds (Fig. 3). Inbreds developed by the University of Illinois–Urbana/Champaign program are visibly distinct from the inbreds developed by the University of Wisconsin–Madison Sweet Corn Breeding Program, especially along the principal component two axis. The distinction between the Illinois and Wisconsin inbreds is also reflected in the PCA when labeled by endosperm type, indicating that Wisconsin and Illinois breeding programs have focused on breeding for different endosperm mutants (Fig. 4). Variance explained by the first principal component is equal to 4.5%, and the variance explained by the second principal component equaled 3.3%. Many of the resistant inbreds share similar ancestry as indicated by the pedigree analysis (Table 1).

LINKAGE DISEQUILIBRIUM. Within the WSCDP, we found that the median (50th percentile) of the 12,012 pre-LD-pruned high-quality SNP markers showed LD decay to $R^2 < 0.1$ levels by ≈ 1 kb. However, there were large variances in the structure of LD and LD persisted at higher percentile cutoffs. Ninety percent of the SNPs reached $R^2 < 0.2$ levels by ≈ 250 kb. Given this, candidate genes were included within a ± 250 kb window of the

GWAS detected loci. Previous studies have also found loci as far away as 5 Mb downstream of the 2.7-Mb PAV on chromosome 6 to be in strong LD with the PAV (Gustafson et al., 2018).

ASSOCIATION ANALYSIS. Genome-wide association was conducted using 420 inbreds scored with SNP markers. Through the R package GWA-Spoly, loci of interest using the general model were identified as SNP marker S6_19987285 and S6_36947510 on chromosome 6 at position and 36,947,510 all above the Bonferroni threshold of 5.13 $-\log_{10}(P)$ [$P = 2.5 \times 10^{-8}$ and 1.6×10^{-8} (Fig. 5)]. These loci are downstream of the PAV spanning from 12.9 to 15.4 Mb. No SNPs within the PAV were present after filtering due to the prevalence of the absence variation in the panel. Due to the rate of LD decay within these samples, a 250-kb window surrounding the two loci was used to identify nearby associated genes (Supplemental Fig. 1). Thirty-one associated gene models were found (Supplemental Tables 2 and 3). Due to the nature of the nearby PAV and previous research on downstream loci being linked with the PAV and causal genes within it, these

genes are associated with the significant loci but are not necessarily candidate genes (Gustafson et al., 2018).

A second GWAS was conducted on only the 360 inbreds without the *Scm1* haplotype within the WSCDP for the symptomatic percentage trait. No significant loci were found (Fig. 6).

Discussion

PHENOTYPIC ANALYSIS. We found 46 inbreds (or 8.2% of the panel) that qualified as resistant to SCMV, meaning totally asymptomatic 10 DAI with SCMV across four pot replications. Samples were inoculated at 14 DAP because younger plants are most susceptible to infection through inoculation (Rosenkranz and Scott, 1978). Many of the resistant inbreds had SCMV-resistance inbred parentage, according to their known pedigrees. Resistance to SCMV is an economically useful trait that has been bred into sweet corn inbreds, so it is unsurprising to find inbreds with known SCMV-resistant parents. However, there were several resistant inbreds such as We02412 and BST4 whose backgrounds reveal no known source of resistance. Of the susceptible inbreds, 509 inbreds were $>10\%$ symptomatic,

Fig. 4. Principal component (PC) analysis of 420 sweet corn inbreds within the Wisconsin Sweet Corn Diversity Panel. Inbreds are labeled with their endosperm mutants' categorization. aeduw = amylose-extender; dull: waxy; se = sugary enhancer; sh2 = shrunken2; sh2i = shrunken2 intermediate; su1 = sugary1.

meaning that among all the plants of each inbred, 10% or more of their plants showed symptoms of SCMV infection. Inbreds B73 and C.I.540 were 3% and 7% symptomatic, respectively. Each inbred had no symptomatic seedlings during the first planting. B73 has two symptomatic seedlings out of the 23 plants that germinated during the second planting. Inbred C.I.540 had one symptomatic seedling out of 22 during the second planting. C.I.540 was screened twice, with the other screening fully resistant with 0% symptomatic plants. C.I.540 and B73 were found to have the *Scm1*-IL793a haplotype. The implied partial resistance does not follow with how SCMV acts in most other inbreds, and the one or two symptomatic plants are more likely due to outcrosses or admixtures in the seed source than due to partial susceptibility. Ninety-six percent of control plants were symptomatic, indicating that our infection methods were effective, and inbreds with 0% symptomatic plants were not resistant due to inoculating error. Notably, however, 21% of the control pots had one or more asymptomatic plants. The asymptomatic plants among the controls were likely due to inoculation error, pollen contamination, or admixture. This indicated that some of the inbreds that had between 50% and 90% symptomatic plants might have had less than 100% symptomatic plants due to inoculation error or a contaminated seed source as opposed to partial

susceptibility. Among the 420 inbreds that had associated GBS information, we were able to identify which inbreds had the *Scm1*-IL793a haplotype. The average percent symptomatic plants were significantly different between the group with the *Scm1*-IL793a haplotype and the group without the haplotype, indicating that the presence of the *Scm1*-IL793a haplotype is associated with more resistant inbreds. However, we expected the number of resistant inbreds with *Scm1*-IL793a haplotype to be higher, considering that seven inbreds without the *Scm1*-IL793a haplotype were resistant. One explanation may be that because we did not have GBS data within the PAV of those seven inbreds, they do have the *Scm1* allele, but it is at linkage equilibrium with the *Scm1*-IL793a haplotype.

POPULATION STRUCTURE.

There were clear signs of population structure within the WSCDP. Within the PCA, the inbreds clustered within the different breeding programs of origin with distinction among inbreds created by the University of Illinois–Urbana/Champaign

and the University of Wisconsin–Madison (Fig. 3). As previously mentioned, these distinctions correlate with the endosperm mutant types, indicating that Wisconsin and Illinois breeding programs have focused on breeding for different endosperm mutants resulting in consistent differences in the genetics of their germplasm and visible population structure (Fig. 4). The clustering within the PCA indicates that many of the resistant inbreds are closely related as indicated by the pedigrees (Table 1).

CANDIDATE GENE DISCOVERY. Most of the SNPs used within the population in this analysis exhibited rapid linkage decay at most loci, likely due to the relatively few markers used within this study ($R^2 < 0.1$ levels by ≈ 1 kb). We found variation in the LD decay rates in different regions and chromosomes. Ninety percent of SNPs were at R^2 of about 0.1×1 Mb, or $R^2 < 0.2 \times 250$ kb, which, in addition to the precedent of previous WSCDP studies, led us to select a 250-kb window for finding associated genes (Baseggio et al., 2019). There were several gene models within the 250-kb window surrounding the two associated-with-resistance loci found in the GWAS study (Supplemental Tables 2 and 3). It is possible that these nearby genes could be used as markers in future studies. However, numerous previous studies confirm the importance of the *Scm1* gene in conferring maize SCMV resistance (DuBle et al., 2000, 2003; Xu et al., 2000;

Table 1. The 46 identified resistant sweet corn inbreds, with any known resistant ancestors, notation of whether the inbred has associated genotyping-by-sequencing (GBS) data, and the inbreds' endosperm mutant type.

Resistant inbred	Known resistant ancestor	Associated GBS data	Endosperm type ^z
2		Yes	<i>su1</i>
83610b		Yes	<i>su1</i>
83612b		Yes	<i>su1</i>
A632		Yes	<i>Field</i>
A632 su/su	A632		<i>su1</i>
AS11	Pa405	Yes	<i>su1</i>
BSQ1	B68 × Silver Queen		<i>su1</i>
BST1			<i>su1</i>
BST2			<i>su1</i>
BST4			<i>su1</i>
C.I.540		Yes	<i>Field</i>
IL696a		Yes	<i>se</i>
IL793a	Pa405	Yes	<i>su1</i>
IL794a	Pa405	Yes	<i>su1</i>
IL795a	Pa405	Yes	<i>su1</i>
IL797a		Yes	<i>su1</i>
IL799b	Pa405	Yes	<i>su1</i>
IL800a	Pa405		<i>su1</i>
ILB5870		Yes	<i>su1</i>
ILB6011	Pa405	Yes	<i>su1</i>
Ma83608		Yes	<i>su1</i>
MDM-15	B68	Yes	<i>su1</i>
MDM-16	B68	Yes	<i>su1</i>
MDM-17	B68	Yes	<i>su1</i>
MDM-18	B68	Yes	<i>su1</i>
MDM-20	B68	Yes	<i>su1</i>
MDM3	B68		<i>su1</i>
MDM-5	B68	Yes	<i>su1</i>
MDM-6	B68	Yes	<i>su1</i>
NY MDM-1	B68	Yes	<i>su1</i>
OH7B		Yes	<i>Field</i>
T62s		Yes	<i>su1</i>
We02412		Yes	<i>se</i>
Wh10140R		Yes	<i>sh2</i>
Wh10173V	Mexican Dent	Yes	<i>sh2</i>
Wh10216V	Mexican Dent	Yes	<i>sh2</i>
Wh10231V	Hawaiian SS	Yes	<i>sh2</i>
Wh11005	Wh92047		<i>sh2</i>
Wh12014	Wh92047		<i>sh2</i>
Wh12048V	Wh92047		<i>sh2</i>
Wh13022A			<i>sh2</i>
Wh13063A	Wh92047		<i>sh2</i>
Wh13070	Wh92047		<i>sh2</i>
Wh13074	Wh92047		<i>sh2</i>
Wh92047	A632	Yes	<i>sh2</i>
Wuh07469i			<i>sh2i</i>

^zEndosperm mutants types are *se* = sugary enhancer; *sh2* = shrunken2; *sh2i* = shrunken2 intermediate; *su1* = sugary1.

Yuan et al., 2003). Additionally, the two identified SNP markers S6_19987285 and S6_36947510 are relatively near the PAV surrounding the *Scm1* gene. Given these two factors, we believe that these associated loci are in LD with the *Scm1* gene but not themselves indicative of other causal genes that are contributing

to SCMV resistance. Note that the two significant loci are not within the 250-kb LD window of *Scm1* nor within the PAV that encompasses *Scm1*. Previous studies have found that an associated locus at 19.4 Mb was in LD with the PAV, so we believe this is the case with our locus at 19.9 Mb as well (Gustafson et al., 2018). We did not find any associated loci closer to the *Scm1*, likely due to the lack of SNP coverage within the PAV spanning from 12.9 to 15.4 Mb. There were no SNPs from our GBS included within the 9.5- to 17.0-Mb positions on chromosome 6 as the commonality of the absence variation made that region difficult to genotype. Numerous studies have shown *Scm1* is a dominant allele, vital to SCMV resistance in maize (Dußle et al., 2000; Melchinger et al., 1998; Użarowska et al., 2009; Xia et al., 1999; Xing et al., 2006). Finding that *Scm1* is present within the resistant inbreds in the WSCDP and that it contributes to resistance corroborates those previous studies.

We did not detect SNP markers associated with *Scm2* in any of the GWAS, despite its documented effects (Dußle et al., 2000, 2003; Xu et al., 2000; Yuan et al., 2003). Even when controlling for the presence of *Scm1* by only analyzing those inbreds that do not have the *Scm1*- IL793a haplotype, association between loci near *Scm2* and resistance was still not seen. This is likely because we only analyzed at one time point very early in development and *Scm2* is associated with sustained resistance over maize's development (Dußle et al., 2000; Xing et al., 2006). Alternatively, this may be due to *Scm2* acting as a modifier to *Scm1* and would only be detectable in a panel enriched for the *Scm1* presence (Użarowska et al., 2009).

Because this was the first screening of sweet corn inbreds for SCMV resistance, this study had shortcomings that could be improved with future research. Planting more replicates of each inbred in a completely randomized design could increase the power of the analysis and increase confidence in percentage of symptomatic plants. The lack of SNP coverage within the PAV containing *Scm1* was a study hinderance. Future studies should take special care to ensure SNP coverage of these area for more confidence in their categorization of inbreds having or not having the *Scm1* gene. Additionally, higher SNP coverage overall would increase the likelihood of finding other genomic regions contributing to SCMV resistance. Finally, inoculating the inbreds at multiple time points and enriching the panel for inbreds with the *Scm1* gene could lead to identifying association with *Scm2* and resistance.

Conclusions

The purpose of this disease screening was to identify and study resistance to SCMV within temperate sweet corn. We used the WSCDP as a representative sample of the diversity found within sweet corn populations. We identified 46 inbreds that were fully resistant to SCMV when evaluated 10 DAI. Many of the inbreds we identified are publicly available and could be used by breeders interested in introducing SCMV resistance into their breeding programs. The role of *Scm1* was confirmed within sweet corn, but we did not detect the effects of *Scm2* or of any novel loci associated with resistance. Future studies to investigate *Scm2* contributions to resistance may use the WSCDP to screen the panel at multiple time points to better measure SCMV response over time. Additionally, targeted genotyping of the resistant lines within the panel would provide better discernment between resistant lines with the *Scm1* gene.

Fig. 5. Manhattan plot of susceptibility to *Sugarcane mosaic virus* genome-wide association study of 420 sweet corn inbreds within the Wisconsin Sweet Corn Diversity Panel. Logarithm of odds value with a 5% Bonferroni threshold identified two loci within chromosome 6 in sweet corn.

Fig. 6. Manhattan plot of susceptibility to *Sugarcane mosaic virus* genome-wide association study of 360 sweet corn inbreds within the Wisconsin Sweet Corn Diversity Panel that do not have the *Scm1- IL793a* haplotype. Logarithm of odds value with a 5% Bonferroni threshold identified no significant loci.

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Supplemental Fig. 1. Linkage disequilibrium (LD) estimates among the 420 sequenced sweet corn inbreds. Mean decay of LD measured as pairwise R^2 from the 12,012 high-quality single-nucleotide polymorphism (SNP) markers over physical distance. Different percentile cutoffs are indicated of the distribution of SNP markers among all chromosomes.

Supplemental Table 1. Phenotypic data for all sweet corn inbreds within screening, with information about planting order (pot label), ratio of sugarcane mosaic virus symptomatic plants, whether the line was sequence (genetic info), and had the haplotype associated with resistance within its sequence (*Scm1*- IL793a haplotype), the inbreds' endosperm mutant type, and the inbreds' program of origin.

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> -IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
350	2	0	TRUE	TRUE	su1	
158	21-396-68(B)	88	TRUE	FALSE	su1	IL
293	257A9	75	TRUE	FALSE	su1	
27	34f	100	TRUE	FALSE	su1	
192	471-U6	100	TRUE	FALSE	su1	USDA
550	81-1	100	TRUE	FALSE	su1	USDA
30	83610b	0	TRUE	TRUE	su1	
196	83612b	0	TRUE	FALSE	su1	
295	A10579	100	TRUE	FALSE	su1	
190	A15	25	TRUE	FALSE	su1	MN
534	A632	0	TRUE	TRUE	field	MN
269	A632 su/su	0	FALSE	NA	su1	MN
153	A648	100	TRUE	FALSE	su1	MN
369	A684su	75	TRUE	FALSE	su1	MN
231	A685su	75	TRUE	FALSE	su1	MN
61	A686su	100	TRUE	FALSE	su1	MN
228	A7	100	TRUE	FALSE	su1	MN
141	AS11	0	TRUE	FALSE	su1	MN
114	B5765 Se	100	TRUE	FALSE	NA	IL
191	B5870	63	TRUE	FALSE	su1	IL
202	B73	7	TRUE	TRUE	field	IA
4	BSQ1	0	FALSE	NA	NA	
6	BST1	0	FALSE	NA	NA	
234	BST2	0	FALSE	NA	NA	
72	BST4	0	FALSE	NA	NA	
185	C12-1	75	TRUE	FALSE	su1	CT
204	C13	100	TRUE	TRUE	su1	CT
254	C13et-123:68	57	TRUE	TRUE	su1	CT
246	C15	75	TRUE	FALSE	su1	CT
76	C1540	0	TRUE	FALSE	field	CT
372	C18	75	TRUE	FALSE	su1	CT
323	C2	75	TRUE	FALSE	su1	CT
294	C22	67	TRUE	FALSE	su1	CT
145	C23	100	TRUE	FALSE	su1	CT
35	C27	75	TRUE	FALSE	su1	CT
132	C27	100	TRUE	FALSE	su1	CT
74	C27red	100	TRUE	FALSE	su1	CT
283	C3	57	TRUE	FALSE	su1	CT
49	C30	100	TRUE	FALSE	su1	CT
314	C31	63	TRUE	FALSE	su1	CT
24	C38	100	TRUE	FALSE	su1	CT
118	C4	88	TRUE	FALSE	su1	CT
64	C40	100	TRUE	FALSE	su1	CT
88	C42	100	TRUE	FALSE	su1	CT
165	C5	60	TRUE	FALSE	su1	CT
229	C5 Ark 1956	38	TRUE	FALSE	su1	CT
94	C5 Cold Hardy Comp	100	FALSE	NA	NA	CT
264	C5-NT	60	TRUE	FALSE	su1	CT
95	C65	100	TRUE	FALSE	su1	CT
87	C68	63	TRUE	FALSE	su1	CT
104	C68 su	50	TRUE	FALSE	su1	CT
80	C68R(x)-552:63	100	TRUE	FALSE	su1	CT
216	C6A	88	TRUE	FALSE	su1	CT
257	C7	100	TRUE	FALSE	su1	CT

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Supplemental Table 1. (Continued)

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> - IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
12	C7e	86	TRUE	FALSE	su1	CT
248	C8 white	43	TRUE	FALSE	su1	CT
353	C81	88	TRUE	FALSE	su1	CT
331	C8pseudo	71	TRUE	FALSE	su1	CT
322	C90	38	TRUE	FALSE	su1	CT
198	CI540	33	TRUE	TRUE	NA	
127	CTS6-1	100	TRUE	FALSE	su1	
116	CTS6-2	63	FALSE	NA	NA	
209	DF14 Su	67	TRUE	TRUE	field	
18	DSU 146	100	FALSE	NA	NA	
205	EP58	100	TRUE	FALSE	su1	SP
250	EP60	86	TRUE	FALSE	su1	SP
227	EP61	57	TRUE	FALSE	su1	SP
128	EP62	100	TRUE	FALSE	su1	SP
194	EP82	100	TRUE	FALSE	su1	SP
313	EP83	100	TRUE	FALSE	su1	SP
58	EP84	100	TRUE	FALSE	sh2	SP
335	EP85	43	TRUE	FALSE	sh2	SP
147	F2	100	FALSE	NA	field	FR
212	F7	100	FALSE	NA	field	FR
517	Fa32B	75	TRUE	FALSE	sh2i	FL
71	Fa56A	100	TRUE	FALSE	sh2i	FL
363	G8	86	TRUE	FALSE	su1	IN
123	GMBAB	100	TRUE	FALSE	su1	
45	GTS1	100	TRUE	FALSE	su1	GA
176	HotevillaAzwhite sweetcorn_1	50	TRUE	FALSE	su1	
144	I45	86	TRUE	FALSE	su1	
281	I817	60	TRUE	FALSE	su1	
33	Ia2003	86	TRUE	FALSE	su1	IA
329	Ia2015 (yar) (A)	50	TRUE	FALSE	su1	IA
69	Ia2076	63	TRUE	FALSE	su1	IA
60	Ia2132	100	TRUE	FALSE	su1	IA
288	Ia2256	33	TRUE	FALSE	su1	IA
91	Ia2256B	56	FALSE	NA	su1	IA
272	Ia2730	20	TRUE	FALSE	su1	IA
206	Ia4189	52	TRUE	FALSE	su1	IA
321	Ia45-1	43	TRUE	FALSE	su1	IA
276	Ia45-2	43	TRUE	FALSE	su1	IA
84	Ia453	63	TRUE	FALSE	su1	IA
274	Ia45-3	14	TRUE	FALSE	su1	IA
8	Ia5125 (non-fire)	88	FALSE	NA	su1	IA
236	Ia5125 su	67	TRUE	FALSE	su1	IA
220	Ia5145	100	TRUE	FALSE	NA	IA
43	Ia5145	NA	TRUE	FALSE	NA	IA
287	Ia5220	71	TRUE	FALSE	su1	IA
73	Ia5261	63	TRUE	FALSE	su1	IA
237	Ia59	50	TRUE	FALSE	su1	IA
126	IaCG4189	100	TRUE	FALSE	su1	IA
261	IaEV3002	57	TRUE	FALSE	su1	IA
325	IaEV3004	40	TRUE	FALSE	su1	IA
222	IaEV3015	25	TRUE	FALSE	su1	IA
347	If825	67	TRUE	FALSE	sh2	
267	If853	40	TRUE	FALSE	sh2	
85	IL101-dgB-2727:62	75	FALSE	NA	NA	IL
22	IL101Q	63	FALSE	NA	NA	IL

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Supplemental Table 1. (Continued)

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> - IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
226	IL101T	38	TRUE	FALSE	su1	IL
362	IL101t su	100	FALSE	NA	NA	IL
86	IL103a	88	TRUE	FALSE	su1	IL
52	IL104g	100	TRUE	FALSE	su1	IL
345	IL107a	67	FALSE	NA	su1	IL
249	IL110K	100	TRUE	FALSE	su1	IL
41	IL112t	100	TRUE	FALSE	su1	IL
357	IL1171	100	TRUE	FALSE	su1	IL
83	IL11D	100	TRUE	FALSE	su1	IL
44	IL124a	100	TRUE	FALSE	su1	IL
235	IL125a	57	FALSE	NA	NA	IL
150	IL125b	100	TRUE	FALSE	su1	IL
277	IL134	50	FALSE	NA	su1	IL
316	IL13B	50	TRUE	FALSE	su1	IL
368	IL14b	71	TRUE	FALSE	su1	IL
359	IL14C	17	TRUE	FALSE	su1	IL
101	IL14H	100	TRUE	FALSE	su1	IL
300	IL14H-1014:74	33	TRUE	FALSE	su1	IL
161	IL14H-588-68(B)	100	TRUE	FALSE	su1	IL
93	IL18b	100	TRUE	FALSE	se	IL
193	IL18c	80	FALSE	NA	NA	IL
266	IL200e	43	TRUE	FALSE	su1	IL
334	IL21f	67	TRUE	FALSE	su1	IL
36	IL279AxRp1d	100	TRUE	FALSE	su1	IL
135	IL27a	100	TRUE	FALSE	su1	IL
99	IL27A-317-68A©	100	TRUE	FALSE	su1	IL
377	IL303b	86	TRUE	FALSE	su1	IL
360	IL318a	75	TRUE	FALSE	su1	IL
119	IL31a	88	TRUE	FALSE	su1	IL
292	IL323a	86	TRUE	FALSE	su1	IL
243	IL325a	17	TRUE	FALSE	su1	IL
164	IL328a	100	TRUE	FALSE	su1	IL
290	IL354a	67	TRUE	FALSE	su1	IL
251	IL366a	67	TRUE	FALSE	su1	IL
173	IL368a	75	TRUE	FALSE	su1	IL
317	IL369b	43	TRUE	FALSE	su1	IL
55	IL370a	100	TRUE	FALSE	su1	IL
201	IL370a (su)	63	TRUE	FALSE	su1	IL
54	IL370b	100	FALSE	NA	NA	IL
155	IL393a	75	TRUE	FALSE	su1	IL
56	IL395a	75	TRUE	FALSE	su1	IL
210	IL430a	100	TRUE	FALSE	su1	IL
188	IL437a	100	TRUE	FALSE	su1	IL
203	IL442a	100	TRUE	FALSE	su1	IL
370	IL442A-431-68(D)	75	TRUE	FALSE	su1	IL
351	IL44b	100	TRUE	FALSE	se	IL
344	IL451-18 Se Se	71	FALSE	NA	NA	IL
48	IL451b	100	TRUE	FALSE	su1	IL
102	IL453b	71	TRUE	FALSE	su1	IL
343	IL454a	86	TRUE	FALSE	su1	IL
121	IL465a	88	TRUE	TRUE	su1	IL
152	IL47A	100	TRUE	FALSE	su1	IL
308	IL515a	43	TRUE	FALSE	su1	IL
311	IL543c	67	TRUE	FALSE	su1	IL
137	IL545	100	TRUE	FALSE	su1	IL
326	IL557a	63	TRUE	FALSE	su1	IL

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Supplemental Table 1. (Continued)

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> - IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
28	IL607a	63	TRUE	TRUE	su1	IL
3	IL611a	100	TRUE	FALSE	su1	IL
20	IL615a	83	TRUE	FALSE	su1	IL
358	IL618b	60	TRUE	FALSE	su1	IL
315	IL648	57	TRUE	FALSE	su1	IL
260	IL648a	67	TRUE	FALSE	su1	IL
239	IL661a	100	TRUE	FALSE	su1	IL
189	IL671a	100	TRUE	FALSE	su1	IL
110	IL673a	100	TRUE	FALSE	su1	IL
26	IL676a	86	TRUE	FALSE	su1	IL
259	IL677a	57	TRUE	TRUE	se	IL
103	IL678a	100	TRUE	FALSE	su1	IL
70	IL678a-se	86	FALSE	NA	NA	IL
275	IL689a	13	TRUE	FALSE	su1	IL
282	IL692c	75	TRUE	TRUE	su1	IL
256	IL696a	0	TRUE	TRUE	se	IL
303	IL699e	63	TRUE	FALSE	su1	IL
89	IL699f	88	FALSE	NA	NA	IL
166	IL7	86	TRUE	TRUE	su1	IL
270	IL701e	50	TRUE	FALSE	su1	IL
245	IL707a	100	TRUE	FALSE	su1	IL
77	IL710a	88	FALSE	NA	NA	IL
113	IL711a	100	FALSE	NA	NA	IL
46	IL713a	50	TRUE	FALSE	su1	IL
180	IL729a	86	TRUE	FALSE	su1	IL
199	IL731	100	TRUE	FALSE	se	IL
291	IL731a (se)	86	TRUE	FALSE	se	IL
258	IL733a	67	TRUE	FALSE	su1	IL
115	IL744a	100	TRUE	TRUE	su1	IL
304	IL747b (se)	33	TRUE	TRUE	se	IL
149	IL751a	100	TRUE	FALSE	su1	IL
151	IL757b	75	TRUE	TRUE	su1	IL
262	IL761a	67	TRUE	TRUE	su1	IL
154	IL761c	71	FALSE	NA	NA	IL
302	IL764b	100	TRUE	TRUE	su1	IL
181	IL765a	88	TRUE	FALSE	su1	IL
241	IL766a (se)	67	TRUE	TRUE	se	IL
162	IL767a	100	FALSE	NA	NA	IL
208	IL767b	88	TRUE	TRUE	su1	IL
301	IL768a (se)	63	FALSE	NA	NA	IL
305	IL769a	100	FALSE	NA	NA	IL
255	IL772a	100	TRUE	TRUE	su1	IL
330	IL772b	57	FALSE	NA	NA	IL
352	IL773a	100	TRUE	FALSE	su1	IL
356	IL774a	75	FALSE	NA	NA	IL
200	IL774b su	86	FALSE	NA	NA	IL
211	IL774d	100	FALSE	NA	NA	IL
336	IL775a	83	TRUE	TRUE	su1	IL
59	IL776a	100	TRUE	FALSE	su1	IL
156	IL777a	100	TRUE	FALSE	su1	IL
75	IL778c	100	TRUE	TRUE	su1	IL
13	IL779a	100	TRUE	TRUE	su1	IL
163	IL781a	88	FALSE	NA	NA	IL
307	IL788a	50	TRUE	FALSE	su1	IL
319	IL789b	63	TRUE	FALSE	su1	IL
238	IL791a	63	TRUE	FALSE	su1	IL

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Supplemental Table 1. (Continued)

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> - IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
373	IL793a	0	TRUE	TRUE	su1	IL
125	IL794a	0	TRUE	TRUE	su1	IL
263	IL795a	0	TRUE	TRUE	su1	IL
371	IL796a	57	TRUE	TRUE	su1	IL
364	IL797a	0	TRUE	TRUE	su1	IL
178	IL798c	88	TRUE	TRUE	su1	IL
214	IL799b	0	TRUE	TRUE	su1	IL
159	IL800a	0	FALSE	NA	su1	IL
324	IL801a	50	TRUE	TRUE	su1	IL
299	IL802b Ht2	100	TRUE	TRUE	su1	IL
124	IL803a	86	TRUE	FALSE	su1	IL
39	IL804a	100	TRUE	TRUE	su1	IL
11	IL86e	88	TRUE	FALSE	su1	IL
37	IL91j	75	TRUE	FALSE	su1	IL
139	ILB5765 se	100	TRUE	FALSE	se	IL
268	ILB5870	0	TRUE	FALSE	su1	IL
273	ILB6011	0	TRUE	TRUE	su1	IL
230	Ind.5817	75	FALSE	NA	su1	
285	IP39	86	TRUE	FALSE	su1	IA
341	Jubileex9017	100	TRUE	FALSE	su1	
17	LUTHER HILL	100	TRUE	FALSE	su1	WI
366	M23	50	TRUE	FALSE	su1	
15	M45	100	TRUE	TRUE	su1	
65	M5	100	TRUE	FALSE	su1	
528	M6388	100	TRUE	FALSE	aeduwX	USDA
523	M6411	88	TRUE	TRUE	aeduwX	USDA
524	M6415	100	TRUE	TRUE	aeduwX	USDA
529	M6421	71	TRUE	TRUE	aeduwX	USDA
224	M822	100	TRUE	FALSE	su1	
106	Ma2402	100	TRUE	FALSE	su1	MA
339	Ma2410-1191	80	TRUE	FALSE	NA	MA
225	Ma32	100	TRUE	FALSE	su1	MA
160	Ma401	100	TRUE	FALSE	su1	MA
172	Ma5125	100	TRUE	FALSE	su1	MA
51	Ma609	100	TRUE	TRUE	su1	MA
148	Ma83608	0	TRUE	FALSE	su1	MA
186	MDM-1	100	TRUE	TRUE	su1	
219	MDM-12	57	TRUE	FALSE	su1	
279	MDM-15	0	TRUE	TRUE	su1	
247	MDM-16	0	TRUE	FALSE	su1	
29	MDM-17	0	TRUE	TRUE	su1	
14	MDM-18	0	TRUE	FALSE	su1	
365	MDM-19	100	TRUE	FALSE	su1	
53	MDM-2	100	TRUE	FALSE	su1	
96	MDM-20	0	TRUE	TRUE	su1	
82	MDM-21	71	TRUE	FALSE	su1	
146	MDM-23	100	TRUE	FALSE	su1	
108	MDM3	0	FALSE	NA	NA	
130	MDM-5	0	TRUE	TRUE	su1	
253	MDM-6	0	TRUE	TRUE	su1	
42	MDM-8	88	TRUE	FALSE	su1	
348	MDM-9	33	TRUE	TRUE	su1	
244	Me1	29	TRUE	FALSE	su1	ME
171	Me1	88	TRUE	FALSE	su1	ME
38	Me100	100	TRUE	FALSE	su1	ME
375	Me121-wb	100	TRUE	FALSE	su1	ME

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Supplemental Table 1. (Continued)

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> -IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
296	Me123	100	TRUE	FALSE	su1	ME
197	Me135	88	TRUE	FALSE	su1	ME
252	Me2	100	TRUE	FALSE	su1	ME
367	Me244Wa	83	TRUE	FALSE	NA	ME
143	Me244-wb	100	TRUE	FALSE	su1	ME
7	Me2rt	86	TRUE	FALSE	su1	ME
133	Mo17	75	TRUE	FALSE	field	MO
280	MR10 (Cristalino Norteno) S6	50	TRUE	FALSE	sh2i	WI
221	MR14	100	TRUE	FALSE	su1	WI
310	ND1	88	TRUE	FALSE	su1	ND
32	NJ112	86	TRUE	FALSE	su1	NJ
195	NJ116Wb	50	TRUE	FALSE	NA	NJ
297	NJ143	0	FALSE	NA	NA	NJ
31	NJ159	50	TRUE	FALSE	su1	NJ
47	NJ618	88	TRUE	FALSE	su1	NJ
271	NJ7Y	25	TRUE	FALSE	su1	NJ
327	NY MDM-1	0	TRUE	TRUE	su1	NY
286	OH43	32	TRUE	FALSE	field	OH
318	OH7B	0	TRUE	FALSE	field	OH
67	OLCOTT	57	TRUE	FALSE	su1	
170	OLCOTT-1067:72	88	TRUE	FALSE	NA	
140	OLCOTT-1069:72	100	TRUE	FALSE	NA	
232	P14	43	TRUE	FALSE	su1	IN
179	P35	86	TRUE	FALSE	su1	IN
349	P39	86	TRUE	FALSE	su1	IN
19	P39	100	TRUE	FALSE	su1	IN
217	P39	100	TRUE	FALSE	su1	IN
289	P39 Goodman-Buckler	86	TRUE	FALSE	su1	IN
81	P39 su	100	TRUE	FALSE	su1	IN
320	P39(New)	67	TRUE	FALSE	su1	IN
57	P39-10-226:61	100	FALSE	NA	NA	IN
332	P39-16-275:61	75	FALSE	NA	NA	IN
62	P39-16-276:61	88	FALSE	NA	NA	IN
68	P39A	83	TRUE	FALSE	su1	IN
129	P39a	100	TRUE	FALSE	su1	IN
355	P39b	100	TRUE	FALSE	su1	IN
338	P39Le-253	100	TRUE	FALSE	su1	IN
265	P39LE-261:61	38	TRUE	FALSE	su1	IN
10	P39-LE-3438:64	88	FALSE	NA	NA	IN
79	P39LE-3502:61	100	TRUE	TRUE	NA	IN
131	P39M94	100	TRUE	FALSE	su1	IN
169	P39M96	100	TRUE	FALSE	su1	IN
187	P39xRp1d	100	TRUE	FALSE	su1	IN
168	P51	100	TRUE	FALSE	su1	IN
142	P51-3289:61	100	TRUE	FALSE	su1	IN
120	P51B	100	TRUE	FALSE	su1	IN
100	P51xRp1d	100	TRUE	FALSE	su1	IN
157	R825	100	TRUE	FALSE	sh2	IL
105	R839	100	TRUE	FALSE	sh2	IL
342	R851	100	TRUE	FALSE	sh2	IL
97	R853 sh2	100	TRUE	FALSE	sh2	IL
50	S4	100	TRUE	FALSE	su1	MN
223	S43	100	TRUE	FALSE	su1	MN
117	SC1441	100	TRUE	FALSE	su1	SC
340	SC6069	100	TRUE	FALSE	su1	SC

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Supplemental Table 1. (Continued)

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> - IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
298	SD176	100	TRUE	FALSE	su1	SD
374	SD226	100	TRUE	FALSE	su1	SD
333	SD245	71	TRUE	FALSE	su1	SD
136	SD277	88	TRUE	FALSE	su1	SD
2	SD42	88	TRUE	FALSE	su1	SD
215	SD469	100	TRUE	FALSE	su1	SD
98	SD592	75	TRUE	FALSE	su1	SD
182	SD789	100	TRUE	TRUE	su1	SD
1	SD829	100	TRUE	FALSE	su1	SD
376	SD883	43	TRUE	FALSE	su1	SD
78	SD884	75	TRUE	FALSE	su1	SD
240	SD908	100	TRUE	FALSE	su1	SD
213	SD909	88	TRUE	FALSE	su1	SD
122	sm*sm*smooth@@	100	FALSE	NA	su1	
233	sm*sm*wrinkled @@	100	FALSE	NA	su1	
34	STRAIN 11D-586-68A	100	TRUE	FALSE	su1	IL
174	Strain 18B-5-64	88	TRUE	FALSE	su1	IL
25	STRAIN 304A-408-68B	100	TRUE	FALSE	su1	IL
309	STRAIN 451B-4-68B	75	TRUE	FALSE	su1	IL
328	STRAIN 465A-421-68B	34	TRUE	TRUE	su1	IL
92	Strain 675A-415-68(B)	100	TRUE	FALSE	su1	IL
5	STRAIN T24-395-68B	88	TRUE	FALSE	su1	IL
361	Strain T32-397-68r	75	TRUE	FALSE	su1	IL
184	STRAIN T33-399-68B	100	TRUE	FALSE	su1	IL
183	STRAIN T35-388-68A	88	TRUE	FALSE	su1	IL
306	T20-2-68(B)	29	TRUE	FALSE	su1	IL
16	T625	0	TRUE	TRUE	su1	TN
111	T62s	0	TRUE	FALSE	su1	TN
346	V775	100	TRUE	FALSE	su1	
90	V781	86	TRUE	FALSE	su1	
354	VIA5492	75	TRUE	FALSE	su1	
242	Vineland576	100	TRUE	TRUE	su1	
207	W 6786	71	TRUE	FALSE	su1	WI
63	W1736	100	TRUE	FALSE	su1	WI
9	W3647	88	TRUE	FALSE	su1	WI
107	W3722	88	TRUE	FALSE	su1	WI
337	W3742	100	TRUE	FALSE	su1	WI
109	W5543	100	TRUE	FALSE	su1	WI
175	W5552	100	TRUE	FALSE	su1	WI
23	W5579	88	TRUE	FALSE	su1	WI
218	W6366	100	TRUE	FALSE	su1	WI
112	W6462	100	TRUE	FALSE	su1	WI
66	W64A	38	TRUE	TRUE	field	WI
312	W6714	57	TRUE	FALSE	su1	WI
284	W6720-1	88	TRUE	FALSE	su1	WI
21	W6720-2	NA	FALSE	NA	NA	WI
134	W6728	100	TRUE	FALSE	su1	WI
177	W6757	88	TRUE	FALSE	su1	WI
138	W6786	100	TRUE	FALSE	su1	WI
167	W6786	100	TRUE	FALSE	su1	WI
40	W7151	63	TRUE	FALSE	su1	WI
451	W822GSe	63	TRUE	FALSE	se	WI
278	W85	75	TRUE	FALSE	field	WI
464	We01407	100	TRUE	FALSE	se	WI
467	We02404	100	TRUE	FALSE	se	WI
462	We02412	0	TRUE	FALSE	se	WI

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Supplemental Table 1. (Continued)

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> - IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
425	We03418a	83	TRUE	FALSE	se	WI
417	We04401	86	FALSE	NA	se	WI
463	We04806	100	FALSE	NA	se	WI
542	We05401	88	TRUE	FALSE	se	WI
563	We05408	100	TRUE	FALSE	se	WI
411	We05410	75	TRUE	FALSE	se	WI
546	We05411	83	TRUE	FALSE	se	WI
540	We06409b	100	TRUE	FALSE	se	WI
559	we07403a	100	FALSE	NA	se	WI
468	We08405	63	FALSE	NA	se	WI
388	We08408	71	TRUE	FALSE	se	WI
421	We08414a	67	TRUE	FALSE	se	WI
466	We08424	57	FALSE	NA	se	WI
483	We09402	75	TRUE	FALSE	se	WI
416	We09413	100	TRUE	FALSE	se	WI
410	We09425	100	TRUE	FALSE	se	WI
482	We10401	48	TRUE	FALSE	se	WI
519	We11401	75	FALSE	NA	se	WI
556	We11404	63	FALSE	NA	se	WI
450	We11405	83	FALSE	NA	NA	WI
543	we11416	88	FALSE	NA	se	WI
557	we12401	100	FALSE	NA	se	WI
446	Wh00033	86	TRUE	FALSE	sh2	WI
505	Wh00050	75	TRUE	FALSE	sh2	WI
495	Wh00051	100	TRUE	FALSE	sh2	WI
561	Wh00065	100	TRUE	FALSE	sh2	WI
497	Wh01001	63	TRUE	FALSE	sh2	WI
383	Wh03031	86	TRUE	FALSE	sh2	WI
480	Wh04020a	50	TRUE	FALSE	sh2	WI
552	Wh05032a	100	TRUE	FALSE	sh2	WI
384	Wh05051	100	TRUE	FALSE	sh2	WI
481	Wh05066a	60	TRUE	FALSE	sh2	WI
493	Wh05070a	50	FALSE	NA	sh2	WI
449	Wh05081b	86	TRUE	FALSE	sh2	WI
551	Wh05081b	100	TRUE	FALSE	sh2	WI
395	Wh07013	NA	TRUE	FALSE	sh2	WI
444	Wh07020a	67	TRUE	FALSE	sh2	WI
440	Wh07029	67	FALSE	NA	sh2	WI
390	Wh07051	NA	TRUE	FALSE	sh2	WI
433	Wh07061a	75	TRUE	FALSE	sh2	WI
456	Wh07061b	75	FALSE	NA	sh2	WI
429	Wh07165R	100	TRUE	FALSE	sh2	WI
432	Wh07166R	80	TRUE	FALSE	sh2	WI
424	Wh08022	88	TRUE	FALSE	sh2	WI
502	Wh08039	100	FALSE	NA	sh2	WI
431	Wh08042	86	TRUE	FALSE	sh2	WI
492	Wh08045	88	FALSE	NA	sh2	WI
428	Wh08092	67	TRUE	FALSE	sh2	WI
562	Wh08114	75	FALSE	NA	sh2	WI
499	Wh08114	88	FALSE	NA	sh2	WI
544	Wh08115b	86	TRUE	FALSE	sh2	WI
381	Wh09009	67	TRUE	FALSE	sh2	WI
409	Wh09033	100	TRUE	FALSE	sh2	WI
426	Wh09061	60	TRUE	FALSE	sh2	WI
427	Wh09068	80	TRUE	FALSE	sh2	WI
454	Wh09075	100	TRUE	FALSE	sh2	WI

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Supplemental Table 1. (Continued)

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> - IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
445	Wh09083	63	TRUE	FALSE	sh2	WI
531	Wh09086	100	FALSE	NA	sh2	WI
541	Wh09087	100	TRUE	FALSE	sh2	WI
420	Wh09117R	100	TRUE	FALSE	sh2	WI
379	Wh09126R	63	TRUE	FALSE	sh2	WI
408	Wh09137R	57	TRUE	FALSE	sh2	WI
412	Wh09141R	75	TRUE	FALSE	sh2	WI
477	Wh10006R	50	TRUE	TRUE	sh2	WI
430	Wh10034R	100	TRUE	FALSE	sh2	WI
484	Wh10035R	68	TRUE	FALSE	sh2	WI
485	Wh10040R	71	FALSE	NA	sh2	WI
393	Wh10046R	63	TRUE	FALSE	sh2	WI
500	Wh10049R	88	TRUE	FALSE	sh2	WI
496	Wh10052R	100	FALSE	NA	sh2	WI
491	Wh10057R	100	FALSE	NA	sh2	WI
400	Wh10062R	83	TRUE	FALSE	sh2	WI
472	Wh10067R	83	FALSE	NA	sh2	WI
512	Wh10067R	100	FALSE	NA	sh2	WI
514	Wh10068R	100	FALSE	NA	sh2	WI
479	Wh10070R	100	FALSE	NA	sh2	WI
435	Wh10080	50	TRUE	FALSE	sh2	WI
536	Wh10085	100	FALSE	NA	NA	WI
537	Wh10088R	86	FALSE	NA	sh2	WI
452	Wh10090R	100	FALSE	NA	sh2	WI
441	Wh10105R	83	FALSE	NA	sh2	WI
415	Wh10110R	100	TRUE	TRUE	sh2	WI
406	Wh10113R	50	TRUE	FALSE	sh2	WI
532	wh10117R	100	FALSE	NA	sh2	WI
506	Wh10127R	50	FALSE	NA	sh2	WI
507	Wh10135R	80	FALSE	NA	sh2	WI
465	Wh10137R	63	FALSE	NA	sh2	WI
436	Wh10140R	0	TRUE	FALSE	sh2	WI
476	Wh10163	23	TRUE	TRUE	sh2	WI
439	Wh10173V	0	TRUE	TRUE	sh2	WI
498	Wh10216V	0	TRUE	TRUE	sh2	WI
407	Wh10231V	0	TRUE	TRUE	sh2	WI
549	Wh10804R	88	TRUE	TRUE	sh2	WI
520	Wh11001	57	FALSE	NA	sh2	WI
402	Wh11005	0	FALSE	NA	sh2	WI
535	Wh11008	100	FALSE	NA	sh2	WI
489	Wh12001	67	FALSE	NA	sh2	WI
469	Wh12004	71	FALSE	NA	sh2	WI
389	Wh12005	100	FALSE	NA	sh2	WI
503	Wh12008	50	FALSE	NA	sh2	WI
458	Wh12014	0	FALSE	NA	sh2	WI
490	Wh12048V	0	FALSE	NA	sh2	WI
396	Wh13022A	0	FALSE	NA	sh2	WI
385	Wh13037	86	FALSE	NA	sh2	WI
478	Wh13040	50	FALSE	NA	sh2	WI
399	Wh13041	86	FALSE	NA	sh2	WI
382	Wh13048	67	FALSE	NA	sh2	WI
447	Wh13053	57	FALSE	NA	sh2	WI
405	Wh13063A	0	FALSE	NA	sh2	WI
459	Wh13064	21	FALSE	NA	sh2	WI
403	Wh13070	0	FALSE	NA	sh2	WI
392	Wh13074	0	FALSE	NA	sh2	WI

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Supplemental Table 1. (Continued)

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> - IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
494	Wh14100V	100	FALSE	NA	sh2	WI
461	Wh92047	0	TRUE	TRUE	sh2	WI
457	Wh9261	67	TRUE	FALSE	sh2	WI
554	Wh93006	100	TRUE	FALSE	sh2	WI
501	Wh93016	100	TRUE	FALSE	sh2	WI
545	Wh93017	100	TRUE	FALSE	sh2	WI
553	Wh94053	100	TRUE	FALSE	sh2	WI
460	Wh95016a	100	TRUE	FALSE	sh2	WI
548	Wh96015	100	FALSE	NA	sh2	WI
471	Wh96034	100	TRUE	FALSE	sh2	WI
470	Wh98044	83	TRUE	FALSE	sh2	WI
555	Wh98044	100	TRUE	FALSE	sh2	WI
418	Wh98063	86	TRUE	FALSE	sh2	WI
378	Wu00801	80	TRUE	FALSE	su1	WI
558	Wu00802	100	TRUE	FALSE	su1	WI
518	Wu0184R	57	FALSE	NA	NA	WI
487	Wu04819	60	TRUE	FALSE	su1	WI
508	Wu10802R	75	TRUE	FALSE	su1	WI
509	Wu99811	86	TRUE	FALSE	su1	WI
510	Wu99823	71	TRUE	FALSE	su1	WI
386	Wuh07177i	63	TRUE	FALSE	sh2i	WI
560	wuh07182i	100	TRUE	FALSE	sh2i	WI
547	Wuh07183i	100	TRUE	FALSE	sh2i	WI
438	Wuh07186i	100	TRUE	FALSE	sh2i	WI
533	Wuh07413	60	TRUE	FALSE	sh2i	WI
423	Wuh07422i	67	TRUE	FALSE	sh2i	WI
521	Wuh07428i	33	FALSE	NA	sh2i	WI
448	Wuh07429i	80	TRUE	FALSE	sh2i	WI
455	Wuh07466i	50	TRUE	FALSE	sh2	WI
486	Wuh07469i	0	TRUE	FALSE	sh2i	WI
443	Wuh07481i	100	TRUE	FALSE	sh2i	WI
522	Wuh07492i	50	FALSE	NA	sh2i	WI
404	Wuh08127ia	75	TRUE	FALSE	sh2i	WI
474	Wuh09093i	50	TRUE	FALSE	sh2i	WI
387	Wuh09097i	60	TRUE	FALSE	sh2i	WI
422	Wuh09098i	75	TRUE	FALSE	sh2i	WI
434	Wuh09144i	86	TRUE	FALSE	sh2i	WI
413	Wuh09149i	100	TRUE	FALSE	sh2i	WI
414	Wuh09150i	75	TRUE	FALSE	sh2i	WI
475	Wuh09153i	33	TRUE	FALSE	sh2i	WI
380	Wuh09164i	86	TRUE	FALSE	sh2i	WI
391	Wuh09170i	60	TRUE	FALSE	sh2i	WI
473	Wuh09174i	75	TRUE	FALSE	sh2i	WI
394	Wuh09183i	100	TRUE	FALSE	sh2i	WI
437	Wuh09188i	67	TRUE	FALSE	sh2i	WI
419	Wuh09191i	100	TRUE	FALSE	sh2i	WI
530	Wuh11801i	75	FALSE	NA	sh2i	WI
539	Wuh11805i	100	FALSE	NA	sh2i	WI
527	Wuh11809i	67	FALSE	NA	sh2i	WI
516	Wuh11810i	100	FALSE	NA	sh2i	WI
526	Wuh11811i	88	FALSE	NA	sh2i	WI
538	Wuh11812i	88	FALSE	NA	sh2i	WI
515	Wuh11813i	100	FALSE	NA	sh2i	WI
453	Wuh11816i	100	FALSE	NA	sh2i	WI
488	Wuh12018i	100	FALSE	NA	sh2i	WI
397	Wuh12019i	100	FALSE	NA	sh2i	WI

(Continued on next page)

Supplemental Table 1. (Continued)

Pot label	Inbred line	Plants symptomatic (%) ^z	Genetic info	<i>Scm1</i> -IL793a haplotype ^y	Endosperm type ^x	Program of origin ^w
511	Wuh12020i	100	FALSE	NA	sh2i	WI
513	Wuh12027i	100	FALSE	NA	sh2i	WI
401	Wuh12035i	83	FALSE	NA	sh2i	WI
504	Wuh12037i	100	FALSE	NA	sh2i	WI
398	Wuh12039i	67	FALSE	NA	sh2i	WI
442	Wuh98032	86	TRUE	FALSE	sh2	WI
525	WVG3	100	TRUE	FALSE	su1	WI

^zPlants symptomatic percentage was determined by taking the number of plants symptomatic for sugarcane mosaic virus and dividing by the total number of plants per pot from all replicates

^y*Scm1*- IL793a haplotype was determined for genotyped inbreds by comparing their haplotype near the *Scm1* locus to IL793a, an inbred with known to have the *Scm1*.

^xEndosperm mutants types are aeduw x = amylose-extender: dull: waxy; se = sugary enhancer; sh2 = shrunken2; sh2i = shrunken2 intermediate; su1 = sugary1.

^wCT = University of Connecticut–Storrs, CT; FL = University of Florida–Gainesville, FL; FR = National Institute of Agricultural Research (Paris, France); GA = University of Georgia–Athens; IA = Iowa State University–Ames, IA; IL = University of Illinois–Champaign; IN = Purdue University (West Lafayette, IN); MA = University of Massachusetts–Amherst; ME = University of Maine–Orono, ME; MN = University of Minnesota (Minneapolis, MN); MO = University of Missouri–Columbia. ND = North Dakota State University of Agriculture and Applied Sciences (Fargo, ND); NJ = Rutgers, The State University of New Jersey (Brunswick, NJ); NY = Cornell University (Ithaca, NY); OH = The Ohio State University (Columbus, OH); SC = Clemson University (Clemson, SC); SD = South Dakota State University (Brookings, SD); SP = Spanish National Research Council (Pontevedra, Spain); TN = University of Tennessee–Knoxville; USDA = U.S. Department of Agriculture (Washington, DC); WI = University of Wisconsin–Madison; NA = not applicable.

Supplemental Table 2. *Zea mays* genes associated with the significant locus S6_19987285 found in the susceptibility-to-sugarcane mosaic virus genome-wide association study.

Associated gene model ^z	Model type ^y	Location	Gene description
GRMZM2G037500	PCD	(Chr6: 19847399.19851546)	gbptf1 (GeBP-transcription factor 1)
AC203396.3_FG003	TE	(Chr6: 19973765.19988320)	
GRMZM2G147637	LC	(Chr6: 19872033.19878792)	
GRMZM2G392516	PCD	(Chr6: 19960725.19967788)	
GRMZM2G384990	LC	(Chr6: 19985350.19988797)	
GRMZM5G856609	LC	(Chr6: 19985470.19988699)	
GRMZM2G533307	LC	(Chr6: 19985815.19988915)	
GRMZM2G533313	LC	(Chr6: 20014034.20017176)	
GRMZM2G058741	TE	(Chr6: 20058058.20061531)	
LOC103629131	PCD	(Chr6: 20213684.20217652)	period circadian protein homolog 2
GRMZM5G802354	PCD		

^zAssociated gene model referenced from Maize B73 Refgen v3.

^yModel types are LC = low confidence; PCD = protein coding; TE = transposable element.

Supplemental Table 3. *Zea mays* genes associated with the significant locus S6_36947510 found in the susceptibility-to-sugarcane mosaic virus genome-wide association study.

Associated gene model ^z	Model type ^y	Location	Gene description
GRMZM2G581005	LC	(Chr6: 36807922.36811018)	
GRMZM2G571740	LC	(Chr6: 36895611.36900999)	
GRMZM2G581004	TE	(Chr6: 36821164.36824302)	
GRMZM2G581002	LC	(Chr6: 36831156.36834257)	
GRMZM2G432999	LC	(Chr6: 36858137.36862112)	2 leaf expansion stage
GRMZM2G532195	TE	(Chr6: 37170575.37173682)	
GRMZM5G858648	TE	(Chr6: 37026108.37033894)	
GRMZM2G179069	PCD	(Chr6: 36945138.36950105)	dof3 (DNA binding with one finger3)
GRMZM2G486245	TE	(Chr6: 36971750.36975125)	
GRMZM5G883412	LC	(Chr6: 37074520.37077940)	
GRMZM5G837567	TE	(Chr6: 37076232.37081427)	
AC197336.3_FG001	TE	(Chr6: 36972387.36975488)	
GRMZM5G876606	LC	(Chr6: 36946563.36949718)	dof3 (DNA binding with one finger3)
GRMZM2G021909	PCD	(Chr6: 37023635.37028921)	
GRMZM2G493573	LC	(Chr6: 37045645.37048735)	
GRMZM2G021931	LC	(Chr6: 37046349.37050104)	
GRMZM2G383019	LC	(Chr6: 37175236.37181956)	
GRMZM2G581000	LC	(Chr6: 36831737.36834834)	
AC187244.4_FG004	LC	(Chr6: 36832550.36837598)	
GRMZM5G858648	TE	(Chr6: 37026108.37033894)	

^zAssociated gene model referenced from Maize B73 Refgen v3.

^yModel types are LC = low confidence; PCD = protein coding; TE = transposable element.