Identification of Small Grains Genotypes Resistant to *Wheat spindle streak mosaic virus*

L. Cadle-Davidson, Department of Plant Pathology, M. E. Sorrells, Department of Plant Breeding and Genetics, S. M. Gray, United States Department of Agriculture–Agricultural Research Service (USDA-ARS), Plant Protection Research Unit, and G. C. Bergstrom, Department of Plant Pathology, Cornell University, Ithaca, NY 14853

ABSTRACT

Cadle-Davidson, L., Sorrells, M. E., Gray, S. M., and Bergstrom, G. C. 2006. Identification of small grains genotypes resistant to *Wheat spindle streak mosaic virus*. Plant Dis. 90:1045-1050.

Once *Wheat spindle streak mosaic virus* (WSSMV) becomes established in a field, the only available control strategy is the planting of resistant genotypes. In this study, we assessed 112 genotypes of winter wheat, rye, triticale, and barley for resistance to WSSMV in a 3-year trial in a field that had been used continuously for WSSMV evaluation for over 20 years. Because resistance to WSSMV reduces the percentage of plants that develop detectable virus titer and symptoms, we collected and analyzed disease incidence data. None of the genotypes was immune to infection. Sixty-two of the regionally adapted genotypes repeatedly expressed resistance to WSSMV, thus providing growers with a choice of cultivars resistant to WSSMV. Because of a significant interaction between genotypes and environment (year), genotypes should be assessed for incidence of symptomatic plants in multiple years, particularly when differentiating intermediate responses from highly susceptible and highly resistant responses.

Additional keywords: *Bymovirus*, disease gradient, logistic regression, *Polymyxa graminis*

Wheat spindle streak mosaic virus (WSSMV), a *Bymovirus* in the family *Potyviridae*, causes significant disease resulting in measurable yield loss in winter wheat in the northeastern United States (16,17,20). WSSMV putatively is vectored by the protozoan plasmodiophorid *Polymyxa graminis*, an obligate parasite of wheat roots that can survive in dry soil without a host for at least 10 years while retaining transmissibility of the viruses it may harbor (1). Transmission occurs shortly after autumn planting (5); however, because of slow systemic viral movement (19), symptoms typically are not expressed until the following spring. In New York, symptom expression begins in early May (stem elongation stages), with typical symptoms appearing as elongated spindle-shaped chlorotic streaks, each surrounding a dark green island. Symptoms fail to develop on emerging leaves when the average temperature exceeds 15°C, but can reinitiate on growth as late as the flag leaf

given conducive weather conditions (L. Cadle-Davidson and G. C. Bergstrom, unpublished). Chemical and cultural control tactics for managing WSSMV have not been identified. Due to the longevity of WSSMV and *P. graminis* in soil even in the absence of a host, the only practical control tactic once WSSMV becomes established in a field is the planting of resistant genotypes.

Resistance to WSSMV is highly heritable and controlled by a few dominant genes (11,12). In a population of recombinant inbred lines derived from a cross between resistant 'Geneva' and susceptible 'Augusta,' one major gene for resistance to WSSMV resided on chromosome arm 2DL (11). Cultivar resistance against the putative vector has not been identified (2,3,22). Presumably, the vector is able to transmit WSSMV to the cortical root tissue of all host genotypes.

Resistance to WSSMV has been characterized as a reduction in the incidence of symptomatic plants but does not necessarily correspond with a reduction in virus titer or symptom severity in infected plants (7). Yield loss has been correlated to the incidence of systemically infected plants (17). In the susceptible cvs. Augusta and Frankenmuth, yield declined as much 32% associated with an increase in wheat spindle streak mosaic (WSSM) disease incidence from 0 to 100% (17). In the breeding line NY6432-10, symptoms were difficult to diagnose, and neither symptom incidence nor viral titer (detected by enzyme-linked immunosorbent assay [ELISA]) had statis-

tically significant correlation with yield loss (17).

Using serological methods, WSSMV has been detected in roots of the resistant wheat cv. Geneva not expressing foliar symptoms, suggesting that resistance blocks neither transmission nor cell-to-cell movement in roots (L. Cadle-Davidson, unpublished data). In a study looking at the incidence of WSSM on individual stems in the field, a small proportion of individual Geneva plants became systemically infected with WSSMV and produced foliar symptoms (7). Highly resistant 'KS92WGRC22' had a low incidence of symptoms on individual plants and, on those plants that were symptomatic, individual stems were asymptomatic (7). Together, these observations suggest that one mechanism of resistance to WSSMV is to restrict the movement of virus into foliar portions of the plant and that this restriction can be overcome in individual plants or stems, resulting in foliar symptoms.

When evaluating resistance, researchers commonly either use a subjective whole-plot severity (or yellowing) scale (11,18) or gather incidence data and convert that to relative ranks or a "severity scale" (9,11). These methods have the advantage of being easily analyzed and are amenable to multiple comparisons analyses, such as Duncan's (versus a check variety) or Fisher's least significant difference. Another valid statistical method is the use of half-normal probability plots, which can be used for analysis of unreplicated trials (14). The downfall of half-normal probability plot analysis is the subjectivity required in the analysis itself (13). Logistic regression also has been employed to analyze incidence data and detect quantitative variation in a trait while maintaining the ability to demarcate discrete relative ratings based on the interpreter's needs (4).

Our aim in this study was to evaluate resistance to WSSMV in regionally adapted cultivars and advanced breeding lines.

MATERIALS AND METHODS

Plant materials. The genotypes included in this study composed two sets of genotypes selected for evaluation by the small grains breeding program at Cornell University, Ithaca, NY. One set included only soft white winter wheat (SWWW) genotypes, the majority being advanced breeding lines. The other set was predomi-

Corresponding author: G. C. Bergstrom
E-mail: gcb3@cornell.edu

Current address for L. Cadle-Davidson: USDA-ARS, Grape Genetics Research Unit, Cornell University, Geneva, NY 14456.

Accepted for publication 13 March 2006.

DOI: 10.1094/PD-90-1045

This article is in the public domain and not copyrightable. It may be freely reprinted with customary crediting of the source. The American Phytopathological Society, 2006.

Table 1. Average percentage of *Wheat spindle streak mosaic virus*-symptomatic stems for each year for genotypes with 3 years' data (check genotypes)

Check	Genotype ^b	Year ^a		
		2000 (%)	2002 (%)	2003 (%)
1	TW97613	62.3	65.7	94.3
2	Roane	74.3	46.7	90.0
3	Pioneer 2548	nd	41.1	76.1
4	Superior	16.7	64.3	91.0
5	A94-1315	48.3	29.0	78.3
6	Sawyer	49.0	27.7	51.0
7	Foster	57.7	36.7	23.3
8	OH 530	29.0	35.7	37.7
9	IL 91-15911	51.0	19.0	31.0
10	Sisson	32.3	25.7	27.7
11	Marilee	8.9	46.7	nd
12	Freedom	27.7	17.7	33.3
13	AC Ron	11.0	26.7	20.0
14	Va 96W-247	32.3	6.7	12.3
15	NYBatavia (MS)	21.0	4.3	23.3
16	Cardinal	nd	2.3	28.9
17	Watford	5.7	0.0	14.3
18	Whitby	1.0	4.3	14.3
19	Harus (MR)	4.0	3.3	11.7
20	Pioneer 25W33	2.3	9.0	7.7
21	Patton	7.7	1.0	10.0
22	Houser	11.0	1.0	5.7
23	Pearl	0.0	1.0	13.3
24	Caledonia	1.0	1.0	10.0
25	Pioneer 2510	10.0	1.0	0.0
26	Trical 815 (Tcl)	7.7	2.3	1.0
27	Mendon	5.7	1.0	3.3
28	Geneva (R)	2.3	3.3	4.0
29	KY86C-61-8	1.0	2.3	5.7
30	Bavaria	0.0	1.0	6.7
31	Hoffman 37	1.0	2.3	4.3
32	Richland	nd	0.0	5.0
33	Presto (Tcl)	3.3	3.3	0.0
34	Cayuga	1.7	1.0	1.7
35	Pioneer 2540	0.0	3.3	1.0
36	RSIBG 1998 (Tcl)	0.0	2.3	0.0
37	Aroostock (rye)	nd	1.1	0.0
38	Pioneer 25R37	nd	1.1	0.0
39	RSI AZ 1998 (Tcl)	nd	0.0	0.0

^a Spring in which symptoms were assessed; average incidence of infected stems for that year based on 3 or 6 replications of 30; nd = not determined.

^b Genotypes included in the study for all 3 years. Wheat, unless otherwise stated; Tcl = triticale. Previously published resistance ratings are in bold: MS = moderately susceptible, MR = moderately resistant, and R = resistant.

Fig. 1. Wheat spindle streak mosaic (WSSM) disease distribution for 'NY88036-8082' in 2003. Point locations represent the middle of each row of the susceptible check NY88036-8082 wheat, which was planted in every sixth row throughout the nursery. Location and corresponding incidence data were entered into a geographic information system and Kriging interpolation was performed. Shading represents a point estimate for the percent incidence of WSSM. The map is oriented with north at its top.

nantly soft red winter wheat (SRWW) cultivars, but included several barley, rye, and triticale cultivars and some SWWW cultivars for comparison across the two sets. In 1999 to 2000, our nursery included the two sets of 40 genotypes, with several of the genotypes being present in both sets. Therefore, up to 18 rows (three replicates, two sets, 3 years) of a single genotype could have been assessed, as was the case for 'Harus' and 'Cayuga.' In subsequent years, as some of the original genotypes were replaced in the breeding program, we chose to retain genotypes to obtain multiple years of data. By the end of the study, we had evaluated 112 different genotypes, 32 of which were evaluated in all 3 years (Table 1).

Field evaluation. The SWWW and SRWW genotypes were planted at Caldwell Field (Williamson very fine sandy loam soil; Ithaca, NY) as two separate experiments in a randomized complete block design with three replications of single, 1-m rows with seed planted at 5 g/m. The WSSMV nursery in Caldwell Field has been planted to winter wheat continuously since the 1980s, and WSSM symptoms have been uniform throughout the nursery in most years.

Each year the WSSMV nursery was plowed, disked, and harrowed, incorporating 10-20-20 NPK at 225 kg ha⁻¹ prior to September planting. Plants were top dressed with ammonium nitrate at 84 kg ha⁻¹ in early April. In early May, during peak symptom expression, the majority of stems was harvested from each row. A subsample was constructed by arbitrarily plucking 30 stems from the pile of harvested stems. Each of the 30 stems was examined in a laboratory setting and rated individually for presence or absence of typical symptoms of WSSM (i.e., elongated chlorotic lesions tapered at each end and with a green island in the center). These highly definitive symptoms were considered diagnostic of WSSM based on positive confirmation of coat protein of WSSMV in symptomatic plants from this nursery when assayed by double-antibody sandwich (DAS)-ELISA over more than a decade of testing (6-8; G. C. Bergstrom and S. M. Gray, *unpublished*). During that same historical period, *Soilborne wheat mosaic virus* was not detected in this nursery by visual inspection or serological tests (G. C. Bergstrom and S. M. Gray, *unpublished*), nor was it detected visually during the period of this study. In the first year of this study, 2000, symptomatic leaves of the check cvs. Pioneer 2548, Mendon, and Geneva tested positive for WSSMV by DAS-ELISA. No serology was performed in subsequent years of this study. In 2001, no viral symptoms developed in the field, and ratings were not taken.

Some of the cultivars in the study had been rated for WSSMV resistance in previous publications and are thus denoted in

Tables 1 and 2 for reference. In studies conducted in New York, cvs. Geneva, Harus, and NYBatavia have been suggested to be resistant, moderately resistant, and moderately susceptible, respectively (11,15–17,21).

Characterizing disease distribution.

To detect and potentially account for non-uniform distribution of WSSMV at Caldwell Field, the susceptible check 'NY88036-8082' was planted in every sixth row for the 2002–03 growing season. Incidence was assessed as described above, and the results were entered into a geographic information system (ArcGIS; ESRI, Redlands, CA) along with the corresponding location data for each row. For the purpose of interpolation, each row was defined by the midpoint of the row, and Kriging analysis was performed (10). The resulting surface was intended to be diagnostic and to suggest whether our statistical analysis ought to take disease distribution into account (Fig. 1).

Statistical analysis. Proc Logistic in SAS (SAS Institute Inc., Cary, NC) was used to calculate a 95% Wald confidence interval for the odds ratio, setting the incidence of each genotype against a check genotype selected when programming. Three scenarios exist for this confidence interval: (i) if the interval contained 1, WSSM incidence was not significantly different than the check genotype; (ii) if the interval contained only numbers less than 1, the genotype had significantly less WSSM incidence than the check; or (iii) if the interval contained only numbers greater than 1, the genotype had significantly greater WSSM incidence than the check (all scenarios at $\alpha = 0.05$).

The analysis was repeated so that each of 39 cultivars evaluated in two or three seasons (Table 1) was the check genotype for one iteration. By encoding a 0 for scenario (i), a –1 for scenario (ii), and a 1 for scenario (iii), the statistical analysis gives rise to a matrix comparing the set of all genotypes with the set of check genotypes (Fig. 2). Summing across comparisons with all check genotypes yields a statistic for relative WSSM incidence or, loosely, susceptibility. Sorting this matrix from resistant (most negative number) to susceptible (largest positive number) for both the set of all genotypes and the set of check genotypes allows for graphic representation of relative resistance. Using this method, a model was analyzed with only genotype and year as predictors because the spatial variability of disease incidence in the WSSMV nursery was negligible.

RESULTS

Disease incidence and distribution.

Disease incidence was high in spring 2000 (20.3% average incidence across all genotypes; 61.6% average for TW97613, Roane, and A94-1315, the three genotypes

Table 2. Summary of the average percentage of *Wheat spindle streak mosaic virus*-symptomatic stems for all genotypes across all years

Genotype ^a	Average (%) ^b	Minimum (%) ^c	Maximum (%) ^d	Sampled rows ^e
TW060-075	90.0	86.7	93.3	3
VAN98W-170WS	85.6	73.3	96.7	3
NY88036-8082	83.2	60.0	100.0	15
Century II	76.7	63.3	90.0	3
TW97613	74.1	46.7	96.7	9
NY89073SP-8110	72.2	40.0	96.7	6
Roane	70.4	33.3	96.7	9
TW97611	70.0	53.3	83.3	3
Pioneer 25R18	67.8	60.0	83.3	3
Pioneer 2548	64.4	40.0	100.0	9
CaledoniaResel-T	57.8	40.0	80.0	3
Superior	56.2	10.0	96.7	9
Va97W-375WS	48.9	40.0	63.3	3
NY89073SP-8145	47.8	0.0	93.3	6
NY89056SP-9060	47.8	36.7	60.0	3
A94-1315	45.0	13.3	83.3	8
Sawyer	42.6	6.7	66.7	9
Genesis 9953	42.2	0.0	76.7	6
NY88048-8185	40.0	16.7	70.0	9
Foster	39.3	13.3	80.0	9
NY89088-9101	36.7	26.7	50.0	3
NY89059SP-8094	36.1	13.3	80.0	6
OH 530	34.1	10.0	66.7	9
IL 91-15911	33.7	6.7	76.7	9
M98*2152	33.3	23.3	40.0	3
CaledoniaResel-SS	31.1	13.3	50.0	3
T123	30.0	13.3	46.7	6
Sisson	28.5	20.0	40.0	9
Pioneer 25R78	26.7	23.3	30.0	3
Freedom	26.3	6.7	53.3	9
TW96202	25.8	13.3	43.3	6
AC Ron	19.3	3.3	50.0	9
NY89088-8152	18.9	0.0	46.7	6
NY89088-9118	17.8	13.3	23.3	3
NY88045-8164	17.8	10.0	26.7	3
Va 96W-247	17.0	0.0	53.3	9
VA97W-375	16.7	3.3	30.0	6
NYBatavia (MS)	16.3	0.0	33.3	9
NY89078SP-8184	16.1	3.3	33.3	6
Cardinal	15.6	0.0	43.3	6
NY89082-7159	15.0	0.0	43.3	6
NY89065SP-8113	13.7	0.0	43.3	9
Marilee	12.2	6.7	23.3	6
VA98W-593	10.6	0.0	23.3	6
NY89063-9126	10.0	10.0	10.0	3
NY88046-8138	10.0	3.3	20.0	5
NY89035SP-8162	7.9	0.0	17.5	6
NY89088-8173	7.8	0.0	16.7	9
NY89088-7401	7.8	0.0	13.3	6
NY91017-8080	7.2	0.0	13.3	6
NY89081-7161	6.8	0.0	26.7	6
Whitby	6.7	0.0	20.0	9
Watford	6.7	0.0	23.3	9
NY89064SP-7139	6.7	0.0	26.7	6
Harus (MR)	6.3	0.0	23.3	18
Pioneer 25W33	6.3	0.0	20.0	9
Patton	6.3	0.0	20.0	9
Houser	5.9	0.0	20.0	9
NY89086SP-9203	5.6	0.0	13.3	6
NY89037SP-8140	5.6	0.0	20.0	6
CaledoniaResel-VT	5.6	3.3	6.7	3
NY89086-7120	5.0	0.0	16.7	6
NY89052-9140	5.0	0.0	13.3	6
Pearl	4.8	0.0	26.7	9

(continued on next page)

^a Wheat, unless otherwise stated. Previously published resistance ratings are in bold: MS = moderately susceptible, MR = moderately resistant, and R = resistant.

^b Average percent incidence: the sum of symptomatic stems in all replications in all years tested divided by the total number of stems assessed. Overall = overall incidence percentage.

^c Minimum percent of symptomatic stems in any replication in any year.

^d Maximum percent of symptomatic stems in any replication in any year.

^e Total number of replications tested, which may be the result of 1 to 3 years as an entry in the trial in one or both of the genotype sets.

Table 2. (continued from preceding page)

Genotype ^a	Average (%) ^b	Minimum (%) ^c	Maximum (%) ^d	Sampled rows ^e
NY89014SP-9220	4.7	0.0	10.0	5
TW044-065	4.4	0.0	10.0	3
NY89066-7131	4.4	0.0	13.3	6
NY89052-7142	4.4	0.0	10.0	6
NY87048W-7388	4.4	0.0	16.7	6
D8006	4.4	3.3	6.7	3
CaledoniaResel-A	4.4	0.0	6.7	3
Trical 815 (triticale)	3.3	0.0	23.3	12
Mendon	3.3	0.0	10.0	9
McCormick	3.3	0.0	10.0	6
NY92145BW-9144W	3.3	0.0	10.0	3
NY91028-9073	3.3	0.0	6.7	3
NY89052SP-9232	3.3	0.0	6.7	6
NY88072-9070W	3.3	0.0	6.7	3
Geneva (R)	3.1	0.0	13.3	17
KY86C-61-8	3.0	0.0	16.7	9
Trical 336 (triticale)	2.8	0.0	6.7	6
Hoffman 37	2.6	0.0	6.7	9
Bavaria	2.6	0.0	13.3	9
Richland	2.5	0.0	13.3	12
Presto (Triticale)	2.2	0.0	6.7	9
NY91028SP-9245W	2.2	0.0	3.3	3
NY88046SP-9230W	2.2	0.0	3.3	3
NY89056SP-7208	2.0	0.0	6.7	5
NY90008-8092	1.7	0.0	3.3	6
Pioneer 2540	1.5	0.0	6.7	9
Cayuga	1.5	0.0	3.3	18
NY93251R-9141W	1.1	0.0	3.3	3
NY88041-9063W	1.1	0.0	3.3	3
Elmira (barley)	1.1	0.0	6.7	6
CaledoniaResel-L	1.1	0.0	3.3	3
Caledonia	1.1	0.0	3.3	15
Pioneer 25R37	0.8	0.0	3.3	12
RSIBG 1998	0.7	0.0	3.3	9
Pioneer 2510	0.7	0.0	3.3	9
TW005-008	0.6	0.0	3.3	6
RSI111	0.6	0.0	3.3	6
McGregor (barley)	0.6	0.0	3.3	6
CaledoniaResel-S	0.6	0.0	3.3	6
Aroostock (rye)	0.6	0.0	3.3	6
U-1273-5-18-8	0.0	0.0	0.0	6
TW006-007	0.0	0.0	0.0	6
RSI AZ 1998 (triticale)	0.0	0.0	0.0	6
Red Caledonia	0.0	0.0	0.0	3
Pioneer 25R23	0.0	0.0	0.0	3
NY89025-9111W	0.0	0.0	0.0	3
McDermott	0.0	0.0	0.0	5
Bobcat (triticale)	0.0	0.0	0.0	3
Overall	17.1	0.0	100.0	738

rated most susceptible across all three years; Table 1) and 2003 (20.6% for all genotypes; 87.5% for the three most susceptible genotypes) and moderate in 2002 (12.0% for all genotypes; 47.1% for the three most susceptible genotypes), but no WSSM disease symptoms were detected in 2001. Over the three sampling years, the average incidence ranged from 0 to 90% for individual genotypes, with the average incidence of all genotypes being 17.1% (Table 2). A relatively uniform disease incidence was observed across the field, with the exception of a slight disease gradient detected near the western border of the plot in 2003 (Fig. 1). Most registered cultivars that currently are grown in the Northeast had relatively low incidence of WSSMV. The more susceptible genotypes in the study were breeding lines and cultivars not grown currently in the Northeast.

Evaluating relative susceptibility. A logistic model was created in SAS using all 3 years' data, with the response variable being the proportion of symptomatic stems and the predictors being genotype, year, and the interaction genotype-year. Genotype and genotype-year both were significant predictors of incidence ($P < 0.001$ each). Table 1 reflects this interaction; for example, in 2002, a year with low average incidence, 'AC Ron' wheat had its highest incidence.

Statistical analysis of the data summarized in Table 2 is represented in Figure 2. The y-axis contains each of the 112 experimental genotypes, and the x-axis represents each of the 39 check genotypes. Genotypes with overlapping bars did not have significant differences in the percent incidence for the 3-year study, nor did they have significantly different incidence than the check genotypes with which the bars

intersect. Therefore, Figure 2 depicts several overlapping incidence classes and shows that 69 of the genotypes tested are significantly more susceptible than the most resistant genotype, RSI AZ 1998 (or that 43 of the genotypes are not significantly more susceptible).

In the current study, Geneva (check cultivar no. 28) did not have significantly different WSSM incidence than the 62 most resistant genotypes, including check cultivars no. 20 to 39 (Fig. 2). Harus (check cultivar no. 19) had significantly lower incidence of WSSM than check cultivars no. 1 to 16, significantly higher incidence than check cultivars no. 27 to 39, and not significantly different incidence than check cultivars no. 17 to 26. NYBatavia (check cultivar no. 15) had significantly lower incidence of WSSM than check cultivars no. 1 to 11, significantly higher incidence than check cultivars no. 17 to 39, and not significantly different incidence than check cultivars no. 12 to 16. These results support the previous rankings of relative resistance of these three cultivars.

DISCUSSION

In New York, nearly every field with a history of small grains production is infested with WSSMV (16), and planting cultivars resistant to WSSMV is the only practical approach to managing the disease once WSSMV is introduced. Geneva, Harus, and NYBatavia previously have been suggested to be resistant, moderately resistant, and moderately susceptible, respectively (11,15–17,21). Therefore, these three cultivars were included as checks in our evaluation of 112 cultivars and advanced breeding lines over 3 years of variable disease incidence. The relative incidence of WSSM in these cultivars supported their previous rankings. Sixty-two of the tested genotypes exhibited a high level of resistance to WSSMV, similar to Geneva, based on consistently low disease incidence.

Our data provide evidence that cultivars planted by New York wheat growers today are more resistant than cultivars commonly grown in the 1980s (15,16). Resistant cultivars such as Caledonia and Richland account for major percentages of New York wheat acreage today and many promising breeding lines also have resistance. However, highly susceptible cultivars also are available, suggesting that vigilance still is warranted in cultivar selection by New York growers. Growers and seed distributors can use our results to select regionally adapted cultivars with resistance to WSSMV.

Resistance evaluation using mechanical inoculation of leaves with WSSMV has not corresponded with resistance evaluation in the field (7), presumably because resistance limits root-to-shoot movement and not movement within the shoots. Be-

Fig. 2. Relative resistance of 112 small grain genotypes to *Wheat spindle streak mosaic virus* (WSSMV) based on disease incidence. Each horizontal bar is the confidence interval for disease incidence of one y-axis genotype relative to disease incidence of x-axis genotypes. The y-axis represents the entire set of 112 genotypes assessed for resistance to WSSMV and sorted from most susceptible (top) to most resistant (bottom). For readability, only 23 genotypes of interest are labeled. The x-axis represents 39 check genotypes (Table 1) sorted from most susceptible (left) to most resistant (right). Genotypes on the y-axis (listed in the order of Table 2) with overlapping bars did not have significantly different percent incidences of wheat spindle streak mosaic disease over the course of the study, relative to the check cultivars represented on the x-axis.

cause protocols for efficient WSSMV inoculation of roots have not been developed (and because evaluation based on mechanical root inoculation also may not correspond with evaluations in the field), resistance still must be assessed in field trials. Although labor-intensive, evaluation of the incidence of WSSM symptoms in field trials is straightforward and the results can be applied directly to genotype selection. In some years, however, in a subset of planting dates, resistance is less effective, resulting in significant genotype-planting date interactions (5). Fur-

thermore, given the environmental sensitivity of symptom expression, reliable evaluation of resistance requires compilation of multiple years of replicated data. Due to the complexity of the pathosystem and its interaction with environment, adjacent individual plants of the same genotype often respond differently to the same conducive environment. Assessing the incidence of WSSM in a population or row takes this variability into account.

Because assessment of incidence is labor intensive, most evaluations of resistance to soilborne viruses use a whole-plot severity (yellowing) scale, which is essentially an index combining percent incidence and individual plant severity. Although powdery mildew and other springtime foliar diseases can complicate visual whole-plot WSSM assessment, when conducted carefully, whole-plot assessments can adequately distinguish WSSMV-susceptible from WSSMV-resistant genotypes. However, in addition to the subjectivity required in classifying relative symptom production, whole-plot assessments may not discern the quantitative variation in WSSMV resistance that we have identified here. By quantifying percent incidence, the subjectivity of the data collection was minimized. Furthermore, classification of the genotypes did not require subjectivity, and minor differences in relative resistance to WSSMV could be discerned. The ability to detect subtle quantitative variation is critical to a fundamental understanding of resistance.

Because resistance to WSSMV is expressed as a reduction in the incidence of symptomatic plants or stems, the appropriate method for evaluating resistance is to analyze incidence. Logistic analysis of our disease incidence data showed a significant interaction between genotype and year, partly due to the effect of soil microenvironment on WSSMV transmission and the age-dependent susceptibility of wheat genotypes (5). This statistical interaction can result from changes in percent incidence in different years for genotypes even if relative susceptibility of genotypes remains the same. This phenomenon is common because resistant genotypes such as Cayuga wheat have low incidence of WSSM symptoms in most years but the incidence in susceptible genotypes such as IL 91-15911 wheat may fluctuate greatly depending on environment and disease pressure. Genotype-year interactions also can result from changes in relative susceptibility of genotypes in different years. One example of this was 'Superior' wheat which, in the first year, had moderate incidence of WSSM but, in the last 2 years, had high incidence. Therefore, our data reemphasize the need for WSSM symptoms to be assessed in multiple environments (locations, years, or both) to increase confidence that a genotype is truly resistant or

to improve the resolution of classification for genetic studies.

ACKNOWLEDGMENTS

This research was supported by Cornell University Hatch Projects NYC153472, NYC153433, and NYC149419; a grant from the New York State IPM Program; a fellowship to L. Cadle-Davidson from the Storkan-Hanes-McCaslin Research Foundation; and a graduate research assistantship to L. Cadle-Davidson from the Department of Plant Pathology, Cornell University, Ithaca, NY. The plots were expertly aligned, planted, and managed by D. Benscher and D. Valenta.

LITERATURE CITED

- Adams, M. J. 1990. Epidemiology of fungally-transmitted viruses. *Soil Use Manage.* 6:184-189.
- Adams, M. J., Jones, P., and Swaby, A. G. 1987. The effect of cultivar used as host for *Polymyxa graminis* on the multiplication and transmission of *Barley yellow mosaic virus* (BaYMV). *Ann. Appl. Biol.* 110:321-327.
- Adams, M. J., Swaby, A. G., and Macfarlane, I. 1986. The susceptibility of barley cultivars to *Barley yellow mosaic virus* (BaYMV) and its fungal vector, *Polymyxa graminis*. *Ann. Appl. Biol.* 109:561-572.
- Cadle-Davidson, L. 2003. Transmission of and resistance to soilborne viruses of wheat. Ph.D. dissertation, Cornell University, Ithaca, NY.
- Cadle-Davidson, L., and Bergstrom, G. C. 2004. The effects of post-planting environment on the incidence of soilborne viruses of small grains. *Phytopathology* 94:527-534.
- Carroll, J. E., Bergstrom, G. C., and Gray, S. M. 1992. Detection of coat protein of *Wheat spindle streak mosaic virus* is positively correlated with characteristic symptom expression. (Abstr.) *Phytopathology* 82:1147.
- Carroll, J. E., Bergstrom, G. C., and Gray, S. M. 2002. Assessing the resistance of winter wheat to *Wheat spindle streak mosaic bymovirus*. *Can. J. Plant Pathol. Rev. Can. Phytopathol.* 24:465-470.
- Carroll, J. E., Gray, S. M., and Bergstrom, G. C. 1995. Use of antiserum to a New York isolate of *Wheat spindle streak mosaic virus* to detect related *Bymoviruses* from North America, Europe, and Asia. *Plant Dis.* 79:346-353.
- Dahlberg, J. A., Bandyopadhyay, R., Rooney, W. L., Odvody, G. N., and Madera-Torres, P. 2001. Evaluation of sorghum germplasm used in US breeding programmes for sources of sugary disease resistance. *Plant Pathol.* 50:681-689.
- Goovaerts, P. 1992. Factorial Kriging analysis—a useful tool for exploring the structure of multivariate spatial soil information. *J. Soil Sci.* 43:597-619.
- Khan, A. A., Bergstrom, G. C., Nelson, J. C., and Sorrells, M. E. 2000. Identification of RFLP markers for resistance to *Wheat spindle streak mosaic bymovirus* (WSSMV) disease. *Genome* 43:477-482.
- Koeveing, V. M., Haufler, K. Z., Fulbright, D. W., Isleib, T. G., and Everson, E. H. 1987. Heritability of resistance in winter wheat to *Wheat spindle streak mosaic virus*. *Phytopathology* 77:742-744.
- Lawson, J., Grimshaw, S., and Burt, J. 1998. A quantitative method for identifying active contrasts in unreplicated factorial designs based on the half-normal plot. *Comput. Stat. Data Anal.* 26:425-436.
- Lenth, R. V. 1989. Quick and easy analysis of unreplicated factorials. *Technometrics* 31:469-473.
- Miller, N. R., Bergstrom, G. C., and Gray, S. M. 1988. Assessment of the occurrence and impact of viral diseases of winter wheat in New York State in 1988. (Abstr.) *Phyto-*

- pathology 78:1509.
16. Miller, N. R., Bergstrom, G. C., and Gray, S. M. 1991. Identity, prevalence, and distribution of viral diseases of winter wheat in New York in 1988 and 1989. *Plant Dis.* 75:1105-1109.
 17. Miller, N. R., Bergstrom, G. C., and Sorrells, M. E. 1992. Effect of *Wheat spindle streak mosaic virus* on yield of winter wheat in New York. *Phytopathology* 82:852-857.
 18. Mueller, D. S., Hartman, G. L., Nelson, R. L., and Pedersen, W. L. 2002. Evaluation of *Glycine max* germplasm for resistance to *Fusarium solani* f. sp. *glycines*. *Plant Dis.* 86:741-746.
 19. Schenk, P., Sohn, A., Adams, M. J., Antoniwi, J. F., Hamacher, J., and Steinbiss, H. H. 1995. Movement of Bymoviruses and functions of RNA2-encoded proteins of *Barley yellow mosaic virus*. *Agronomie* 15:387-391.
 20. Slykhuis, J. T., and Barr, D. J. S. 1978. Confirmation of *Polymyxa graminis* as a vector of *Wheat spindle streak mosaic virus*. *Phytopathology* 68:639-644.
 21. Sorrells, M. E., and Bergstrom, G. C. 1998. Registration of 'NYBatavia' wheat. *Crop Sci.* 38:551-551.
 22. Yili, R., Zou, W. H., and Chen, J. P. 1990. Quantitative assay of resting spores of *Polymyxa graminis* on barley roots. *Ann. Appl. Biol.* 117:343-347.