Identification of Small Grains Genotypes Resistant to *Soilborne wheat mosaic virus*

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ABSTRACT

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Soilborne wheat mosaic virus (SBWMV) was detected in New York in 1998 for the first time and has been associated with yield loss where identified. We assessed 115 regionally adapted small grains genotypes for resistance to SBWMV over four growing seasons. Resistance to SBWMV reduces the percentage of plants that develop detectable viral titer and symptoms. Logistic regression was used to analyze disease incidence data and was compared with a general linear model for categorizing relative resistance to SBWMV. Logistic regression facilitated assessment of the effects of small sample size, low disease incidence, and nonuniform disease distribution. By increasing sample size from 20 to 30 stems per replicate, the number of resistance categories was increased through improved resolution of intermediate resistance classes. In environments with low disease incidence, the number of genotypes categorized as susceptible decreased while intermediate genotypes appeared to be resistant in the analysis. Inclusion of disease distribution data as covariates in a spatially balanced experiment did not increase the power of the logistic analysis. No genotype assessed in multiple years was immune to infection. However, 41 of the regionally adapted genotypes tested repeatedly expressed strong resistance to SBWMV, providing growers a choice of cultivars resistant to SBWMV.

Soilborne wheat mosaic virus (SBWMV) infects wheat, rye, triticale, and barley, causing disease and yield loss on those crops sown in the autumn (12,13). SBWMV first was detected in New York State in 1998 and since then has been spreading in the Finger Lakes region, where autumn-sown small grains are important in crop rotations with vegetables and other field crops. Prior to 1998, small grains bred in New York State did not undergo testing for resistance to SBWMV.

In New York, SBWMV symptom expression begins in late April, with typical chlorotic symptoms appearing in patches of plants where machinery first entered the field, reflecting recent introduction of the virus. Symptoms on individual leaves are a genotype-specific mosaic (13,21). Symptoms fail to develop on emerging leaves when the average temperature exceeds 20°C (11), but can reinitiate on new growth as flag leaves emerge given conducive weather conditions (L. Cadle-

Davidson and G. C. Bergstrom, *unpublished*). Wet autumns and prolonged cool periods in spring are conducive for disease development.

SBWMV presumably 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). A controlled environment study using infested field soil suggested that optimal SBWMV transmission requires at least 24 h of matric potentials greater than –20 kPa (wetter than field capacity) any time after seedling emergence but before soil temperatures drop below 7°C (7). Suboptimal soil temperature or moisture in the autumn results in less efficient transmission and, therefore, reduced disease incidence the following spring (6). Furthermore, cool weather in the spring promotes the development and maintenance of symptoms and disease severity.

Currently, the only feasible control tactic for SBWMV, once inoculum has become established in a field, is the planting of resistant cultivars (11). Resistance to SBWMV appears to be controlled by one to three genes (4,14,15,17,18,20). Resistance most often is expressed as a reduction in the incidence of symptomatic plants, but not necessarily as reduced titer or severity in infected plants (5,10,16,19). Because cultivar resistance against the putative vector has not been identified (2,3,22), SBWMV is likely to be transmitted to the cortical root tissue of most host

genotypes. Therefore, the resistance observed involves the inability of SBWMV to move from colonized roots to foliar tissue and result in symptoms (16). Resistance evaluation using mechanical inoculation of leaves does not translate to field resistance, because resistance appears to be directed against root-to-shoot movement and not systemic movement in the shoots (16).

In this study, we evaluated host resistance to SBWMV in 115 wheat, rye, triticale, and barley genotypes based on incidence data. Additionally, we identified the effects of spatial heterogeneity, small sample sizes, and low disease pressure on resistance evaluation, and an experimental design was suggested to overcome these potential problems.

MATERIALS AND METHODS

Plant materials. The genotypes included in this study composed two sets 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 predominantly soft red winter wheat (SRWW) cultivars, but included several barley, rye, triticale, and SWWW cultivars. In 1999 to 2000, our nursery included the two sets of 40 genotypes, including several genotypes that were included in both sets. In subsequent years, we added genotypes of interest. By the end of the study, we had evaluated 115 genotypes.

Field evaluation. The SWWW and SRWW genotypes were planted in a grower's field nursery in Trumansburg, NY (Conesus gravelly silt loam soil) 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. In 2001–02, limited resources reduced the sample size to 20 stems from each of the two replicate blocks with the highest disease incidence. The SBWMV nursery in Trumansburg was planted to susceptible winter wheat continuously from 1997 to 2002 and expressed consistent natural occurrence of SBWM symptoms every year. In the last two growing seasons of the study (2001–02 and 2002–03), the locations of the blocks were dispersed in a spatially balanced, nonrandom design in order to increase the likelihood that both nurseries encountered strong disease pressure.

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Each year the SBWMV nursery was plowed, disked, and harrowed, incorporating 10-20-20 NPK at 225 kg ha⁻¹ prior to September planting. In early May, during peak symptom expression, stems were harvested from a majority of plants in each row. Thirty stems were subsampled arbitrarily and rated individually for expression of viral symptoms in a laboratory setting. In the first year of the study, visual assessment was verified using enzyme-linked immunosorbent assay (ELISA) to detect SBWMV coat protein as per the manufacturer's instructions (Agdia, Elkhart, IN). Sap for ELISA was extracted from roots of individual plants using a rolling leaf press and diluted in 0.5 ml of phosphate-buffered saline (PBS). Samples with absorbance values greater than five times the uninfected control were considered to be positive for SBWMV.

Disease incidence and distribution. The first 2 years of resistance assessments suggested that disease pressure was not distributed uniformly across the SBWMV nursery. To verify this observation, we planted the susceptible wheat cv. Harus in every sixth row in each of the last 2 years. Using the incidence of soilborne wheat mosaic (SBWM) disease on Harus, we then developed a disease incidence map across the field for each of the last 2 years using a geographic information system (ArcGIS; ESRI, Redlands, CA). The mid-point of each row was assigned a point location along with the corresponding SBWM incidence. A Kriging analysis was used for interpolating between Harus rows (9). The resulting surface was intended to be diagnostic and to suggest whether the statistical analyses ought to take disease gradients into account.

Statistical analysis. The logistic analysis and visual representation as described by Cadle-Davidson et al. (8) was used to define resistance classes and assign genotypes to them. Criteria for delineating relative resistance categories can be devised based on the end use; however, in this article, "discrete resistance classes" were used to compare objectively the power of each analysis. For identifying the maximal number of discrete resistance classes, the interval that included the most susceptible genotype based on disease incidence was used as the first class. The next most susceptible genotype that had significantly lower percent incidence than the first genotype (non-overlapping bars) then was identified. This defined the second class, and the process was continued until no genotypes had significantly lower percent incidence than the previous genotype.

RESULTS

Disease incidence and distribution. The average percent incidence of SBWM over 4 years ranged from 0 to 83% for individual genotypes, with the average incidence of all genotypes being 16.6%

Table 1. Four-year summary statistics for percent incidence of *Soilborne wheat mosaic virus* (SBWM) for each genotype tested

Cultivar	Average (%) ^a	Min (%) ^b	Max (%) ^c	Sampled rows ^d	Years tested ^e
NY89088-8152	83.0	63.3	90.0	5	3,4
NY89088-8173	71.7	10.0	95.0	7	3,4
NY88046-8138	69.7	23.3	100.0	5	3,4
NY87048W-7388	68.7	20.0	95.0	5	3,4
NY89088-7401	68.3	43.3	95.0	5	3,4
NY89088-9118	61.1	60.0	63.3	3	4
VA98W-593	59.7	3.3	100.0	5	3,4
NY89035SP-8162	59.0	10.0	95.0	5	3,4
NY89063-9126	56.7	26.7	73.3	3	4
Genesis 9953	51.0	10.0	95.0	5	3,4
VA97W-375	49.7	16.7	90.0	5	3,4
Sisson	49.1	10.0	95.0	11	1,2,3,4
TW044-065	48.9	23.3	63.3	3	4
TW97613	48.3	10.0	100.0	11	1,2,3,4
TW006-007	48.0	13.3	100.0	5	3,4
Va 96W-247	47.1	10.0	95.0	11	1,2,3,4
CaledoniaResel-VT	45.6	13.3	80.0	3	4
RSI111	45.3	0.0	90.0	5	3,4
Freedom	42.7	0.0	100.0	11	1,2,3,4
NY89088-9101	42.2	16.7	56.7	3	4
Trical 336 (triticale)	39.8	13.3	60.0	8	2,3,4
RSI AZ 1998 (triticale)	39.4	13.3	80.0	8	2,3,4
NY89065SP-8113	39.0	20.0	65.0	7	3,4
Harus	36.7	0.0	100.0	30	1,2,3,4
NY89025-9111W	35.6	13.3	80.0	3	4
Pioneer 2548	34.6	0.0	80.0	8	2,3,4
Cardinal	34.4	0.0	95.0	11	1,2,3,4
Watford	33.6	0.0	90.0	11	1,2,3,4
Aroostock (rye)	31.5	0.0	90.0	8	2,3,4
Marilee	31.3	0.0	100.0	8	1,2,3
Presto (triticale)	30.0	0.0	80.0	11	1,2,3,4
NY88045-8164	28.9	23.3	40.0	3	4
Mendon	25.8	0.0	100.0	11	1,2,3,4
Pioneer 25W33	25.5	0.0	80.0	11	1,2,3,4
NY89078SP-8184	25.0	6.7	60.0	5	3,4
NY89073SP-8110	24.0	0.0	66.7	5	3,4
RSIBG 1998 (triticale)	22.4	0.0	85.0	11	2,3,4
CaledoniaResel-L	20.0	3.3	40.0	3	4
Bobcat (triticale)	15.6	0.0	43.3	3	4
Puma (rye)	15.6	10.0	23.3	3	2
AC Ron	13.9	0.0	46.7	11	1,2,3,4
Pioneer 2540	13.9	0.0	100.0	11	1,2,3,4
Va97W-375WS	12.2	3.3	26.7	3	4
M98*2152	11.1	0.0	26.7	3	4
NY88048-8185	9.8	0.0	33.3	7	3,4
NY89073SP-8145	9.0	0.0	20.0	5	3,4
RSI 111 (triticale)	8.9	3.3	13.3	3	2
Roane	8.6	0.0	73.3	11	1,2,3,4
Trical 815 (triticale)	7.5	0.0	10.0	4	1,2,3
NY89037SP-8140	7.0	0.0	30.0	5	3,4
NY88036-8082	6.7	0.0	26.7	7	3,4
OH 530	6.1	0.0	16.7	11	1,2,3,4
D8006	6.0	0.0	20.0	5	2,3
NY89059SP-8094	5.7	0.0	13.3	5	3,4
NY91028-9073	5.6	0.0	13.3	3	4
VAN98W-170WS	5.6	0.0	13.3	3	4
RSI815	4.8	0.0	15.0	9	1,2,3,4
Pioneer 25R37	4.6	0.0	23.3	13	2,3,4
CaledoniaResel-A	4.4	0.0	6.7	3	4
CaledoniaResel-T	4.4	0.0	10.0	3	4
Superior	4.4	0.0	20.0	11	1,2,3,4
McCormick	4.3	0.0	10.0	5	3,4
NYBatavia	4.2	0.0	16.7	11	1,2,3,4
NY89086SP-9203	4.0	0.0	10.0	5	3,4
TW005-008	4.0	0.0	10.0	5	3,4

(continued on next page)

^a Average percent incidence: the sum of infected stems in all replications in all years tested divided by the total number of stems assessed.

^b Minimum percentage of infected stems in any replication in any year.

^c Maximum percentage of infected stems in any replication in any year.

^d Total number of replications tested, which may be the result of 1 to 4 years as an entry in the trial.

^e Calendar years in which a given genotype was assessed for SBWM incidence: 1 = 2000, 2 = 2001, 3 = 2002, and 4 = 2003.

Table 1. (continued from preceding page)

Cultivar	Average (%) ^a	Min (%) ^b	Max (%) ^c	Sampled rows ^d	Years tested ^e
U-1273-5-18-8	4.0	0.0	10.0	8	2,3,4
Pearl	3.9	0.0	15.0	11	1,2,3,4
Foster	3.8	0.0	10.0	11	1,2,3,4
Pioneer 2510	3.6	0.0	20.0	11	1,2,3,4
Patton	3.5	0.0	23.3	11	1,2,3,4
Whitby	3.5	0.0	15.0	10	1,2,3,4
NY92145BW-9144W	3.3	0.0	10.0	3	4
TW97611	3.3	0.0	6.7	6	1,2
Elmira (Barley)	2.9	0.0	10.0	8	2,3,4
NY89086-7120	2.7	0.0	10.0	5	3,4
Hoffman 37	2.6	0.0	20.0	11	1,2,3,4
Cayuga	2.5	0.0	20.0	21	1,2,3,4
NY90008-8092	2.5	0.0	5.0	4	3
TW96202	2.5	0.0	15.0	8	1,2,3
NY89052-7142	2.3	0.0	5.0	5	3,4
NY89081-7161	2.3	0.0	6.7	5	3,4
Sawyer	2.3	0.0	10.0	11	1,2,3,4
CaledoniaResel-SS	2.2	0.0	3.3	3	4
NY89056SP-9060	2.2	0.0	6.7	3	4
Pioneer 25R18	2.2	0.0	6.7	6	1,2
Pioneer 25R23	2.2	0.0	6.7	3	4
Pioneer 25R78	2.2	0.0	6.7	3	4
NY89052SP-9232	2.0	0.0	5.0	5	3,4
NY89056SP-7208	2.0	0.0	10.0	5	3,4
NY89064SP-7139	2.0	0.0	5.0	5	3,4
NY89066-7131	2.0	0.0	5.0	5	3,4
Houser	1.7	0.0	10.0	11	1,2,3,4
NY89052-9140	1.7	0.0	5.0	5	3,4
T123	1.7	0.0	5.0	5	3,4
Bavaria	1.5	0.0	10.0	11	1,2,3,4
Geneva	1.3	0.0	13.3	22	1,2,3,4
McGregor (barley)	1.3	0.0	10.0	8	2,3,4
NY89082-7159	1.3	0.0	6.7	5	3,4
A94-1315	1.2	0.0	5.0	11	1,2,3,4
IL 91-15911	1.2	0.0	10.0	11	1,2,3,4
KY86C-61-8	1.1	0.0	6.7	11	1,2,3,4
NY88072-9070W	1.1	0.0	3.3	3	4
NY91028SP-9245W	1.1	0.0	3.3	3	4
NY93251R-9141W	1.1	0.0	3.3	3	4
TW060-075	1.1	0.0	3.3	3	4
NY89014SP-9220	1.0	0.0	5.0	5	3,4
NY91017-8080	1.0	0.0	5.0	5	3,4
CaledoniaResel-S	0.7	0.0	3.3	5	3,4
Century II	0.6	0.0	3.3	6	1,2
Caledonia	0.5	0.0	5.0	20	1,2,3,4
Richland	0.5	0.0	5.0	10	3,4
McDermott	0.0	0.0	0.0	6	4
NY88041-9063W	0.0	0.0	0.0	3	4
NY88046SP-9230W	0.0	0.0	0.0	3	4
Red Caledonia	0.0	0.0	0.0	3	4
Overall	16.6	0.0	100.0	811	...

(Table 1). Of the samples rated as symptomatic in the first year of the study, 97% of the samples tested positive for SBWMV coat protein using ELISA. Nonsymptomatic plants were not tested for presence of SBWMV and, in subsequent years, we did not confirm our visual assessment with ELISA. Out of 28,270 stems assessed for soilborne virus symptoms in the SBWMV nursery in the 4 years of this study, only three stems had characteristic symptoms of the common soilborne virus *Wheat spindle streak mosaic virus* (WSSMV). These three stems tested positive for the presence of WSSMV by ELISA.

Below average disease incidence occurred in spring 2000 and 2001, with the average incidence across all genotypes being 4.6 and 9.6%, respectively. The plot

layout further complicated resistance analysis and comparisons in these first 2 years, because it was discovered later that all SWWW replications were planted together in a portion of the field that was shown to have low disease pressure. Thus, by not distributing replications throughout the field, it first appeared that more SWWW genotypes had high levels of resistance to SBWMV (average incidence of 1.6% in 2000 and 3.1% in 2001) when compared with the SRWW genotypes (6.6% in 2000 and 12.3% in 2001), which were located in a portion of the field with relatively high disease pressure. Strong disease pressure occurred in the springs of 2002 and 2003 (25.6 and 16.8% average incidence, respectively), although one portion of the field still had relatively low

SBWM incidence (Fig. 1, 2002 and 2003). Within our small nursery (30 by 15 m), we observed a gradient of incidence for our susceptible check from 17.5 to 100% in 2002 and from 0.0 to 100% in 2003.

Statistical analysis. Initially, only the last 2 years' data (2002 and 2003) were analyzed in order to understand how the disease gradient affected the results. A subset of 42 genotypes with at least 3 years' data was used to compare analyses from the first 2 years to the last 2 years. Analyzing ratio data (experimental genotype to nearest row of Harus) using a general linear model in SAS with Fisher's protected least significant difference for multiple comparisons analysis of square-root (ratio) resulted in wide intervals of nonsignificance with only two discrete resistance classes, as defined in Materials and Methods (Table 2). Use of a logistic regression model, which recognizes each plant as an observation instead of each row, resulted in narrower intervals and increased from two to four the number of discrete resistance classes (Table 2). To incorporate our knowledge of disease gradients in the field, we added the number of symptomatic Harus plants in the nearest row as a covariate reflecting disease gradient. This additional information provided for a slightly better statistical model by decreasing the width of the intervals but did not alter the number of discrete resistance classes (Table 2).

In order to compare data between the 2 years with low disease incidence and the 2 years with high disease incidence, we used a logistic model containing year and genotype as predictors. In the years with low incidence, only three discrete resistance classes could be discerned, and only 12 of 42 genotypes were significantly more susceptible than the most resistant genotype, compared with 16 genotypes under high disease incidence in the last 2 years (Table 2). Thus, the first 2 years' data gave the false impression that four genotypes were resistant or moderately resistant. Analysis of all 4 years together resulted in four discrete resistance classes and 18 genotypes being significantly more susceptible than the most resistant genotype (Table 2).

To determine whether small sample size reduced the power of resistance assessment, we analyzed the first 2 years' results after extrapolating our data to sample sizes of 20 instead of 30 for three replications over each of 2 years. This analysis does not change the percent incidence or relative susceptibility, but did result in wider intervals of nonsignificance and reduced the number of disease resistance classes from three to two (Table 2). Furthermore, the smaller sample size resulted in three moderately susceptible genotypes being classified as resistant.

Choice of statistical method, accounting for disease gradient, low disease incidence, and extra years of repetition, affected the

Fig. 1. Soilborne wheat mosaic virus (SBWM) distribution on 'Harus' wheat in spring 2002 (above) and 2003 (below). Point locations represent the middle of each row of the susceptible check Harus 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 SBWM. Both maps are oriented with south at the top.

Table 2. Comparison of statistical models and data sets using the same subset of cultivars

Model ^a	Years ^b	No. of DRC ^c	Average interval ^d	No. susceptible ^e
GLM	... 2002, 2003	2	17.7	16
Logistic	... 2002, 2003	4	13.4	16
Logistic, covariate ^f	... 2002, 2003	4	13.0	18
Logistic	2000, 2001 ...	3	15.2	12
Logistic, $n = 20^g$	2000, 2001 ...	2	20.4	9
Logistic	2000, 2001, 2002, 2003	4	11.5	18

^a Statistical model used for analysis, either general linear model (GLM) or logistic model. In both cases, the predictors used were genotype and year unless otherwise noted.

^b Year of the spring when incidence data was collected.

^c Number of discrete resistance classes (DRCs), as described in the Materials and Methods.

^d Average number of check cultivars not significantly different in soilborne wheat mosaic (SBWM) disease incidence than the test cultivar.

^e Number of cultivars (out of 42) that had significantly higher SBWM disease incidence than the cultivar with the lowest SBWM disease incidence.

^f For this analysis, the incidence of SBWM on the closest row of 'Harus' wheat was used as a covariate in the statistical model.

^g Data used in the analysis one line above was extrapolated from sample size of 30 to 20 and reanalyzed.

relative ranks (Table 3). The risks of drawing conclusions from only 2 years of data also are revealed in Table 3. For example, 'Superior' and 'Roane' wheat were rated

among the most resistant cultivars in the first 2 years of the study (under low disease pressure) but were rated among the more susceptible cultivars in the final 2 years.

Evaluating relative susceptibilities. Finally, we analyzed data from all 4 years for all genotypes using Proc Logistic (SAS) with the predictors 'year' and 'genotype' but not accounting for the disease gradient. The result suggests four significantly discrete resistance classes. Of the 115 genotypes tested, 41 were not significantly more susceptible than the most resistant check genotype, Caledonia (Fig. 2).

DISCUSSION

With SBWMV being recently introduced to Tompkins County, NY and spreading to surrounding counties, this study was essential for providing a control strategy for small grains producers in the region. We found that resistance is available in many cultivars adapted to the Northeast. These have been recommended to growers and adopted in areas at risk to infection by SBWMV. Cultivars should be tested in other locations or against other strains of SBWMV. The limited geographical infestation in New York probably is limited also in genetic diversity. Our data provide a large number of potential check cultivars for other environments.

This study also provided insights into the optimal conduct of field studies involving soilborne pathogens. Although the maps in Figure 1 reflect similar disease distributions in 2002 and 2003, their differences support the utility of a spatially balanced design, which increased the likelihood that multiple replications of all genotypes would encounter high disease pressure. The spatially balanced, randomized complete block design used in the final 2 years of the study was sufficient to account for the nonuniform disease distribution in our nursery. Statistical models that included disease distribution data provided no added benefit. However, when analyzing the logistic models with and without susceptible check cv. Harus as a covariate (Tables 1 and 2), the important point is interpreting which model could improve the accuracy of genotype classification where disease gradients confound the scoring. In this study, the two models led to the same conclusions about resistance of cultivars to SBWMV. Thus, we conclude that the simplest model is the best model in this case.

Different environments (years) changed not only the relative resistance ranks but also the number of cultivars in the highest resistance category. A previous study found that, in some years, in a subset of planting dates, resistant genotypes developed disease, resulting in significant genotype-planting date interactions (6). In the current study, moderately resistant cultivars appeared to be more resistant in years with low disease incidence. Given the spatial patchiness of virus or vector as well as the environmental sensitivity of transmission and symptom expression, reliable evaluation

Table 3. Relative ranks of genotypes for data sets and analyses represented in Table 2

Genotype ^a	GLM		Logistic		
	2002-03 ^b	2002-03 ^c	2002-03, covar. ^d	2000-01 ^e	2000-03 ^f
Elmira (barley)	6	6	6	23	10
McGregor (barley)	6	6	6	19	3
Aroostock (rye)	33	31	30	28	30
Presto (Tcl)	30	32	33	34	33
RSI 815 (Tcl)	17	16	9	23	16
RSI AZ 1998 (Tcl)	29	28	27	39	34
RSIBG 1998 (Tcl)	26	28	30	30	27
Trical 336 (Tcl)	35	30	27	40	35
Trical 815 (Tcl)	21	16	9	26	22
A94-1315	13	8	9	2	4
AC Ron	27	27	29	20	26
Bavaria	8	11	17	2	6
Caledonia	1	1	3	1	1
Cardinal	33	32	36	36	35
Cayuga	8	11	9	2	10
Foster	22	16	19	18	16
Freedom	37	36	39	36	39
Geneva	1	3	1	2	2
Harus	40	36	37	35	38
Hoffman 37	13	15	15	2	10
Houser	8	8	15	2	6
IL 91-15911	1	3	3	12	6
KY86C-61-8	1	3	3	12	4
Mendon	30	32	32	27	29
NYBatavia	19	19	24	12	23
OH 530	24	24	23	20	24
Patton	13	19	19	12	16
Pearl	19	19	22	2	16
Pioneer 2510	23	19	19	2	13
Pioneer 2540	8	14	6	31	27
Pioneer 2548	35	35	34	32	32
Pioneer 25R37	1	3	1	33	16
Pioneer 25W33	30	38	35	22	31
Roane	27	26	25	2	25
Sawyer	13	8	9	12	9
Sisson	38	38	38	42	42
Superior	25	25	26	2	21
TW97613	42	42	42	36	40
U-1273-5-18-8	8	11	9	25	13
Va 96W-247	40	40	40	41	40
Watford	38	41	41	28	35
Whitby	17	19	17	17	13

^a All genotypes are wheat unless otherwise noted; Tcl = triticale.

^b Relative resistance ranking (1 to 42, most resistant to most susceptible) resulting from analyzing genotype:Harus data as a general linear model (GLM).

^c Relative resistance ranking resulting from analyzing a logistic model without accounting for disease gradient, using the last 2 years' data.

^d Relative resistance ranking resulting from analyzing a logistic model while accounting for disease gradient with Harus as a covariate, using the last 2 years' data.

^e Relative resistance ranking resulting from analyzing a logistic model without accounting for disease gradient, using the first 2 years' data.

^f Relative resistance ranking resulting from analyzing a logistic model without accounting for disease gradient, using all 4 years' data.

of resistance requires compilation of multiple years of replicated data.

In field trials, sampling time often is compromised by weather or lack of resources, and researchers are forced to sample fewer individuals or fewer replicates. This was the case in spring 2002, when there was capacity to assess only two replications of 20 individuals ($n = 20$, $\text{rep} = 2$). In order to visualize the effect of this constraint, we reanalyzed the first 2 years of data ($n = 30$, $\text{rep} = 3$) after extrapolating the same percentages to smaller sample sizes but retained all three replications ($n = 20$, $\text{rep} = 3$). The results, shown in Tables 1 and 2, reflect but underestimate the loss of power using the smaller sample size in

2002. Of course, the sample size required is dependent upon the variance of disease incidence, but the sample size of 30 was significantly better than 20 for assessing SBWMV incidence data in this study.

Figure 2 may be interpreted to reflect four discrete clusters of response to SBWMV. These response clusters may have a quantitative genetic basis consistent with published findings that resistance in individual cultivars is conditioned by one to three genes (4,14,15,17,18,20). Although the current study was not designed to investigate the inheritance of resistance, the objective and quantitative nature of the method developed could be particularly useful in such studies.

Fig. 2. Relative resistance of 115 small grains genotypes to *Soilborne wheat mosaic virus* (SBWMV) 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 115 genotypes assessed for resistance to SBWMV and sorted from most susceptible (top) to most resistant (bottom). For readability, only 29 genotypes of interest are labeled. The x-axis represents 38 check genotypes sorted from most susceptible (left) to most resistant (right). Genotypes on the y-axis with overlapping bars did not have significantly different percent incidences of SBWM over the course of the study, relative to the check cultivars represented on the x-axis.

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