Heritability of Resistance in Winter Wheat to Wheat Spindle Streak Mosaic Virus

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Portion of a thesis submitted by the first author in partial fulfillment of the requirements for the M.S. degree, Michigan State University.
Michigan Agricultural Experiment Station Journal Series Article 12151.
We wish to thank Karen L. Klomparens, Director, Center for Electron Optics, for help with electron microscopy.
Accepted for publication 10 November 1986 (submitted for electronic processing).

ABSTRACT

Van Koevering, M., Haufler, K. Zagula, 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.

The heritability of resistance to wheat spindle streak mosaic virus in winter wheat was studied using a seven-parent diallel analysis. Parents and F_1 progenies were evaluated for disease reaction based on virus particle counts determined by immunosorbent electron microscopy. Resistance was found to be a highly heritable trait controlled by a few dominant genes. A two-locus genetic model was proposed to account for the observed resistance classification of the parents and the patterns of inheritance exhibited in the F_1 progenies.

One of the most prominent virus diseases of winter wheat in the United States and Canada is wheat spindle streak mosaic (WSSM). The disease was first reported in the United States in Michigan 15 yr ago (17), and since then it has been found in most winter wheat-growing regions (2,3,10–12,18).

Accurate yield loss data are somewhat limited, but estimates of crop loss because of WSSM range from 2 to 59% (2,6,13,16). Although a few older winter wheat cultivars show some resistance to wheat spindle streak mosaic virus (WSSMV) (10,12,13,16,18), most commercially grown cultivars are susceptible (2,8,12). Because recommended cultural practices such as crop rotation and late planting have not been effective in controlling the disease, it is important to identify germ plasm and to develop cultivars resistant to WSSMV.

Diallel crosses have been used to study the heritability of resistance to viruses in wheat (4,5) and other crops. In the analysis of winter wheat tolerance to barley yellow dwarf virus, heritability of tolerance was low, with additive genetic effects (high general-combining ability) more important than nonadditive and reciprocal effects in determining tolerance. Results from the analysis of a five-parent winter wheat diallel cross indicated that resistance to soilborne wheat mosaic virus was monogenic dominant over susceptibility (5). Although diallel analysis has some limitations (1), it does provide estimates of genetic parameters that serve as a basis for selection procedures used in a plant breeding program.

Haufler and Fulbright (8) recently identified several soft winter wheat cultivars and experimental breeding lines with resistance to WSSMV. Disease ratings were based on symptom expression and virus content of leaves using immunosorbent electron microscopy (ISEM) (7). Screening the germ plasm revealed differential cultivar reactions to WSSMV, ranging from susceptible to resistant. Because the genetic mechanisms controlling expression of resistance to WSSMV are unknown, a diallel mating design using parents with known differential reactions to the virus was used to study the mode of inheritance and gene action of resistance to WSSMV. A preliminary report of a portion of this work has been presented (15).

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MATERIALS AND METHODS

Three Michigan cultivars and four advanced experimental lines from the Michigan State University wheat breeding program, all showing differential reactions to WSSMV, were chosen as parents in a diallel mating design. The parental pedigrees, which originated from widely diverse genetic backgrounds, included both red and white soft winter wheat (Table 1). The seven parents were crossed in all possible combinations to produce 42 F_1 progenies. Preliminary analysis revealed nonsignificant maternal effects; therefore, reciprocal crosses were not included in the diallel analysis. The remaining 21 F_1 progenies and seven parents were evaluated for disease reaction based on numbers of virus particles in leaf tissue using ISEM (8).

Soil infested with the fungal vector *Polymyxa graminis* Led. was collected from a field in Saranac, MI, where wheat previously had shown severe WSSM symptoms. Four parts of infested soil were mixed with one part sterilized sand to increase soil aeration and drainage. Sterilized wooden flats were filled with the infested soil mixture, and seeds from the 21 F_1 progenies and seven parents were planted in a randomized block design in rows with five seeds per row. Each flat represented a single replication and contained 30 entries, including 7 parental rows, 21 F_1 progeny rows, and 2 control rows of a known susceptible cultivar (Ionia). Three replications were planted in November 1983. Seeds were germinated in the greenhouse, and seedlings were kept at 20 ± 3 C

TABLE 1. Winter wheat cultivars and lines used as parents in diallel examining inheritance of resistance to wheat spindle streak mosaic virus

Experimental or CI No.	Common name	Origin	Kernel color	Reaction[a]
CI 14469	Ionia	USA	White	S
CI 17831	Augusta	USA	White	S
CI 17287	Tecumseh	USA	White	MS
B4145	Experimental line	USA, New Zealand	White	MR
B7321	Experimental line	Russia	Red	MR
B6018	Experimental line	USA, Japan	White	R
B9028	Experimental line	Yugoslavia, Mexico	Red	R

[a] S = susceptible, MS = moderately susceptible, MR = moderately resistant, and R = resistant. Disease rating (reaction) is based on virus particle counts as described previously (8).

for 20 days. Flats were then placed in an outdoor cold frame to vernalize for 3 mo. In February 1984, the vernalized plants were transferred to a growth chamber with a 10-hr photoperiod of 135 μE $m^{-2}s^{-1}$ and average day/night temperatures of 10/8 C (14).

Plants were allowed to grow for 2 wk, after which one of the three replicates was sampled every 3 wk and prepared for ISEM. Possible statistical variation because of time of sampling was partitioned by replications in a randomized block design. Each entry was sampled three times during a 9-wk period. Only lower leaves from randomly chosen plants were harvested to maintain uniform sampling procedure. Because plants differed in the amounts of leaf tissue during the early sampling periods, a ratio of 0.15 g of leaf tissue per 1.0 ml of 0.06 M sodium phosphate buffer (pH 7.0) was used for all samples. Virus was extracted from harvested leaves and ISEM was performed as described previously (7). An estimate of the total number of virus particles per sample was made by averaging numbers of particles observed on 10 randomly chosen 300-mesh grid squares.

Virus particle counts were averaged over replications and analyzed according to Hayman's additive-dominance diallel model (9) using the log transformation $y = \log(x+1)$. Components of the model include additive variance (D), dominance variance (H_1), and the covariation of the additive and dominance effects (F). Parental array variances (V_r), covariances (W_r), and their differences ($W_r - V_r$) were calculated from the results shown in Table 2; these main statistics were used to test the adequacy of the proposed additive-dominance model. A covariance-variance graph was constructed by plotting the parental array values of covariances for all observed variances.

RESULTS AND DISCUSSION

WSSMV particle counts for parents and F_1 progenies ranged from 0 to nearly 400 particles per grid square (Table 2). Plants with counts of > 70 particles per grid square were considered susceptible to WSSMV, those with counts between 21 and 70 particles were rated moderately susceptible, those with 1–20 particles were rated moderately resistant, and those with 0 particles were rated resistant to WSSMV (8). F_1 progeny resulting from crosses between susceptible parents (as in Augusta × Ionia) were also susceptible to WSSMV based on virus particle counts (Table 2), whereas crosses between resistant parents (as in B7321 × B6018) produced resistant F_1 progeny with few to no virus particles. Crosses between susceptible and resistant parents (as in Augusta × B9028) produced resistant F_1 progeny, whereas crosses between parents with intermediate reactions to WSSMV (as in Tecumseh × B7321) also produced resistant F_1 progeny. These data indicate that genes for resistance to WSSMV are dominant.

The preliminary analysis of variance of parents and F_1 progenies showed highly significant differences between genotypes (*data not shown*). These results were expected because the parents were chosen for their differential reactions to WSSMV. Broad-sense heritability for resistance to WSSMV was very high (0.98), indicating that variation was the result of genetic rather than environmental factors. There were no significant differences between replications.

The products of the main statistics and their corresponding multipliers (9) were used to calculate the components of variation and their standard errors (Table 3). Both additive variance (D) and overall dominance variance (H_1) contributed significantly to the overall genetic variance as indicated by estimates approaching a value of 1.0. In addition, $D - H_1$ was not significantly different from zero, indicating nearly complete dominance. The negative F value for WSSMV resistance indicated that a greater frequency of recessive alleles was found among the parental arrays. Also, the mean degree of dominance was $\sqrt{H_1/D} = 1.0266$, indicating nearly complete dominance (a value of 1.0 represents complete dominance).

Based on the regression of covariance on variance for the parental arrays (Fig. 1), the pattern of inheritance of resistance to WSSMV was one of complete dominance. An order of dominance among the parental arrays was apparent: those parents nearest the origin of the line (B6018, B9028, and B4145) contained a greater proportion of dominant alleles for resistance to WSSMV, whereas

TABLE 2. Virus particle counts for parents and F_1 progeny in diallel analysis of resistance to wheat spindle streak mosaic virus[a]

Parental number and name	1	2	3	4	5	6	7
1 Augusta	99						
2 Ionia	183	360					
3 B4145	6	3	11				
4 Tecumseh	145	225	3	16			
5 B6018	4	3	0	1	1		
6 B7321	62	78	1	8	1	19	
7 B9028	1	4	2	0	1	0	0

[a] Particle counts are means per grid square obtained by examining 60 grid squares for each parent and F_1 progeny.

TABLE 3. Components of variation and their standard errors for diallel analysis of resistance to wheat spindle streak mosaic virus

Notation[a]	Estimate	Standard error	P
D	0.7681	±.0439	<.001
F	−0.2351	±.1054	.05-.01
H_1	0.8095	±.1058	<.001
$\sqrt{H_1/D}$	1.0266	±.0918	>.10

[a] D = additive variance, F = covariance of additive and dominance effects, and H_1 = dominance variance.

TABLE 4. Genic model proposing allelic combinations to represent phenotypic variation in parental and F_1 progeny reactions to wheat spindle streak mosaic virus

	$A_1A_1B_1B_1$	$A_1A_1B_2B_2$	$A_2A_2B_1B_1$	$A_3A_3B_1B_1$	$A_2A_2B_2B_2$	$A_3A_3B_2B_2$
$A_1A_1B_1B_1$	1[a]					
$A_1A_1B_2B_2$	1	11				
$A_2A_2B_1B_1$	1	1	19			
$A_3A_3B_1B_1$	1	3	8	16		
$A_2A_2B_2B_2$	2	6	62	145	99	
$A_3A_3B_2B_2$	6	3	78	225	183	360
Parents	B6018 B9028	B4145	B7321	Tecumseh	Augusta	Ionia

[a] Numbers are virus particle counts as reported in Table 2.

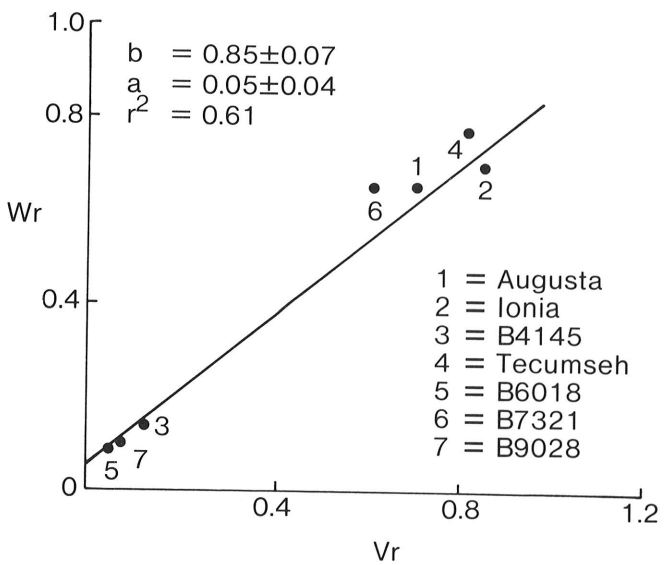

Fig. 1. Regression of parental array covariances (W_r) on variances (V_r). Three parents clustered nearest origin are similar in reaction to wheat spindle streak mosaic virus, whereas four farthest from origin also show similar reactions to virus. Slope is significant at $P < 0.01$.

parents farthest from the origin (B7321, Tecumseh, Augusta, and Ionia) contained a greater proportion of recessive alleles and were more susceptible to WSSMV. The coefficient of determination (r^2) was 0.61, indicating that genes for resistance to WSSMV were mostly dominant.

The seven parents used in the diallel fell into six distinct classes based on virus particle counts. A two-locus genetic model is proposed to account for this classification of parents and the patterns of inheritance exhibited in the F_1 progenies based on the virus particle count data (Table 4). The model has three alleles (A_1, A_2, and A_3) at the A locus and two alleles (B_1 and B_2) at the B locus. The A_1 allele is completely dominant to A_2 and A_3 and codes for resistance to WSSMV. Thus, parents with this allele (such as B6018 and B9028) show virus resistance. The A_2 and A_3 alleles show additive gene action, so that parents with the A_3 allele (such as Ionia) are more susceptible than parents containing the A_2 allele (such as B7321). The B locus also exhibits additive gene action, so that parents with homozygous B_2 alleles (such as Ionia and Augusta) are more susceptible to WSSMV than parents with homozygous B_1 alleles. The heterozygote B_1B_2 lies nearly midway between homozygous parents, expressing additivity. In addition, there is an additive by additive epistatic interaction between locus A and B in the absence of the A_1 allele.

Resistance to WSSMV appears to be a highly heritable trait controlled by a few dominant genes with some additive effect. Based on the proposed model, cultivars with A_1 or B_1 alleles show some resistance to WSSMV. This model is currently being tested by analyzing the segregating F_2 and F_3 generations. Genes for resistance to WSSMV are presently being incorporated into commercial wheat cultivars through our wheat breeding program.

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