Inheritance of Resistance to Maize Dwarf Mosaic Virus in Maize Inbred Line Oh7B

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ABSTRACT

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The inheritance of reaction to maize dwarf mosaic virus (MDMV) was studied in progenies derived from crosses between the resistant maize inbred line Oh7B and two susceptible lines, Oh43 and Pa91. Field-grown seedlings at one- to three-leaf stage were mechanically inoculated with a johnsongrass isolate of MDMV; no natural inoculum was present. Reactions of parental, F_1, F_2, and F_3 plants at the postanthesis stage were scored on a scale of 1–7. Scores of 1–4 were considered resistant, and 6 and 7 were susceptible. Class 5 plants could be considered either resistant or susceptible, but from our analyses we concluded that they belong in the resistant class. The F_3 progenies were from randomly selected, classified F_2 plants. Three procedures were used to interpret genetic control of MDMV reactions: direct classification of F_2 plants, determining F_2 phenotypes from mean scores of F_3 rows, and determining F_2 genotypes from inspection of scores of individual F_3 plants. The three methods gave similar segregation ratios. We conclude that reaction to MDMV in Oh7B is conditioned by one dominant gene.

Additional key words: corn viruses, fate map, maize chlorotic dwarf virus, reaction classes.

Several studies on the inheritance of resistance of maize (*Zea mays* L.) to maize dwarf mosaic virus (MDMV) have been reported, but in most cases, naturally occurring johnsongrass (*Sorghum halepense* (L.) Pers.) was the primary source of inoculum (2,6,8,9,11). In those reports, the recorded reactions to MDMV were probably confounded with reactions to maize chlorotic dwarf virus (MCDV), which usually occurs as a coinfecting virus of johnsongrass and which spreads to maize, producing mixed infections with MDMV (1,12). Symptoms of severe stunting, sterile ears, and striking yellowing or reddening, which are characteristic of maize infected naturally where johnsongrass is present, cannot be reproduced when maize is artificially inoculated with MDMV (5). Thus, studies on inheritance of resistance to MDMV conducted prior to 1972 with field-grown maize are of questionable genetic value, even though they may be very useful in maize-breeding because they probably reflect reactions of maize lines to both MDMV and MCDV. Under these conditions, resistance to MDMV has been reported to be inherited oligogenically or polygenically with the estimated number of genes for resistance varying from one to 10 in a single inbred (2,3). Other workers reported only that reaction to MDMV was under genetic control, that resistance was partially dominant to dominant, or that relatively few major genes conditioned resistance (2,6,8,9,10). Dollinger et al (2) reported that inbred Oh43 contributed a susceptible gene, and we stated (5) that the intermediate resistance of inbreds Va-LE8 and T8 was recessive to the susceptibility of Pa91 and Oh43.

Recognition of MCDV has made possible more definitive genetic studies of reaction of maize to viruses. Findley et al (4) concluded that inbred Oh07 has one dominant gene that conditions resistance to MDMV strains and that Pa405 has two dominant genes that condition resistance to MDMV strain B when progenies are mechanically inoculated, but only one dominant gene that conditions resistance when progenies are aphid inoculated. They also found that Pa405 is monogenic for resistance to strains A, D, E, and F of MDMV. Roane et al (14) reported that one dominant gene was present in inbred Oh7B and that the results obtained with inbred T8 could not be explained simply. A more critical analysis of the inheritance of resistance to MDMV in Oh7B is presented here. The problems inherent in genetic interpretation of the MDMV-maize interactions are discussed.

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MATERIALS AND METHODS

Based on our previous experience with inbred lines and F_1 hybrids (5), we chose to study inheritance in crosses between the resistant inbred Oh7B and the susceptible inbreds Oh43 and Pa91. Only rarely does Oh7B show any response to MDMV; Oh43 and Pa91 develop almost completely susceptible responses. Crosses among these inbreds were made during the winter of 1972 in a Florida breeding nursery; F_1 plants were grown and selfed in a nursery near Blacksburg, VA, in the summer of 1972. In 1973, a planting of parental lines, F_1, and F_2 was grown, inoculated in the one- to three-leaf stage with MDMV, and each plant was scored for reaction to virus. Numerous randomly selected F_2 plants were selfed, and at harvest the MDMV score of each harvested plant was recorded on the seed envelope. In 1974, 34 seeds from each selfed F_2 ear were planted, and all plants were inoculated as before. Thus, the individual F_3 plant scores were recorded and, accordingly, the score of the F_2 plant, which produced the F_3 seed, was also known. There was no johnsongrass, and there had been no known natural occurrence of MDMV or MCDV in the nursery area.

Virus for inoculations was produced and prepared as described by Jones and Tolin (7). The MDMV had been maintained in a plant of johnsongrass grown from seed and was presumed at the time to be strain A. Inoculations of plants at the one- to three-leaf stage of growth were made with artist's airbrushes (5). A new scale, necessitated by a need to describe numerically the response of each plant, was devised for scoring reactions to MDMV. The scale is illustrated in Fig. 1; all plants were classified after anthesis. In this scale, plants in classes 1–4 were undoubtedly resistant, those in class 5 could be considered either resistant or susceptible, and those in classes 6 and 7 were susceptible.

RESULTS

Responses of parental lines to MDMV are shown in Table 1. Inbred Oh7B was immune to MDMV, as there were no infected plants. Inbred Oh43 had six (8%) uninfected plants, and Pa91 had one (1%). Apparently, these were escapes because in most previous experiences with these lines, all plants had shown some evidence of

infection. The mean scores of Oh43 and Pa91 clearly place them in the susceptible category.

In F_1 hybrids (Table 1), the dominance of resistance from Oh7B was nearly complete in combination with Pa91, and somewhat less complete with Oh43. The three plants of Pa91 × Oh7B in reaction class 7 may have come from selfed seeds, since Pa91 was the seed parent. Likewise, the four plants of Oh43 × Oh7B in reaction classes 6 and 7 could have been selfs of Oh43. However, in a population of F_1 plants, inbred plants would have been recognized by their lack of vigor. Inbreds Oh43 and Pa91 combined to give a mean nearly equal to the parental midpoint of 6.6. The eight plants in reaction class 1 suggest that some susceptible plants in all crosses may have escaped infection.

Although F_1 dominance relations are important to the maize breeder, segregation in F_2 and F_3 generations is vital to genetic analysis. The behavior of the F_2 progenies in this study was examined from three approaches: by classifying plants as uninfected or infected, by scoring F_2 plants and placing them in phenotypic classes, and by random selfing among scored F_2 plants and classifying a sample of F_3 progeny from each selfed plant (Table 2). By classifying the entire F_2 of Oh43 × Oh7B and Pa91 × Oh7B into uninfected vs infected (class 1 vs classes 2–7), a fit to the 9:7 ratio ($P > 0.3$) was obtained. This is the procedure followed by Findley et al (4), but it is traditional to recognize low and high infection types and to partition the data accordingly. Any suppression of symptom expression would be an indication of some mechanism for resistance; therefore, it seems more plausible to group the data into the classes 1–4:5–7 or 1–5:6–7. For either of these groupings, the entire F_2 of Oh43 × Oh7B fits a 3:1 ratio only for the 1–5:6–7 grouping, and the entire F_2 of Pa91 × Oh7B deviates slightly from a 3:1 ratio for all groupings (Table 2).

When we selfed the F_2 plants and classified the F_3 progenies from each, we obtained a better fit to the 3:1 ratio, but for these data to be meaningful, the population of selfed F_2 plants should be representative of the entire F_2 generation. For both crosses, the χ^2 tests show that they were representative of the entire F_2 generation ($P > 0.8$). For the selfed F_2 of both crosses, the best fit was for a 3:1 ratio with classes grouped into 1–5:6–7 (Table 2); however, the populations fitted the 3:1 ratio with class 5 placed in either phenotype.

The F_2 parents can be classified by inspection of the class frequencies observed in their F_3 rows as homozygous low infection type, heterozygous, and homozygous high infection type. In addition, a mean score can be calculated for each row. A low infection type would produce a mean score <4.5 or <5.5, depending on the grouping employed. Calculation and interpretation of the mean score require no judgment. A fit to a 3:1 ratio was obtained for both crosses, but the different groupings gave reciprocal high P values for the Oh43 × Oh7B cross (Table 3). For mean row scores to be useful, the scores given to F_2 parent plants should be highly correlated with the mean scores of F_3 rows. Although significant correlations were obtained from each cross, the values for r were not impressive (Table 3). Upon examination of individual F_3 classification data, we found that the cause of low r values was the disagreement between F_2 and F_3 results for about one third of the plants examined (Table 4).

For the cross Oh43 × Oh7B, some F_2 plants given a score of 1 or 2 did not segregate in F_3 as expected (Table 4). Plants in class 1 could be homozygous for low reaction or could be heterozygous; 18 F_3 rows were classified as homozygous 1–4, and 18 were heterozygous. Twelve class 1 F_2 plants behaved as if they should have been classified as 6–7 (plants 38–49, Table 4). Plant 37 (Table 4) gave an F_3 mean <4.5 but by inspection appeared susceptible. One class 6 and 13 class 7 F_2 plants (plants 60–73, Table 4) yielded progenies that behaved as if they should have been classified as 1–4. This accounts for the relatively low correlation coefficient (r = 0.268, Table 3). The agreement between classification by mean scores and by inspection of class frequencies in F_3 suggests that either of these methods was more reliable for classifying F_2 plants than was direct classification of them (Table 4). From the inspection method for R:H:S, 1:2:1, $\chi^2 = 0.146$, $P > 0.9$; from the mean score for R:S, 3:1, $\chi^2 = 0.016$, $P > 0.9$ or 1.317, $P > 0.2$, depending upon the category in which class 5 is placed (Tables 3 and 4).

For the cross Pa91 × Oh7B, nine F_2 plants in classes 1 and 2 (plants 38–45, 47; Table 5) did not segregate as expected in the F_3 and five class 7 F_2 plants (plants 53–57, Table 5) segregated as if they should have been classified 1–4 (Table 5). Thus, fewer plants showed segregations inconsistent with their reaction classes in this cross than in Oh43 × Oh7B, and therefore, the correlation coefficient was larger (r = 0.564). The agreement between classification by mean scores and by inspection of class frequencies in F_3 is close and again appears to be reliable (Table 5). For R:H:S,

TABLE 1. Frequency of plants of parental maize lines and F_1 progeny in seven classes of reaction to maize dwarf mosaic virus

Inbred or hybrid	Reaction class and frequency[a]									Parental midpoint
	1	2	3	4	5	6	7	N	$\bar{X}$	
Oh7B	98[b]	0	0	0	0	0	0	98	1.0	
Oh43	6[b]	0	0	0	7	2	61	76	6.3	
Pa91	1[b]	1	0	0	0	7	88	97	6.8	
Oh43 × Oh7B	69[b]	0	8	13	0	1	3	94	1.8	3.7
Pa91 × Oh7B	90[b]	0	0	0	0	0	3	93	1.2	4.9
Oh43 × Pa91	8[b]	0	0	0	0	1	89	98	6.5	6.6

[a]See Fig. 1 for description of reaction classes.
[b]Number of plants in each class; N = total number of plants.

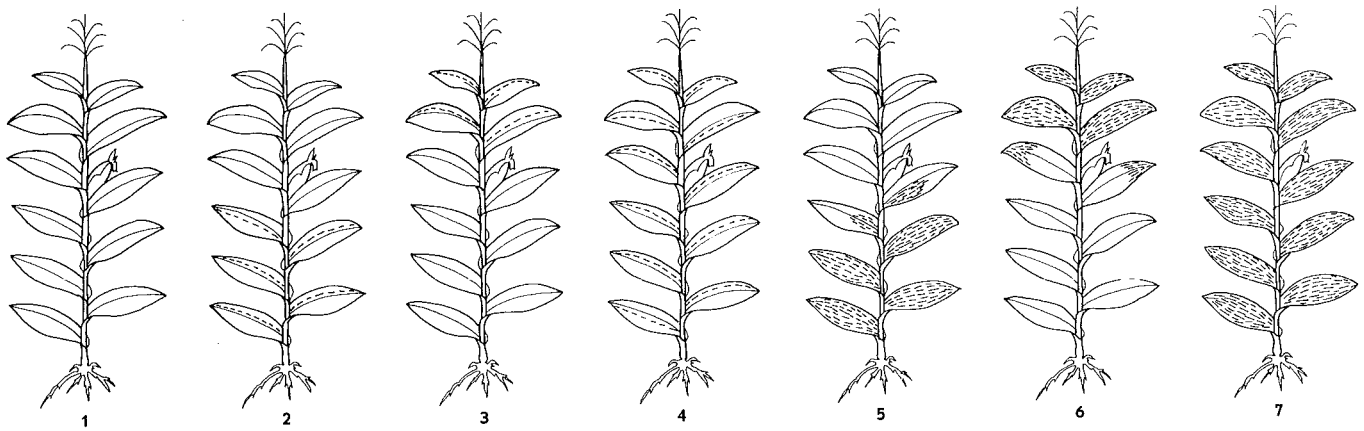

Fig. 1. Classes of responses of maize to maize dwarf mosaic virus: **1,** no response; **2,** lower leaves with a narrow band of mottled tissue; **3,** upper leaves with a narrow band of mottled tissue; **4,** most or all leaves with a narrow band of mottled tissue; **5,** lower leaves mottled with typical symptoms of MDM; upper leaves appearing healthy; **6,** upper leaves mottled with typical symptoms, lower appearing healthy; presumed to have escaped early infection; **7,** all leaves mottled with typical symptoms. Classes 1–4, resistant; class 5, probably resistant; and classes 6 and 7, susceptible.

1:2:1, from the inspection method $\chi^2 = 4.91$, $P > 0.05$; from the mean score R:S, 3:1, $\chi^2 = 3.52$, $P > 0.05$, or 1.21, $P > 0.2$, depending upon the category in which class 5 is placed (Tables 3 and 5).

DISCUSSION

When this study was initiated, most inheritance studies in maize with MDMV had been made in fields infested with johnsongrass or were based upon reactions of F_1 plants (2,6,8,9,11). Conventional genetic techniques had been applied in only one study (2), but that study was confounded by the presence of MCDV. There was a need for further investigation of Mendelian inheritance of resistance to MDMV. As our work was under way, Findley et al (4) reported a study apparently conducted in the absence of MCDV, in which they inoculated plants by a method similar to ours. Resistance to MDMV strain A in each of the inbreds Oh07 and Pa405 was conditioned by a single dominant gene. However, they observed a continuing increase in the percentage of infected plants for three dates of observations from 12 August to 9 September. Their genetic analysis of inbred Oh07 following mechanical inoculation appears valid only for the 12 August observations. In our study, no changes in response to virus infection were observed after anthesis. Findley et al (4) apparently based their conclusions upon the percentage of infected plants and gave no consideration to the different types of response among infected plants. In an earlier report, Dollinger et al (2) had employed a rating scale in which the effects of MDMV and MCDV were confounded. Because a variety of responses to infection by both MDMV and MCDV have been observed, it is difficult to separate with certainty the effect of one virus in a mixed infection. Scott and Rosenkranz (15) studied the inheritance of reaction to Mississippi corn stunt (which may have been MCDV) and to MDMV in experiments whereby they inoculated with MDMV but relied upon natural inoculation for stunt. They were unable to fit these data to known genetic ratios for either disease. Since others have reported that one to several genes may condition the reaction to MDMV or a virus complex (3,4,9,11,13), data were needed from a study where natural inoculum would not interfere and where artificial inoculation followed by mature plant expression of reactions would allow careful plant classification among segregating populations. A new scale was needed to describe plant responses only to MDMV on a numerical basis. The scale we devised (Fig. 1) may provide a tool or the impetus needed to separate genetic differences.

In studying inheritance of virus-host interactions, it is important to classify F_2 plants for response to virus infection and to record the reactions of their F_3 progenies. We followed such a procedure. Although it is neither difficult nor expensive to grow, inoculate, and adequately classify maize F_2 populations, the logistics of providing for adequate F_3s are very limiting. Therefore, it was important to demonstrate that the F_2 plants we selfed were representative of the entire F_2 population. We achieved this in both crosses studied (Table 2). Thus, we expected that any conclusions drawn from F_3 data should substantiate those drawn from F_2 data.

The segregation ratios obtained by classifying F_2 plants (Table 3), by classifying F_3 plants in order to determine F_2 phenotypes from mean scores of F_3 rows, and by inspection of the data from individual F_3 plants to determine the genotypes of individual F_2 plants (Tables 3–5) were all similar and mutually supportive. These data suggest that the reaction to MDMV in inbred Oh7B is conditioned by one dominant gene. However, some F_2 plants, which were placed in reaction class 1, produced F_3 progenies classified mostly in reaction classes 6 and 7, and some F_2 plants in class 7 produced F_3 progenies mostly in classes 1–4 (Tables 4 and 5). The frequencies of these reversals had a balancing effect and thus did not disturb the 3:1 ratio, but they caused the correlation coefficients to be small (Table 3). They also caused us to conclude that greater reliance should be placed upon data from F_3 plants whether one

TABLE 2. Observed frequencies of entire F_2 progeny and selfed F_2 progeny[a] and expected (Exp.) frequencies of selfed F_2 progeny in seven classes of reaction of maize to maize dwarf mosaic virus

Pedigree	Reaction classes[b] 1	2	3	4	5	6	7	N	P value for χ^2 with classes grouped: 1:2–7	1–4:5–7	1–5:6–7	Ratio tested (R:S)
Oh43 × Oh7B												
Entire F_2	93	0	2	6	9	1	44	155	<0.01	>0.01	>0.2	3:1
Selfed F_2	49	0	2	5	3	1	22	82	<0.01	>0.1	>0.7	3:1
Exp. selfed F_2 $\chi^2 = 2.997$ $P > 0.8$[c]	49.2	0	1.1	3.2	4.8	0.5	23.3	82				
Pa91 × Oh7B												
Entire F_2	89	1	0	2	9	3	46	150	<0.01	<0.01	>0.02	3:1
Selfed F_2	46	1	0	0	4	1	17	69	>0.1	>0.1	>0.8	3:1
Exp. selfed F_2 $\chi^2 = 3.046$ $P > 0.8$[c]	40.9	0.5	0	0.9	4.1	1.4	21.2	69				

[a] F_2 Plants for self pollination were randomly selected.
[b] See Fig. 1 for description of reaction classes.
[c] Probability that Selfed F_2 is representative of Entire F_2.

TABLE 3. Direct classification of selfed F_2 maize plants for reaction to maize dwarf mosaic virus, classification of selfed F_2 as determined from mean scores of F_3 rows, and correlation of direct classification scores of F_2 plants with mean scores of their F_3 rows

Pedigree	Direct classification of selfed F_2[a] Class grouping	Frequency	Ratio tested	P	Classification of selfed F_2 as determined from mean scores of F_3 rows Class grouping	Frequency	Ratio tested	P	Correlation of selfed F_2 plant scores with mean scores of F_3 rows (r)
Oh43 × Oh7B	1–5:6–7	59:23	3:1	>0.5	1–5.5:5.5–7	66:16	3:1	>0.2	
	1–4:5–7	56:26	3:1	>0.1	1–4.5:4.5–7	61:21	3:1	>0.9	
									0.268*[b]
Pa91 × Oh7B	1–5:6–7	51:18	3:1	>0.8	1–5.5:5.5–7	53:16	3:1	>0.7	
	1–4:5–7	47:22	3:1	>0.1	1–4.5:4.5–7	45:22	3:1	>0.05	
									0.564**[b]

[a] See Fig. 1 for description of reaction classes.
[b] * = statistically significant, ** = statistically highly significant.

uses mean row scores or inspection of reaction classes of individual plants. The reversal of class 1 F_2 plants to class 7 in F_3 plants might be explained by assuming that some class 1 F_2 plants escaped infection. On that basis, there should have been more class 6 plants; ie, those that escaped artificial inoculation but were later inoculated by aphids and thus mottled only in the upper leaves. Only one class 6 plant was observed in each F_2, but such plants were common in F_3. It is also unlikely that the inoculation process was highly efficient for most plants but inefficient for specific ones, since susceptible parental lines were infected at high percentage levels. There is no apparent explanation for the reversal of class 6 and 7 F_2 plants to class 1 in F_3. It is ironic that if we had reported only F_2 and F_3 segregations without following scored F_2 plants into the F_3 generation, we would have had no trouble explaining our data.

Reaction class 5 is an enigma. A plant that is apparently developing into a class 7 stops developing symptoms, and no virus can be detected in its asymptomatic leaves. Does such a plant belong in the resistant or susceptible group of reaction classes? Seven class 5 plants were observed in the F_2 populations (Tables 4 and 5). Plant 57 (Table 4) produced progenies characteristic of a homozygous class 1 parent. Plants 58 and 59 (Table 4) and plants 48 and 49 (Table 5) were heterozygous and therefore should have been in classes 1–4, but plants 50 and 51 (Table 5) were more like class 6–7 plants in the reactions of their F_3 progenies. Where to place class 5 plants remains unsolved; fortunately, class 5 plants are infrequent. Perhaps class 5 is comparable to the mesothetic (X) infection type of rusts. We did not monitor environmental factors, but the temperature or light conditions may have been so unfavorable for a brief period that the virus could not replicate in certain plants.

Reaction classes 2–4 are also enigmas. Obviously, the ontogeny of a grass plant is different from that of a dicot. How can a linear band of virus-infected tissues exist in a leaf of an otherwise virus-free plant, and what restricts the virus to a band in that leaf and subsequent leaves? Possibly, a somatic mutation for susceptibility occurs in the meristem too late for it to produce susceptible tissue for all subsequent leaf-blade tissue. Such an hypothesis could be consistent with the "fate-map" concept (16), whereby "Each of the meristem derivatives functioning as a leaf progenitor cell will be responsible for producing all of the blade tissue in a sector running the length of the leaf and occupying (on the average) 1/32 of the leaf width." This hypothesis may explain some of the striping we observed, but the mutation often must involve the meristem for more than one leaf. Most of the plants that striped (classes 2–4) were heterozygous. In F_1 progeny of Oh43 × Oh7B, 21 (22%) plants

TABLE 4. Classification of selfed Oh43 × Oh7B F_2 maize plants for reaction to maize dwarf mosaic virus and classification of their progenies in F_3 rows

F_3 row no.	F_2 plant score[a]	Reaction classes[b] 1	2	3	4	5	6	7	N	Mean score	Classification by: Mean score[c]	Inspection[d]
1	1	24	0	0	0	0	0	0	24	1.0	R	R
2	1	24	0	0	0	0	0	0	24	1.0	R	R
3	1	27	0	0	0	0	0	0	27	1.0	R	R
4	1	28	0	0	0	0	0	0	28	1.0	R	R
5	1	30	0	0	0	0	0	0	30	1.0	R	R
6	1	32	0	0	0	0	0	0	32	1.0	R	R
7	1	33	0	0	0	0	0	0	33	1.0	R	R
8	1	20	0	1	0	0	0	0	21	1.1	R	R
9	1	24	0	0	1	0	0	0	25	1.1	R	R
10	1	26	0	1	0	0	0	0	27	1.1	R	R
11	1	30	0	0	1	0	0	0	31	1.1	R	R
12	1	31	0	1	0	0	0	0	32	1.1	R	R
13	1	26	0	0	2	0	0	0	28	1.2	R	R
14	1	29	0	0	2	0	0	0	31	1.2	R	R
15	1	23	0	3	1	0	0	0	27	1.3	R	R
16	1	28	0	1	2	0	0	0	31	1.3	R	R
17	1	29	0	1	2	0	0	0	32	1.3	R	R
18	1	29	0	0	0	0	0	2	31	1.4	R	H
19	1	22	0	0	0	0	2	1	25	1.6	R	H
20	1	22	1	6	1	1	0	0	31	1.7	R	R
21	1	24	0	0	0	0	3	1	28	1.8	R	H
22	1	21	0	0	0	0	0	4	25	2.0	R	H
23	1	23	0	0	1	0	0	5	29	2.1	R	H
24	1	27	0	0	0	0	4	3	34	2.1	R	H
25	1	23	0	0	0	0	1	5	29	2.2	R	H
26	1	18	0	0	0	0	0	5	23	2.3	R	H
27	1	14	0	0	0	0	1	4	19	2.5	R	H
28	1	23	0	0	0	0	2	6	31	2.5	R	H
29	1	22	0	0	0	0	2	9	33	2.9	R	H
30	1	15	0	0	1	1	0	7	24	3.0	R	H
31	1	19	0	0	0	0	1	11	31	3.3	R	H
32	1	18	0	0	0	0	1	11	30	3.4	R	H
33	1	19	1	0	1	0	0	13	34	3.4	R	H
34	1	16	0	1	1	0	1	10	29	3.4	R	H
35	1	13	0	2	2	0	1	9	27	3.6	R	H
36	1	13	0	1	2	0	1	12	29	3.9	R	H
37	1*	10	0	0	0	4	0	10	24	4.2	R	S
38	1*	9	0	0	1	0	1	12	23	4.5	S	S
39	1*	10	0	0	4	0	1	16	31	4.6	S	S
40	1*	7	0	1	3	0	0	16	27	5.0	S	S
41	1*	6	0	1	2	0	1	16	26	5.2	S	S
42	1*	6	0	0	0	0	1	15	22	5.3	S	S
43	1*	5	0	0	0	0	1	14	20	5.5	S	S
44	1*	3	0	0	0	0	3	21	27	6.2	S	S
45	1*	3	0	0	0	0	0	24	27	6.3	S	S
46	1*	2	0	0	0	0	0	28	30	6.6	S	S
47	1*	1	0	0	0	0	0	19	20	6.7	S	S
48	1*	1	0	0	0	0	2	21	24	6.7	S	S
49	1*	0	0	0	0	0	0	16	16	7.0	S	S
50	3	26	0	1	0	0	1	2	30	1.6	R	H
51	3	18	0	4	1	1	0	5	29	2.6	R	H
52	4	24	0	3	3	0	0	0	30	1.5	R	R
53	4	11	1	2	9	1	1	1	26	2.8	R	H
54	4	20	0	0	0	0	1	11	32	3.2	R	H
55	4	15	1	0	2	0	0	11	29	3.5	R	H
56	4	12	0	0	5	1	0	10	28	3.8	R	H
57	5*	31	0	0	0	0	0	0	31	1.0	R	R
58	5	15	1	3	1	0	0	4	24	2.5	R	H
59	5	10	1	0	0	0	0	7	18	3.4	R	H
60	6*	8	0	1	1	0	0	7	17	3.8	R	H
61	7*	19	0	1	3	0	0	3	26	1.7	R	H
62	7*	21	0	0	0	0	0	3	24	1.8	R	H
63	7*	23	0	1	0	0	0	4	28	1.9	R	H
64	7*	23	0	1	0	0	0	6	30	2.3	R	H
65	7*	21	0	0	0	0	1	5	27	2.3	R	H
66	7*	20	0	0	0	0	0	6	26	2.4	R	H
67	7*	19	0	1	0	0	1	6	27	2.6	R	H
68	7*	14	0	0	2	0	0	7	23	3.1	R	H
69	7*	15	0	0	4	0	1	7	27	3.2	R	H
70	7*	15	0	2	1	0	0	13	31	3.7	R	H
71	7*	15	0	0	1	0	0	13	29	3.8	R	H
72	7*	10	2	0	5	1	1	11	30	4.1	R	H
73	7*	8	0	1	1	0	0	10	20	4.3	R	H
74	7	4	0	0	0	0	2	14	20	5.7	S	S
75	7	5	0	0	0	1	1	21	28	5.8	S	S
76	7	4	0	0	0	0	3	25	32	6.2	S	S
77	7	2	0	0	0	0	4	19	25	6.4	S	S
78	7	1	0	0	0	0	0	22	23	6.7	S	S
79	7	0	0	0	0	0	0	18	18	7.0	S	S
80	7	0	0	0	0	0	0	26	26	7.0	S	S
81	7	0	0	0	0	0	0	27	27	7.0	S	S
82	7	0	0	0	0	0	0	32	32	7.0	S	S

[a] * = rows in which the distribution disagreed with the F_2 classification.

[b] For a description of reaction classes, see Fig. 1.

[c] For rows with mean scores >4.5, it was judged that the F_2 plant should have been classified susceptible; such F_2 plants rated 1 may have escaped infection.

[d] For rows with more plants in classes 5–7 than in 1–4, it was judged that the F_2 plant should have been classified susceptible.

TABLE 5. Classification of selfed Pa91 × Oh7B F_2 maize plants for reaction to maize dwarf mosaic virus and classification of their progenies in F_3 rows

F_3 row no.	F_2 plant score[a]	Reaction classes[a] 1	2	3	4	5	6	7	N	Mean score	Classification by: Mean score[c]	Inspection[d]
1	1	22	0	0	0	0	0	0	22	1.0	R	R
2	1	29	0	0	0	0	0	0	29	1.0	R	R
3	1	30	0	0	0	0	0	0	30	1.0	R	R
4	1	31	0	0	0	0	0	0	31	1.0	R	R
5	1	33	0	0	0	0	0	0	33	1.0	R	R
6	1	31	0	0	0	0	0	0	31	1.0	R	R
7	1	26	0	0	0	0	0	1	27	1.2	R	H
8	1	29	0	1	1	0	0	0	31	1.2	R	R
9	1	30	0	0	0	0	1	0	31	1.2	R	H
10	1	31	0	0	0	0	0	1	32	1.2	R	H
11	1	30	0	1	1	0	0	0	32	1.2	R	R
12	1	32	0	0	2	0	0	0	34	1.2	R	R
13	1	16	0	7	2	0	0	0	25	1.8	R	R
14	1	17	4	6	0	0	0	1	28	1.8	R	H
15	1	29	1	0	1	0	1	3	35	1.8	R	H
16	1	24	0	0	0	0	0	4	28	1.9	R	H
17	1	24	0	0	0	0	0	5	29	2.0	R	H
18	1	27	0	0	0	0	2	4	33	2.0	R	H
19	1	28	0	0	0	0	0	6	34	2.1	R	H
20	1	20	3	1	3	0	0	3	30	2.1	R	H
21	1	23	0	0	0	0	0	6	29	2.2	R	H
22	1	16	1	2	1	0	1	3	24	2.3	R	H
23	1	17	0	0	0	0	2	3	22	2.3	R	H
24	1	25	0	0	1	0	1	6	33	2.3	R	H
25	1	25	0	1	0	0	2	5	33	2.3	R	H
26	1	24	1	1	0	0	0	7	33	2.4	R	H
27	1	20	0	1	3	0	1	5	30	2.5	R	H
28	1	26	0	0	0	0	1	8	35	2.5	R	H
29	1	10	0	12	7	0	1	0	30	2.7	R	H
30	1	17	1	3	0	0	0	9	30	3.0	R	H
31	1	17	1	3	2	0	0	8	31	3.0	R	H
32	1	17	0	0	2	1	0	9	29	3.2	R	H
33	1	17	1	1	4	0	0	9	32	3.2	R	H
34	1	19	0	0	0	0	4	10	33	3.4	R	H
35	1	16	0	0	3	0	0	12	31	3.6	R	H
36	1	16	1	1	1	0	0	14	33	3.7	R	H
37	1	10	0	1	8	0	0	12	31	4.2	R	H
38	1*	8	0	1	2	0	0	12	23	4.5	S	S
39	1*	6	0	0	4	0	1	15	25	5.1	S	S
40	1*	7	0	0	0	0	3	18	28	5.4	S	S
41	1*	4	0	2	3	3	2	16	30	5.4	S	S
42	1*	5	0	0	2	0	4	19	30	5.7	S	S
43	1*	2	0	0	0	0	4	17	23	6.3	S	S
44	1*	1	0	0	0	0	6	20	27	6.6	S	S
45	1*	1	0	0	0	0	3	27	31	6.7	S	S
46	2	34	0	1	0	0	0	0	35	1.1	R	R
47	2*	6	1	0	2	0	0	10	19	4.5	S	S
48	5	25	0	0	0	0	0	7	32	2.3	R	H
49	5	18	1	0	2	0	0	5	26	2.4	R	H
50	5	6	0	0	0	0	2	18	26	5.5	S	S
51	5	4	0	1	0	0	1	20	26	5.9	S	S
52	6	5	0	0	0	0	2	23	30	5.9	S	S
53	7*	16	0	3	4	0	0	7	30	1.6	R	H
54	7*	25	0	1	1	0	0	5	32	2.1	R	H
55	7*	20	0	2	2	0	0	9	33	2.9	R	H
56	7*	15	0	0	2	0	0	14	31	3.9	R	H
57	7*	16	0	0	0	0	1	14	31	3.9	R	H
58	7	10	0	1	4	0	0	19	34	4.8	S	S
59	7	7	0	0	2	1	0	20	30	5.3	S	S
60	7	3	0	0	0	0	6	17	26	6.1	S	S
61	7	4	0	0	0	0	2	23	29	6.1	S	S
62	7	3	0	0	0	0	6	22	31	6.2	S	S
63	7	2	0	0	1	0	0	28	31	6.5	S	S
64	7	3	0	0	0	0	0	30	33	6.5	S	S
65	7	2	0	0	0	0	0	25	27	6.6	S	S
66	7	0	0	0	0	0	4	21	25	6.8	S	S
67	7	0	0	0	0	0	2	29	31	6.9	S	S
68	7	0	0	0	0	0	3	29	32	6.9	S	S
69	7	0	0	0	0	0	0	22	22	7.0	S	S

[a] * = rows in which the distribution disagreed with the F_2 classification.
[b] For a description of reaction classes, see Fig. 1.
[c] For mean row scores >4.5, it was judged that the F_2 plant should have been classified susceptible; such F_2 plants rated 1 may have escaped infection.
[d] For rows with more plants in classes 5–7 than in 1–4, it was judged that the F_2 plant should have been classified susceptible.

striped; in the F_2 eight (5%) plants striped. Seven of these F_2 striped plants were selfed (Tables 1 and 2) and four of them appeared to be heterozygous (Table 4). No striping appeared in F_1 plants of Pa91 × Oh7B, and two were found in the F_2, but neither of these was selfed. According to the fate-map concept (16), somatic mutation of the dominant allele to the recessive would permit bands of susceptible tissue to develop in an otherwise resistant plant. When stripes are examined closely, it can be seen that the edge of the stripe is usually midway between two secondary veins. The stripe is not restricted by secondary veins themselves, as is the tendency for gray leaf spot lesions or other fungus infections. Thus, the host tissue in the stripe must be susceptible, whereas the remainder of the tissue is resistant. This condition lends more credence to the fate-map concept.

Although resistance in Oh7B appears to be conditioned by a single dominant gene, expression of maize-virus interactions may be complicated by ontogenetic traits characteristic of Gramineae but not of dicots. Interpretation of the reaction classes we describe and assignment of a gene symbol to inbred Oh7B will be given further consideration.

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