Genetics of Reaction to Maize Dwarf Mosaic Virus Strain A in Several Maize Inbred Lines

C. W. Roane, S. A. Tolin, and H. S. Aycock

Professor emeritus and professor, Department of Plant Pathology, Physiology, and Weed Science, and assistant professor, Department of Crop and Soil Environmental Sciences, Virginia Polytechnic Institute and State University, Blacksburg 24061.
Present address of third author, 5776 Vesper Drive, South Beloit, IL 61080.
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ABSTRACT

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Nine maize inbred lines resistant to maize dwarf mosaic virus strain A (MDMV-A) were studied in hybrid combinations for inheritance of reaction to MDMV-A in F_1 and F_2. Plants growing in a field free of natural inoculum were mechanically inoculated at the two- to four-leaf stage. Three to four weeks later they were scored as healthy or symptomatic in response to virus. This was done to detect early symptoms not always apparent after anthesis. After anthesis plants were scored again but on a scale of 1–7; 1 = no infection, and 7 = nearly completely mottled. Plants that had symptoms before but not after anthesis were scored 2. Resistant inbred lines B68, Oh1EP, Oh7B, and Pa405 (the B68 group) behaved homogeneously for virus reactions as lines and the F_1 of crosses with susceptible line Va50 produced only type 1 plants. In the F_2, resistance was monogenic and completely dominant. Since no infected plants were observed in F_2 of crosses of lines within the B68 group, resistance occurred at one locus. The symbol *Rmd1* is suggested for this locus. The resistant inbred lines A239, Va53, Va85, and VaOM73 (the Va53 group) produced only type 1 plants, but in the F_1 of crosses with Va50, produced some susceptible plants; however, in F_2 all segregated monogenically. In the F_2 of crosses within the Va53 group and between the Va53 and B68 groups, susceptible segregates appeared. However, since the Va53 group behaved variably in F_1 combination with Va50 it could not be ascertained that genes of the Va53 group were allelic with or different from the resistance gene of the B68 group. Va35 is unstable as a line and produced inconclusive results; in the F_2 of Va50 × Va35, a 1:1 ratio was obtained instead of the expected 3:1. Inconclusive results were also obtained in the crosses Va35 × Pa405 and Va35 × A239.

Additional keywords: corn, potyvirus, resistance.

In 1983 it was reported that resistance to maize dwarf mosaic virus (MDMV) in the maize inbred line Oh7B was conditioned by one dominant gene (6). From the literature that was reviewed through early 1983, it seemed that most genetic studies with MDMV were done with natural inocula from johnsongrass (*Sorghum halepense* (L.) Pers.). Consequently, results and interpretations were probably confounded by interaction with maize chlorotic dwarf virus (MCDV). A seven-part diagrammatic scale was presented for classifying the observed plant responses. By accepting the restricted display of symptoms (types 2–5) as evidence for resistance, a departure was taken from traditional studies of inheritance of resistance to viruses by accepting some symptomatic plants as being resistant. Some reversals of behavior between F_2 plants and their F_3 progenies were also reported. It was pointed out that these reversals were detected by the pedigree procedures employed, and had those procedures not been used, the F_2 and F_3 data would have supported the hypothesis for one dominant gene for resistance without question. Type 5 plants caused concern as to whether their response should be interpreted as resistance, susceptibility, or heterozygosity. The F_2 and F_3 data in Tables 4 and 5 in (6) did not satisfactorily resolve the problem.

Scott and Rosenkranz (9) had described a method for assessing the number of genes conditioning reaction to MDMV based on healthy vs. symptomatic plants and the length of the incubation period. Although each experiment included six hybrid combinations and apparently a susceptible inbred check, resistant inbred parents and their F_1 hybrids were not included. Data were recorded every 2–3 days (9) or daily (7) after symptoms began to appear, for about 4 wk, until the increase in number of diseased plants became negligible. The cumulative data were used to calculate the number of genes conditioning resistance. Their assumptions were that resistance "genes are independent, no dominance, and that each allele has an equal effect" (9). Thus, a quantitative approach was applied at the outset. Rosenkranz and Scott (7) reiterated these assumptions and argued strongly for their validity and against the use of a rating scale. Of the 10 inbred lines evaluated by Rosenkranz and Scott in Mississippi (7), only Pa405 and Va35, reported to have five genes and one gene conditioning resistance, respectively, were included in the experiments reported here.

Mikel et al (3) reported on the genetics of MDMV reaction in dent-sweet corn crosses in which dent inbred lines Pa405, B68, Oh1EP, and Ga209 were used as sources of resistance. They inoculated with a mixture of MDMV-A and MDMV-B and proposed hypotheses of complex gene interaction involving three genes conditioning resistance in both B68 and Pa405. In a preliminary report (4), it was concluded that resistance is conditioned by single dominant genes in the inbred lines B68, Oh1EP, Oh7B, and Va85 and that the genes are allelic. In this report, we provide evidence for these conclusions, and contrast our results to the previous reports of others in which it was concluded that resistance to MDMV is a multigenic trait (1,3,7,9).

MATERIALS AND METHODS

Inoculum production and inoculation procedures were those described previously (2,6). The same MDMV culture was used in all the studies and was maintained in a greenhouse in johnsongrass, grown originally from seed. The culture was verified as MDMV-A by Dr. Raymond Louie, USDA-ARS, and the Ohio Agricultural Research and Development Center, Wooster, OH. Plots were located at Blacksburg, VA, in an area free of johnsongrass and of naturally occurring MDMV. Plants were inoculated in the two- to four-leaf stage scored 3–4 wk after inoculation, and again shortly after anthesis. The first scoring was particularly important for recognizing restricted infections that could be overlooked after anthesis. Plants were classified

as healthy or with symptoms of stripe or uniform mosaic. The second scoring was according to the previously published 1–7 scale (6). Recently, the percentage of symptomatic tissue has also been estimated; plant types 2 and 3 display less than 8% symptomatic tissue; type 4, less than 15%; types 5 and 6, 20–40%; and type 7, more than 50%, usually 70–80%. Plant types 4 and 5 occurred at very low frequency. Plant types 1–4 were classified as resistant, types 6 and 7 as susceptible. Type 5 plants were considered resistant, but their frequency was so low that classifying them as resistant or susceptible did not affect segregation ratios significantly. Plants with symptoms early but not after anthesis were rated type 2.

Field plots consisted of parental lines, F_1, and F_2 plants of each cross and all plants were inoculated. The integrity of each F_1 family was maintained. In the F_2, data from different F_1 families were pooled only if the chi-square test for heterogeneity was not significant.

The resistant parental inbred lines were selected from several sources because they had been used previously by others in genetics of maize-MDMV interactions, were widely used in the hybrid maize industry, or were of potential importance in the Virginia maize breeding project. The susceptible line Va50 was chosen because plants infected with MDMV produce a bright yellow mottle that persists until plants become senescent. All lines had been inoculated, selected, and inbred for at least two generations before they were incorporated into the genetic study. As all appeared homogeneous for plant type and reacted uniformly from generation to generation, they were presumed to be homozygous for response to MDMV-A (Table 1). Although Va35 could not be stabilized for a type 1 reaction, its variable behavior was consistent from generation to generation of inbreeding.

RESULTS

The numbers of parental line and F_1 progeny plants in each reaction type in response to inoculation with MDMV-A are shown in Table 1. In combination with the susceptible line Va50, the F_1 of lines B68, Oh1EP, Oh7B, Pa405, and VaOM73 produced only resistant plants. Of these, low-level reaction types occurred in some plants in crosses with Oh7B and VaOM73. The F_1 of crosses of inbreds lines A239, Va35, Va53, and Va85 with Va50 produced some susceptible plants. Since, as a line, Va35 produced type 6 and 7 plants, these types were expected to occur in its F_1. As lines, A239, Va53, and Va85 produced no symptomatic plants, therefore, susceptible plants were not expected in the F_1 of crosses with Va50 if resistance is dominant.

The F_1 of resistant × resistant inbred lines produced no symptomatic plants (Table 1). However, based on responses of parents and F_1 of resistant × susceptible crosses, infected plants could have appeared in the F_1 of A239 × Va35 and A239 × Va53.

TABLE 1. Distribution of parental maize line plants and plants of their F_1 progenies in seven reaction types to maize dwarf mosaic virus strain A

	Reaction type[a]							
	1	2	3	4	5	6	7	*N*
Inbred lines								
A239	19	0	0	0	0	0	0	19
B68	112	0	0	0	0	0	0	112
Oh1EP	31	0	0	0	0	0	0	31
Oh7B	76	0	0	0	0	0	0	76
Pa405	82	0	0	0	0	0	0	82
Va35	44	2	0	0	0	1	4**[b]	51
Va53	36	0	0	0	0	0	0	36
Va85	55	0	0	0	0	0	0	55
VaOM73	31	0	0	0	0	0	0	31
Va50 (common susc.)	0	0	0	0	0	0	80	80
F_1 Susceptible × resistant crosses								
Va50 × A239	18	1	3	0	0	2	5**[c]	29
Va50 × B68	17	0	0	0	0	0	0	17
Va50 × Oh1EP	32	0	0	0	0	0	0	32
Va50 × Oh7B	50	3	0	0	0	0	0*	53
Va50 × Pa405	42	0	0	0	0	0	0	42
Va50 × Va35	26	0	0	0	0	1	5**[c]	32
Va50 × Va53	12	0	0	1	0	1	1**[c]	15
Va50 × Va85	24	1	0	0	0	3	0**	28
Va50 × VaOM73	25	1	3	0	0	0	0*	29
F_1 Resistant × resistant crosses								
B68 × Oh7B	35	0	0	0	0	0	0	35
B68 × Pa405	69	0	0	0	0	0	0	69
B68 × VaOM73	23	0	0	0	0	0	0	23
Oh1EP × B68	24	0	0	0	0	0	0	24
Va85 × B68	36	0	0	0	0	0	0	36
Oh1EP × Pa405	16	0	0	0	0	0	0	16
Oh7B × Pa405	52	0	0	0	0	0	0	52
Va35 × Pa405	16	0	0	0	0	0	0	16
Va85 × Pa405	82	0	0	0	0	0	0	82
VaOM73 × Pa405	51	0	0	0	0	0	0	51
Va85 × Oh7B	30	0	0	0	0	0	0	30
Va85 × VaOM73	42	0	0	0	0	0	0	42
Va53 × VaOM73	25	0	0	0	0	0	0	25
Va53 × A239	37	0	0	0	0	0	0	37
Va35 × A239	41	0	0	0	0	0	0	41

[a]Reaction types: 1 = no symptoms, 2 = narrow streaks in leaves below the ear, 3 = narrow streaks in leaves above the ear, 4 = narrow streaks in leaves above and below the ear, 5 = mottled leaves only below the ear, 6 = mottled leaves only above the ear, 7 = mottled leaves above and below the ear.

[b]*Some plants symptomatic but considered resistant. **Some plants considered susceptible.

[c]An unexpected fit to the 3:1 ratio, resistant:susceptible.

In the F_2 of resistant × susceptible crosses, plants were grouped by reaction types into the phenotypes resistant and susceptible, with reaction types 1–5 being resistant and 6 and 7 being susceptible; they were also grouped into the phenotypes healthy (type 1) and infected (types 2–7), and in each phenotypic grouping frequencies were tested for a fit to the 3:1 ratio (Table 2). For convenience, the crosses are assigned a cross number (×no.). When tested as resistant:susceptible, all except ×6 segregated satisfactorily into a 3:1 ratio; ×6 responded as if it were a backcross. When tested as healthy:infected, only ×6 and ×8 failed to segregate into a 3:1 ratio.

In crosses of resistant × resistant lines, no infected plants appeared among the F_2 of B68 × Oh7B (×10), B68 × Pa405 (×11), Oh1EP × B68 (×13), Oh1EP × Pa405 (×20), and Oh7B × Pa405 (×21) (Tables 2 and 3). Thus, these lines, designated the B68 group, must have a resistance gene at a common locus. However, some crosses between lines of the B68 group with those of the Va53 group behaved inconsistently. Va85 × B68 (×14) produced one infected type 3 resistant plant, Va85 × Oh7B (×16) segregated 15:1, and Va85 × Pa405 (×17) produced two infected plants, one of which was type 7 susceptible ($N = 453$). B68 × VaOM73 (×12) produced eight infected plants, only two of which were type 7 susceptible ($N = 234$), and thus segregated 15:1 for healthy vs. infected but not for resistant vs. susceptible. VaOM73 × Pa405 (×19) did not segregate ($N = 469$). Va85 × VaOM73 (×18) produced two infected plants, one of which was type 7 susceptible ($N = 425$). This appears strange since these lines produced infected plants in the F_1 of their crosses with Va50, but only Va50 × Va85 produced susceptible plants (Table 1); furthermore, this cross did not segregate 3:1, healthy:infected, in the F_2 (Table 2). It is probable that Va85 and VaOM73 have a gene locus in common with the B68 group.

The behavior of Va35 cannot be explained. As a line and in F_1 combination with Va50, Va35 is variable (Table 1). In F_2, Va50 × Va35 (×6) segregated 1:1, and with A239 (×23) it segregated into an acceptable fit to 15:1 for healthy vs. infected (Table 2). This is not surprising since the F_1 of both Va35 and A239 segregated unexpectedly 3:1 in combination with Va50 (Table 1).

Va53 was stable as a line but segregated in the F_1 of Va50 × Va53 (×7). In F_2, Va53 segregated 3:1 with Va50 (×7), and it segregated with A239 (×24) and VaOM73 (×25) but not digenically. With Oh7B (×15), it did not segregate (Tables 2 and 3). Because A239 behaved erratically, it rather than Va53 caused the appearance of susceptible plants in ×24. Thus, Va53 probably

TABLE 2. Distribution of F_2 plants of 25 maize crosses in seven reaction types in response to inoculation with maize dwarf mosaic virus strain A and chi-square tests for goodness of fit to indicated ratio

Cross no.	Pedigree	Reaction type[a] 1	2	3	4	5	6	7	N	No. F_1 families	Segregating Res:Susc. 1–5:6–7	χ^2	P	Segregating Healthy:Inf. 1:2–7	χ^2	P
Susceptible × resistant:												3:1			3:1	
1	Va50 × A239	134	0	2	0	0	10	42	188	2	136:52	0.71	>0.30	134:54	1.39	>0.20
2	Va50 × B68	188	0	0	1	0	23	40	252	2	189:63	0.00	>0.99	188:64	0.02	>0.80
3	Va50 × Oh1EP	219	1	5	2	0	24	59	310	3	227:83	0.52	>0.30	219:91	3.14	>0.05
4	Va50 × Oh7B	139	4	2	1	0	13	31	210	2	166:44	1.83	>0.10	159:51	0.06	>0.80
5	Va50 × Pa405	314	17	1	7	2	18	97	456	5	341:115	0.01	>0.90	314:125	2.83	>0.05
6	Va50 × Va35	160	7	4	7	0	22	143	343	3	178:165[b]	···	<0.01	160:183[b]	···	<0.01
7	Va50 × Va53	205	3	1	4	0	22	42	277	3	213:64	0.53	>0.30	205:72	0.15	>0.70
8	Va50 × Va85	131	2	5	4	0	18	33	193	2	142:51	0.21	>0.50	131:62	5.22	<0.02
9	Va50 × VaOM73	212	0	1	0	2	29	36	280	3	215:65	0.48	>0.30	212:68	0.08	>0.70
Resistant × resistant:												15:1			15:1	
10	B68 × Oh7B	191	0	0	0	0	0	0	191	2	191:0	···	···	191:0	···	···
11	B68 × Pa405	503	0	0	0	0	0	0	503	5	503:0	···	···	503:0	···	···
12	B68 × VaOM73	226	5	0	0	1	0	2	234	3	232:2	11.6	<0.01	226:8	3.20	>0.05
13	Oh1EP × B68	85	0	0	0	0	0	0	85	1	85:0	···	···	85:0	···	···
14	Va85 × B68	131	0	1	0	0	0	0	132	1	132:0	···	···	131:1	6.8	<0.01
15	Va53 × Oh7B	74	1	0	1	0	0	0	76	1	76:0	···	···	74:2	1.70	>0.10
16	Va85 × Oh7B	260	5	1	6	1	1	12	286	4	273:13	1.42	>0.20	260:26	3.94	>0.02
17	Va85 × Pa405	451	0	0	1	0	0	1	453	4	452:1	28.1	<0.01	451:2	26.1	<0.01
18	VaOM73 × Pa405	423	1	0	0	0	0	1	425	4	424:1	26.1	<0.01	423:2	24.2	<0.01
19	VaOM73 × Pa405	469	0	0	0	0	0	0	469	4	469:0	···	···	469:0	···	···
20	Oh1EP × Pa405	242	0	0	0	0	0	0	242	2	242:0	···	···	242:0	···	···
21	Oh7B × Pa405	720	0	0	0	0	0	0	720	6	720:0	···	···	720:0	···	···
22	Va35 × Pa405	282	3	0	1	0	0	1	287	2	286:1	17.1	<0.01	282:5	9.95	<0.01
23	Va35 × A239	139	0	0	1	0	0	3	143	1	140:3	4.21	>0.02	139:4	2.91	>0.05
24	Va53 × A239	120	0	0	0	0	0	2	122	1	120:2	4.43	>0.02	120:2	4.43	>0.02
25	Va53 × VaOM73	133	0	1	1	0	0	1	136	1	135:1	7.06	<0.01	133:3	3.80	>0.02

[a] Reaction types are as described in Table 1 and in Materials and Methods.
[b] Appears to segregate 1:1; no explanation for this behavior, inconsistent with Va35 × Pa405 (×22), and Va35 × A239 (×23).

TABLE 3. Summary of maize F_2 crosses shown in Table 2[a]

Inbred line	A239	B68	Oh1EP	Oh7B	Pa405	Va35	Va53	Va85	VaOM73
Va50	[1]3:1	[2]3:1	[3]3:1	[4]3:1	[5]3:1	[6]1:1	[7]3:1	[8]3:1	[9]3:1
A239	×	···	···	···	···	[23]Seg	[24]Seg	···	···
B68		×	[13]R	[10]R	[11]R	···	···	[14]R	[12]Seg
Oh1EP			×	···	[20]R	···	···	···	···
Oh7B				×	[21]R	···	[15]R	[16]Seg	···
Pa405					×	[22]Un	···	[17]Un	[19]R
Va53							×	···	[25]Un
Va85								×	[18]Un

[a] Superscripts refer to cross number shown in Table 2; R = all resistant, Seg = segregating, Un = uncertain classification because of occurrence of only 1 susceptible plant in F_2 (Table 2) and one of the lines shows a lack of complete dominance in F_1 of the cross with Va50 (Table 1).

has a major gene allelic with that of the B68 group, but it is modified by other genes, is environment sensitive, or lacks sufficient penetrance to behave invariably as a major gene.

DISCUSSION

By using a 1–7 scale to describe reaction types of postanthesis plants and partitioning the reaction types into either resistant and susceptible, 1–5:6–7, or healthy and infected, 1:2–7, it was found that eight of nine lines studied were monogenic dominant for reaction to MDMV-A. In addition, inbred lines B68, Oh1EP, Oh7B, Pa405, and probably Va53 and Va85 have their genes at a common locus. Lines A239, Va53, and VaOM73 behaved erratically, although there were only two crosses among these lines, Va53 × A239 (×24), and Va53 × VaOM73 (×25). These lines and Va35, which was in a class of its own, may have been poor choices as parents for a genetic study, even though they may be very useful in producing MDMV-resistant hybrids.

The data from crosses of susceptible × resistant inbreds provide clear-cut evidence that resistance is monogenic dominant. In all crosses, intermediate reaction types 3–5 (with 8–40% symptomatic tissue) are of low frequency. As a result, no evidence appears for epistasis or additive gene effects. For the locus in the B68 group, we propose the symbol *Rmd*1 with its recessive allele *rmd*1, resistant and susceptible to MDMV-A. If an additional locus occurs, it is in A239.

Scott and Rosenkranz (9) have proposed a different scheme for counting genes conditioning reaction to MDMV. They inferred that it is invalid to use a scale to classify plant reactions because each step on the scale should be related to the number of alleles contributing an increment of resistance. To accept their premise would preclude dominance and epistasis, and mandate gene independence and additive effects. Their reasoning is that each allele delays symptom expression by some period of time and, therefore, expresses an additive gene effect. Both dominance and epistasis are common in genetics of reaction to pathogens and linkage is not uncommon. In this study, genes in the B68 group were completely dominant. Additive effects are characteristic of quantitative inheritance and are relatively uncommon when pure strains of pathogens and cultivars are used. Scott and Rosenkranz included Va35 in their first report (9) and Pa405 in their second report (7); both of these lines are included in the current study. For Va35, Scott and Rosenkranz reported 81% of 561 plants as symptomatic; we report 14% of 51 as symptomatic and 10% as susceptible. They concluded Va35 has one gene conditioning resistance. Our work was inconclusive. However, in this study, 90% of the parental plants were resistant and they expected 88% to be symptomatic. These differences could be attributed to environmental factors or to differences in inbred line purity; therefore, any comparison is probably meaningless.

For Pa405, Rosenkranz and Scott (7) included no parental plants in the field as checks, but from inoculation of an unknown number of plants in the greenhouse, 100% remained healthy. We reported that 100% of 82 plants remained healthy in the field. They conclude that Pa405 has five genes conditioning reaction to MDMV-A; we conclude that one gene conditions the reaction.

Mikel et al (3) derived a three-gene hypothesis to explain the segregations obtained from Pa405 × susceptible line crosses, in which the presence of one gene conferred resistance in the presence of either of the other two. The expected ratio was 45 resistant: 19 susceptible plants (actually, healthy: symptomatic) whether the reaction was with MDMV-A inoculum or with a mixture of MDMV-A and -B. For Pa405 crosses and with a mixture of strains A and B for the F_2 they obtained a fit to the 45:19 ratio in 16 computations (3, Table 6); 13 of the 16 also fit a 3:1 ratio. In 2 yr (1980, 1982), the totals fit both the 45:19 and 3:1. As they found for the 3-yr total 1980–1982, the year totals could be pooled and be homogeneous for a 45:19 ratio (3, Table 6). Where only strain A was used in the inoculum (3, Table 9), a satisfactory fit was also obtained for a 45:19 ratio. For the B68 model, Mikel et al (3) expected an F_2 ratio of 27 resistant: 37 susceptible. An allele of each gene had to be present to confer resistance. This was dubbed the B68 model. Two sets of data were provided that cause one to doubt the validity of the Pa405 and B68 models. In a cross of Pa405 × B68, the F_2 segregated 11 resistant: 8 susceptible plants; in a reciprocal cross the F_2 segregated 162 resistant: 1 susceptible (3, Table 4). If their models are correct, it is difficult to explain the high frequency susceptible plants in a 19-plant population. The genes in Pa405 alone could reduce the expected frequency of susceptibles to 5.6; certainly the B68 genes would have reduced the frequency of susceptibles to almost zero. In our cross of B68 × Pa405, $N = 456$, no infected plants were observed (Table 2). Although Mikel et al made use of inbred lines Oh1EP and Ga209, they limited their comments about them to, "Although they carry similar genes for resistance to MDMV, the genes in Pa405 appear more completely dominant than those in B68, Oh1EP, or Ga209." In our studies, B68, Oh1EP, and Pa405 performed equally well and had completely dominant genes (Table 2). The use of a mixture of MDMV-A and -B by Mikel et al may have precipitated their complex hypotheses (3). Scheifele and Wernham (8) have shown that Pa405 is resistant to MDMV-A and -B, and Findley et al (1) reported Pa405 has one dominant gene conditioning resistance to -A and two dominant genes conditioning reaction to -B. Although the use of the virus mixtures may be desirable in breeding for virus resistance, as experienced with other pathogens, it often leads to imprecise genetic interpretations. Inheritance studies with different strains of soybean viruses in which a gene-for-gene system was hypothesized suggests that such a system may be expected to occur with different strains of MDMV (5). Wernham and Scheifele suggested such is the case (10).

The genetics of reaction to MDMV has proved to be very complex. From report to report, there is very little evidence to support a single hypothesis where one inbred line is common to each study. Methods of recording phenotypes, differences in environment, and choice of susceptible parents may have differential effects on the outcome of the experiments. At Blacksburg, VA, for example, daytime temperatures rarely exceed 35C for the duration of a corn growing season and nighttime temperatures are usually in the range of 15–26 C. Under this temperature regime, plants retain vivid symptoms of MDMV infection well toward senescence.

By our methods and in our environment, we conclude that corn inbred lines B68, Oh1E, Oh7B, and Pa405 (the B68 group) each have single dominant gene conditioning reaction to MDMV-A, and that these genes are either allelic or very closely linked. We further conclude that inbred lines A239, Va53, Va85, and VaOM73 (the Va53 group) probably are monogenic, but for some reason their genes do not provide homogeneous resistance as do those of the B68 group. Because of their inability to produce homogeneous F_1 progenies, F_2 data with them are suspect. For example, Va85 segregated in the F_2 of the cross with Oh7B (×16), but not with B68 (×13); with Pa405 (×24) and VaOM73 (×18) large chi-square values were produced. Va53 segregated with A239 and VaOM73 and produced significant chi-square values, but did not segregate with Oh7B (×18). Va35 crossed with Va50 segregated 1:1, but with Pa405 did not segregate. How could this happen?

Our results from intercrossing B68, Oh1EP, and Pa405, much more so than the results obtained by Mikel et al (3), clearly indicate these lines have an allelic gene. In addition, complete dominance of the genes in B68, Oh1EP, and Pa405 is apparent from our results but not from those of Mikel et al (3). Findley et al (1) observed complete dominance of the resistance in Pa405 but not in Oh7B; from our results the resistance in Oh7B is completely dominant.

Inbred line Pa405 is the most widely studied line in the genetics of reaction with MDMV-A. It has been reported as having one dominant gene (1), three genes (3), five genes (7), and one gene again in our study. In no two cases were the results obtained by crossing Pa405 to the same susceptible inbred line. Genetic background must also be added to environment and scoring methods as a source of variation in MDMV genetics.

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