Effects of Genotype and Inoculation Protocols on Resistance Evaluation of Maize to Maize Dwarf Mosaic Virus Strains

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ABSTRACT

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Responses of maize inbreds A239, A375, B14, CG1, CI.44, Ky61:2335, Ky66:2500, Oh28, M14, Pa32, Pa405, and Va35 to inoculation protocols with maize dwarf mosaic virus (MDMV) strains A and B were determined. Protocols included age of test plant at inoculation, number of repeated inoculations per plant, number of rubs per inoculation, and inoculum dilutions. Tests involving four inoculations with two rubs at 4-day intervals resulted in the greatest number of infected plants in all inbreds. Except for the number of repeated rub inoculations per plant with MDMV-A, cluster analysis of area under the disease progress curve separated inbreds into more groupings than did cluster analysis of final readings. MDMV-B was generally not more virulent than MDMV-A except in the number of repeated rub-inoculation tests involving inbreds A239, B14, M14, and Va35. Inbred Pa405 was most resistant to both strains. The significance of variation in host response to different inoculation protocols in studies of disease resistance evaluation is discussed.

Additional key words: corn viruses, methodology, tolerance, *Zea mays*.

Researchers breeding for disease resistance or studying genetic mechanisms for resistance in maize (*Zea mays* L.) to maize dwarf mosaic virus (MDMV) are aware of variations in host responses to virus infection. Many variables have been reported to influence host response to infection (1–4,7–9,11,13,16,22–26). To obviate variations caused by natural infections of maize by MDMV, use of mechanical inoculation and inoculum from greenhouse-grown plants is now common (4,9,14,16–18). Several classifications of host reactions (e.g., a disease scale of 1–9 or 1–7 or time for symptom development instead of a positive or negative reaction) to infection by MDMV (4,9,16–18,20) are used for genotype evaluation. Despite many and varied attempts, the different hypotheses on the genetic mechanisms of disease resistance are not universally accepted (3,16–18). One preliminary step toward resolving these variations is to develop a capability to produce consistent host responses to virus inoculations. The lack of this capability may have hampered progress in studies of genetic mechanisms for disease resistance to MDMV in maize. This paper reports on four inoculation protocols and their effects on responses of 12 inbred lines to MDMV infection. A preliminary report of this work has been published (12).

MATERIALS AND METHODS

Maize inbreds A239, A375, B14, CG1, CI.44, Ky61:2335, Ky66:2500, Oh28, M14, Pa32, Pa405, and Va35 were used to test effects of inoculation protocols. To ensure inbred homogeneity, individual plants were selfed for five to 10 (usually eight or nine) generations. At the last selfed generation, 10–20 seeds from the same ear were planted to produce a seed lot for tests. Seeds of inbreds were planted in wood flats (30 × 46 × 8 cm) filled with autoclaved Wooster silt loam and peat moss mix (6:1, v/v). Uniform plant growth and stands were obtained. Twelve to 15 seeds of an inbred were planted in each of five rows in a flat, and each inbred was replicated four times in a test for each virus strain. During October to April, natural light in the greenhouse was supplemented with 12 hr of fluorescent light from 2.4-m fixtures and temperatures were maintained at 18–25 C. Plants were watered with warmed water as needed and fertilized weekly with a 20-20-20 (NPK) solution. In all tests except where plants were inoculated more than once (number of repeated inoculations = repeated inoculation test) or when more than 10 days old (age test), plants were in the two- to three-leaf stage and 8–16 cm tall when inoculated. Virus inoculum (1:10, w/v) was prepared from Oh28 maize seedlings inoculated when 10 days old and harvested 14–21 days after inoculation. Plants were inoculated with a virus-silicon carbide mixture (600-mesh, 0.25%, w/v), using foam pads (1.5 × 3.8 cm) placed between tongs of modified battery clips calibrated to apply a force of approximately 40 g/cm^2. At inoculation, all leaves (two or three) of each plant, except those in the rub tests, were inoculated with two rubs, and six plants were inoculated before pads were recharged with virus inoculum. Because of growth responses to seasonal changes, actual plant age sometimes varied ±2 days from the scheduled plant age at inoculation time. Plants were diagnosed for virus symptoms beginning 7–10 days after inoculation and thereafter at weekly intervals up to 10 wk. Included in the diagnoses were notations of whether symptoms were local lesions on inoculated leaves or systemic infections that were limited or general and consisted of mosaics, mottles, or flecks and streaks.

Inoculation protocols included inoculation of plants with two rubs at 10, 14, 18, or 22 days after planting (age test); two to four repeated inoculations of plants with two rubs each at 10 and 14, at 10, 14, and 18, and at 10, 14, 18, and 22 days after planting, respectively (repeated inoculation test); rub inoculation of the same leaves one, three, or six times at 10 days after planting (rub test); and use of inoculum diluted (w/v) 1:10, 1:40, 1:80, 1:160, or 1:320 to inoculate plants with two rubs each at 10 days after planting (dilution test).

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MDMV-A and MDMV-B were collected in Ohio and maintained in maize. Strain identity was monitored each time inoculum was prepared for a test by also inoculating maize inbreds Oh28 and N20, *Sorghum bicolor* (L.) Moench cv. Atlas, and *Triticum aestivum* L. cv. Monon. Differential symptoms on Atlas sorghum identified MDMV-A and MDMV-B (5).

Each test consisted of a randomized factorial with three factors: inbred, inoculation treatment, and replicate. Analysis of variance (ANOVA) was used to assess the effect of each factor and interaction. Both the disease incidence at final reading (FR) and area under the disease progress curve (ADPC) (21) of weekly disease incidence readings were analyzed. Analysis of ADPC quantified the rate of symptom development. To standardize ADPC values for comparison among treatments in a test, the calculated value was divided by the number of days of observation and multiplied by 100, converting the proportion into a whole number. After ANOVA, FR and ADPC means were separated by Scott-Knott cluster analysis (15,19) into nonoverlapping groups.

RESULTS

FR and ADPC analyses. On the basis of ANOVA, inbred, inoculation treatment, and their interactions were significant ($P < 0.05$) for every test with both virus strains and for both FR and ADPC. Therefore, means of the interaction of inbred and inoculation treatment were separated into clusters. For any test, a maximum of seven nonoverlapping clusters were found. Inbreds classified in cluster 1 or 7 were most susceptible or most resistant, respectively. For presentation purposes, the means and appropriate cluster for each inbred were partitioned by treatment level.

Tests with MDMV-A. *Test plant age.* In cluster analysis of the means of disease incidence at FR (FR cluster analysis), the level of resistance and/or immunity in some inbreds (e.g., CI.44, Ky66:2500, Pa32, and Pa405) was high throughout the test range (Fig. 1). In contrast, a high level of susceptibility at all four test ages was only found in inbred Oh28. The level of resistance in some inbreds (e.g., A239, B14, M14, and Va35) was higher at 10 days of age than at 14, 18, and 22 days of age at the time of inoculation. In cluster analysis of ADPC (ADPC cluster analysis), similar trends were observed for various ages of inbreds.

Repeated inoculations. In FR cluster analysis of inbreds inoculated at 10 days after planting and again at 14, 18, and 22 days, the level of susceptibility of nine of 12 inbreds was uniformly high at all inoculation times (Fig. 2). The level of resistance and/or immunity in two inbreds (Pa32 and Pa405) was not altered by repeated inoculations. In one inbred, Ky66:2500, the level of resistance changed from moderately high to very low as the number of repeated inoculations increased from one to four times. In the ADPC cluster analysis, the level of resistance in all inbreds except Ky66:2500 remained unchanged in response to repeated inoculation.

Number of rubs per inoculation. FR cluster analysis of inbreds in the number of rubs test (Fig. 3) showed that an increased number of rubs significantly increased virus transmission most in inbred

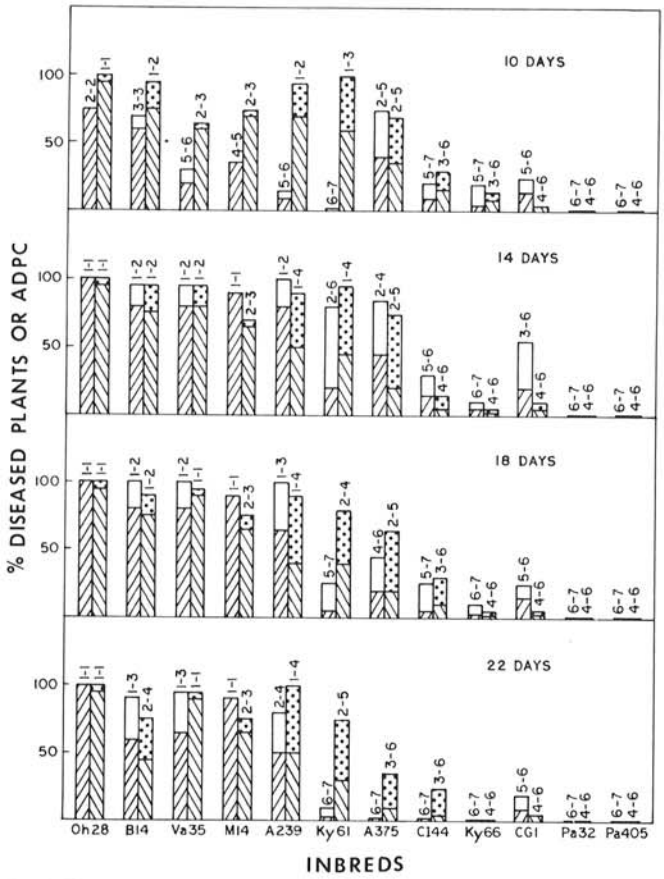

Fig. 1. Responses of inbreds at different ages to inoculation with maize dwarf mosaic virus (MDMV) strain A or B; first and second bar in each set of an histogram for an inbred, respectively. Ky61 and Ky66 are inbreds Ky61:2335 and Ky66:2500, respectively. Numbers above each bar represent cluster designation by Scott-Knott analyses on final readings (FR) or area under disease progress curve (ADPC). Inbreds at cluster 1 were most susceptible and inbreds at cluster 7 were most resistant. MDMV-A = □ and ▨ for FR and ADPC, respectively. MDMV-B = ⁙ and ▩ for FR and ADPC, respectively.

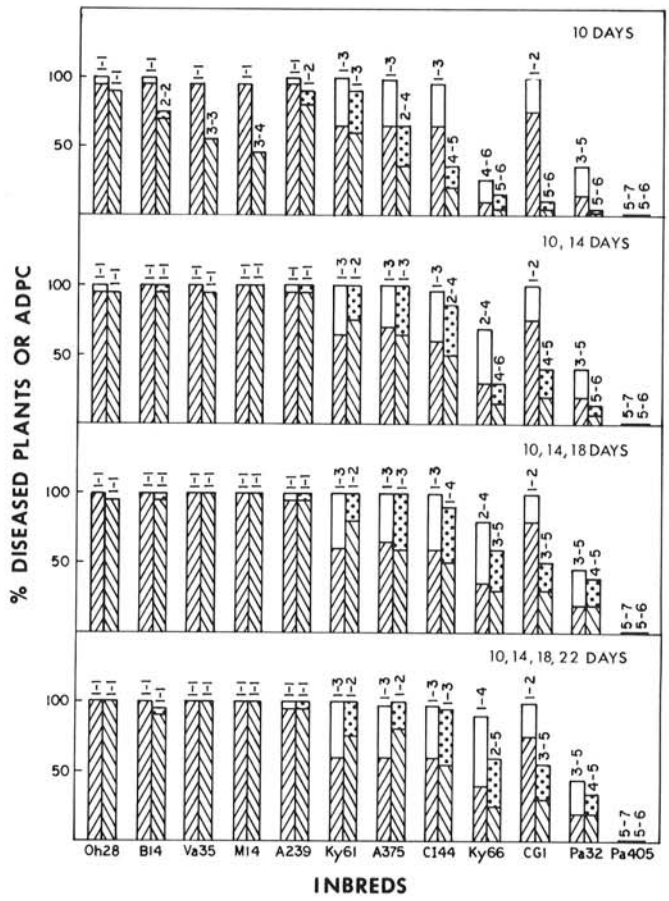

Fig. 2. Responses of inbreds to number of repeated inoculations at 10; 10 and 14; 10, 14, and 18; and 10, 14, 18, and 22 days after planting with maize dwarf mosaic virus (MDMV) strain A or B; first and second bar in each set of an histogram for an inbred, respectively. Ky61 and Ky66 are inbreds Ky61:2335 and Ky66:2500, respectively. Numbers above each bar represent cluster designation by Scott-Knott analyses on final reading (FR) or area under disease progress curve (ADPC). Inbreds at cluster 1 were most susceptible and inbreds at cluster 7 were most resistant. MDMV-A = □ and ▨ for FR and ADPC analyses, respectively. MDMV-B = ⁙ and ▩ for FR and ADPC, respectively.

A239 and least in inbreds B14, M14, and Va35. ADPC cluster analysis showed similar trends.

Inoculum dilutions. In the test with 1:10 dilution of inoculum (Fig. 4), seven of 12 inbreds were classified by FR cluster analysis into the most susceptible group and usually with the highest disease incidence. Greatest differentiation of inbred host response to inoculation occurred at dilutions of 1:40 and 1:80 and generally with the seven inbreds classified as most susceptible at the 1:10 dilution. The responses of three inbreds (A375, Ky61:2335, and Va35) changed significantly at each higher dilution up to 1:160 or 1:320. In ADPC analysis, the greatest separation among inbreds also occurred at the 1:40 dilution. Symptom development in inbreds A375 and Ky61:2335 at 1:10 inoculum dilution was slower than in other susceptible inbreds (e.g., A239, B14, M14, Oh28, and Va35) similarly classified by FR analysis. ADPC analysis classified inbreds A239, B14, M14, and Va35 into the same group at 1:10 and 1:80 but found statistical differences among these inbreds at 1:40.

Tests with MDMV-B. *Test plant age.* In FR cluster analysis of disease incidence means, two inbreds (A239 and Oh28) were always very susceptible at any age (Fig. 1). At the other extreme, two inbreds (Pa32 and Pa405) were immune. Inbreds Ky61:2335 and B14 were relatively more resistant when inoculated at 22 than at 10 days. Conversely, Va35 was more susceptible, whereas the level of resistance in inbreds CG1, M14, Pa32, and Pa405 was not altered by plant age. The ADPCs of inbred A239 at all ages tested were smaller than those of Oh28, although the inbreds were similarly classified by FR cluster analysis. ADPCs of inbred A375 were also significantly smaller than those of inbreds similarly classified in cluster 2 by FR cluster analysis. For example, at 10 days, inbreds M14 and A375 were classified by FR into cluster 2 but into cluster 3 and 5, respectively, by ADPC analysis.

Repeated inoculations. Four repeated inoculations of inbreds at 4-day intervals significantly changed all but four (CG1, Ky66:2500, Pa32, and Pa405) inbred classifications in the FR analysis to cluster 1 (Fig. 2). Expression of susceptibility of inbred M14 was most affected by repeated inoculation, changing from a cluster 3 (44.7% infection) to a cluster 1 classification (100% infection) after inoculations at 10 and again at 14 days. To some degree, a similar trend was observed in inbreds CG1, CI.44, Ky66:2500, and Pa32. Of particular interest is the first significant shift of host response of inbred Pa32 from 0% infection in the age test to 40.6% in the frequency of inoculation test. Resistance in inbred Pa405 was not modified by repeated inoculation. ADPC cluster analyses showed that symptom development in inbred CI.44 at repeated inoculations on days 10, 14, 18 and 10, 14, 18, and 22 was slower than in inbreds similarly classified as cluster 1 by FR. Symptom development was slower in inbreds A375 and Ky61:2335 but not to the same degree as in inbred CI.44.

Number of rubs per inoculation. FR cluster analysis of the number of rub inoculations (Fig. 3) showed CG1 and CI.44 differed statistically between three and six and among one, three, and six rubs, respectively, whereas inbreds Ky66:2500, Pa32, and Pa405 (classified as resistant) remained unchanged as did inbreds classified as susceptible. Different rates of symptom development were revealed by ADPC analysis, occurring mostly with susceptible inbreds. Symptom development in inbreds Ky61:2335 and A375 was always slower than other similarly classified inbreds

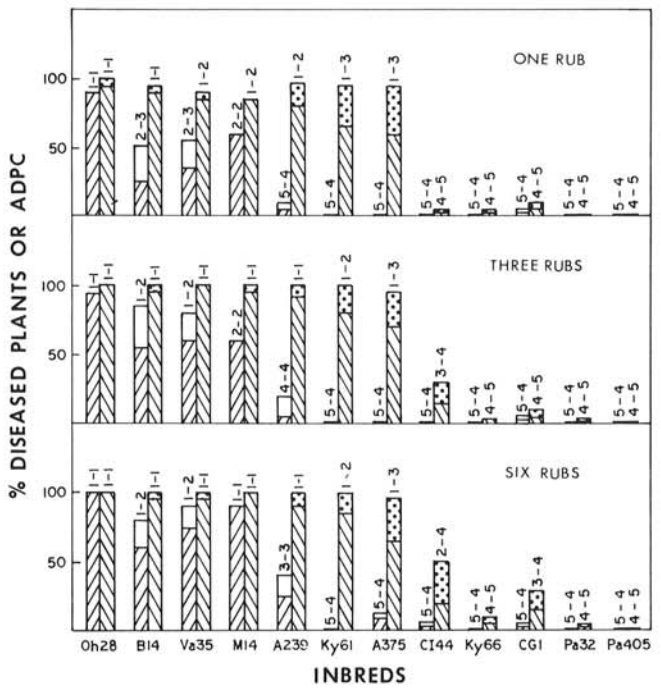

Fig. 3. Responses of inbreds to one, three, or six rub inoculations with maize dwarf mosaic virus strain A or B; first and second bar in each set of an histogram for an inbred, respectively. Ky61 and Ky66 are inbreds Ky61:2335 and Ky66:2500, respectively. Numbers above each bar represent cluster designation by Scott-Knott analyses on final reading (FR) or area under disease progress curve (ADPC). Inbreds at cluster 1 were most susceptible and inbreds at cluster 7 were most resistant. MDMV-A = □ and ▨ for FR and ADPC, respectively. MDMV-B = ⁙ and ▩ for FR and ADPC, respectively.

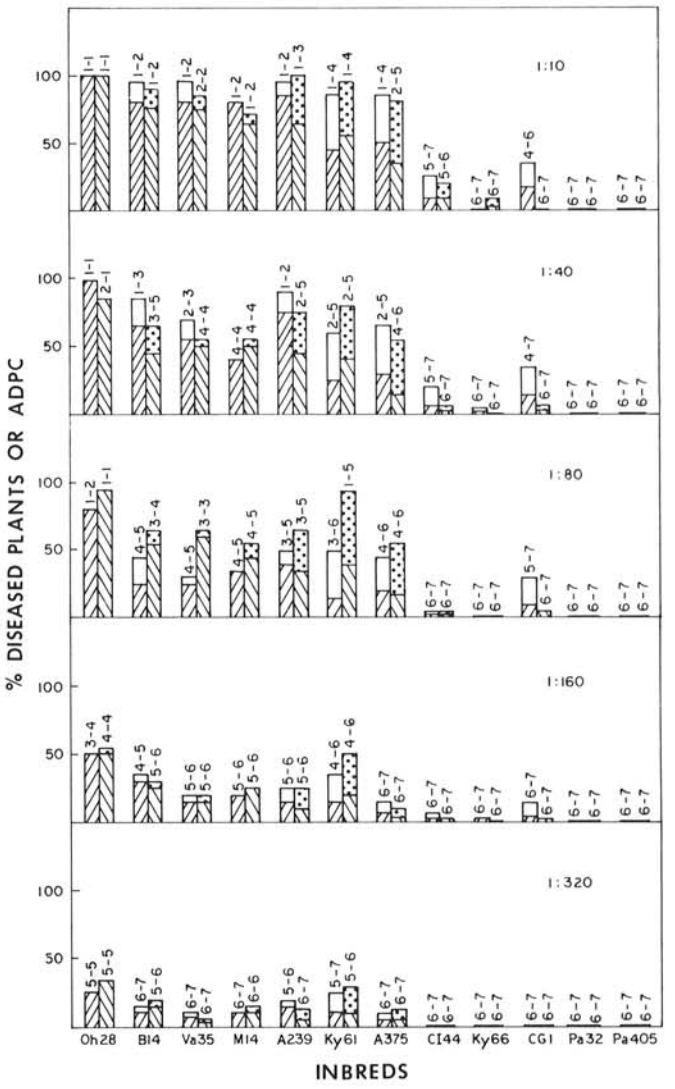

Fig. 4. Responses of inbreds to inoculation with diluted inoculum of maize dwarf mosaic virus (MDMV) strain A or B; first and second bar in each set of an histogram for an inbred, respectively. Ky61 and Ky66 are inbreds Ky61:2335 and Ky61:2500, respectively. Numbers above each bar represent cluster designation by Scott-Knott analyses on final reading (FR) or area under disease progress curve (ADPC). Inbreds at cluster 1 were most susceptible and inbreds at cluster 7 were most resistant. MDMV-A = □ and ▨ for FR and ADPC, respectively. MDMV-B = ⁙ and ▩ for FR and ADPC, respectively.

at all rub inoculations. Only CI.44 showed an increased rate with additional rub inoculations.

Inoculum dilutions. Changes in FR classification with changes in virus titer were significant in most inbreds (Fig. 4). At some dilution, the frequency of infection of susceptible inbreds was reduced. Regardless of inoculum dilution, however, resistance in inbreds CG1, Ky66:2500, Pa32, and Pa405 was not affected. ADPC cluster analyses showed significant differences between inbreds A375 and Va35 at 1:10 and 1:40 but statistically similar levels of disease incidences (i.e., 79 and 85%, respectively for the 1:10 dilution). Rate of disease development, however, in A375 was slower than in Va35 at dilutions of 1:10, 1:40, 1:80, and 1:160.

Inbred responses to MDMV-A and MDMV-B. Inbred Pa405 was most resistant to MDMV-A and MDMV-B in all tests. Inbred Pa32 was susceptible to MDMV-A and MDMV-B only in the repeated inoculation test, and even then, the disease incidence was less than 50%. Percentage of infection for the remaining 10 inbreds exceeded 90 with one virus in at least one of the four tests, occurring most often in the repeated inoculation test. Inbred CG1 appeared more susceptible to MDMV-A than to MDMV-B, and this was most accentuated in the repeated inoculation test (i.e., 100 vs. 56% for MDMV-A and MDMV-B, respectively). Ky66:2500 also appeared more susceptible to MDMV-A; the highest percentage of infected plants (91 vs. 61% to MDMV-A and MDMV-B, respectively) occurred in the repeated inoculation test. In addition to Pa405, inbreds CG1, Ky66:2500, and Pa32 were more resistant than the other inbreds to both MDMV-A and MDMV-B.

Symptoms. Three classes of symptoms to MDMV infections were observed: 1) local lesions observed only on inoculated leaves; 2) unlimited systemic virus invasion into newly emerged leaves expressed as mottles and mosaics; and 3) limited systemic virus invasion into newly emerged leaves expressed as short chlorotic spots, streaks, spindles, and flecks on portions of some leaves. Inbred Pa32 inoculated with MDMV-A typically expressed local lesions on inoculated leaves. Inbreds M14 and Oh28 generally expressed mosaic symptoms on all leaves in all tests with strains A and B. Inbreds A239, A375, B14, Ky61:2335, Ky66:2500, and Va35 expressed varying degrees of limited systemic invasion by strains A and B in some tests.

DISCUSSION

Identifying variables that may influence infection and disease development is a prerequisite for studies of maize virus disease resistance. The methods used to study variables and the criteria for evaluation of host responses are also critical because they influence the interpretation of results (10).

The inoculation treatments tested did not resolve causes for variations among tests resulting from undefined variables, e.g., temperature, relative growth rates among inbreds, nutritional status of plants, or inoculation sites. These or other similar effects apparently affected inbreds A239, Ky61:2335, and Va35 in tests on the effect of age of test plants at 10 days with MDMV-A. The relative susceptibilities of these inbreds to MDMV-A in this particular test were lower than their responses to MDMV-A at the same test age and similar inoculum level in tests involving MDMV-A inoculum dilutions and repeated inoculations. On the basis of overall reactions of these inbreds in the other tests, these three inbreds were very susceptible to MDMV-A.

The inoculation protocol resulting in highest levels of infection was two rubs per inoculation repeated at four 4-day intervals. The mechanism(s) responsible for this may be the addition of more virus at each inoculation time, the inoculation of more suitable infection courts not found in a single inoculation, a response to a changed host physiology in aging or wounding from previous inoculations, or any combinations of these factors.

No generalities were found as reported by others (9,23) that permitted prediction of an inbred response to infection by these MDMV strains. A positive or negative response to one strain did not ensure a similar response to the other strain. Similarly, an inbred's response to any one inoculation protocol was not necessarily the same as that to another inoculation protocol. Although not valid for statistical analysis, it appeared that the classifications of inbreds treated at a similar dilution of inoculum (1:10), age (10–14 days old), and amount of rubbing (two times) were dissimilar when these same conditions were compared in the different inoculation protocols. This variation suggests that at times inbred response to inoculation was more influenced by unidentified variables than by the test protocol. A rub-inoculation test at some standardized plant age, virus dilution, and over a period of time could help substantiate this type of variation and indicate possible solutions.

Statistical analyses. The relative status of resistance of an inbred was influenced by the method of data analysis. ANOVA of data from the FR and then mean separation by Scott-Knott's cluster analysis were most useful for quantitative assessment of inbred susceptibility to MDMV. In the absence of immunity to virus infection or because of the presence of other more desirable agronomic traits, limiting the assessment of an inbred performance to only a final reading may be too severe a selection criterion. Almost all inbreds were classified as susceptible to MDMV-A or MDMV-B by FR analysis in the repeated inoculation test but were separated into different classes by ADPC analysis. In these cases, ADPC analysis was more useful because it identified inbreds with slower symptom development, whereas FR analysis did not differentiate these inbreds. In this sense, ADPC analysis was similar to the disease index rating system of Kuhn and Smith (9). Under field conditions in northern Ohio, epiphytotics of maize dwarf mosaic do not occur until past mid-July (1). Thus, inbreds that possess traits limiting the invasion of MDMV may be just as suitable as an immune inbred for disease control because plant growth and grain production would be completed before the disease were expressed or became economically damaging.

Virus strains and symptom development. Previous experience (W. R. Findley, J. K. Knoke, and R. Louie, *unpublished*) with MDMV-A and MDMV-B indicated that strain B was usually more infectious. More inbreds and more individuals of an inbred were infected when inoculated with strain B than with A. However, mosaic symptoms caused by MDMV-B usually were milder and more difficult to diagnose than those caused by MDMV-A. In tests involving age of test plants at time of inoculation, repeated inoculations at 4-day intervals, and dilution of inoculum, MDMV-B was not consistently more infectious than MDMV-A. Only in the rub-inoculation test was MDMV-B generally more infectious to more inbreds than MDMV-A. In this test, MDMV-B was consistently more infectious to inbreds A239, B14, M14, and Va35 than was MDMV-A. In no instance was MDMV-A more infectious than MDMV-B. Although trends were observed, because treatments instead of virus were the main comparison in these tests, no statistical inferences about the two strains can be made. Furthermore, because factors such as virus titer was not controlled between tests, a higher rate of infectivity by one strain may just as likely to result from differences in virus titer as from differences in virulence.

Resistant and moderately resistant inbreds generally developed local lesions on inoculated leaves followed by limited systemic symptoms of streaks and flecks. Susceptible inbreds usually did not have local lesions and systemic symptoms were mosaics and mottles. However, type of symptom was equally prone to variations among different tests.

Inbreds. Selection of the 12 inbreds in this study was based on the varying degrees of resistance to maize dwarf mosaic observed under field and greenhouse test conditions in Ohio (R. Louie, *unpublished*). No common genetic basis (6) among inbreds was found to aid in predicting their responses to MDMV-A or MDMV-B infection. MDMV was reported as early as 1964 (26), and it probably existed for some time before that. However, the presence or absence of MDMV had little or no impact on the resistance selection processes until that time. There was no correlation between areas where MDMV was prevalent and locations where inbreds with high levels of resistance were developed. The most resistant or nearly immune inbreds were Pa32 and Pa405, both developed in Pennsylvania. Ky66:2500, which has

a fair degree of resistance, came from an indigenous MDMV area. An equally resistant inbred, CG1, was developed in Canada. More recently, because of an awareness of maize dwarf mosaic, good progress has been made in developing inbreds with acceptable levels of resistance and tolerance. T232 and Ga209 are examples of virus-tolerant inbreds developed from southern areas, where MDMV is constantly a problem.

Disease resistance selection strategy. Isolating and identifying the specific gene(s) for MDMV resistance have been difficult. Different researchers (4,16–18) have formulated different genetic hypotheses to explain MDMV resistance. There could be as many gene systems and modifiers as the different test systems proposed. However, different methods used to inoculate plants, different virus strains, different times for reading host reactions, use of disease severity or disease incidence to measure host reaction, and different statistical analyses may modify the apparent genetic expression of disease resistance. This study demonstrated a method of inoculation that would eliminate most disease escapes and allow for the detection of genes not easily affected by variation in environmental conditions. By comparison of data obtained from repeated inoculations with data from other methods, inbreds with genes that are modified by different environmental conditions would also be identified. Also, it showed the importance of methods used to monitor and evaluate resistance under those conditions.

To test elite inbreds for use as sources of major resistant genes not readily affected by different environmental conditions, I recommend inoculating plants by using inoculum diluted not more than 1:10 (w/v) and supplemented with 0.25% 600-mesh silicon carbide, rubbing the leaf surfaces of 10-day-old plants twice with a force of approximately 40 g/cm^2, and repeating the inoculations when plants are 14, 18, and 22 days old. Preferably, the inbreds also should be retested over a period of time. This method will remove many individuals that might, under less rigorous conditions, escape infection. Survivors also may be retested to confirm the selection for disease resistance. In practice, some researchers (4,9,16–18) already regularly inoculate plants more than once in their screening procedures. We also have used a four inoculation series (R. Louie and J. K. Knoke, *unpublished*) with the solid-stream method of inoculation (14) successfully in field plot evaluations. This present study demonstrated the efficacy of such a practice. In testing elite lines, these slight modifications in procedures will reduce the numbers of escapes and improve selection efficiency.

In conclusion, the various hypotheses (e.g., a one-gene, two-gene, etc.) reported for disease resistance in maize to MDMV (4,16–18) may result from determinable genetic differences among different inbreds used as parents. However, the different expressions of resistance may as likely be due to undetermined variability among some inbreds under different test conditions. Among factors affecting a test for virus resistance in inbreds, some more critical ones include virus strains, virus titer, age of test plants, number of rubs during inoculation, and number of repeated inoculations. The most rigorous test of resistance demonstrated in this study was the repeated inoculation protocol. This protocol can be adapted by researchers performing such tests as a standard procedure. However, such standardization will not eliminate all variability from inoculation procedures. What remains is to assess undefined variability statistically and to incorporate this variability into the analysis of the data.

LITERATURE CITED

1. Abt, J. J. 1983. Epidemiology of maize dwarf mosaic in northern Ohio sweet corn. M.S. thesis. Ohio State University, Columbus. 195 pp.
2. Anzola, D., Romaine, C. P., Gregory, L. V., and Ayers, J. E. 1982. Disease response of sweet corn hybrids derived from dent corn resistant to maize dwarf mosaic virus. Phytopathology 72:601-604.
3. Findley, W. R., Louie, R., and Knoke, J. K. 1985. Breeding corn for resistance to corn viruses in Ohio. Pages 52-67 in: Proc. Annu. Corn Sorghum Res. Conf. Publ. 39. Am. Seed Trade Assoc., Washington, DC.
4. Findley, W. R., Louie, R., Knoke, J. K., and Dollinger, E. J. 1976. Breeding corn for resistance to virus in Ohio. Pages 123-128 in: Proc. Int. Maize Virus Dis. Colloq. Workshop. L. E. Williams, D. T. Gordon, and L. R. Nault, eds. Ohio Agric. Res. Dev. Cent., Wooster.
5. Gordon, D. T., and Williams, L. E. 1970. The relationship of a maize virus isolate from Ohio to sugarcane mosaic virus strains and the B strain of maize dwarf mosaic virus. (Abstr.) Phytopathology 60:1293.
6. Henderson, C. B. 1980. Maize Research and Breeders Mannual No. IX. 8th rev. Illinois Foundation Seeds, Inc., Champaign. 111 pp.
7. Jones, R. K., and Tolin, S. A. 1972. Concentration of maize dwarf mosaic virus in susceptible and resistant corn hybrids. Phytopathology 62:640-644.
8. Knoke, J. K., and Louie, R. 1981. Epiphytology of maize virus diseases. Pages 92-102 in: Viruses and Viruslike Diseases of Maize in the United States. D. T. Gordon, J. K. Knoke, and G. E. Scott, eds. South. Coop. Ser. Bull. 247.
9. Kuhn, C. W., and Smith, T. H. 1977. Effectiveness of a disease index system in evaluating corn for resistance to maize dwarf mosaic virus. Phytopathology 67:288-291.
10. Kulkarni, R. N., and Chopra, V. L. 1982. Environment as the cause of differential interaction between host cultivars and the pathogenic races. Phytopathology 72:1384-1386.
11. Loesch, P. J., and Zuber, M. S. 1967. An inheritance study of resistance to maize dwarf mosaic virus in corn (*Zea mays* L.). Agron. J. 59:423-426.
12. Louie, R. 1985. Variations in maize inbred response to maize dwarf mosaic virus inoculation treatments. (Abstr.) Phytopathology 75:1354.
13. Louie, R., Findley, W. R., and Knoke, J. K. 1976. Variation in resistance within corn inbred lines to infection by maize dwarf mosaic virus. Plant Dis. Rep. 60:838-842.
14. Louie, R., Knoke, J. K., and Reichard, D. L. 1983. Transmission of maize dwarf mosaic with solid-stream inoculum. Plant Dis. 67:1328-1331.
15. Madden, L. V., Knoke, J. K., and Louie, R. 1982. Considerations for the use of multiple comparison procedures in phytopathological investigations. Phytopathology 72:1015-1017.
16. Mikel, M. A., D'Arcy, C. J., Rhodes, A. M., and Ford, R. E. 1984. Genetics of resistance of two dent corn inbreds to maize dwarf mosaic virus and transfer of resistance into sweet corn. Phytopathology 74:467-473.
17. Roane, C. W., Tolin, S. A., and Genter, C. F. 1983. Inheritance of resistance to maize dwarf mosaic virus in maize inbred line Oh7B. Phytopathology 73:845-850.
18. Rosenkranz, E., and Scott, G. E. 1984. Determination of the number of genes for resistance to maize dwarf mosaic virus strain A in five corn inbred lines. Phytopathology 74:71-76.
19. Scott, J. A., and Knott, M. 1974. A cluster analysis method for grouping means in the analysis of variance. Biometrics 30:507-512.
20. Scott, G. E., Rosenkranz, E. E., and Nelson, L. R. 1969. Host reaction to maize dwarf mosaic virus from Mississippi and Ohio. Plant Dis. Rep. 53:933-935.
21. Shaner, G., and Finney, R. E. 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. Phytopathology 67:1051-1056.
22. Thompson, D. L., and Hebert, T. T. 1970. Development of maize dwarf mosaic symptoms in eight phytotron environments. Phytopathology 60:1761-1764.
23. Toler, R. W., and Miller, F. R. 1983. Effect of various combinations of inoculation pressure and concentration of varietal disease response of sorghum following spray gun inoculation with maize dwarf mosaic virus. Crop Sci. 23:83-85.
24. Tu, J. C., and Ford, R. E. 1968. Influence of host nutrition on susceptibility of, multiplication in, and symptom expression by corn infected by maize dwarf mosaic virus. Phytopathology 58:1343-1348.
25. Tu, J. C., and Ford, R. E. 1969. Effect of temperature on maize dwarf mosaic virus infection, incubation, and multiplication in corn. Phytopathology 59:699-702.
26. Williams, L. E., and Alexander, L. J. 1965. Maize dwarf mosaic, a new corn disease. Phytopathology 55:802-804.