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The Linkage of Molecular Markers to a Gene Controlling the Symptom Response in Maize to Maize Dwarf Mosaic Virus

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Restriction fragment length polymorphism (RFLP) analysis was used to identify a major gene for symptom response (resistance) to MDMV strain A (MDMV-A) on chromosome 6 of the maize inbred Pa405. RFLP analysis of individual backcross plants of the genotypes (Pa405 × yM14) × yM14 and (Pa405 × K55) × K55, inoculated with MDMV-A, mapped this resistance gene to a region near the centromere of chromosome 6. This gene is tightly linked to and located between the RFLP marker loci, UMC85 and BNL6.29. This gene is essential for any resistance response because all plants lacking this gene rapidly developed generalized mosaic symptoms. In yM14 backcross plants, this gene both delayed symptom appearance and reduced symptom severity. In K55 backcross plants, this gene caused a similar delay in symptom appearance; however, the delayed symptoms were more variable and more severe than in the yM14 backcross plants, indicating that the effect of this gene on symptom development can be influenced by the genotype of the susceptible parents. We propose to designate this gene *Mdm1* for maize dwarf mosaic virus resistance, gene 1.

Additional keywords: *Zea mays*, corn, potyvirus.

Maize dwarf mosaic virus (MDMV) is a widespread and prevalent pathogen of maize in the United States (Gordon *et al.* 1981). In previous genetic studies of MDMV resistance, researchers have attempted to fit progenies of resistant × susceptible crosses into MDMV-induced symptom response classes predicted by various models of gene number and dominance effects (Findley *et al.* 1977; Scott and Rosenkranz 1982; Roane *et al.* 1983; Mikel *et al.* 1984). A coherent synthesis of the results from these studies into a model for the genetic basis of resistance to MDMV is complicated by disease escapes, the use of different resistant and susceptible parental lines, mixtures of MDMV strains, environmental variables inherent in conducting field trials at different locations, and in particular, the use of different symptom classification systems. For example, plants having limited symptoms induced by MDMV have been classified as resistant, intermediate, or susceptible depending on the predilection of the investigator.

The inbred Pa405 is one of the best known sources of resistance to MDMV; it has a symptomless response to inoculation with MDMV strains A and B (Lei and Agrios 1986; Louie 1986). The results of experiments using Pa405 as the resistant parent have been interpreted to support models for resistance conditioned by one (Findley *et al.* 1977), two (Findley *et al.* 1977), three (Mikel *et al.* 1984), or five (Rosenkranz and Scott 1984) genes. It is clear that further progress in understanding MDMV resistance will not be made until the individual gene or genes affecting resistance are identified, their chromosomal map positions determined, and the effects of adding these loci to previously susceptible lines evaluated under controlled conditions.

Chromosome translocation stocks have been used to test specific chromosomes for the presence of resistance genes (Scott and Nelson 1971; Findley *et al.* 1973; Scott and Rosenkranz 1973). Although the results of these studies differ in detail, chromosome 6 has been repeatedly associated with resistance. In addition, linkage of MDMV resistance to the chromosome 6 gene for yellow endosperm (*Y1*) has been reported (Scott 1986). Any attempt to extend this result to a rigorous placement of an MDMV resistance gene(s) on the maize genetic map using morphological traits as genetic markers is greatly restricted by the effect of most morphological markers on the ability to score symptoms induced by MDMV and by the difficulties associated with crossing multiple markers into a specific background without affecting the response to virus infection.

These problems can be circumvented by using restriction fragment length polymorphisms (RFLPs) for genetic markers (Helentjaris *et al.* 1986a, 1986b). RFLPs, representing the differences in lengths of DNA fragments generated by restriction enzyme digestion and being homologous to specific single copy DNA probes, have been used to construct genetic maps of many plant species including: maize (Helentjaris *et al.* 1986a, 1986b), tomato (Helentjaris *et al.* 1986a; Bernatzky and Tanksley 1986), lettuce (Landry *et al.* 1987), potato (Bonierbale *et al.* 1988), and rice (McCouch *et al.* 1988). Once an RFLP map has been constructed for a plant species, then the position of a gene(s) controlling any trait capable of being scored can be placed onto the RFLP map by determining the linkage of expression of that trait with the segregation of the parental alleles of flanking RFLP marker loci.

Young *et al.* (1988) have demonstrated the utility of RFLP analysis in mapping genes for virus resistance in tomato. In maize, RFLPs are present between inbred lines,

and the segregation of alleles of RFLP marker loci in the progeny of crosses of MDMV resistant and susceptible lines can be determined without affecting the ability to score virus symptoms on the same plants.

The goals of this study were to use RFLP analysis to: 1) confirm the presence of a major gene for resistance to MDMV strain A (MDMV-A) on chromosome 6 of Pa405, 2) obtain a chromosomal map position for this gene, and 3) determine the effect of this gene on symptom expression induced by MDMV.

MATERIALS AND METHODS

Pa405 was chosen for the resistant parental line because plants of this inbred have been the most resistant to the development of symptoms induced by MDMV-A in field and greenhouse trials (Mikel *et al.* 1984; Findley *et al.* 1985; Louie 1986 and his unpublished observations). The inbreds K55 and yM14 (a white endosperm conversion of M14) were used as susceptible parental lines because under controlled inoculation and screening procedures (Louie 1986 and his unpublished observations), plants of these lines consistently had generalized mosaic virus symptoms when inoculated with MDMV-A. In addition, both susceptible inbreds have the recessive *y1* allele at the yellow endosperm locus (*Y1*, chromosome 6, proximal long arm, map position 17, [Hoisington *et al.* 1988]). In backcrosses to the susceptible parent, the genotype at *Y1* can be unambiguously identified (white kernels = *y1*/*y1* plants, yellow kernels = *Y1*/*y1* plants), permitting the integration onto the chromosome 6 genetic map of linkages established with the chromosome 6 RFLP marker loci.

Seeds were planted, one per pot, and the plants maintained in the greenhouse. Beginning at the two- or three-leaf stage, each plant was rub-inoculated twice with MDMV-A on four dates at 2–3-day intervals (Louie 1986). Plants were observed daily and then rated for symptoms induced by MDMV-A at 3–6-day intervals starting approximately 1 wk after the initial inoculation (Table 1). The date for the first symptom reading and the intervals between ratings were dependent on the rapidity of symptom development and the growth rates of the plants.

The dates on which symptoms were present and the extent of virus symptoms (generalized mosaic on upper leaves, limited chlorotic streaks on otherwise normal leaves, and so forth) were noted. In experiment 1, inoculated lower and uninoculated upper leaves of all symptomless plants at the fourth rating (22 days after the initial inoculation) were bioassayed for MDMV-A by rub inoculation of Oh28 maize test seedlings with the juice extracted from those samples (Knoke *et al.* 1974). All plants were positive for virus on the inoculated leaves, indicating that there were no escapes from the inoculation procedure. All symptomless plants were negative for virus on the upper leaves, confirming the accuracy of the visual scoring.

After the final rating, DNA was extracted from the youngest leaves by the CTAB procedure (Saghai-Maroof *et al.* 1984). In experiment 1, 3 g of fresh leaf tissue was ground with mortar and pestle in liquid N_2, and 8 ml of CTAB extraction buffer was added. In experiments 2 and 3, leaf tissue was quick-frozen with liquid N_2, lyophilized, and ground with a Wiley mill (40 mesh). Three-tenths gram of lyophilized tissue was used per 8 ml of CTAB extraction buffer. The RFLP probes used in this study were UMC85, UMC59, UMC21 (Hoisington *et al.* 1988), and BNL6.29 (Burr *et al.* 1988). All probes were single copy *Pst*I inserts in pUC plasmids. Southern hybridizations to the DNAs of the parental inbreds and F_1s indicated that suitable probe-enzyme combinations for resolving Pa405 from K55 and yM14 alleles were UMC85 and UMC59-*Hin*dIII, BNL6.29-*Eco*RV, and UMC21-*Bam*HI.

For Southern analysis, 5 μg of DNA (not adjusted for residual RNA) was digested with 20 units of the appropriate restriction enzyme and electrophoresed on 0.8% agarose, Tris-acetate (40 mM Tris, 20 mM $NaC_2H_3O_2$, 2 mM Na_2EDTA, pH 7.8) gels. The DNA in the gel was denatured, neutralized, and transferred to nylon membranes (Zetaprobe, Bio-Rad, Richmond, CA). The membranes were baked at 80° C under vacuum for 2 hr and prehybridized for 4–6 hr at 65° C in 50 mM Tris, pH 8.0, 5× SSC, 2× Denhardt's solution, 10 mM Na_2EDTA, 0.2% sodium dodecyl sulfate (SDS), and 62.5 μg/ml of salmon sperm DNA. Gel-purified inserts or linear plamids were labeled by random priming (Feinberg and Vogelstein 1983). The membranes were hybridized in the same solution as above with the addition of a denatured probe at 0.5 to 2.0×10^6 cpm/ml and 10% dextran sulfate. After hybridization at 65° C for 40 to 48 hr, the membranes were washed five times with 2× SSC, 0.1% SDS at 65° C and then five times with 0.1× SSC, 0.1% SDS at 65° C. Autoradiograms were prepared by exposing the membranes to XAR-5 (Eastman Kodak, Rochester, NY) film at −70° C with intensifying screens.

RESULTS AND DISCUSSION

The MDMV-A induced symptom response of Pa405, K55, yM14, F_1s, and backcrosses to susceptible parents was examined in four experiments. The data for the appearance of virus symptoms in days after initial inoculation are summarized in Table 1. Virus symptoms were not observed in any of the Pa405 plants, while all K55 and yM14 plants developed generalized mosaic symptoms within the first two readings. Of the Pa405 × yM14 F_1 plants, 20 were symptomless at all ratings and one plant showed very limited, chlorotic streaks at the final reading in experiment 1. These results indicate that resistance from Pa405 was dominant in Pa405 × yM14 F_1 plants. The results were not as uniform with K55 × Pa405 F_1 plants. For example, in experiment 2 all six K55 × Pa405 F_1 plants developed virus symptoms by 19 days after initial inoculation, but in experiments 3 and 4 the K55 × Pa405 F_1 plants were either symptomless (7 of 10) or developed symptoms 24–31 days after initial inoculation (3 of 10). The resistance from Pa405 apparently did not have the same high degree of dominance in the F_1 with K55 as with yM14.

The responses of the backcross plants were divided into two reaction classes: the initial reaction (ratings 1 and 2) and the delayed reaction (ratings 3 or later). The reasons for this classification are twofold: all the K55 and yM14 plants developed virus symptoms within the first two ratings

of each experiment, and there was a low number of plants in which virus symptoms were first detected at the third rating (none in experiments 1 and 3, three plants in experiment 2, and four plants in experiment 4). Although the plants from the yM14 or K55 backcrosses gave similar initial responses to MDMV-A, the delayed responses (ratings 3 or later) were very different.

The majority of the (Pa405 × yM14) × yM14 plants that were resistant in the initial response were also symptomless at all subsequent ratings (experiment 1, 27 of 40; experiment 3, 14 of 21; and experiment 4, 22 of 25). In addition, the development of delayed virus symptoms affected only a very limited portion of the leaf area. In contrast, very few (Pa405 × K55) × K55 plants were symptomless at all ratings (experiment 2, 0 of 44; experiment 3, 2 of 20; and experiment 4, 8 of 21). Furthermore, the extent to which delayed symptoms were expressed on individual (Pa405 × K55) × K55 plants was greater and more variable than the delayed symptoms on (Pa405 × yM14) × yM14 plants. These plants had either full mosaic

Table 1. Number of plants first showing maize dwarf mosaic virus strain A symptoms at each rating

Genotypes	Number of plants with symptoms[a]								Symptomless[b]	Total
	Days after initial inoculation									
Experiment 1	7	11	15	22	28					
Pa405	0	0	0	0	0				9	9
yM14	8	1	0	0	0				0	9
Pa405 × yM14	0	0	0	0	1				9	10
(Pa405 × yM14) × yM14 yellow kernels	1	0	0	10	3				24	38
(Pa405 × yM14) × yM14 white kernels	34	2	0	0	0				3	39
	Days after initial inoculation									
Experiment 2	6	8	12	15	19	23				
Pa405	0	0	0	0	0	0			6	6
K55	5	1	0	0	0	0			0	6
K55 × Pa405	0	0	0	4	2	0			0	6
(Pa405 × K55) × K55 yellow kernels	3	1	3	28	9	1			0	45
(Pa405 × K55) × K55 white kernels	35	7	0	3	0	0			0	45
	Days after initial inoculation									
Experiment 3	8	11	14	18	22	25	28	31		
Pa405	0	0	0	0	0	0	0	0	4	4
yM14	4	0	0	0	0	0	0	0	0	4
K55	2	2	0	0	0	0	0	0	0	4
K55 × Pa405	0	0	0	0	0	0	1	1	2	4
Pa405 × yM14	0	0	0	0	0	0	0	0	4	4
(Pa405 × K55) × K55 yellow kernels	0	1	0	2	5	4	5	0	2	19
(Pa405 × K55) × K55 white kernels	1	18	0	0	0	0	1	0	0	20
(Pa405 × yM14) × yM14 yellow kernels	0	0	0	0	2	3	1	0	13	19
(Pa405 × yM14) × yM14 white kernels	14	5	0	0	0	0	0	1	1	21
	Days after initial inoculation									
Experiment 4	11	14	17	20	24					
Pa405	0	0	0	0	0				6	6
yM14	7	1	0	0	0				0	8
K55	5	3	0	0	0				0	8
K55 × Pa405	0	0	0	0	1				5	6
Pa405 × yM14	0	0	0	0	0				7	7
(Pa405 × K55) × K55 yellow kernels	2	1	2	3	7				8	23
(Pa405 × K55) × K55 white kernels	18	3	0	0	1				0	22
(Pa405 × yM14) × yM14 yellow kernels	0	0	2	1	0				21	24
(Pa405 × yM14) × yM14 white kernels	21	2	0	0	0				1	24

[a]The date of each virus rating is given in days after initial inoculation. The actual dates for ratings were determined by the rapidity of symptom appearance on the inbred controls in each experiment.
[b]The plants in the symptomless column did not show virus symptoms at any rating.

symptoms or limited symptoms that progressed to mosaic, or they showed partial recovery from mosaic to limited symptoms.

There was an association of initial symptom response and kernel color for (Pa405 × yM14) × yM14 and (Pa405 × K55) × K55 backcross plants (Table 2). This association indicates that there is a gene or genes in Pa405 located on chromosome 6 near *Y1* affecting symptom response to MDMV-A. To determine a map position for this gene, it was necessary 1) to show that plants in the exceptional classes (yellow kernel, susceptible; white kernel, resistant) arose from recombination between *Y1* and the resistance gene, 2) to show that the genotype of plants in parental classes corresponds to their class assignment, and 3) to define the linkage of the resistance gene with flanking markers. To meet these criteria, the plants in experiments 1, 2, and 3 were analyzed using the RFLP probes UMC85, UMC59, UMC21 (Hoisington *et al.* 1988), and BNL6.29 (Burr *et al.* 1988). They were all located on chromosome 6 near *Y1*.

Table 2. The association of kernel color with the initial response to inoculation with maize dwarf mosaic virus strain A

	Genotype	Kernel color	Resistant[a]	Susceptible[b]
Experiment 1	(Pa405 × yM14) × yM14	Yellow	37	1
		White	3	36
Experiment 2	(Pa405 × K55) × K55	Yellow	41	4
		White	3	43
Experiment 3	(Pa405 × K55) × K55	Yellow	18	1
		White	1	19
	(Pa405 × yM14) × yM14	Yellow	19	0
		White	2	19
Experiment 4	(Pa405 × K55) × K55	Yellow	20	3
		White	1	21
	(Pa405 × yM14) × yM14	Yellow	24	0
		White	1	23

[a]Resistant = plants that did not show any virus symptoms at the first two virus ratings in each experiment.
[b]Susceptible = plants with virus symptoms at the first two ratings of each experiment.

By analyzing the alleles in backcross plants by multiple three point tests (Table 3), the gene order consistent with a minimal number of crossover events was determined to be: UMC85, gene for initial symptom response, BNL6.29, *Y1*, UMC59, and UMC21 (Table 3). This gene order of UMC85, UMC59, and UMC21 is consistent with the published RFLP map (Hoisington *et al.* 1988). In addition to determining a map position for a gene for symptom response, our data place *Y1* and BNL6.29 on the UMC RFLP map (Fig. 1). According to this gene order, all plants in the exceptional classes for initial response and kernel color were recombinant for *Y1* and UMC85. Symptom response was very tightly linked to and mapped between the RFLP markers UMC85 and BNL6.29 with less than 1% recombination detected between the gene for symptom response and either UMC85 or BNL6.29. RFLP analysis of 236 backcross plants revealed only one recombination between the locus for resistance and UMC85 and two recombinations between the locus for resistance and BNL6.29. An example of the Southern hybridization results demonstrating the linkage of this initial resistance to symptom development to the Pa405 allele of UMC85 is shown in Figure 2.

The results from the RFLP analyses of 236 backcross plants rated for symptom responses to MDMV-A infection are completely consistent with a single major dominant gene located on chromosome 6 in Pa405 controlling the

Table 3. Number of crossovers between each neighboring pair of genes or marker loci[a]

	Gene and marker order[b]				
	UMC85 — R/S	— BNL6.29	— *Y1*	— UMC59	— UMC21
Genotype	(Crossovers per number of backcross plants[c])				
Experiment 1					
(Pa405 × yM14) × yM14	0/71	0/71	3/71	3/71	28/71
Experiment 2					
(Pa405 × K55) × K55	1/90	1/90	6/90	0/90	32/90
Experiment 3					
(Pa405 × yM14) × yM14	0/36	0/36	2/36	1/36	17/36
(Pa405 × K55) × K55	0/39	1/39	1/39	1/39	11/39
Total	1/236	2/236	12/236	5/236	88/236

[a]The parental genotypes were: Pa405 = Pa405-specific alleles with the RFLP probes UMC85, UMC59, UMC21, BNL6.29, resistance to initial symptom response (no virus symptoms at ratings 1 and 2 [Table 1]), and yellow kernel color; yM14 and K55 = yM14- or K55-specific alleles with the RFLP probes (virus symptom response at the first two ratings) and white kernel color.
[b]The indicated gene order requires the lowest number of recombinational events to explain the genotypes of the backcross individuals. R/S indicates the gene for resistance or susceptibility to initial symptom development. The data were analyzed as a multiple set of three point crosses. First, the alleles identified at UMC85, UMC59, and UMC21 indicated a gene order of UMC85-UMC59-UMC21 consistent with the published RFLP map (Hoisington *et al.* 1988). The marker locus for BNL6.29 was closely linked to UMC85, and three point analysis indicated an order of UMC85-BNL6.29-UMC59. Expression of kernel color was most closely linked to UMC59, and three point analysis indicated an order of BNL6.29-*Y1*-UMC59. Initial symptom response to maize dwarf mosaic virus was closely linked to the Pa405 alleles of both UMC85 and BNL6.29, and three point analysis indicated an order of UMC85-resistance-BNL6.29.
[c]DNA was isolated from all plants in experiments 1, 2, and 3. In experiment 1, three plants did not yield DNA suitable for Southern analysis; one of these plants was white kernel-resistant. In addition, one DNA preparation from a backcross plant in experiment 3 was lost. Three of the (Pa405 × yM14) × yM14 plants from yellow kernels in each of experiments 1 and 3 had only a Pa405 allele at one or more RFLP loci. The simplest explanation for these plants is that they are from contaminating F_2 seed. These six plants were eliminated from the analysis. The results of the RFLP analysis for all (Pa405 × K55) × K55 plants were consistent with expectations for backcross plants.

initial symptom response. In backcrosses to K55 and yM14, this gene caused at least a delay in the appearance of virus symptoms. In crosses to yM14, this gene apparently also decreased the extent of delayed symptoms. Finally, this gene was required for any resistance response because all plants lacking this gene rapidly developed mosaic symptoms. We propose to name this gene *Mdm1* (MDMV resistance, gene 1). The chromosomal map position determined for *Mdm1* relative to *Y1* and the RFLP markers is shown in Figure 1. This gene must be located near the centromere of chromosome 6 but is not as yet assigned to a chromosome arm. This is the first published assignment of a chromosomal map position in maize for any gene affecting resistance to any viral disease of maize.

The genetic basis for the delayed symptoms observed in this study is difficult to define from these experiments. In analyzing the progenies of crosses between resistant and susceptible inbreds, both Roane *et al.* (1983) and Mikel *et al.* (1984) reported that some inoculated plants had symptoms of limited streaks similar to the delayed-limited symptoms we observed in many (Pa405 × yM14) × yM14 plants. Roane *et al.* (1983) classified these plants as resistant and concluded that resistance was controlled by one dominant gene. Mikel *et al.* (1984) placed plants showing any level of virus symptoms into the susceptible class and arrived at a three gene (one essential plus either of the other two) model for complete resistance. While the development of delayed-limited symptoms in initially resistant *Mdm1*/*mdm1* plants is consistent with the need for additional loci to convert yM14 to the completely resistant response of Pa405, the possible effects of semi-dominance, level of penetrance, genotype-environment interactions, and the genetic distinctness of limited and symptomless plants must also be considered.

The percentage of infected plants, time of symptom appearance, and the extent of delayed symptoms on (Pa405 × K55) × K55 plants varied among experiments, indicating that all variables associated with virus symptom expression have not been completely controlled in our greenhouse screening procedures. In K55 × Pa405 plants, the delay of symptom appearance by *Mdm1* is dominant but the entire resistance response apparently is not, because many of the K55 × Pa405 plants subsequently developed delayed symptoms. Thus, the effect of *Mdm1* on delayed symptoms is affected by the genotype of the susceptible parent. In the majority of genetically defined examples of virus resistance in plants, cultivar resistance is controlled by a single gene (Fraser 1986). Examples of oligogenic resistance are rare and can be difficult to distinguish from monogenic resistance affected by genotype-specific modifiers and genotype-environment interactions (Fraser 1987). Our data demonstrate that initial symptom response to MDMV-A is controlled by a single gene which we have designated *Mdm1*. Our data also suggest that genotype-specific effects influence delayed symptoms in plants with *Mdm1*.

Establishing the linkage of RFLP marker loci to *Mdm1* will aid in both basic and applied research on MDMV resistance. The ability to follow *Mdm1* in crosses based on RFLP associations will assist maize breeders in developing new virus-resistant lines. Plants with *Mdm1* can be identified by RFLP analysis, circumventing ambiguities caused by variable symptom response and differences in the level of resistance in specific genetic backgrounds. The

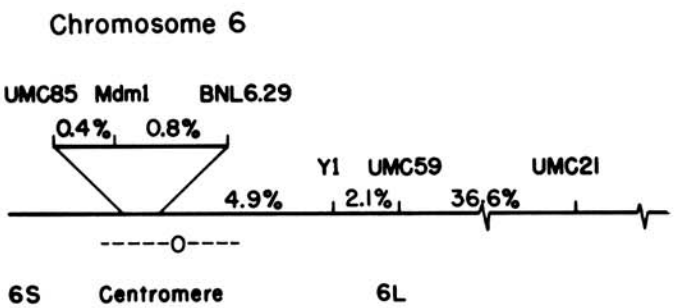

Fig. 1. Chromosomal map determined for the region of maize chromosome 6 containing the Pa405 gene for initial symptom response to inoculation with maize dwarf mosaic virus strain A (MDMV-A). We have designated this gene *Mdm1* (MDMV resistance, gene 1). The map distances are the percentage of backcross plants that were recombinant between the markers (Table 3).

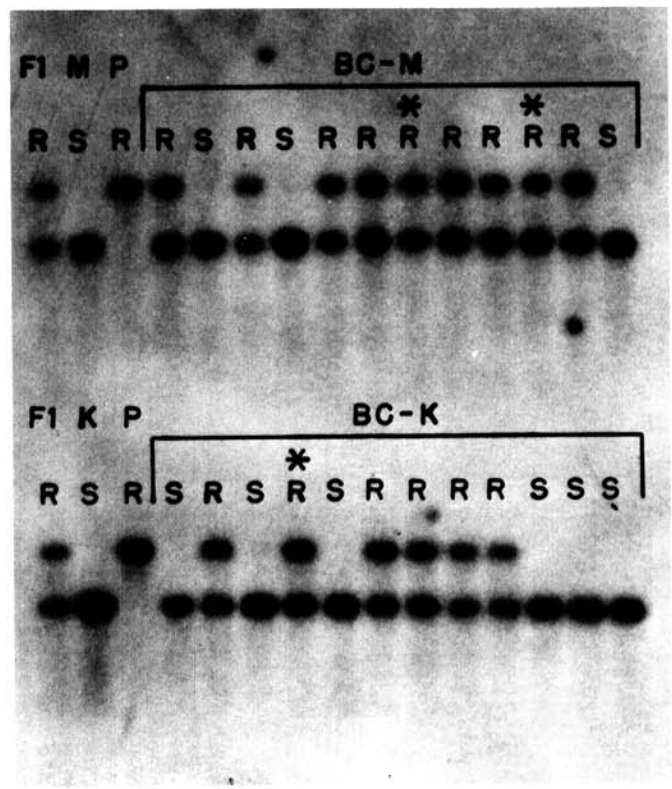

Fig. 2. Linkage of Pa405 allele of UMC85 and resistance to initial symptom development: Southern hybridization analysis of DNA from plants in experiment 3 using the restriction fragment length polymorphism (RFLP) probe UMC85. All DNA samples were digested with the restriction enzyme *Hin*dIII and hybridized with the probe UMC85 as described in the text. Top lanes: F_1 = Pa405 × yM14, M = yM14, P = Pa405, and BC-M = (Pa405 × yM14) × yM14. Bottom lanes: F_1 = K55 × Pa405, K = K55, P = Pa405, and BC-K = (Pa405 × K55) × K55. R = plants scored resistant to initial symptom development; S = plants scored as susceptible to initial symptom development. The * indicates plants from white kernels that were resistant. All plants assayed in this hybridization that were resistant were associated with the presence of the Pa405-specific allele of UMC85, indicating the tight linkage between the resistance gene and the RFLP locus UMC85.

close linkage of RFLP marker loci with *Mdm1* provides a starting point for obtaining a physical map of chromosome 6 surrounding *Mdm1*. With recent advances in mapping and cloning large segments of genomic DNA, it may be possible to use pulse field gradient electrophoresis (Schwartz and Cantor 1984) and yeast artificial chromosome vectors (Burke *et al.* 1987) to "walk" to *Mdm1*.

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