

Inheritance of Resistance to Lettuce Mosaic Virus in Safflower

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ABSTRACT

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The safflower cultivar LMVFP-1 possesses a high level of resistance to lettuce mosaic virus (LMV). Mechanical inoculation of either young or adult plants differentiated the high level of resistance from lower levels. A mild, nonlethal form of systemic necrosis that developed subsequent to local lesions was characteristic of high resistance. The reactions of the F₁, F₂, and BC₁ progenies derived from crosses between LMVFP-1 and a

mosaic-susceptible cultivar, and between LMVFP-1 and a cultivar that developed local lesions and severe systemic necrosis, indicate that the local lesion reaction is conditioned by a single dominant gene, and that the recessive allele conditioning the mosaic reaction is epistatic to a second independently dominant gene that conditions the mild vs systemic necrosis reaction.

Lettuce mosaic virus (LMV) causes a disease of safflower (*Carthamus tinctorius* L.) in Arizona and California that is serious, particularly in breeding nurseries (1). All safflower cultivars tested have reacted to LMV either by the production of local lesions or the development of systemic mosaic (2). Intermediate reactions have not been observed. Systemic necrosis usually develops in plants that produce local lesions. The necrosis may affect the roots, stems, leaves, and flower buds and results in reduced yield of seed (1). Local lesion production is dominant to the systemic mosaic reaction and it is conditioned by a single gene (3). Susceptibility to systemic necrosis caused by LMV has limited the use of several safflower cultivars in breeding programs. In the high-yielding cultivar VFR-1 (4), which has combined resistance to several diseases, LMV causes a severe and usually lethal systemic necrosis.

During routine screening tests of new safflower germ plasm, we observed one cultivar that reacted to LMV by producing local lesions that subsequently developed very mild systemic necrosis. This cultivar, released as LMVFP-1, exhibited a high level of resistance to LMV in field tests in Arizona and California under conditions of natural infection (5). The purpose of this study was to further characterize the reaction of this cultivar to LMV and to determine the inheritance of the reaction.

MATERIALS AND METHODS

Tests were run in controlled-environment rooms with 14,000 lux (1,300 ft-c) of light (cool-white fluorescent, supplemented by incandescent) for 12 hr/day. Light and dark temperatures of 28 and 20 C, respectively, were maintained. Plants were grown, six plants per pot, in steamed soil in porous 15.2-cm-diameter clay pots. Insects were controlled by weekly fumigations.

LMV was maintained on safflower plants of the mosaic-reacting cultivar Nebraska 10 (N10) by inoculating cotyledons of 7-day-old plants. Inoculum was prepared by triturating infected leaves from 4-wk-old plants in an equal amount (w/v) of 0.01 M phosphate buffer, pH 7.2. The inoculum was rubbed with cheesecloth on Carborundum-dusted leaves. The reaction of plants to mechanical inoculation with LMV was used to evaluate levels of resistance and to determine inheritance.

In evaluation tests, the reactions of cultivar LMVFP-1 were

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compared with those of cultivars VFR-1, Pacific 7 (P7), and Nebraska 10 (N10). The cotyledons of 7-day-old plants, the youngest two leaves of 3-wk-old plants, and youngest three leaves of 6-wk-old plants were inoculated.

In the inheritance study, LMVFP-1 was used as the female parent in crosses with N10 and with VFR-1. Parents, F₁ and F₂ generations, and progenies from backcrosses of F₁ hybrids to both parents were tested. Cotyledons of 7-day-old plants were inoculated. New leaves were inoculated 10-14 days later if no symptoms resulted from the first inoculation.

RESULTS

Evaluation tests. The reactions of 100 or more plants of each of the four cultivars at each of the three ages were observed until the plants were either dead or mature. All of the N10 plants developed systemic mosaic in 7-12 days after inoculation and survived to maturity. Local lesions developed on the inoculated leaves of LMVFP-1, VFR-1, and P7, and subsequently the plants developed systemic necrosis. The local lesions that developed on LMVFP-1 4-5 days after inoculation were light brown, averaged 1.0 mm in diameter, and enlarged very little with age. Lesions that averaged 1.75 mm in diameter developed in 5-6 days on VFR-1 and P7. They were dark brown and with aging enlarged to as much as 4.0 mm.

Systemic necrosis appeared 1-4 days after local lesions and it affected the leaves, stems, and, in older plants, flower buds. Its development on VFR-1 and P7 was extensive; it girdled stems, caused whole leaves to collapse, and resulted in death of plants. Systemic necrosis was much less extensive on LMVFP-1, and none of the plants were killed.

Plant age had no effect on type of cultivar reaction and little effect on time required for symptom appearance after inoculation. Subsequent symptom development, however, particularly that of systemic necrosis, was most rapid and severe on the youngest plants.

Inheritance. The F₁ plants of the cross LMVFP-1 × N10 reacted to mechanical inoculation with LMV by developing local lesions on the inoculated leaves followed by mild systemic necrosis. Segregation in the F₂ generation for local lesions with mild systemic necrosis, local lesions with severe systemic necrosis, and systemic mosaic was in good agreement with a 9:3:4 (modified dihybrid) ratio (Table 1).

The F₁ plants of the cross LMVFP-1 × VFR-1 reacted with local lesions and mild systemic necrosis. Segregation for mild and severe systemic necrosis in the F₂ generation was in good agreement with a

TABLE 1. Reaction of Nebraska 10, VFR-1, and LMVFP-1, the F₁ hybrids, F₂, and backcross populations of safflower to lettuce mosaic virus

Parent or cross	Number of plants			Chi-square	P
	Local lesions with systemic necrosis		Mosaic		
	Mild	Severe			
N10	0	0	100		
VFR-1	0	100	0		
LMVFP-1	100	0	0		
LMVFP-1 × N10 F ₁	100	0	0		
LMVFP-1 × N10 F ₂	226	72	99	0.116 ^a	0.95-0.90
(LMVFP-1 × N10) × LMVFP-1	100	0	0		
(LMVFP-1 × N10) × N10	46	42	88	1.511 ^b	0.50-0.30
LMVFP-1 × VFR-1 F ₁	100	0	0		
LMVFP-1 × VFR-1 F ₂	231	81	0	0.154 ^c	0.70-0.50
(LMVFP-1 × VFR-1) × VFR-1	138	144	0	0.127 ^d	0.80-0.70
(LMVFP-1 × VFR-1) × LMVFP-1	100	0	0		

^aGoodness of fit to a 9:3:4 ratio.

^bGoodness of fit to a 1:1:2 ratio.

^cGoodness of fit to a 3:1 ratio.

^dGoodness of fit to a 1:1 ratio.

3:1 ratio, with mild necrosis ranging from predominant to extensive.

Data from the BC₁ progenies from each of the crosses substantiated the F₂ results and the hypothesis that both the local lesions vs systemic mosaic reaction and the mild vs severe systemic necrosis reaction are conditioned primarily by single gene pairs exhibiting complete dominance. When the gene conditioning the systemic mosaic reaction was homozygous recessive it was epistatic to the gene conditioning the systemic necrosis reaction.

DISCUSSION

The reactions of LMVFP-1, VFR-1, and P7 to LMV indicate that the local lesion reaction does not limit systemic spread of the virus. Resistance of LMVFP-1 consists of a mild reaction to systemic spread of the virus and results in minor amounts of necrosis and damage.

The safflower cultivars I studied differed in systemic necrosis reactions much like bean cultivars do to systemic mottle caused by either pod mottle virus or southern bean mosaic virus (6) which produce local lesions and are immune to systemic infection (7). Such cultivars are considered to be commercially resistant because damage in the field is negligible. A similar type of resistance may exist in safflower germplasm. Klisiewicz (3) found three plant

introduction safflower accessions which reacted with local lesions and only a small percentage of the plants developed systemic necrosis. He was able by selection to reduce the percentage of plants that developed necrosis. Although immunity to systemic necrosis would be desirable, LMVFP-1 has a very high level of resistance in the field (5). The more severe reaction of LMVFP-1 in growth-room tests was not surprising because hosts of other virus frequently also react more severely after mechanical inoculation in the greenhouse than in the field (6).

LITERATURE CITED

1. Klisiewicz, J. M. 1965. Identity of viruses from safflower affected with necrosis. *Plant Dis. Rep.* 49:451-454.
2. Klisiewicz, J. M. 1966. Reactions of safflower to lettuce mosaic virus. *Phytopathology* 56:1354-1356.
3. Klisiewicz, J. M. 1967. Inheritance of reactions in safflower to lettuce mosaic virus. *Phytopathology* 57:789-790.
4. Thomas, C. A. 1971. Registration of VFR-1 safflower germplasm. *Crop. Sci.* 11:606.
5. Thomas, C. A., Zimmerman, L. H., and Urie, A. L. 1978. Registration of LMVFP-1 safflower germplasm. *Crop Sci.* 18:1099.
6. Zaumeyer, W. J., and Harter, L. L. 1943. Two new virus diseases of beans. *J. Agric. Res.* 67:305-328.
7. Zaumeyer, W. J., and Thomas, H. R. 1948. Pod mottle, a new virus disease of beans. *J. Agric. Res.* 77:81-96.