

A Common Gene for Resistance to Bean Yellow Mosaic Virus and Watermelon Mosaic Virus 2 in *Pisum sativum*

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ABSTRACT

Genetic studies of inheritance of resistance and susceptibility to bean yellow mosaic virus (BYMV) and watermelon mosaic virus 2 (WMV-2) in *Pisum sativum* with F_1 , F_2 , and F_3 populations and clones of some F_2 families established that a common gene, *mo mo*, governed resistance to BYMV and WMV-2. Thirty-two cultivars possessing the *mo mo* genotype for resistance to BYMV were also completely resistant to WMV-2, and 32 cultivars with the susceptible *Mo Mo* genotype were also

Additional key words: serology, virus relationship.

Reaction of *Pisum sativum* L. to watermelon mosaic virus 2 (WMV-2) is variously reported in the literature, presumably because of differences in susceptibility of the cultivars tested (1, 2, 3, 4, 6, 8, 10, 11). Van Regenmortel et al. (10) failed to infect a given cultivar with six isolates of the virus from South Africa. Natural infection of a number of cultivars grown commercially in western Japan was reported by Inouye (3), but no mention was made of resistance. Schroeder and Provvidenti (8) indicated that susceptibility and resistance in pea depended upon the cultivar used and that resistance was conditioned by the same gene that conferred resistance to bean yellow mosaic virus (BYMV). This paper confirms that earlier report and describes other properties of WMV-2, notably temperature relations in the different genotypes, that may relate it to BYMV (9).

MATERIALS AND METHODS.—Mechanical inoculations followed a technique previously described for BYMV on pea (7). Aphid transmissions were accomplished by caging healthy pea aphids, *Acyrtosiphon pisum* Harris, on WMV-2-infected Ranger pea plants for 24 hr, and allowing the insects to migrate freely to adjacent healthy plants of the same cultivar. Recovery tests of WMV-2 from test plants were made on *Cucurbita pepo* 'Zucchini Elite', which responded with systemic infection, and on *Chenopodium amaranticolor*, which developed local chlorotic lesions. Three isolates of WMV-2 were used: NY69-81B, isolated in 1969 from infected *C. maxima* 'Blue Hubbard' at Auburn, N.Y.; 69-109, supplied by D. E. Purcifull, Florida; and 70-1, supplied by K. A. Kimble, California. Only one isolate of BYMV, isolate 1, originally obtained from D. J. Hagedorn, Wisconsin, was used in these studies. Genetic populations used in these studies were obtained from crosses of BYMV-resistant Bonneville (*mo mo*) with BYMV-susceptible Ranger (*Mo Mo*). Except where otherwise noted, all inoculated plants were incubated at 28 C.

RESULTS AND DISCUSSION.—*Symptoms of WMV-2 in pea.*—Symptoms of WMV-2 in susceptible pea, sim-

ilar to those reported by Inouye (3), appeared initially as a veinal prominence followed by a mild mottle of indefinitely shaped chlorotic areas located along and between the veins. Leaf laminae were often slightly undulate, and curved downward from the midrib. A grayish desiccation of the tissue gradually developed at margins of older leaves and extended over the blade, often into the petioles (Fig. 1). Pod symptoms, mild deformation with areas devoid of a waxy surface, resembled those incited by BYMV. Plants were stunted. Temperatures of 16, 22, and 28 C did not affect symptom expression of WMV-2 in pea, but resulted in disease incubation periods of 15, 12, and 8 days, respectively. All three isolates of WMV-2 incited identical symptoms.

Reactions of BYMV-susceptible and -resistant genotypes to WMV-2.—Pea accessions representing domestic cultivars and foreign introductions of globally widespread origins were tested for reactions to BYMV and the three isolates of WMV-2. Thirty-two lines homozygously susceptible to BYMV were similarly susceptible to WMV-2. An equal number of BYMV-resistant lines were also uniformly resistant to all three isolates of WMV-2. All susceptible varieties, with two notable exceptions, showed a similar degree of stunting with WMV-2. One exception, G460 (a selection from P.I. 201497), exhibited only mild symptoms. The second, G-91 (an enation mosaic-resistant Midfreezer cultivar), developed severe foliage and stem necrosis, culminating in the early death of plants.

Genetic evidence for dual role of mo mo against BYMV and WMV-2.—Corroboration of the dual role of *mo mo* in resisting infections by WMV-2 and BYMV in pea at 28 C was obtained from inoculations with the two viruses of F_1 , F_2 , and F_3 population halves from crosses between *mo mo* and *Mo Mo* genotypes. Clones of some F_2 populations were also tested by inoculating the mother plants with WMV-2 and the rooted cuttings with BYMV. Data summarized in Table 1 show F_1 plants (*Mo mo*) to be completely susceptible to both viruses, and the F_2 populations segre-

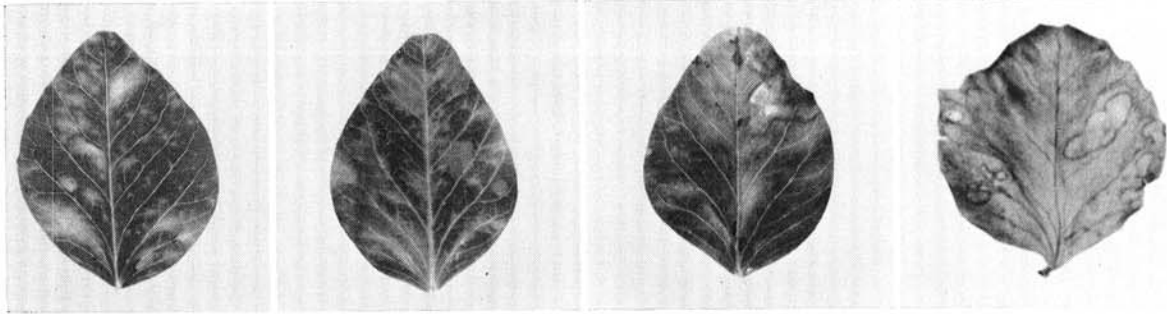


Fig. 1. Typical symptom development of WMV-2 in *Pisum sativum* 'Ranger' (*Mo Mo*) at 28 C, ranging from mild mottling along and between veins to gradual desiccation from leaf margins inward to the eventual necrosis of older leaflets.

gating in a ratio of one resistant:three susceptible, with clonal plants reacting identically to each virus. A common gene for resistance to both viruses was further substantiated by the homozygous resistant reactions of progenies of F₃ plants from 22 families of plants selected as *mo mo* on the basis of their reactions in the F₂ to one or the other virus.

Temperature relations of WMV-2 in Mo mo and mo mo genotypes.—Temperature relations in pea of heterozygotes and resistant homozygotes with WMV-2 were generally comparable to those observed with BYMV (9). *Mo mo* plants inoculated with WMV-2 and incubated at 28 C developed symptoms identical to those of *Mo Mo* plants. After three-four successive leaves on such plants developed typical symptoms, the plants were removed to 16 C. Symptoms continued to appear on the ensuing growth of the susceptible *Mo Mo* plants used as controls, but not on the *Mo mo* plants. After the heterozygotes developed four-five leaves, they were returned to a 28-C regime where typical symptoms appeared on the new growth that developed at the higher temperature. The leaves on the same plants which had developed without symptoms at 16 C now developed a necrosis similar to that described for *Mo Mo* plants at 28 C. In this respect, WMV-2 differed from BYMV in that symptomless tissue of BYMV-inoculated *Mo*

mo plants remained so when removed to the higher temperature, although new growth developed typical symptoms.

Homozygous resistant plants (*mo mo*) inoculated with WMV-2 and incubated at 28 C showed no symptoms, nor was the virus recovered, regardless of the isolate used. If the incubation temperature was kept at 30 C or above, however, symptoms similar to those described for susceptible plants appeared at the ninth node about 15 days after inoculation, a phenomenon similar to that for BYMV on the *mo mo* genotype (5). Unlike the thermal strains of BYMV, which were always milder in *mo mo* than in *Mo Mo* plants, the thermal strains of WMV-2 incited more severe symptoms in either genotype and usually killed the plants. The WMV-2 thermal strain, recovered from such plants, incited equally severe symptoms on all genotypes above 22 C. At 16 C, no symptoms were observed in *mo mo* plants, but infection occurred, as evidenced by a severe necrosis of such plants when removed to 28 C. Although severe on pea, thermal strains were mild on squash and cucumber; nevertheless, they retained their ability to infect pea *mo mo* genotypes when maintained by serial transfer in either *Mo Mo* genotypes or susceptible cucurbits.

Relationship of WMV-2 to BYMV in pea.—Although

TABLE 1. Segregations in F₁, F₂ and F₃ for resistance and susceptibility in *Pisum sativum* to watermelon mosaic virus 2 and bean yellow mosaic virus at 28 C

Cross	Families	Viruses	Individuals		Expect. ratio	Goodness of fit, P	Heterogeneity, P
			Resistant	Susceptible			
	no.		no.	no.			
Ranger (<i>Mo Mo</i>)	—	< BYMV	0	72			
		< WMV-2	0	72			
Bonneville (<i>mo mo</i>)	—	< BYMV	72	0			
		< WMV-2	72	0			
(Ranger × Bonneville) F ₁	8	< BYMV	0	24			
		< WMV-2	0	24			
(Ranger × Bonneville) F ₂	Clones	< BYMV	6	24	1:3	.54	
		< WMV-2	6	24	1:3	.54	
(Ranger × Bonneville) F ₂	2	< BYMV	13	32	1:3	.64	
		< WMV-2	9	35	1:3	.49	0.76
(Ranger × Bonneville) F ₃	22	< BYMV	878	0			
		< WMV-2	872	0			

both viruses are flexuous rods and are vectored to pea by the aphid, *Acyrtosiphon pisum*, cross-protection tests with susceptible Ranger did not indicate a relationship. Van Regenmortel et al. (10), however, showed a distant serological relationship between the two viruses. The production of thermal strains by both viruses is unique, and further suggests relationship. The possibility of two closely linked genes functioning separately against the two viruses is remotely possible but highly improbable, because all cultivars and foreign introductions from widely scattered areas of the world and the large F₃ populations of the genetic studies did not indicate gene linkage. A common genetic basis for susceptibility and resistance to the parasitic action of the two viruses, as described in this paper, might imply a functionally close parasitic relationship not easily demonstrable by conventional methods. In future virus classifications, a common gene that governs parasitic action in a given host might well be considered of some importance in determining relationships among viruses.

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