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Inheritance of resistance to peanut mottle virus in *Phaseolus vulgaris*

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ABSTRACT: One-hundred-and-eleven bean (*Phaseolus vulgaris*) cultivars of domestic and foreign origin reacted identically to the N and the M strains of peanut mottle virus (PMV). Seventy-eight cultivars (70 percent) developed chlorotic or necrotic local lesions, without systemic infection (resistant). Thirty cultivars (27 percent) were infected with local chlorotic or necrotic lesions followed by systemic necrosis and death (susceptible). Three cultivars (3 percent) yielded resistant and susceptible plants (heterogeneous populations). In F_1, F_2, and reciprocal backcross populations derived from crosses between PMV-resistant and -susceptible selections of the cultivar Royalty Purple Pod, resistance to the N strain was conferred by a single, but incompletely dominant gene, designated *Pmv*. No seed transmission of PMV could be demonstrated in progenies of susceptible cultivars because of premature death. The virus was not transmitted in seed of F_2 intermediate resistant plants.

DURING the summer of 1983, a bean field of Royalty Purple Pod located in central New York was found to be severely affected by a disease caused by peanut mottle virus (PMV)[12]. A high percentage of the plants exhibited foliar chlorosis and necrosis, apical and stem necrosis, then died prematurely. Greenhouse tests revealed that this cultivar and the closely related Royal Burgundy were heterogeneous populations for their reaction to PMV[5,12].

The source of the viral infection in bean was traced to a neighboring PMV-infected peanut (*Arachis hypogaea* L.) field[12]. Peanut has been reported to be a major source of this virus[1,2,6,7,9] that, under field conditions, is efficiently spread by aphid species in a non-persistent manner[3]. However, peanuts are seldom grown on a commercial scale in New York State, hence the presence of PMV in one bean field should be considered an isolated and perhaps unusual event. Conversely, in other regions where peanuts are cultivated extensively, this virus can be a serious threat to susceptible bean cultivars[2]. The purpose of this study was to search for sources of resistance to PMV in cultivars of *Phaseolus vulgaris* L., and determine the mode of inheritance.

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Materials and Methods

One-hundred-and-eleven bean cultivars of domestic and foreign origin were tested with the N and M strains of PMV[11], which were available from a previous study[12]. The identity of these strains had been ascertained using electron microscopy, serology, and diagnostic species. An antiserum to PMV was obtained from C. W. Kuhn, University of Georgia. Sixteen plants of each line were inoculated mechanically with each strain by rubbing the Carborundum dusted primary leaves with extracts from PMV-infected Bonneville pea plants. Inocula were prepared by macerating infected leaf tissue with 0.05 M phosphate buffer (K^+) (p*H* 8.5). Inheritance studies were conducted with the N strain using F_1, F_2, and reciprocal backcross populations derived from crosses between two selections of Royalty Purple Pod (RPP), a resistant (RPP-14) and a susceptible (RPP-15) selection. Irrespective of symptoms, each test plant was assayed for PMV infection using ELISA (enzyme-linked immunosorbent assay), or in some cases, by back inoculations to Black Turtle Soup beans. Plants were considered resistant if they were free of symptoms and pertinent assays were negative. All test plants were maintained in an insect-free greenhouse at 28–30° C.

Results

The following cultivars were found to be susceptible: Aurora, Antigua, Arriaga, Black Turtle Soup, Blanco Japonese, Bush Blue Lake 94, Dubbele Witte, Green McCaslan, Greensleeves, Jamapa, Kentucky Wonder, Kentucky Wonder Wax, Midnight, Negro Pacoc, Negro Patzicia, Pico, Pioneer, ICTA Quetzal, Rabia de Goto, San Martin, Savor, Scotia, Spartan Half Runner, ICTA Suchitán, Sulphur, Sutton Pink, ICTA Tamazulapa, Tennessee Green Pod, ICTA Jutiapan, and Xacpael. Plants of these cultivars reacted to both strains of PMV with local chlorotic or necrotic lesions, veinal browning, systemic mottle, apical and stem necrosis, and death.

Resistant cultivars included: Amanda, Astro, Avalanche, Barbuni, Black Valentine, Bluecrop, Bonanza, Bountiful, Brittle Wax, Burly, Bush Blue Lake 47, Bush Blue Lake 274, Bush Blue Lake GV2, Cacahuate, California Light Red Kidney, Canario 101, Canario 107, Checkmate, Cherokee Wax, Contender, Commodore, Del Ray, Dwarf Horticultural, Early Gallatin, Executive, French Horticultural, Gaelic, Gourmand, Horizon, Kamiakin, Kardinal, King Horticultural, Gator Green, Giant Stringless Pod, Golden Crop, Goldrush, Great Northern U.I. 31, Great Northern U.I.123, Great Northern 1140, Harvester, Hi Style, Jacob's Cattle, Landreth Stringless Green Pod, Lazer, Improved Golden, Jubila, King Horn Wax, Michelite 62, Monroe, Pencil Pod Wax, Pencil Pod Black Pod Wax, Pinto 111, Pinto 114, Provider, Redkloud, Redkote, Red Mexican 34, Red Mexican 35, Roma, Romano, Ruddy, Sacramento Red Kidney, Sanilac, Slendergreen, Slenderette, Slim Green, Spartan Arrow, Sprite, Soldier, Stringless Green Refugee, Tenderlake, Tendercrop, Tendergreen, Tenderette, Topcrop, Top Notch Golden Wax, Vitagreen, and White Kidney. With both strains of PMV, plants of these cultivars responded with localized infection ranging from inconspicuous to prominent chlorotic or necrotic lesions with or without veinal browning, but the virus failed to move systemically.

Cultivars exhibiting heterogeneous reactions: Royalty Purple Pod, Royal Burgundy, and Picker. The percentage of systemically resistant plants in these cultivars varied with the seed lots and ranged from 8 to 30%. Susceptible plants died prematurely.

Inheritance of resistance: Following inoculation with the N strain of PMV, plants of RPP-14 reacted with necrotic lesions and veinal browning confined to the inoculated leaves (systemically resistant). Plants of RPP-15 developed a similar local reaction, but the virus moved systemically, causing chlorotic mottle, apical and stem necrosis, wilting, and death (susceptible). Plants of (RPP-14 × RPP-15)F_1 responded with local necrotic lesions and systemic chlorotic patches exhibiting prominent browning of veins and veinlets. Affected leaves showed malformation, and some abscissed prematurely. Most of the pods were distorted, with green streaks over the purplish color. Plants of F_2 populations segregated approximately in the ratio of 1:2:1. Thus, one-fourth of the plants developed only local infection and produced pods of normal size, shape, and color (resistant). Another one fourth reacted with local and systemic necrosis and died prematurely (susceptible). Half of the population responded with local necrotic spots and systemic chlorotic areas in which necrosis spread dendritically along veins and veinlets. However, the intensity of these symptoms varied from plant to plant and within a given plant. Most of the pods were malformed and exhibited color break, and plants were slightly stunted (incompletely resistant). Further evidence of incomplete dominance was obtained in the backcrosses to the resistant and susceptible parents. Progeny of (RPP-14 × RPP-15) × RPP-14, segregated in a ratio of 1 systemically resistant to 1 with intermediate resistance, whereas that of (RPP-14 × RPP-15) × RPP-15, segregated 1 intermediate resistant to 1 susceptible. The data in Table I reveal that resistance to PMV in Royalty Purple Pod is conditioned by a single but incompletely dominant gene, to which the symbol *Pmv* (peanut mottle virus) is assigned. Since a number of PMV-resistant bean cultivars are susceptible to bean common mosaic virus (BCMV), blackeye cowpea mosaic virus (BlCMV), and cowpea aphid-borne mosaic virus (CabMV), *Pmv* must be considered a distinct genetic entity, independently inherited from the genes (*I*, *Bcm*, and *Cam*) conferring resistance to these viruses[13,14].

Seed transmission tests: Plants of susceptible cultivars inoculated with PMV died prematurely, thus, no seed were available for virus transmission studies. No virus was detected in 435 plants derived from seed of F_2 plants classified

as intermediate resistant. These F_3 plants were grown in a greenhouse, and all were assayed for viral infection.

Discussion

PMV is a destructive pathogen causing diseases of economic importance in beans, cowpeas (*Vigna unguiculata* (L.) Walp.), lupines (*Lupinus albus* L. and *L. angustifolius* L.), peanuts, and soybeans (*Glycine max* (L.) Merr.)[1,2,6,7,9,10]. Symptoms caused by PMV in beans can be easily attributed to those incited by other well known bean viruses: e.g., the necrosis-inducing strains of BCMV[14]. This may well explain the paucity of reports on its natural occurrence in the bean. Bock et al.[4] isolated PMV from the lima bean (*P. lunatus* L.) grown in the highlands of Kenya. Behncken and McCarthy[2] found it infecting bean fields in Queensland (Australia), where cultivars bred for local conditions were particularly affected. Symptoms were identical to those observed in New York, consisting of foliar chlorosis, necrosis, stunting, and premature death. Pods that were forming at the time of infection shrivelled and produced no seed, whereas those that were well formed at the time of infection yielded normal seed. These researchers also reported a low percentage of PMV transmission in seed of Australian cultivars, but they attributed the spread of PMV into bean fields to the migration of aphids from nearby infected peanut fields[2]. Most researchers are in agreement regarding infected peanut seed as the major source of this virus[1,2,6,7,9,12]. In peanuts, the incidence of seed transmission ranges from 0.3 percent to 8.5 percent[1], but a lower percentage (<1 percent) also occurs in cowpeas[7], and two lupine species[10]. The virus also is known to infect some forage legumes and weed species[3,8].

Effective control of PMV in beans can be achieved by: 1) complete isolation of bean fields from peanut fields; 2) planting of buffer crops between bean and neighboring peanut fields; 3) destruction of volunteer peanut plants from previous crops; and 4) use of resistant cultivars. This investigation has shown that a large number of the cultivars tested (70 percent) were resistant to PMV, but a significant number (30 percent) were susceptible or contained both resistant and susceptible individuals. Consequently, only resistant cultivars should be considered for areas in which this virus is prevalent. Resistance to PMV is simply inherited and the *Pmv* gene can be employed very effectively in a breeding program utilizing the backcross method.

Table I. Segregation ratios in cross and backcross populations of two selections of the bean cultivar Royalty Purple Pod (RPP), resistant (RPP-14) and susceptible (RPP-15), to the N strain of peanut mottle virus

Populations	No. plants			Expected ratio	Goodness-of-fit (*P*)
	resistant	intermediately resistant	susceptible		
RPP-14	36	0	0		
RPP-15	0	0	40		
(RPP-14 × RPP-15)					
F_1	0	25	0		
F_2	40	104	42	1:2:1	0.40
BC (F_1 × RPP-14)	30	36	0	1:1	0.41
BC (F_1 × RPP-15)	0	33	29	1:1	0.57

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