Theor Appl Genet (1987) 74:595–600

Inheritance of resistance to potato y viruses in *Phaseolus vulgaris* L.

1. Two independent genes for resistance to watermelon mosaic virus-2

M. M. Kyle [1] and R. Provvidenti [2]

[1] Department of Plant Breeding, Bradfield Hall, Cornell University, Ithaca, NY 14853, USA; and Department of Horticultural Science, N.Y.S. Agricultural Experiment Station, Geneva, NY 14456, USA
[2] Department of Plant Pathology, N.Y.S. Agricultural Experiment Station, Geneva, NY 14456, USA

Received April 21, 1987; Accepted May 22, 1987
Communicated by G. S. Kush

Summary. Resistance to watermelon mosaic virus-2 in *Phaseolus vulgaris* L. is conferred by two distinct dominant alleles at independent loci. Based on segregation data one locus is designated *Wmv*, the other, *Hsw*. The dominant allele *Wmv* from cv. Great Northern 1140 prevents systemic spread of the virus but viral replication occurs in inoculated tissue. In contrast, *Hsw* confers both local and systemic resistance to WMV-2 below 30 C. At higher temperatures, plants that carry this allele in the absence of modifying or epistatic factors develop systemic veinal necrosis upon inoculation with the virus that results in rapid death. Pathotype specificity has not been demonstrated for either allele; both factors confer resistance to every isolate tested. A temperature-sensitive shift in epistasis is apparent between dominant alleles at these loci. Because *Hsw* is very tightly linked if not identical to the following genes for hypersensitivity to potyviruses *I*, (bean common mosaic virus), *Bcm*, (blackeye cowpea mosaic virus), *Cam*, (cowpea aphid-borne mosaic virus) and *Hss* (soybean mosaic virus), parental, reciprocal dihybrid F_1 populations, and selected F_3 families were inoculated with each of these viruses and held at 35 C. F_1 populations developed vascular necrosis completely or primarily limited to inoculated tissue, while F_3 families from WMV-2-susceptible segregates were uniformly susceptible to these viruses. The relationship between *Hsw*, *Wmv* and other genes for potyvirus resistance suggest patterns in the evolution of resistance and viral pathogenicity. Characterization of the resistance spectrum associated with each factor provides an additional criterion to distinguish genes for plant virus resistance.

Key words: Plant virus resistance – WMV-2 – *Phaseolus vulgaris* L. – *I* gene

Introduction

Although the genetics of resistance to obligate fungal plant pathogens has received considerable attention, similar understanding of the relationships among genes for plant virus resistance is less complete. Our studies in bean and pea have identified a number of single genes for resistance to plant potyviruses (Kyle et al. 1986 b; Provvidenti et al. 1982, 1983; Provvidenti 1987 a, b). This family, named for the type member potato virus Y, is the largest and probably most destructive grouping of plant viruses and is also characterized by extensive interrelationships among its members (Hollings and Brunt 1981). The identification of genes for resistance to potyviruses, and their organization in the genome reveal patterns in the evolution of host resistance and viral pathogenic diversity. The objective of the present study was to further define the genetic basis for resistance to watermelon mosaic virus-2 (WMV-2) in bean, and to evaluate the relationship between resistance to this potyvirus and resistance to related potyviruses.

Watermelon mosaic virus-2, one of fourteen or more potyviruses that infect the common bean, causes significant economic damage to cucurbit crops worldwide and has been recovered from a number of leguminous species including *Phaseolus vulgaris* L. (Edwardson 1974). The virion is a flexuous rod that encapsidates a monopartite single-stranded RNA genome of approximately 10,000 nucleotides (Purcifull and Hiebert 1984). Although most leading commercial bean cultivars are resistant to WMV-2, this virus remains a potential threat where susceptible cultivars are grown because of the ubiquity of overwintering hosts and the presence of efficient vectors.

Provvidenti (1974) screened 280 bean lines, 160 domestic varieties and breeding lines and 120 plant introductions for resistance to WMV-2 and identified two classes of resistance. One class, represented by cv. Great Northern 1140 (GN 1140), did not develop systemic symptoms of infection at any temperature. The second type of response, apparent in Black Turtle-1 (BT-1), a selection from cv. Black Turtle Soup, was a hypersensitive reaction resulting in rapid veinal necrosis and apical death at temperatures above 30 C. Local responses to mechanical inoculation were not determined and the allelic relationship of these factors was not defined, therefore gene symbols were not assigned. The present study provides evidence to designate two independent dominant genes for resistance to WMV-2 in *P. vulgaris*, *Hsw*, *Wmv*, and defines the relationship between *Hsw*, *Wmv*, and four other dominant factors, *I* (Ali 1950), *Hss* (Kyle et al. 1986 b), *Bcm* and *Cam* (Provvidenti et al. 1983) for potyvirus resistance in bean.

Materials and methods

Germplasm and genetic populations

Genetic studies were based on populations that derive from the two resistant parental lines, GN 1140 and BT-1, and a second selection from the cv. Black Turtle Soup, BT-2 (Provvidenti 1983), that is susceptible to WMV-2. Each resistant parent was crossed with BT-2 and with each other to generate F_1, F_2, testcross, and reciprocal backcross populations. These populations and selected F_3 families were tested at 25 C and 35 C for local and systemic response to WMV-2. Parental lines, F_1, and selected F_3 families were also inoculated with four related potyviruses, bean common mosaic virus (BCMV), blackeye cowpea mosaic virus (B1CMV), cowpea aphid-borne mosaic virus (CAbMV), and soybean mosaic virus (SMV).

Viral cultures and inoculation

Inocula for mechanical transmissions were prepared from infected foliar tissue homogenized in a 0.05 M K_2HPO_4 buffer pH 8.8. Viral cultures were maintained on the following hosts: WMV-2 isolate 62–76 on *Cucurbita pepo* cv. Seneca Hybrid, BCMV NY 15 type strain from M. A. Silbernagel, Prosser, WA and NY 15 isolate NY 68–95 (Kyle and Provvidenti 1987) on *P. vulgaris* cv. California Light Red Kidney, SMV isolate 76-6 on *P. vulgaris* BT-2, and B1CMV-F1a and CAbMV-Mor on *Vigna unguiculata* cv. California Blackeye. The absence of contamination was monitored by immunodiffusion, host indexing, and symptomatology on a range of susceptible genotypes. Antisera were prepared by Uyemoto et al. (1972) to BCMV, by Taiwo and Gonsalves (1982) to B1CMV and CAbMV, and obtained from Drs. H. A. Scott, U. of Arkansas (WMV-2) and J. B. Sinclair, University of Illinois (SMV).

Seedlings at the primary leaf stage were dusted with 400 mesh Carborundum, rubbed with inoculum, rinsed, and held for 10 days after inoculation either in the greenhouse at 25–28 C or at 35 C in a growth chamber illuminated 12 h daily with incandescent and fluorescent lights. Test plants were examined periodically and scored for the presence of pinpoint necrotic lesions, systemic veinal necrosis or mosaic symptoms. Mock-inoculated and uninoculated controls were included routinely. In some cases, back inoculations were used to discern latent infection, local or systemic.

Results

Reaction of parental lines to WMV-2 at 25 C and 35 C

At 25 C, plants of GN 1140 and BT-1 were completely resistant to systemic infection with WMV-2. However, the virus was recovered from primary leaves of GN 1140 which developed a mild mottle 10–14 days after inoculation. In contrast, BT-1 remained free from local infection. The susceptible parent, BT-2, developed a prominent systemic mosaic similar to symptoms incited by another potyvirus, bean yellow mosaic virus (BYMV).

At 35 C, the response of GN 1140 and BT-2 was similar to that at 25 C, but BT-1 developed pinpoint necrotic lesions on inoculated leaves within three days. Necrosis spread rapidly through the vascular tissue resulting in stem streaks and apical death. The temperature-sensitivity of this distinctive response to WMV-2 is limited to mechanical or aphid transmission. If BT-1 or other hypersensitive lines were approach-grafted to a systemically infected plant, systemic necrosis appeared regardles of ambient temperature.

Reaction of F_1, F_2 and selected F_3 populations to WMV-2 at 25 C

The response of F_1 populations from the crosses (BT-2 × GN 1140) and (BT-2 × BT-1) was identical to the respective resistant parent upon inoculation with WMV-2, consistent with full dominance from each source. Segregation data from F_2 populations were consistent with the 3 resistant: 1 susceptible ratio expected for resistance conferred by a single factor (Table 1).

Reciprocal F_1 populations from the cross (GN 1140 × BT-1) were also completely resistant to WMV-2 at 25–28 C, hence no maternal effect was observed. Under these conditions, expression of resistance to WMV-2 in reciprocal F_1 populations was identical to the BT-1 parent. When (GN 1140 × BT-1) F_2 populations were inoculated with WMV-2 and held at 25–28 C, segregation data suggested a 15 resistant: 1 susceptible ratio, indicating the existence of two independent dominant genes. Progenies of susceptible F_2 plants were tested to confirm homozygosity at both loci.

We propose the symbol *Wmv* for the gene derived from GN 1140. This gene prevents systemic spread of WMV-2 at any temperature, while allowing the virus to replicate in inoculated leaves. For the factor found in BT-1, we propose the designation *Hsw* for the domi-

Table 1. Segregation data for WMV-2 resistance in populations derived from *Phaseolus vulgaris* cv. BT-1, BT-2 and GN 1140 at 25C

Populations	No. of plants[a]		Expected ratio	Goodness of fit P
	R	M		
BT-1	15	0		
GN 1140	15[b]	0		
BT-2	0	15		
(BT-2×BT-1)F_1	36	0		
(BT-2×BT-1)F_2	148	44	3 : 1	0.456
(BT-2×GN)F_1	14[b]	0		
(BT-2×GN)F_2	121[b]	37	3 : 1	0.660
(GN×BT-1)F_1	26	0		
(GN×BT-1)F_2	111	7	15 : 1	0.891

[a] R = resistant, no systemic recovery of virus; M = systemic mosaic

[b] Local infection, apparent as mild mottling of inoculated tissue, was evident

Table 2. Segregation data for WMV-2 resistance in populations derived from *Phaseolus vulgaris* cv. BT-1, BT-2, and GN 1140 at 35C

Populations	No. of plants				Expected ratio	Goodness of fit P
	R[a]	LN	SN	M		
BT-1	0	18	0	0		
GN 1140	19	0	0	0		
BT-2	0	0	20	0		
(GN×BT-1)F_1	0	20	0	0		
(BT-1×GN)F_1	0	20	0	0		
GN×(GN×BT-1)F_1	26	17	0	0	1 : 1	0.177
BT-1×(GN×BT-1)F_1	0	19	22	0	1 : 1	0.655
BT-2×(GN×BT-1)F_1	15	11	19	16	1 : 1 : 1 : 1	0.545
	R or LN		*SN*	*M*		
(GN×BT-1)F_2	39		12	5	12 : 3 : 1	0.568

[a] R = resistant, no systemic infection; LN = local necrosis; SN = systemic necrosis; M = mosaic

nant temperature-sensitive necrotic response to WMV-2. The absence of local infection in F_1 plants heterozygous at both loci indicated that under these conditions, *Hsw* is completely epistatic to *Wmv*.

Reaction of (GN 1140×BT-1) F_1, F_2, testcross and backcross populations to WMV-2 at 35 C

Twenty plants from reciprocal F_1 populations (GN 1140×BT-1) were inoculated with WMV-2 and held at 35 C. Within 72 h numerous pinpoint necrotic lesions developed on inoculated leaves. However, the virus did not move systemically. Thus, the response of the dihybrid F_1 was intermediate, including both necrosis associated with the allele *Hsw* and systemic resistance conferred by *Wmv*. These results indicate a temperature-dependent shift in epistasis at these loci. At 25 C, *Hsw* is completely epistatic to *Wmv;* at 35 C *Wmv* becomes partially epistatic to *Hsw*. This observation was utilized to confirm hypotheses regarding the inheritance of resistance to WMV-2 and the relationship between these factors.

By screening at 35 C, two additional classes were distinguished in populations segregating at the *Wmv* and *Hsw* loci. Based on segregation data presented in Table 2, the following genotypes can be assigned to these classes: plants which show no systemic spread of the virus and no local necrosis (R) (*Wmv/Wmv* or *Wmv/wmv hsw/hsw*), local necrosis without systemic spread (LN) (*Wmv/Wmv* or *Wmv/wmv Hsw/Hsw* or *Hsw/hsw*), systemic necrosis (SN) (*wmv/wmv Hsw/Hsw* or *Hsw/hsw*), and mosaic (M) (*wmv/wmv hsw/hsw*). Thus, the ratio obtained when F_1 plants, heterozygous at both loci, are testcrossed to BT-2 fits the 1 R : 1 LN : 1 SN : 1 M ratio expected for two dominant genes assorting independently.

The backcross to the GN 1140 parent segregated 1 R : 1 LN (Table 2) which supports the hypothesis that necrosis is not observed in plants of the genotype (*Wmv/Wmv* or *Wmv/wmv hsw/hsw*), while individuals with at least one dominant allele at each locus develop local necrosis. The low goodness-of-fit $P = 0.18$ is probably due to incomplete penetrance of the *Hsw* allele. The complete absence of individuals with mosaic or systemic necrosis is as predicted, since each plant in this backcross population should carry at least one dominant allele at the *Wmv* locus.

Conversely, the backcross (BT-1×(GN 1140×BT-1)) segregated 1 SN : 1 LN. This is consistent with the prediction that each individual in this population should carry at least one dominant allele at the *Hsw* locus while segregating 1 *Wmv/wmv*: 1 *wmv/wmv*. F_2 data were recorded in three classes with an expected ratio of 12 : 3 : 1. Segregation data from these populations inoculated with WMV-2 confirm that resistance to this virus is conferred by two independent factors, *Hsw* and *Wmv*.

Reaction of (GN 1140×BT-1) F_1 and selected F_3 families to four related legume potyviruses

Hsw and *Wmv* cannot be distinguished on the basis of WMV-2 pathotype specificity; however, they can be differentiated with other legume potyviruses. When

Table 3. Local and systemic reactions of parental lines and F_1 populations of *Phaseolus vulgaris* to WMV-2, BCMV NY 15, BlCMV, CAbMV, and SMV

Population	Temperature	WMV-2	BCMV	BICMV	CAbMV	SMV
GN 1140	25, 35C	M/R[a,b]	M/R	M/S	M/R	M/R
BT-1	25C	R/R	R/R	R/R	R/R	SN
	35C	SN	SN	SN	SN	SN
BT-2	25, 35C	M	M	M	M	M
(BT-2 × BT-1) F_1	25C	R/R	R/R	R/R	R/R	SN
	35C	SN	SN	SN	SN	SN
(GN × BT-1) F_1	25C	R/R	R/R	R/R	R/R	LN
	35C	LN	LN[c]	LN	LN	LN

[a] Local/systemic reaction
[b] R = resistant, no virus recovered; LN = local necrosis; SN = systemic necrosis; S = latent infection; M = mosaic
[c] An occasional plant collapsed with systemic necrosis

BT-1 or any line that is hypersensitive to WMV-2 was inoculated with BCMV NY 15, BlCMV, CAbMV or SMV, an identical hypersensitive response was observed which uniformly cosegregated with *Hsw* in large populations (Kyle et al. 1986a). GN 1140 never developed systemic or local necrosis with any of these viruses regardless of incubation conditions after inoculation. However, local infection of inoculated leaves was observed with BCMV NY 15, CAbMV, or SMV; BlCMV was recovered systemically (Table 3).

Because of the epistatic interaction of the alleles *Hsw* and *Wmv* with WMV-2, it was of interest to determine whether *Wmv* similarly limits systemic hypersensitivity to the four potyviruses invariably associated with hypersensitivity to WMV-2 conferred by *Hsw*. F_1 plants from the cross (GN 1140 × BT-1) were individually inoculated with BCMV, BlCMV, CAbMV, and SMV and held at 35 C for 10 days, then transferred to the greenhouse for three weeks. Although there was no evidence that *Wmv* is associated with multiple virus resistance, the F_1 did not develop systemic necrosis with BlCMV, CAbMV or SMV, and only an occasional plant inoculated with BCMV NY 15 isolate NY 68–95 collapsed with systemic necrosis. With each virus, the necrotic reaction was completely or primarily limited to inoculated tissue.

When eleven WMV-2-susceptible F_3 families were inoculated with the four potyviruses, every individual in each family developed symptoms of systemic viral infection. These results indicate that every F_2 plant susceptible to WMV-2 and therefore homozygous recessive at *Wmv* and *Hsw*, were also homozygous the following recessive factors for potyvirus susceptibility: *i, cam, bcm, smv,* and *hss*. Among WMV-2-susceptible lines, Provvidenti (1974) noted an "unusual range of susceptibility" to potyviruses including BCMV, BYMV, SMV and a cowpea seedborne mosaic virus. Results obtained from these segregating populations are consistent with this report.

Discussion

These experiments clearly establish that resistance to WMV-2 in *P. vulgaris* is conferred independently by dominant alleles at two unlinked loci. Based on segregation data from these experiments, we have designated the loci *Wmv* and *Hsw*. The dominant allele at *Wmv* prevents systemic spread of WMV-2 at any temperature. However, local infection of inoculated tissue does occur in these genotypes. At temperatures below 30 C, *Hsw* confers local and systemic resistance to WMV-2. However, at higher temperatures, lines carrying *Hsw* without modifying or epistatic factors collapse with systemic veinal necrosis. Neither allele, *Hsw* or *Wmv*, exhibits pathotype specificity since both factors confer resistance to every isolate tested to date (Provvidenti 1974; unpublished results). Previous work has established that *Hsw* is very tightly linked if not identical to the following dominant factors for viral hypersensitivity in Lamprecht's linkage group III, *I*, (BCMV), *Bcm*, (BlCMV), *Cam*, (CAbMV) and *Hss* (SMV) (Kyle and Dickson 1987; Kyle et al. 1986b). Further experiments are in progress to elucidate the genetics of hypersensitivity to these five potyviruses in bean. The linkage relations of *Wmv* have not been defined but there is no evidence that resistance to potyviruses, other than WMV-2, is associated with this allele. In light of this observation, the similarity of response of the (GN 1140 × BT-1) F_1 to inoculation at 35 C with BCMV, BlCMV, CAbMV, and SMV was noteworthy since *Wmv* confers resistance to WMV-2 alone. The uniformity of the F_1 response to these five viruses could be explained if *Wmv* is actually interacting with a

single gene from BT-1 conferring hypersensitivity to five viruses including WMV-2, rather than five distinct but tightly linked factors.

Studies of the inheritance of resistance to potyviruses in bean, pea and soybean have revealed relationships among these genes. Two distinct patterns are emerging, both illustrated by the results presented here concerning alleles for resistance to WMV-2 in bean. First, several pairs of clearly distinct genes for resistance to the same potyvirus have been described recently. Buss et al. (1985) reported two unlinked dominant genes for resistance to peanut mottle virus in *Glycine max*. In pea, Provvidenti has identified two pairs of unlinked recessive genes, one pair (*sbm-2, sbm-3*) for resistance to pea seed-borne mosaic virus-L1 (PSbMV-L1), and another for clover yellow vein virus (CYVV) resistance (*cyv, cyv-2*) (Provvidenti 1987a, b). None of these recessive factors in pea are pathotype-specific, therefore they can only be distinguished genetically. *Wmv* and *Hsw* have been identified as a pair of independent genes for resistance to WMV-2 in bean that are not pathotype-specific, but that can be distinguished phenotypically at both low and high temperatures. Furthermore, a temperature-sensitive shift in epistasis between dominant alleles at these loci is evident in the F_1 from the dihybrid cross inoculated with WMV-2. Identification of conditions which allow genotypes with dominant alleles at both loci to be distinguished from either parental genotype provided a particular advantage in genetic analysis of these loci. Conditional epistasis has not been reported previously for plant virus resistance genes (Fraser 1986).

A second pattern emerging from studies of genetic factors that determine the outcome of potyviral infection concerns the association of resistance to one virus with similar resistance to related viruses. As mentioned above, *Hsw* invariably cosegregates with hypersensitivity to BCMV, BlCMV, CAbMV, and SMV (Kyle and Dickson 1987; Kyle et al. 1986a, b). Cosegregation of resistance to two or more potyviruses has been noted in several crop species including *Solanum* spp., pepper, bean, and pea (Cockerham 1970; Cook 1960; Kyle et al. 1986c; Provvidenti et al. 1983). In pea, resistance to WMV-2 and BYMV is conferred by a single recessive gene, *mo*, on chromosome 2 (Schroeder and Provvidenti 1971). Moreover, recent results indicate that one of each pair of genes for resistance to CYVV and PSbMV-L1 are also very tightly linked to *mo*. Thus, a cluster of at least three factors *mo, cyv,* and *sbm-2* have been identified in pea that confer resistance to four potyviruses including WMV-2. Recessive resistance to BCMV NL-8 and pea mosaic virus (PMV) also cosegregates with these genes (unpublished results).

It is interesting to note that resistance to WMV-2 is associated with resistance to BCMV in both bean and pea. Although these two viruses are somewhat related serologically and identical in particle morphology, they differ considerably with respect to biological properties. WMV-2 has a very broad host range including at least 160 species in 23 dicotyledonous families and is not transmitted through seed or pollen in legumes (Purcifull and Hiebert 1984). Both cytoplasmic and nuclear inclusion bodies are present in infected tissue, therefore it has been classified to subgroup III (Christie and Edwardson 1977). Bean common mosaic virus is transmitted through seed and pollen in *P. vulgaris* and has a narrow host range primarily limited to legumes (Bos 1971; Drijfhout 1978). Only cytoplasmic pinwheel inclusions are observed in BCMV-infected tissue (Christie and Edwardson 1977), which may indicate significant differences in genome structure between WMV-2 and BCMV. Nevertheless, the similarity of the temperature-sensitive hypersensitive response to these viruses associated with *Hsw* in bean, and the association of resistance with identical inheritance in both bean and pea suggests some relationship between these pathogens not revealed by other criteria. To evaluate this possibility, heteroduplexes are being constructed for analysis in the electron microscope from pairwise hybridization of genomic RNA with full-length cDNA from WMV-2, BCMV, and related potyviruses affected by resistance genes which map at or tightly linked to the *Hsw* locus. This approach has been used successfully for a number of bacterial and animal virus families, e.g. the polyomavirus family (Vogel et al. 1986). Results should reveal both the extent and arrangement of sequence homology between these two pathogens and may confirm the existence of relationships suggested by Mendelian analysis of genes for plant virus resistance.

Acknowledgements. The authors wish to acknowledge Drs. M. H. Dickson, G. A. Marx and N. F. Weedon for critical review of the manuscript, and David Benscher for technical assistance. M. M. K. was supported by the National Science Foundation Graduate Fellowship Program.

References

Ali MA (1950) Genetics of resistance to the common bean mosaic virus in the bean (*Phaseolus vulgaris* L.). Phytopathology 40:69–79

Bos L (1971) Bean common mosaic virus. CMI AAB Descriptions of Plant Viruses No 73

Buss GR, Roane CW, Tolin SA, Vinardi TA (1985) A second dominant gene for resistance to peanut mottle virus in soybean. Crop Sci 25:314–316

Christie RG, Edwardson JR (1977) Light and electron microscopy of viral inclusions. Fla Agric Exp Stn Mono Ser 9

Cockerham G (1970) Genetical studies on resistance to potato viruses X and Y. Heredity 25:309–348

Cook AA (1960) Genetics of resistance in *Capsicum anuum* to two virus diseases. Phytopathology 50:364–367

Drijfhout E (1978) Genetic interaction between *Phaseolus vulgaris* L. and bean common mosaic virus and its strains. Agric Res Rep 872. Centre for Agricultural Publishing and Documentation, Pudoc, Wageningen

Edwardson JR (1974) Host ranges of the potato virus Y family. Fla Agric Exp Stn Mono Ser 5

Fraser REF (1986) Genes for resistance to plant viruses. CRC Crit Rev Plant Sci 3:257–294

Hollings M, Brunt A (1981) Potyviruses. In: Kurstak E (ed) Handbook of plant virus infections: comparative diagnosis. Elsevier/North Holland Biomedical Press, New York, pp 731–807

Kyle MM, Dickson MH (1987) Linkage of hypersensitivity to five viruses with the *B* locus in bean. J Hered (in press)

Kyle MM, Dickson MH, Provvidenti R (1986a) Linkage analysis of hypersensitive resistance to four viruses in *Phaseolus vulgaris* L. Bean Improv Coop Annu Rep 29: 80–81

Kyle MM, Dickson MH, Provvidenti R, Munger HM (1986b) *I*, a major gene for broad spectrum virus resistance in *Phaseolus vulgaris* L. HortScience 21:874

Kyle MM, Provvidenti R (1987) A severe isolate of bean common mosaic virus NY 15. Bean Improv Coop Annu Rep 30:87–88

Kyle MM, Provvidenti R, Dickson MH (1986c) A gene and clustered gene family for broad spectrum virus resistance in bean and pea. Agron Abstr, p 69

Provvidenti R (1974) Inheritance of resistance to watermelon mosaic virus-2 in *Phaseolus vulgaris*. Phytopathology 64: 1448–1450

Provvidenti R (1983) Two useful selections of the bean cultivar Black Turtle Soup for viral identification. Bean Improv Coop Annu Rep 26:73–75

Provvidenti R (1987a) Inheritance of resistance to clover yellow vein virus in *Pisum sativum*. J Hered 78:126–128

Provvidenti R (1987b) Inheritance of resistance to a lentil strain of pea seedborne mosaic virus in *Pisum sativum*. J Hered 78: (in press)

Provvidenti R, Gonsalves D, Ranalli P (1982) Inheritance of resistance to soybean mosaic virus in *Phaseolus vulgaris*. J Hered 73:302–303

Provvidenti R, Gonsalves D, Taiwo MA (1983) Inheritance of resistance to blackeye cowpea mosaic and cowpea aphid-borne mosaic viruses in *Phaseolus vulgaris*. J Hered 74: 60–61

Purcifull D, Hiebert E (1984) Watermelon mosaic virus-2. CMI AAB Descriptions of Plant Viruses No 293

Schroeder WT, Provvidenti R (1971) A common gene for resistance to bean yellow mosaic virus and watermelon mosaic virus-2 in *Pisum sativum*. Phytopathology 61:846–848

Taiwo MA, Gonsalves D (1982) Serological grouping of blackeye cowpea mosaic and cowpea aphid-borne mosaic viruses. Phytopathology 72:583–589

Uyemoto JK, Provvidenti R, Schroeder WT (1972) Serological relationship and detection of bean common and bean yellow mosaic viruses in agar gel. Ann Appl Biol 71: 235–242

Vogel F, Rhode K, Scherneck S, Bastien C, Delmas V, Feunteun J (1986) The hamster papovavirus: evolutionary relationships with other polyomaviruses. Virology 154: 335–343