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Inheritance of resistance to potyviruses in *Phaseolus vulgaris* L. III. Cosegregation of phenotypically similar dominant responses to nine potyviruses

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Abstract We have identified monogenic dominant resistance to azuki bean mosaic potyvirus (AzMV), passionfruit woodiness potyvirus-K (PWV-K), zucchini yellow mosaic potyvirus (ZYMV), and a dominant factor that conditioned lethal necrosis to Thailand Passiflora potyvirus (ThPV), in *Phaseolus vulgaris* 'Black Turtle Soup 1'. Resistance to AzMV, PWV-K, ZYMV, watermelon mosaic potyvirus, cowpea aphid-borne mosaic potyvirus, blackeye cowpea mosaic potyvirus, and lethal necrosis to soybean mosaic potyvirus and ThPV cosegregated as a unit with the *I* gene for resistance to bean common mosaic potyvirus.

Key words Plant virus resistance · Potyvirus · *I* gene *Phaseolus vulgaris* · BCMV

Introduction

There are many examples of single dominant and recessive genes in plants known to confer resistance to plant viruses. In many cases, these genes are specific for a particular virus or viral strain (for reviews see Fraser 1986, 1990). We have initiated studies to identify plant genes or tightly-linked gene clusters that confer resistance to more than one distinct virus. In addition to the obvious practical significance of host genes for broad-spectrum plant virus resistance, they may be useful in addressing questions about mechanisms of viral-host interactions.

The clearest examples of monogenic broad-spectrum viral resistance reported thus far involve members of the *Potyviridae*, the largest family of plant viruses (Kyle and Provvidenti 1993 b). Potyviruses often have relatively narrow overlapping host ranges, and it is common that two or three different potyviruses will cause economically significant losses in a single crop species. Thus, breeding for multiple resistance to potyviruses has been a major goal in the improvement of a number of crop species. While the remarkable biological continuum of variants and strains in the *Potyviridae* has made unequivocal taxonomic assignments difficult (Bos 1992), potyviruses can be arranged into clear subgroups based on coat-protein (CP) sequence similarity (Shukla and Ward 1988; Ward and Shukla 1991; Rybicki and Shukla 1992; Ward et al. 1992). In a phylogenetic analysis of CP sequences, one subgroup was identified to contain bean common mosaic potyvirus (BCMV) Serotypes A and B, soybean mosaic potyvirus (SMV), watermelon mosaic potyvirus (WMV), zucchini yellow mosaic potyvirus (ZYMV), Thailand Passiflora potyvirus (ThPV) (Benscher et al. 1993), and passionfruit woodiness virus-K (PWV-K) (Gough and Shukla 1992). HPLC peptide profile data of azuki bean mosaic potyvirus (AzMV) CP and blackeye cowpea mosaic potyvirus (BlCMV) CP demonstrated that these viruses also fall in this same subgroup (McKern et al. 1992). It is plausible that viruses with considerable similarity in sequence may also share processes involved in pathogenicity. If so, then single genes, or clusters of related genes with conserved function in plants, could interrupt these common processes, resulting in a resistant response to several related viruses.

An example of simply-inherited broad-spectrum viral resistance occurs in *Phaseolus vulgaris* genotypes that contain the *I* gene (Kyle 1988). Previous work has demonstrated that *P. vulgaris* 'Black Turtle Soup' selection BT-1 contains the *I* gene for dominant resistance to BCMV (Ali 1950; Provvidenti 1983) and also dominant resistance to four potyviruses that are related to BCMV, namely WMV, BlCMV, cowpea aphid-borne mosaic potyvirus (CAbMV), and PWV-K (Provvidenti et al. 1983; Kyle and Provvidenti 1987 a; Provvidenti et al. 1992; Provvidenti 1993). Subsequently, it has been shown that in a small population of large-seeded beans the dominant resistant responses to BCMV, WMV, BlCMV and CAbMV cosegregated in resistant by susceptible crosses (Kyle and Dick-

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son 1988). Finally, a dominant necrotic reaction to SMV that is phenotypically very similar to breakdown of resistance at high temperature by BCMV, WMV, BlCMV, and CAbMV, also cosegregated with the *I* gene (Kyle and Dickson 1988; Kyle and Provvidenti 1993 a). The objective of the present study was to determine if resistance to additional related potyviruses was associated with the *I* gene in *P. vulgaris*.

Materials and methods

Germplasm and genetic populations

Parental lines included two selections from *P. vulgaris* 'Black Turtle Soup' (Provvidenti 1983); Black Turtle 1 (BT-1) which is resistant to BCMV NY15, BlCMV, CAbMV, and WMV, and Black Turtle 2 (BT-2) which is susceptible to these viruses (Provvidenti et al. 1983; Kyle and Provvidenti 1987 a). To determine the mode of inheritance and possible linkage of the responses to AzMV, ThPV, PWV-K, and ZYMV, reciprocal crosses were made between BT-1 and BT-2 to generate F_1, F_2, F_3 and reciprocal backcross populations.

For evaluation of linkage between the *I* gene, which confers temperature-dependent resistance to serotype-B isolates of BCMV, and the responses to the other potyviruses, individual F_3 families were produced from randomly-selected (BT-2×BT-1) F_2 plants. A set of at least 12 individuals from each F_3 family was planted and inoculated with a single virus. Consequently, each F_3 family was screened with each of the nine viruses, BCMV NY 15, BlCMV, CAbMV, WMV, SMV, AzMV, ThPV, PWV-K, and ZYMV.

Viral isolates and inoculation

Viral isolates of BCMV NY15 isolate NY 68–95 (ATCC PV897) (Kyle and Provvidenti 1987 b), PWV-K, ThPV, WMV isolate NY 62-76, SMV isolate NY 76-6, BlCMV-Fl, and CAbMV-Mor were obtained from R. Provvidenti, NYSAES, Cornell University, Geneva, NY and maintained on *P. vulgaris* 'BT-2'. An isolate of AzMV was provided by M. Silbernagel, USDA WSU-IAREC, Prosser, WA. and was maintained on *P. vulgaris* 'BT-2' and 'California Light Red Kidney'. ZYMV-CT from R. Provvidenti, Geneva, NY was maintained on *Cucurbita pepo* 'PMR Caserta'. The purity of the viral isolates was monitored routinely with ELISA, host index tests, and evaluation of characteristic symptomatology on a range of susceptible host genotypes.

Inoculum was prepared for each of the viruses by grinding systemically infected tissue in 50 mM potassium phosphate buffer, pH 8.4, with carborundum added directly to the inoculum. Inoculum was applied using a pestle to fully-expanded primary leaves. Plants were held either in the greenhouse at about 25°C with supplemental lighting or at high temperature in a growth chamber at 33–35°C with 16 h of fluorescent and incandescent illumination. Test plants were scored visually 7–10 days after inoculation for the presence of necrotic lesions, systemic veinal necrosis, or systemic mosaic. Uninoculated and susceptible controls were routinely included. Virus was recovered from test plants by back inoculation to *P. vulgaris* 'BT-2' or *C. pepo* 'PMR Caserta'. To evaluate the nature of resistance, BT-1 plants were approach-graft inoculated with systemically-infected BT-2 plants. BT-1 plants were visually scored for the presence of vascular necrosis in upper trifoliates 15–20 days after grafting.

The F_1, F_2, backcross and testcross populations inoculated with ZYMV were maintained at 33–35°C where symptoms on the inoculated leaves of susceptible plants were more prominent. In addition to visual assessment of ZYMV disease symptoms, plants were assayed using direct double-antibody sandwich ELISA (Clark and Adams 1977) for the presence of viral CP in the inoculated leaves 10 days after inoculation. Sap was extracted in 1 ml of phosphate-buffered saline (PBS) with a motor-driven stainless-steel tissue extractor. Each sample was centrifuged at 10000 rpm for 7 min using a microcentrifuge. A 100 µl aliquot of sap from each sample was added to wells of a microtiter plate previously coated for at least 12 h at 4°C with ZYMV immunoglobin from D. Purcifull, University of Florida. After at least 12 h at 4°C, 100 µl of antibody conjugated to alkaline phosphatase was added and the plates were incubated as before. Finally, phosphatase substrate (Sigma) was added. After plates had developed, they were read in a Biorad Model 2550 EIA Reader.

Results

Inheritance of resistance to AzMV

Reaction of parental lines to AzMV. At either 25°C or 33°C, the susceptible parental line, BT-2, developed chlorotic lesions on inoculated leaves, followed by systemic infection resulting in severe mosaic, stunting and distortion of upper trifoliate leaves. At 25°C, BT-1 plants were completely resistant to both local and systemic infection. No symptoms of any type were observed, and virus was not recovered from inoculated or uninoculated tissue. At 35°C, however, BT-1 plants developed pinpoint necrotic lesions on inoculated leaves by 3–4 days post-inoculation (dpi). Necrosis then spread rapidly through the vascular tissue, resulting in dark stem streaks and apical death (Table 1). When healthy BT-1 plants were approach-graft inoculated to AzMV-infected BT-2 plants, systemic necrosis developed on BT-1 plants, regardless of ambient temperature.

Reaction of F_1, F_2 and reciprocal backcross populations to AzMV. The phenotypic response of reciprocal (BT-1× BT-2) F_1 populations to mechanical inoculation with AzMV was indistinguishable from the response of the BT-1 parent (Table 2 a) and the F_2 populations segregated approximately three resistant to one susceptible plant at 25°C. These data are consistent with a single dominant gene for resistance to AzMV. This was further supported by data

Table 1 Local and systemic response of parental lines and F_1 populations of *P. vulgaris* to mechanical inoculation with BCMV NY15, AzMV, ThPV, PWV-K, and ZYMV

Population	Temperature	BCMV NY15	AzMV	ThPV	PWV-K	ZYMV
BT-1	25°C	R[a]	R	SN	R	R
	35°C	SN[b]	SN	SN	R	R
BT-2	25°C/35°C	M[c]	M	M	M	CL/R[d]
(BT-2×BT-1) F_1	25°C	R	R	SN	R	R
	35°C	SN	SN	SN	R	R

[a] R, resistant, no virus recovered from inoculated or uninoculated tissue
[b] SN, pinpoint necrotic lesions on inoculated leaves, followed by lethal systemic veinal necrosis
[c] M, systemic mosaic or mottle
[d] CL/R, chlorotic lesions on inoculated leaves, no systemic infection

Table 2a Segregation data for AzMV resistance in populations derived from *P. vulgaris* 'BT-1' and 'BT-2' at 25°C

Populations	No. of plants		Expected ratio	Goodness of fit
	R[a]	M[b]		$P_{\alpha=0.05}$
BT-1	25	0		
BT-2	0	25		
(BT-2×BT-1) F_1	18	0		
(BT-1×BT-2) F_1	16	0		
(BT-2×BT-1) F_2	100	26	3:1	0.26
BT-2×(BT-2×BT-1)	13	17	1:1	0.48
BT-1×(BT-2×BT-1)	25	0		

[a] R, resistant, virus cannot be recovered from inoculated or uninoculated tissue
[b] M, systemic mosaic

Table 2b Segregation data for lethal systemic veinal necrosis to ThPV in populations derived from *P. vulgaris* 'BT-1' and 'BT-2' at 25°C

Populations	No. of plants		Expected ratio	Goodness of fit
	SN[a]	M[b]		$P_{\alpha=0.05}$
BT-1	25	0		
BT-2	0	25		
(BT-2×BT-1) F_1	22	0		
(BT-1×BT-2) F_1	14	0		
(BT-2×BT-1) F_2	68	24	3:1	0.81
BT-2×(BT-2×BT-1)	19	21	1:1	0.76
BT-1×(BT-2×BT-1)	12	0		

[a] SN, lethal systemic veinal necrosis

Table 2c Segregation data for PWV-K resistance in populations derived from *P. vulgaris* 'BT-1' and 'BT-2' at 25°C

Populations	No. of plants		Expected ratio	Goodness of fit
	R[a]	M[b]		$P_{\alpha=0.05}$
BT-1	25	0		
BT-2	0	25		
(BT-2×BT-1) F_1	19	0		
(BT-1×BT-2) F_1	13	0		
(BT-2×BT-1) F_2	83	24	3:1	0.55
BT-2×(BT-2×BT-1)	18	18	1:1	0.98
BT-1×(BT-2×BT-1)	8	0		

[a] R, resistant, virus cannot be recovered from inoculated or uninoculated tissue
[b] M, systemic mottle

Table 2d Segregation data for local infection of ZYMV in populations derived from *P. vulgaris* 'BT-1' and 'BT-2' at 35°C

Populations	No. of plants		Expected ratio	Goodness of fit
	R[a]	CL[b]		$P_{\alpha=0.05}$
BT-1	25	0		
BT-2	0	25		
(BT-2×BT-1) F_1	25	0		
(BT-1×BT-2) F_1	16	0		
(BT-2×BT-1)F_2	58	21	3:1	0.75
BT-2×(BT-2×BT-1)	13	11	1:1	0.69
BT-1×(BT-2×BT-1)	14	0		

[a] R, resistant, virus cannot be recovered from inoculated or uninoculated tissue
[b] CL, chlorotic lesions on inoculated leaf and virus recovered, no systemic infection

from testcross and backcross populations (Table 2 a). Plants of the backcross [BT-1×(BT-2×BT-1)] remained free of local and systemic infection, while plants of the testcross [BT-2×(BT-2×BT-1)] segregated approximately one resistant plant to one susceptible plant.

Inheritance of lethal systemic necrosis to ThPV

Reaction of parental lines to ThPV. Infection of BT-2 by ThPV caused chlorotic lesions on the inoculated leaves by 5 dpi, followed by a systemic mottle and leaf curling. BT-1 plants inoculated with ThPV developed necrotic lesions on inoculated leaves followed by lethal systemic veinal necrosis within 5 days. This response was observed in BT-1 plants held at 25°C, 35°C, and also in BT-1 plants approach-graft inoculated with ThPV-infected BT-2 plants (Table 1).

Reaction of F_1, F_2 and reciprocal backcross populations to ThPV. (BT-2×BT-1) F_1 plants inoculated with ThPV developed lethal veinal necrosis identical to the response observed in BT-1 (Table 2 b). No maternal effect was noted in reciprocal F_1 plants. Inoculated F_2 plants segregated approximately three plants with lethal necrosis to one plant with systemic mosaic, suggesting that the lethal necrotic response to ThPV is conditioned by a single dominant gene. Testcross individuals [BT-2×(BT-2×BT-1)] segregated approximately one plant with lethal necrosis to one plant with systemic symptoms, while all individuals in the [BT-1×(BT-2×BT-1)] backcross population developed lethal systemic veinal necrosis (Table 2 b).

Inheritance of resistance to PWV-K

Reaction of parental lines to PWV-K. In a manner similar to the viruses described above, PWV-K systemically infected BT-2 plants causing chlorotic spotting and some necrotic spots on inoculated leaves, followed by systemic mottle and leaf curling. BT-1 plants were completely resistant to PWV-K at 25°C and 35°C. No symptoms were observed and virus was not recovered from inoculated or uninoculated leaves (Table 1). Nevertheless, when BT-1 plants were inoculated via approach-graft with a PWV-K-infected BT-2 plant, lethal systemic veinal necrosis was observed in the BT-1 plant.

Reaction of F_1, F_2 and reciprocal backcross populations to PWV-K. (BT-2×BT-1) F_1 plants remained free of local

and systemic symptoms after inoculation with PWV-K similar to the BT-1 parent (Table 2 c). No maternal effect was noted. The F_2 population segregated approximately three resistant to one susceptible plant, while the backcross [BT-1×(BT-2×BT-1)] progeny were all resistant to PWV-K, and the testcross [BT-2×(BT-2× BT-1)] progeny segregated approximately one resistant to one susceptible plant (Table 2 c). These data support the hypothesis that resistance to PWV-K is conferred by a single dominant gene.

Inheritance of resistance to ZYMV

Reaction of parental lines to ZYMV. Primary leaves of BT-2 plants inoculated with ZYMV developed local chlorotic ringspots that turned necrotic 7–10 dpi. Symptoms on inoculated leaves were more prominent and developed more quickly at 35°C. ZYMV CP was detected in the inoculated leaves of BT-2 plants using ELISA, and virus was recovered in back-inoculation tests. The infection, however, remained local. Virus could not be recovered from the petioles of BT-2 inoculated leaves, uninoculated leaves, or the stem. In contrast, in BT-1 plants, viral CP was not detected using ELISA and virus was not recovered from the inoculated leaves or uninoculated tissue (Table 1). In all cases the presence or absence of visible symptoms corresponded exactly with the detection of viral CP using ELISA.

Reaction of F_1, F_2 and reciprocal backcross populations to ZYMV. Reciprocal (BT-2×BT-1) F_1 populations were resistant to local infection by ZYMV, while F_2 populations segregated approximately three resistant plants to one plant with local infection (Table 2 d). The backcross [BT-1×(BT-2×BT-1)] plants were resistant to ZYMV, while testcross [BT-2×(BT-2×BT-1)] populations segregated approximately one resistant plant to one plant with local infection (Table 2 d). These data are consistent with a single dominant gene for resistance to ZYMV.

Linkage analysis of dominant resistance and/or lethal systemic necrosis to selected potyviruses

To evaluate the relationship between the dominant gene, *I*, that confers resistance to BCMV Serotype B, and the dominant responses to AzMV, ThPV, PWV-K, and ZYMV, 98 (BT-2×BT-1) F_3 families were screened with each of these viruses to determine whether the family was homozygous resistant/necrotic, segregating, or homozygous susceptible to each virus. No families that differed in response to one or more of the five viruses were observed, and segregation followed a 1:2:1 ratio as expected. Thus, all 17 families that were homozygous for the dominant *I* gene, based on their responses to BCMV NY15, were also homozygous for dominant resistance to AzMV, PWV-K and ZYMV and homozygous for the dominant response of systemic necrosis to ThPV (Table 3 a). Similarly, in the 58 families where the *I* gene was segregating, the dominant responses to the other four potyviruses were also segregating. Finally, the 23 families that were susceptible to BCMV were also uniformly susceptible to AzMV, ThPV, PWV-K, and ZYMV (Table 3 a). These results suggest that there is at least tight linkage, and do not rule out identity, of the genetic determinants of the responses to these five potyviruses.

Table 3 a Reaction of 98 (BT-2×BT-1) F_3 families to inoculation with BCMV NY15, AzMV, ThPV, PWV-K, and ZYMV

No. of F_3 families			Expected ratio	Goodness of fit
R/SN[a]	Seg[b]	Susc[c]		$P_{\alpha=0.05}$
17	58	23	1: 2: 1	0.14

[a] All families resistant to BCMV NY15 were also resistant to AzMV, PWV-K, ZYMV, and systemic necrosis was observed with ThPV
[b] All families found segregating for resistance to BCMV NY 15 were also segregating for resistance or systemic necrosis to the other four viruses
[c] All families susceptible to BCMV NY15 were uniformly susceptible to the other four viruses

Table 3 b Reaction of 1 000 (BT-2×BT-1) F_3 families to inoculation with BCMV NY15, BlCMV, CAbMV, and WMV

No. of F_3 families			Expected ratio	Goodness of fit
R[a]	Seg[b]	Susc[c]		$P_{\alpha=0.05}$
237	515	248	1: 2: 1	0.57

[a] All families resistant to BCMV NY15 were also resistant to BlCMV, CAbMV, and WMV
[b] All families found segregating for resistance to BCMV NY15 were also segregating for resistance to the other three viruses
[c] All families susceptible to BCMV NY15 were uniformly susceptible to the other three viruses

In a separate linkage study of 1000 (BT-2×BT-1) F_3 families, resistance to BCMV, WMV, BlCMV, and CAbMV always cosegregated (Table 3 b). Of these 1000 F_3 families, 381 for which seed was available were also screened with SMV. Dominant systemic veinal necrosis to SMV always segregated with resistance to BCMV, WMV, BlCMV, and CAbMV in these families (Kyle 1988).

Discussion

We have identified single dominant gene(s) in *P. vulgaris* BT-1 that altered plant response to four potyviruses, AzMV, ThPV, PWV-K, and ZYMV, and that segregated as a unit with the *I* gene for resistance to BCMV Serotype B. In a separate linkage study using a larger population, resistance to WMV, BlCMV, CABMV, and lethal veinal necrosis to SMV also segregated as a unit with the *I* gene (Kyle 1988). These results are consistent with the possibility that a single gene is responsible for conditioning resistance and/or lethal necrosis to a set of nine potyviruses, BCMV, WMV, BlCMV, CAbMV, AzMV, ThPV, SMV,

PWV-K, and ZYMV, although the existence of separate tightly-linked genes certainly cannot be ruled out. Multiple genes for resistance to sets of fungal pathogens have been found to map to a single locus (Bennetzen and Hulbert 1992), and recent cloning of the *Pto* gene for resistance to *Pseudomonas syringae* pv *tomato* in tomato revealed the functional gene to be one member of a gene family clustered at the *Pto* locus (Martin et al. 1993, Salmeron et al. 1994). We are currently screening diverse germplasm from both centers of diversity for *P. vulgaris* that include independent sources of the *I* gene (Kelly 1988) in an attempt to identify a recombinant genotype.

The phenotypic responses of BT-1 plants mechanically inoculated with each of the nine potyviruses differed depending on which virus was used and fell into three classes (Table 1). However, when approach-graft inoculation was used, veinal necrosis appeared in every case. This necrosis is not a necessary outcome of infection of BT-1 plants with any potyvirus, but rather a specific response to a unique set of potyviruses since there are potyviruses that do not cause necrosis on BT-1 plants (e.g., bean yellow mosaic potyvirus). In contrast to BT-1, all potyviruses in this study can infect BT-2 plants resulting in typical systemic mosaic or mottle symptoms, except for ZYMV, which accumulated in inoculated leaves but not in uninoculated tissue. In a study to examine the partial host range of ZYMV, no genera within the Leguminosae became systemically infected (Wang et al. 1992).

As mentioned above, after mechanical inoculation, three phenotypic classes were distinguished on BT-1 plants that were not apparent after approach-graft inoculation. The first class was represented by AzMV where temperature-dependent resistance was observed. At low temperature (25°C), BT-1 plants were resistant to mechanical inoculation, but above 33°C pinpoint necrotic lesions and veinal necrosis developed on inoculated leaves, followed by systemic veinal necrosis and plant death. The response to AzMV was identical to the temperature-dependent resistance in BT-1 to three potyviruses, BlCMV, CAbMV, and WMV, studied previously (Provvidenti et al. 1983; Kyle and Provvidenti 1987 a) and to the resistance conferred by the *I* gene to BCMV Serotype B (Ali 1950).

The second class of response in BT-1 plants occurred after mechanical inoculation with ThPV, SMV, or BCMV Serotype A (Drijfhout 1978; Kyle and Provvidenti 1993 a). A rapid systemic veinal necrosis occurred regardless of ambient temperature. The temperature-independent veinal necrotic response to these three potyviruses was phenotypically identical to that observed at high temperature in BT-1 plants inoculated with AzMV, BCMV Serotype B, BlCMV, CAbMV, and WMV. The third type of response to a potyvirus observed in the BT-1 parent is complete local and systemic resistance to mechanical inoculation with PWV-K and ZYMV. These are the first potyviruses reported thus far to which BT-1 is systemically resistant when mechanically inoculated at both low and high temperature. However, when PWV-K was introduced directly into the phloem of BT-1 plants via approach-graft inoculation, a vascular veinal necrosis occurred similar to that observed with the other viruses. Although genetically linked, the mechanistic relationship between the necrotic response and the resistance is unknown.

We have not assigned gene symbols to the gene(s) conferring resistance to PWV-K, AzMV, and ZYMV and lethal necrosis to ThPV, although the inheritance of the response to each particular virus is unambiguous. The current convention for gene designation has been to assign a gene symbol to each host factor that controls the response to a particular virus (e.g., Provvidenti and Hampton 1992). In the present case, the linkage and similar phenotypes suggest that we cannot rule out identity of some or all the factors controlling response to these viruses. Also, proposed revisions of the taxonomic status of viruses within the Potyviridae group would alter the designation of the host resistance genes. For example, Khan et al. (1993) proposed that BlCMV should be considered a strain of BCMV, based on CP sequence and serological similarities, and recent sequence and serological analyses of AzMV CP suggest that this virus could also be considered a strain of BCMV (Tsuchizaki and Omura 1987; McKern et al. 1992; Mink and Silbernagel 1992; CW Collmer personal communication). Until the taxonomic status of the pathogens has stabilized and the number of functional host genes are known, we believe that it is inappropriate to assign symbols to the gene(s) conferring resistance to AzMV, PWV-K, and ZYMV, and lethal necrosis to ThPV.

The set of potyviruses reported so far that have an altered response on BT-1 plants all fall into the same subgroup based on CP sequence similarity (McKern et al. 1992; Rybicki and Shukla 1992; Khan et al. 1993). We expect that viruses with similar nucleotide sequences may share common features of pathogenicity, and therefore could be similarly affected by a single host gene or a cluster of tightly-linked related genes. It is interesting that viral response on BT-1 plants should correlate with a set of viruses grouped solely on CP sequence similarity, given that the CP comprises only a small portion of the entire viral genome, especially in view of evidence of recombination among potyviral isolates (Cervera et al. 1993). It is possible that the CP itself is the viral determinant of the resistant and necrotic responses on BT-1 plants, or else that homology between CPs may reflect similar patterns of divergence throughout the viral genome.

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References

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

Bennetzen JL, Hulbert SH (1992) Organization, instability and evolution of plant disease resistance genes. Plant Mol Biol 20: 575–580

Benscher D, Pappu SS, Niblett CL, Rybicki EP, Bird J (1993) Biological and molecular characterization of potyviruses from *Passiflora*. Phytopathology 83:1422

Bos L (1992) Potyviruses, chaos or order? Arch Virol Suppl 5:31–46

Cervera M, Riechmann J, Martin M, Garcia J (1993) 3′-Terminal sequence of the plum pox virus PS and o6 isolates: evidence for RNA recombination within the potyvirus group. J Gen Virol 74: 329–334

Clark M, Adams A (1977) Characteristics of the microplate method of enzyme-linked immunosorbent assay for the detection of plant viruses. J Gen Virol 34:475–483

Drijfhout E (1978) Genetic interaction between *Phaseolus vulgaris* and bean common mosaic virus with implications for strain identification and breeding for resistance. Agricultural Research Reports 872, Pudoc, Wageningen

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

Fraser R (1990) The genetics of resistance to plant viruses. Annu Rev Phytopathol 28: 179–200

Gough KH, Shukla DD (1992) Major sequence variations in the N-terminal region of the capsid protein of a severe strain of passionfruit woodiness potyvirus. Arch Virol 124:389–396

Kelly JD (1988) Is there more than one source of the *I* gene? Bean Improv Coop Annu Rep 31:148–149

Khan J, Lohuis D, Goldbach R, Dijkstra J (1993) Sequence data to settle the taxonomic position of bean common mosaic virus and blackeye cowpea mosaic virus isolates. J Gen Virol 74: 2243–2249

Kyle M (1988) The *I* gene and broad-spectrum plant virus resistance in *Phaseolus vulgaris* L. PhD thesis, Cornell University

Kyle MM, Dickson MH (1988) Linkage of hypersensitivity to five potyviruses with the *B* locus for seed coat color in *Phaseolus vulgaris* L. J Hered 79:308–311

Kyle MM, Provvidenti R (1987 a) Inheritance of resistance to potato y viruses in *Phaseolus vulgaris* L. I. Two independent genes for resistance to watermelon mosaic virus-2. Theor Appl Genet 74:595–600

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

Kyle MM, Provvidenti R (1993 a) Inheritance of resistance to potyviruses in *Phaseolus vulgaris* L. II. Inheritance, linkage relations, and utility of a gene for lethal systemic necrosis to soybean mosaic virus. Theor Appl Genet 86:189–196

Kyle MM, Provvidenti R (1993 b) Genetics of broad-spectrum virus resistance in bean and pea. In: Kyle MM (ed) Resistance to viral diseases of vegetables: genetics and breeding. Timber Press, Portland, Oregon, pp 153-166

Martin GB, Brommonschenkel SH, Chunwongse J, Frary A, Ganal MW, Spivey R, Wu T, Earle ED, Tanksley SD (1993) Map-based cloning of a protein kinase gene conferring disease resistance in tomato. Science 262:1432–1436

McKern N, Shukla D, Barnett O, Vetten H, Dijkstra J, Whittaker L, Ward C (1992) Coat protein properties suggest that azuki bean mosaic virus, blackeye cowpea mosaic virus, peanut stripe virus, and three isolates from soybean are all strains of the same potyvirus. Intervirology 33:121–134

Mink G, Silbernagel M (1992) Serological and biological relationships among viruses in the bean common mosaic virus subgroup. Arch Virol Suppl 5:397–406

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 (1993) Resistance to three strains of passionfruit woodiness virus in *Phaseolus vulgaris*. Bean Improv Coop Annu Rep 36:137–138

Provvidenti R, Hampton RO (1992) Sources of resistance to viruses in the *Potyviridae*. Arch Virol Suppl 5:189–211

Provvidenti R, Monllor AC, Niblett CL, Bird J, Gough KH (1992) Host differentiation of potyviruses infecting passionfruit (*Passiflora edulis*) in Puerto Rico and Australia. Phytopathology 82: 610

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

Rybicki EP, Shukla DD (1992) Coat-protein phylogeny and systematics of potyviruses. Arch Virol Suppl 5:139–170

Salmeron JM, Barker SJ, Carland FM, Mehta AY, Staskawicz BJ (1994) Tomato mutants altered in bacterial disease resistance provide evidence for a new locus controlling pathogen recognition. Plant Cell 6:511–520

Shukla D, Ward C (1988) Amino-acid sequence homology of coat proteins as a basis for identification and classification of the potyvirus group. J Gen Virol 69:2703–2710

Tsuchizaki T, Omura T (1987) Relationships among bean common mosaic virus, blackeye cowpea mosaic virus, azuki mosaic virus, and soybean mosaic virus. Ann Phytopathol Soc Japan 53: 478–488

Wang H, Gonsalves D, Provvidenti R, Zitter T (1992) Comparative biological and serological properties of four strains of zucchini yellow mosaic virus. Plant Dis 76:530–535

Ward, C, Shukla D (1991) Taxomomy of potyviruses: current problems and solutions. Intervirology 32: 269–296

Ward C, McKern N, Frenkel M, Shukla D (1992) Sequence data as the major criterion for potyvirus classification. Arch Virol Suppl 5:283–297