A Complementation of Two Genes Originating from Susceptible *Capsicum annuum* Lines Confers a New and Complete Resistance to Pepper Veinal Mottle Virus

C. Caranta, A. Palloix, K. Gebre-Selassie, V. Lefebvre, B. Moury, and A. M. Daubèze

First, second, fourth, fifth, and sixth authors: INRA, Station d'Amélioration des Plantes Maraîchères; third author: INRA, Station de Pathologie Végétale, domaine Saint Maurice, B.P. 94, 84143 Montfavet, Cedex, France.
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ABSTRACT

Caranta, C., Palloix, A., Gebre-Selassie, K., Lefebvre, V., Moury, B., and Daubèze, A. M. 1996. A complementation of two genes originating from susceptible *Capsicum annuum* lines confers a new and complete resistance to pepper veinal mottle virus. Phytopathology 86:739-743.

A doubled haploid (DH) line completely resistant to pepper veinal mottle virus (PVMV) was recovered from the F_1 hybrid between two *Capsicum annuum* lines, Perennial and Florida VR2, whereas both of these lines were susceptible to PVMV. These observations suggest that resistance to PVMV results from a complementary action of genes originating from Perennial and Florida VR2. F_2 plants were obtained by crossing individual DH lines from a hybrid (Perennial × Yolo Wonder) with Florida VR2. In 14 F_2 families, the segregation ratios observed for PVMV resistance were consistent with the complementation of two recessive genes, each one coming from one of the parents. One gene from Florida VR2 was identical or tightly linked to $pvr2^2$ (a recessive gene conferring complete resistance to potato virus Y [PVY] pathotypes [0 and 1]). The other gene from Perennial was linked to a restriction fragment length polymorphism marker localized on chromosome Pourpre of the pepper map and was tentatively named *pvr6*. The $pvr2^2$ gene alone conferred complete resistance to PVY(0) and (1), and in the genomic region around the PVMV-linked marker, we previously mapped genes also involved in potyvirus resistance. Complementation between factors already involved in potyvirus resistance conferred an enlarged spectrum of resistance to a distantly related pathogen.

Additional keywords: epistasis, molecular markers, multi-potyvirus resistance.

Pepper veinal mottle virus (PVMV), a member of the genus *Potyvirus*, is one of the most damaging pepper (*Capsicum annuum* L.) diseases in West African pepper crops (4). This virus is transmitted nonpersistently by aphids and also infects other Solanaceae, such as tomato and eggplant. Several strains have been identified (5) according to host range and symptoms induced in susceptible species. PVMV is serologically distinct from potato virus Y (PVY) (5), the type member of the potyvirus family, and from the other potyviruses infecting pepper around the world: chili veinal mottle virus (CVMV), tobacco etch virus (TEV), pepper mottle virus, and potyvirus E (17,19).

Until now, only partial resistance against PVMV (1,32) has been described. In our laboratory, a doubled haploid (DH) line, DH801, completely resistant to PVMV was obtained from an F_1 hybrid between two *C. annuum* cultivars, Perennial and Florida VR2, that are susceptible to PVMV. However, these two pepper lines possess resistance factors against other potyviruses: Florida VR2 possesses a recessive allele conferring resistance to PVY pathotypes (0) and (1) (allele vy^2 [17], renamed $pvr2^2$ [28]); Perennial is resistant to PVY(0), potyvirus E, and CVMV and is partially resistant to PVY(1,2) (7). Palloix (27) suggested that the absolute resistance to PVMV in DH801 resulted from a complementary action of gene(s) from Perennial with the $pvr2^2$ gene from Florida VR2.

The resistance of Perennial to potyviruses was studied further by Caranta and Palloix (7) with a DH progeny from the F_1 hybrid between Perennial and a susceptible *C. annuum* line, Yolo Wonder. Resistance to two PVY pathotypes and potyvirus E was quantitatively expressed in DH segregations and controlled by several recessive genetic factors; resistance to CVMV was conferred by two independent genes. The same DH progeny was used to construct a molecular linkage map of pepper (22). Molecular markers were used to map genetic factors (quantitative trait loci [QTL] or major genes) involved in potyvirus resistance. Some of these factors confer specific resistance to only one potyvirus or PVY pathotype, whereas others confer resistance to several potyviruses (6,8). It is not known if one (or several) of these genes in Perennial would complement the $pvr2^2$ allele from Florida VR2 to confer PVMV resistance, as suggested by the preceding observations.

In the first part of this paper, recombination of potyvirus resistance factors from Perennial with the $pvr2^2$ allele was obtained by crossing individual DH lines from the hybrid Perennial × Yolo Wonder with Florida VR2 to obtain several F_2 families carrying different sets of potyvirus resistance factors from Perennial with the $pvr2^2$ allele. We report the genetic analysis of PVMV resistance in these families. In the second part of the study, we use biological tests and molecular markers to map the loci involved in PVMV resistance and to determine if these loci are involved in other potyvirus resistance. We report evidence consistent with complementation between the $pvr2^2$ allele (or a closely linked factor) involved in resistance to PVY(0) and (1) and a locus linked to the restriction fragment length polymorphism (RFLP) marker TG57 on the chromosome Pourpre. In the same genomic region, we previously have localized other genes involved in potyvirus resistance.

MATERIALS AND METHODS

Plant material. The *C. annuum* line Perennial was provided by J. Singh (Punjab University, Ludhiana, India); Yolo Wonder and Florida VR2 are inbred bell pepper lines from California and

Corresponding author: C. Caranta; E-mail address: caranta@avignon.inra.fr

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Florida, respectively. For genetic analysis, 23 DH lines (obtained by anther culture from an F_1 hybrid between Perennial and Yolo Wonder [12]) were crossed with the line Florida VR2. The 23 F_1 hybrids were selfed to produce 23 F_2 families. Florida VR2 has the recessive *pvr2*^{2} allele from the progenitor PI264281 (10). DH line DH801, derived from the F_1 of a cross between Perennial and Florida VR2, was used as the PVMV-resistant control.

Viral strains and inoculation procedure. To screen the parental response to PVMV, three strains originating from pepper isolates were used: the "standard" strain provided by A. A. Brunt, originating from Ghana, a PVMV strain from Cameroon (26), and a PVMV strain from the Ivory Coast (provided by J. C. Thouvenel, ORSTOM, Montpellier, France). The strains were maintained by the Bos technique (3) and multiplied on susceptible pepper cultivar Yolo Wonder. The purity of the viral strains was monitored routinely with double-antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA) and differential host index tests (19). At least 150 plantlets per F_2 family were mechanically inoculated at the 1-leaf stage (3-week-old plants) with the Ivory Coast strain. Inoculum and inoculation procedure were as described previously (7).

Resistance evaluation. Plants were scored for PVMV symptoms 5 weeks after inoculation. Plants without obvious symptoms were evaluated for presence/absence of the virus by the DAS-ELISA method (9). Tests were considered positive when the absorbance value of the sample was at least three times greater than the average value of the healthy control. Only PVMV-resistant plants were reinoculated with PVY(1) to check for the *pvr2*^{2} allele. DAS-ELISA tests also were performed to verify the presence/absence of PVY. PVMV-susceptible plants were not reinoculated to assess PVY(1) susceptibility because of the occurrence of heteroencapsidation between the two potyviruses (23). Anti-PVMV antiserum was supplied by R. Nono-Wondim (AVRDC, Acusha, Tanzania). Segregation data were compared to theoretical segregation ratios by a chi-square goodness-of-fit test (24).

DNA extraction and RFLP procedure. Total DNA was extracted from approximately 1 g of fresh young leaves of F_2 plants resistant to PVMV. The DNA extraction procedure was adapted from that of Bernatzky and Tanksley (2). The tissue was ground in a mortar with 5 ml of extraction buffer, pH 8.25 (100 mM Tris, 0.35 M Sorbitol, 5 mM EDTA, 2 M NaCl, and 70 mM $Na_2S_2O_5$). The mixture was transferred to a 50-ml screw-cap centrifuge tube, adjusted to 1% CTAB (hexadecyltrimethylammonium bromide, Sigma Chemical Company, St. Louis), 1 M NaCl, and 25 mM EDTA, and heated to 65°C for 20 min. After lysate extraction (with chloroform/octanol, 24:1 vol/vol), a step of precipitation of polysaccharides was added: the aqueous phases were slowly mixed with a 1/3 volume of 100% ethanol (if mixing is not complete, local concentrations of ethanol may become high enough to precipitate DNA), refrigerated in ice for 15 min, and spun at 2,700 × *g* for 10 min at 20°C (25). The aqueous phase was mixed with a 2/3 volume of 100% ethanol to precipitate the DNA. The precipitate was washed in 76% ethanol and 10 mM ammonium acetate at pH 7.0. The cesium chloride/ethidium bromide centrifugation was omitted (A. Blattes, *unpublished data*).

Plant DNA (10 µg) was digested with restriction enzyme *Eco*RI (Boehringer Mannheim, Meylan, France). A Southern blotting procedure was performed as described by Lefebvre et al. (22). The tomato genomic probe, TG57, used in this study was provided by S. D. Tanksley (Cornell University, Ithaca, NY).

To the 84 molecular markers (RFLPs and randomly amplified polymorphic DNA [RAPD]) of the integrated pepper linkage map (22), we added 60 markers, mostly RAPDs, in the DH progeny derived from Perennial × Yolo Wonder. The new map includes 136 markers mapped on 13 linkage groups, spans 1,110.7 centimorgans (cM) (20), and is estimated to cover about 80% of the pepper genome (6). Thanks to the location of four markers (*C*, *L*, *up*, and r45S) for which the assignment to a trisomic has been described already (29,33), four linkage groups were assigned to chromosomes Brun, Noir, Jaune, and Pourpre, respectively. The pepper chromosomes were conventionally designated by French color names by Pochard (29).

To identify markers linked to the PVMV resistance factor from Perennial, DH lines that generated PVMV-resistant F_2 plants were considered resistant, and DH lines that generated only susceptible F_2 plants were considered susceptible. Linkage between the Perennial resistance factor and the molecular markers of the map was assessed by a χ^2 test for independence (24).

Recombination frequencies and standard deviation between the PVMV resistance gene and the molecular marker were calculated by the maximum likelihood method (14). Recombination frequencies were converted to centimorgans by the Kosambi mapping function (20).

RESULTS

Genetics of resistance to PVMV in F_2 families. Three weeks after inoculation, the three PVMV strains induced characteristic symptoms (i.e., systemic vein clearing and mosaics on leaves) on *C. annuum* lines Yolo Wonder, Florida VR2, F_1 Perennial × Yolo Wonder, and F_1 Perennial × Florida VR2. The susceptibility of the F_1 Perennial × Florida VR2 indicates the recessive nature of the resistance. Perennial also was susceptible to the three PVMV strains: 5 weeks after inoculation, all the plants expressed symptoms, and virus was detected with DAS-ELISA tests when they were inoculated with the Cameroon and Ivory Coast strains. When Perennial was inoculated with the standard strain, symptoms observed 5 weeks after inoculation were less severe, and 3 plants (among the 20 inoculated) were virus-free. The resistant control, DH801, never showed any symptoms after mechanical inoculation with PVMV. These visual evaluations were confirmed by DAS-ELISA serological tests.

To analyze the genetic basis of the resistance, the Ivory Coast strain, which induced the most severe symptoms, was used to screen virus resistance in F_2 families. The reaction of plants in each F_2 family and the suggested segregation ratios are presented in Table 1. Among the 23 F_2 families, 9 were totally susceptible to PVMV (i.e., no resistant plants were found 5 weeks after inoculation), and 14 had resistant progeny. In these 14 F_2 families, segregation ratios observed for PVMV resistance (proportion of resistant and susceptible plants) were consistent with two recessive genes involved in PVMV resistance (χ^2_{1df}[1 R:15 S ratio] ranging from 0.0 to 1.88). Because of the susceptibility of the

TABLE 1. Segregation data of the different F_2 families of pepper (*Capsicum annuum*) for reactions against pepper veinal mottle virus and goodness-of-fit to two gene ratios using the χ^2 test

F_2 family[a]	Number of plants		χ^2	
	Resistant	Susceptible	(1 R:15 S ratio)	Probability
DH215 × Flo	16	168	1.88	0.17
DH218 × Flo	13	179	0.08	0.77
DH221 × Flo	15	175	0.88	0.35
DH242 × Flo	8	171	0.97	0.33
DH246 × Flo	12	184	0.00	0.94
DH248 × Flo	9	190	0.74	0.39
DH249 × Flo	11	179	0.00	0.95
DH251 × Flo	11	165	0.00	1.00
DH268 × Flo	10	178	0.28	0.60
DH270 × Flo	10	174	0.21	0.65
DH274 × Flo	7	136	0.45	0.50
DH284 × Flo	15	182	0.63	0.43
DH286 × Flo	10	171	0.16	0.69
DH2123 × Flo	10	185	0.23	0.64
Sum	157	2,437	0.17	0.68

[a] DH = doubled haploid lines derived from the Perennial × Yolo Wonder F_1 hybrid. Flo = Florida VR2.

parental lines, it was likely that one recessive gene from Perennial (via the DH lines) interacted with one recessive gene from Florida VR2 to confer resistance against PVMV (Fig. 1).

Characterization of the gene involved in PVMV resistance from Florida VR2. As hypothesized by Palloix (27), the gene controlling PVMV resistance from Florida VR2 was expected to be *pvr2*^{2}, which confers resistance to PVY(0) and (1), or a tightly linked factor. Perennial also is resistant to PVY(0) but susceptible to PVY(1). To determine if *pvr2*^{2} is involved in PVMV resistance, the 157 F_2 plants resistant to PVMV (from 14 F_2 families) and the parental lines were inoculated with PVY(1). Five weeks after inoculation, none of the F_2 plants showed PVY symptoms, and DAS-ELISA tests confirmed that the plants were virus-free. The parental line, Florida VR2, also was virus-free, whereas PVY(1) was detected in the leaves of Perennial and the parental DH lines. Moreover, the *pvr2* locus was mapped further with RAPD markers (8); PVMV-resistant and -susceptible plants were checked for RAPD markers and indicated the presence of this locus from Florida VR2 (C. Caranta, *unpublished data*). These observations suggest that resistance to PVY(1) and PVMV are controlled by the same genetic factor or by closely linked factors.

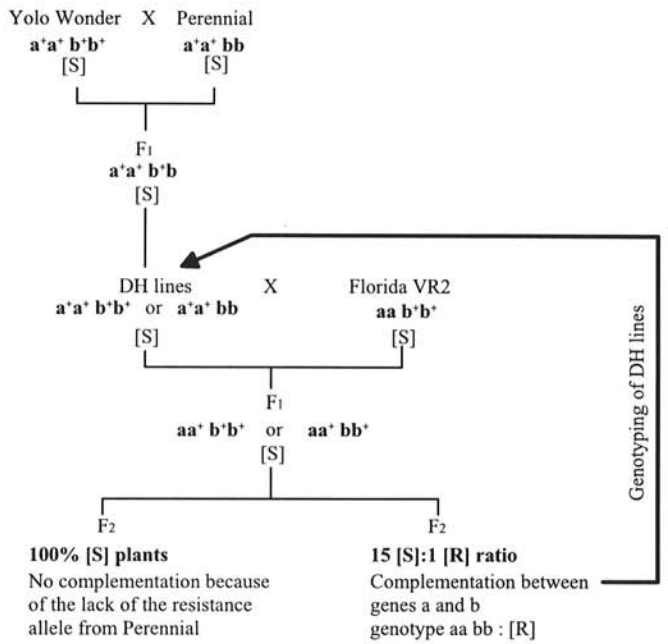

Fig. 1. Pedigree of the F_2 families used to study the genetic basis of pepper veinal mottle virus (PVMV) resistance in pepper (*Capsicum annuum*). a = recessive resistance allele from line Florida VR2, a$^+$ = susceptible allele from Florida VR2, b = recessive resistance allele from line Perennial, and b$^+$ = susceptible allele from Perennial. [S] = PVMV-susceptible phenotype and [R] = PVMV-resistant phenotype.

Identification of the gene from Perennial. The next step in the analysis was to map the PVMV resistance gene from the DH lines. DH lines that generated PVMV-resistant F_2 plants should possess the complementary resistance factor against PVMV (and the Perennial allele at an expected linked marker), whereas DH lines that generated only PVMV-susceptible F_2 plants should not possess the resistance factor (and the Yolo Wonder allele at a linked marker). Associations between the PVMV resistance factor segregating among 23 DH lines and the 144 markers of the map were assessed by two-way contingency tables. Significant deviation from the expected ratio was detected for one marker, TG57, mapping to chromosome Pourpre (χ^2_{1df} = 12.17, P = 0.00049). The nearest flanking marker was mapped at 19.7 cM from TG57; thus, TG57 was the only marker significantly linked to the phenotype. Among the 14 DH lines generating PVMV-resistant F_2 plants, 13 possessed the Perennial allele at the TG57 locus, and among the 9 DH lines generating PVMV-susceptible F_2 plants, 8 possessed the Florida allele at the same locus. To validate linkage between TG57 and the PVMV resistance factor, 26 F_2 plants resistant to PVMV were checked for their genotype at the TG57 locus. Susceptible plants of the same F_2 families were not tested for their genotypes at the TG57 locus: these may have the resistance factor from Perennial and lack *pvr2*^{2} and would be susceptible. Among the resistant plants, 21 were scored as homozygous type Perennial, and 5 were scored as heterozygous (Fig. 2). Linkage between TG57 and the factor conferring resistance to PVMV was estimated to be 9.8 ± 9.0 cM. The restricted number of individuals observed does not allow a precise evaluation of this distance.

DISCUSSION

Evaluation of the F_2 families obtained by crossing DH lines (derived from an F_1 hybrid between Perennial and Yolo Wonder) with Florida VR2 provided evidence that complete resistance against PVMV was conferred by two recessive genes. All the

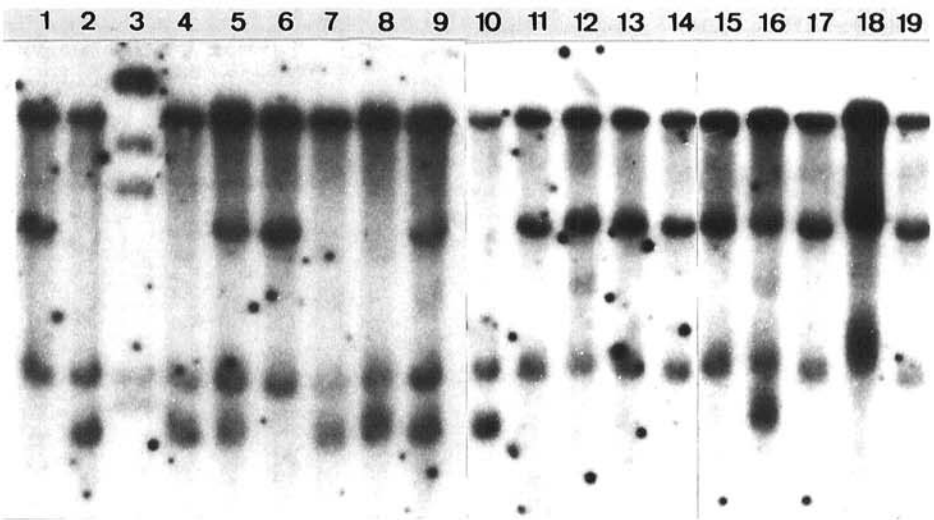

Fig. 2. Southern analysis with probe TG57 of *Eco*RI-digested DNA from pepper line Perennial (lane 1), pepper line Florida VR2 (lane 2), pepper veinal mottle virus (PVMV)-susceptible F_2 plants (lanes 4 through 10) and PVMV-resistant F_2 plants (lanes 11 through 19). Lane 3, DNA size marker λ *Hin*dIII. Lanes 4 through 10, PVMV-susceptible plants did not possess the TG57 Perennial allele (lanes 4, 7, and 8) or were in an heterozygous state (lanes 5 and 9). Lane 6, the plant was recombinant. Lanes 11 through 19, PVMV-resistant plants were homozygous for the TG57 Perennial allele, except lane 16, which was a heterozygous recombinant individual.

parental lines were susceptible to PVMV, suggesting the resistance resulted from a complementation between two genes, each coming from one of the parents. The presence of plants that escaped infection by the standard strain can be related to the results of Poulos et al. (30) who found that Perennial may be resistant to this strain under glasshouse conditions at the 4-leaf stage. Of the 23 F_2 families assessed, only 14 produced PVMV-resistant plants; total susceptibility of some F_2 families was probably due to the lack of one resistance gene from DH lines. The combination of a biological and a molecular approach enabled us to characterize these two genes. One of them, coming from Florida VR2, seems to be *pvr2*^{2} or a closely linked factor and the other, coming from Perennial, is linked to TG57, a molecular marker localized on chromosome Pourpre of the intraspecific pepper map.

The presence of the *pvr2*^{2} allele in the PVMV-resistant F_2 individuals was assessed by reinoculation with PVY(1). All PVMV-resistant plants also were resistant to PVY(1) and only the Florida VR2 parent was resistant through pseudoimmunity to this PVY pathotype (due to the presence of the recessive *pvr2*^{2} allele). Thus, the PVMV-resistant plants are expected to be homozygous for this allele. However, this also could result from cross-protection of the plants due to preinoculation with PVMV. Until now, no cases of cross-protection between PVMV and PVY have been reported in pepper (23). Moreover, the use of RAPDs linked to *pvr2* confirmed the role of this PVY resistance factor (or a closely linked factor) in PVMV resistance (C. Caranta, *unpublished data*). Unlike the resistance factor from Florida VR2, we had no a priori information about an individual effect of the complementary factor from Perennial. By crossing individual DH lines (from the hybrid between Perennial and Yolo Wonder) with Florida VR2, we were able to localize the resistance factor near the TG57 locus. These DH lines were used to construct the molecular map. Linkage between the PVMV resistance factor and TG57 (distance of 9.8 cM) was observed on DH lines and was confirmed on F_2 resistant plants. This recessive resistance factor is tentatively named *pvr6*, according to the nomenclature used for potyvirus-resistance genes in pepper (28).

To our knowledge, few examples of interactions (epistasis) between virus-resistance genes have been reported in the literature. *Phaseolus vulgaris* contains several virus-specific resistance genes (*bc* genes) against bean common mosaic potyvirus. For full expression, each *bc* resistance gene requires an epistatic effect from an additional recessive allele at a separate locus, *bc*-u. Plants homozygous at the *bc*-u locus show no detectable resistance, and plants homozygous at *bc*-1 are partially resistant (K. L. Day in Fraser [15]). In the same species, resistance to azukini mosaic virus was conferred by an interaction between two unlinked genes that are phenotypically indistinguishable from each other; one of these genes also seemed to be associated with resistance to soybean mosaic virus and watermelon mosaic virus (13). Thus, in common bean as well as in pepper, complex interactions between potyvirus-resistance genes seem to occur.

In pepper, the genetic factors or the genomic regions controlling PVMV resistance were individually involved in resistance to other potyviruses. The *pvr2*^{2} allele conferred recessive strain-specific resistance to PVY(0) and (1) and to common strains of TEV (10, 16). The second interacting genetic factor was mapped close to the TG57 locus; in the same genomic region, we previously localized a QTL for partial resistance to potyvirus E (6,8). PVMV resistance factors are involved or closely linked to factors involved in other potyvirus resistances. However, this remains an original example of gene complementation conferring an enlarged spectrum of resistance to distantly related pathogens. From a practical point of view, these results show that the association of several potyvirus-resistance factors can generate new resistances against other potyviruses.

Colocalization of potyvirus-resistance genes has been reported in *Cucurbita moschata* (18), pea (31), bean (13), and pepper (10, 11). This phenomenon may result from a pleiotropic effect of a single locus or tightly linked genes. In case of pleiotropic effects, the gene in question could be triggered by a common sequence (amino acid or RNA) of a viral domain shared by the different potyviruses (21). If broad-spectrum resistance results from clustering of resistances genes, these genes could be structurally related and may share a common mode of action. A QTL of resistance to potyvirus E and an interacting resistance gene to PVMV were localized in the same genomic region around the TG57 locus, providing circumstantial evidence that quantitative and qualitative resistance may share a common genetic basis.

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