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C. Caranta · A. Palloix

Both common and specific genetic factors are involved in polygenic resistance of pepper to several potyviruses

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Abstract Absolute resistance to potato virus Y pathotype 0 (PVY 0), potyvirus E and chili veinal mottle virus (CVMV) and a partial resistance to potato virus Y pathotype 1,2 (PVY 1,2) were found in an Indian pepper line, 'Perennial'. In the doubled haploid (DH) progeny from the F_1 of a cross 'Perennial' by 'Yolo Wonder', resistance to CVMV was confered by two independent genes, one with a clear dominant effect. Resistance to PVY and potyvirus E was quantitatively expressed and controlled by several recessive genetic factors. Genetic analysis showed that fewer resistance factors were necessary to explain resistance to PVY (0) and potyvirus E than resistance to PVY(1,2). Genetic correlations between resistances to the different potyviruses in the DH progeny showed that most of genetic factors involved in PVY(0) resistance appear to be also involved in potyvirus E resistance, and some of these polyvalent factors may be also involved in PVY(1,2) resistance but, in this case, additional specific genes were necessary. One of the two CVMV resistance genes seems to be implicated in potyvirus E resistance. Thus, the polygenic resistance of 'Perennial' to these potyviruses was due both to polyvalent genetic factors, i.e. factors that apparently interact with several viruses, and strain-specific genetic factors.

Key words *Capsicum annuum* L. · Potyviruses · Multivirus resistance · Polygenic inheritance · Doubled haploid progenies

Introduction

Numerous potyviruses infect pepper (*Capsicum annuum* L.) crops around the world. This group is the largest and economically most important of plant viruses (Bos 1992) and is transmitted by aphids in a non-persistant manner. According to serological criteria, at least five potyviruses have been reported to infect pepper: potato virus Y (PVY) is widespread throughout most of the areas where peppers are cultivated, while tobacco etch virus (TEV) and pepper mottle virus (PeMV) occur mainly in North and Central America and in the Caribbean. Pepper veinal mottle virus (PVMV) infects pepper in Africa, and chili veinal mottle virus (CVMV) has been reported in Asia (For review see Green and Kim 1991). Variability is very important for several of these pathogens. For instance, the PVY strains have been grouped into three pathotypes (0,1 and 1,2) with respect to their interaction with host resistance genes (Gebre Selassie et al. 1985). Another potyvirus, designated potyvirus E, was isolated from *Portulacca oleracea* in the south of France and was also shown to infect pepper under experimental conditions. This potyvirus has peculiar antigenic properties: it does not show any serological relationship with the other potyviruses except for a weak immunodiffusion reaction with PVY and PVMV antisera (Gebre-Selassie et al. 1983).

There is a tremendous variability of genetically determined responses to the infection of pepper by these potyviruses. Up to now, only monogenic resistance systems that limit or prevent systemic infection of the plant have been used in cultivar development. These systems are controlled by an allelic series at the *et* locus (for review see Greenleaf 1986) or the *vy* locus (according to Gebre-Selassie et al. 1983). In this allelic series, all of the resistant alleles are recessive over the susceptible ones and the alleles are strain-specific (Gebre-Selassie et al. 1985). More recently, resistance has been characterized in the Mexican line 'Criollo de Morelos 334'. A dominant gene independant from the recessive loci was shown to control resistance to the three known PVY pathotypes and to PeMV (Chaine-Dogimont 1993). However, none of these monogenic systems confered resistance to CVMV, PVMV or potyvirus E.

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C. Caranta (✉) · A. Palloix
Institut National de la Recherche Agronomique (I.N.R.A.), Station d'Amélioration des Plantes Maraîchères, Domaine Saint Maurice, BP94, 84143 Montfavet cedex, France

In addition to these monogenic systems, a more complex resistance was found in an Indian pepper line 'Perennial'. This line does not develop symptoms after inoculation with the three pathotypes of PVY, CVMV nor with potyvirus E. This line also possesses resistance factors against PVMV (Gebre-Selassie et al. 1986) and cucumber mosaic virus (CMV) (Nono Wondim et al. 1993). Preliminary genetic studies suggest that resistance to potyviruses in 'Perennial' is under polygenic control and is quantitatively expressed (Pochard et al. 1983).

Genetic analysis of multiple virus resistance is difficult since classical F_2 or backcross individuals cannot be tested by several viruses. However, the development of doubled haploid (DH) progenies can break down this barrier and allows the segregation of multivirus resistance to be studied. A DH progeny was obtained from an intraspecific F_1 hybrid between 'Perennial' and a potyvirus-susceptible genotype (Dumas de Vaulx et al. 1981).

The objective of the investigation presented in this paper was to dissect the multiresistance of pepper to potyviruses into genetic and phenotypic components. The inheritance of resistance to three potyviruses (PVY pathotypes, CVMV and potyvirus E) is described, and we report results that suggest that there may be common genetic factors with both quantitative and qualitative effects on host response using genetic correlations among the DH progeny.

Materials and methods

Plant material

Pepper plants were grown in a sterilized peat soil mixture in the greenhouse using standard horticultural practices. The material consisted of 94 DH lines obtained at INRA Montfavet (France) from the F_1 hybrid of a cross between a homozygous line 'Perennial' (obtained from J. Singh, Punjab University, Ludhiana, India) and 'Yolo Wonder', a homozygous line susceptible to all potyviruses.

Viral strains and inoculation

The potyvirus strains used in this study are described in Table 1. They were maintained by the Bos technique (Bos 1969) and multiplied on susceptible pepper varieties. The CVMV strain was obtained from S. K. Green (AVRDC, Taïwan). The purity of the viral strains was monitored routinely with DAS-ELISA and differential hosts index tests. Inocula were prepared from 1 g (fresh weight) of infected foliar tissue ground with 4 ml potassium phosphate buffer (0.03 *M*, pH = 7) containing 0.2% of diethyldithiocarbamate, 80 mg active charcoal and 80 mg Carborundum (400 mesh). For inoculation, cotyledons of 3 week-old seedlings were manually rubbed with inoculum extract and rinsed with water 5 min after rubbing. After inoculation, the plants were grown either in the greenhouse or in a growth chamber (22 °C, 12 h light per day).

Virus detection and assay

Serological tests were performed using the doubled antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA) (Clark and Adams 1977) 5 weeks after inoculation to verify the presence/absence of the virus in DH lines. Absorbance values at 405 nm were measured with a spectrophotometer (Titertek Multiskan PLUS) after a 2-h incubation. The tests were considered to be positive when the absorbance value of the sample was at least 3 times greater than the average value of the healthy control. DAS-ELISA was also used to detect viruses in inoculated 'Perennial' leaves. Antisera against PVY, potyvirus E and CVMV were kindly supplied by Dr. K. Gebre-Selassie and Dr. H. Lot (Plant Pathologie Station, Institut National de la Recherche Agronomique, Montfavet, France).

Resistance evaluation

Resistance was assessed by scoring the 94 DH lines for symptoms intensity (SI) every 7th day for 5 weeks after inoculation. The SI scale ranged from 1 to 3 with : 1 = no symptom, 2 = mild mosaic and 3 = severe mosaic and/or systemic necrosis. The Area Under the Symptoms Progress Curve (AUSPC) value was determined for each DH line using the following formula:

$$\text{AUSPC} = \sum_i [(x_i + x_{i+1})/2].t_i$$

with x_i = Sum of SI value of 15 plants at date i
and t_i = time (in days) between scoring date i and scoring date $i+1$

Genetic analysis

Three independant tests on 20 plants per DH line were conducted for each viral strain. Analyses of variance (GLM program, SAS institute 1988) were performed on the mean score of 20 plants per DH line and per replicate to estimate phenotypic variance components. Due to the homozygoty of DH lines, genetic variance is equal to additive variance, in the absence of epistatic effects, and narrow-sense heritability (h_n^2) values for each trait were assessed using the following formula:

$$h_n^2 = \sigma_A^2/(\sigma_A^2 + \sigma_E^2/n)$$

where σ_A^2 is the additive variance, σ_E^2 is the environmental variance and n is the number of DH lines. In the case of DH progeny, narrow-sense heritability is equal to broad-sense heritability.

The number of segregating genes (k') affecting virus resistance was estimated by dividing the square of the deviation of the most extreme DH line (L1) from the population mean (μ) by the additive variance (Choo and Reinbergs 1982a):

$$k' = (\text{L1} - \mu)^2/\sigma_A^2$$

Genetic and environmental correlations between resistances were obtained using the variances-covariances matrix (PROC GLM option MANNOVA procedure, SAS Institute 1988) according to the Falconer (1981) formula:

$$r_G = \text{cov}_{xy}/\sqrt{\text{cov}_{xx}\text{cov}_{yy}}$$

Table 1 Origin of viral strains used in this study

Virus	Pathotype	Strain name	Isolated from	Origin
PVY	P(0)	To 72	*Lycopersicon* sp.	France INRA 1972
	P(1,2)	Son 41	*Solanum nigrum*	France INRA 1972
Potyvirus E	–	POON 17E	*Portulaca oleracea*	France INRA 1977
CVMV	–	Taïwan	*Capsicum* sp.	Taïwan AVRDC

where x and y are the two traits under consideration, cov_{xy} is the covariance of the two traits and cov_{xx} and cov_{yy} are the variances of each trait. To test the relationships between resistance to CVMV and resistance to the other viruses, we compared the mean AUSPC values of the CVMV-resistant lines to the mean AUSPC of the CVMV-susceptible lines using the Student's t-test.

Results

Reaction of parental lines to potyviruses

The parental line, 'Perennial', never showed symptoms of any type after mechanical inoculation with PVY(0), PVY(1,2), CVMV potyvirus E. Ten days post-inoculation, 'Yolo Wonder' developed mosaic on the youngest leaves of plants inoculated by PVY(0), PVY(1,2) and potyvirus E that became very severe, while plants inoculated with CVMV developed chlorosis and veinal necrosis. To confirm the visual evaluation, we assessed the parental lines for the presence of viral antigen by DAS-ELISA (Table 2). Five weeks post-inoculation, the susceptible line 'Yolo Wonder' was strongly positive. In 'Perennial', PVY(0), CVMV and potyvirus E were never detected in the inoculated leaf, nor in the apex, whereas despite the absence of symptoms, PVY(1,2) was weakly detected in the lower and upper leaves. Absorbance values in leaves from 'Perennial' were about one-third of those observed for similar samples from 'Yolo Wonder'.

Inheritance of PVY(0), PVY(1,2) and potyvirus E resistance

The behaviour of F_1 individuals from the cross 'Perennial' × 'Yolo Wonder' was similar to that of 'Yolo Wonder', indicating the recessive nature of the resistance to PVY(0), PVY(1, 2) and potyvirus E (Fig. 1). In the DH progeny, the ratio between resistant and susceptible lines as determined by DAS-ELISA (19-75, 0-94 and 5-89 for PVY(0), PVY(1, 2) and potyvirus E, respectively) did not fit with simple genetic models. Thus, the DH lines were assessed for resistance using the AUSPC criterion. The distribution of DH lines in AUSPC classes was continuous between resistant and susceptible parents (Fig. 1). The quasi-bimodal distributions suggested the intervention of major factors in resistance to PVY(0) and potyvirus E, whereas the distribution of DH lines for resistance to PVY(1, 2) indicated a more complex genetic basis. Moreover, no DH lines were found to be as resistant as the resistant parent 'Perennial'.

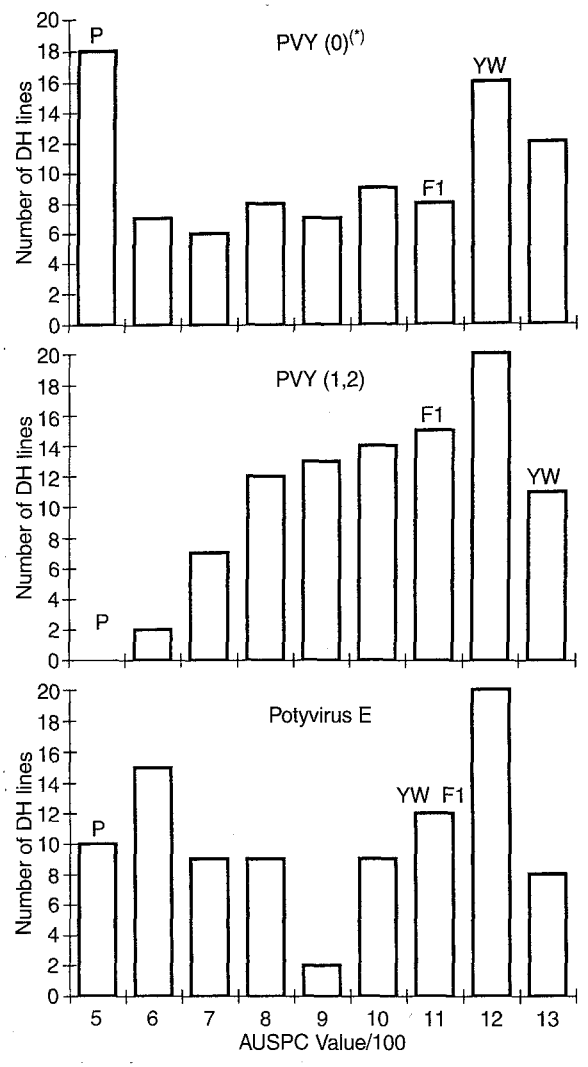


Fig. 1 Distribution of the DH lines assessed by the AUSPC criterion for PVY(0), PVY(1,2) and potyvirus E resistance (*P* 'Perennial', *YW* 'Yolo Wonder', F_1 hybrid 'Perennial' × 'Yolo Wonder' (*) Because of missing data, only 91 DH lines were assessed for PVY(0) resistance

The narrow-sense heritability estimates obtained in this study for all potyviruses were high (Table 3). These values attest to our confidence in the estimation of resistance by a phenotypic evaluation. Estimates of the number of segregating factors (Choo and Reinberg 1982a) for resistance to PVY(0), potyvirus E and PVY(1,2) were 2.7, 3.1 and 7.7 respectively (Table 3).

Table 2 DAS-ELISA absorbance means and standard errors in leaves from 'Perennial', 'Yolo Wonder' and F_1 ('Perennial' × 'Yolo Wonder') 5 weeks after inoculation with PVY(0), PVY(1,2), potyvirus E and CVMV

	PVY(0)	PVY(1,2)	Potyvirus E	CVMV
Healthy control	0.150	0.143	0.108	0.138
Perennial	0.182 ± 0.028	0.681 ± 0.254	0.114 ± 0.002	0.121 ± 0.043
Yolo Wonder	1.968 ± 0.546	2.445 ± 0.113	2.109 ± 0.331	2.176 ± 0.322
F_1 (Perennial × Yolo Wonder)	Necrotic	2.212 ± 0.324	2.168 ± 0.234	0.170 ± 0.403

Table 3 Variance components (σ_A^2 and σ_E^2), heritability (h_n^2) and estimated number of genetic factors involved in PVY(0), PVY(1,2) and potyvirus E resistance

Virus	PVY(0)	PVY(1,2)	Potyvirus E
σ_A^2	76 933	32 873	65 968
σ_E^2	8 392	9 376	11 498
h_n^2	0.96	0.90	0.95
μ	856	975	864
L1	1 313	473	1 313
k'	2.7	7.7	3.1

Inheritance of CVMV resistance

F_1 progeny of the 'Perennial' × 'Yolo Wonder' cross was found to be similar to 'Perennial', i.e. without any symptom, indicating that resistance to CVMV may be due to dominant factors.

It was not possible to assess resistance using the AUSPC criterion because no clear differences were observed between DH lines for period of time between inoculation and expression of symptoms. The DH lines fall into two clear-cut phenotypic groups of either with and without symptoms lines: 63 lines were resistant to CVMV, and 27 were susceptible. The simplest genetic hypothesis fitting best with the segregations observed in the DH progeny was that CVMV resistance is controlled by two unlinked genes that confer independantly an absolute resistance [χ^2(3:1 ratio) = 1.2, $P = 0.27$].

Correlations between resistances

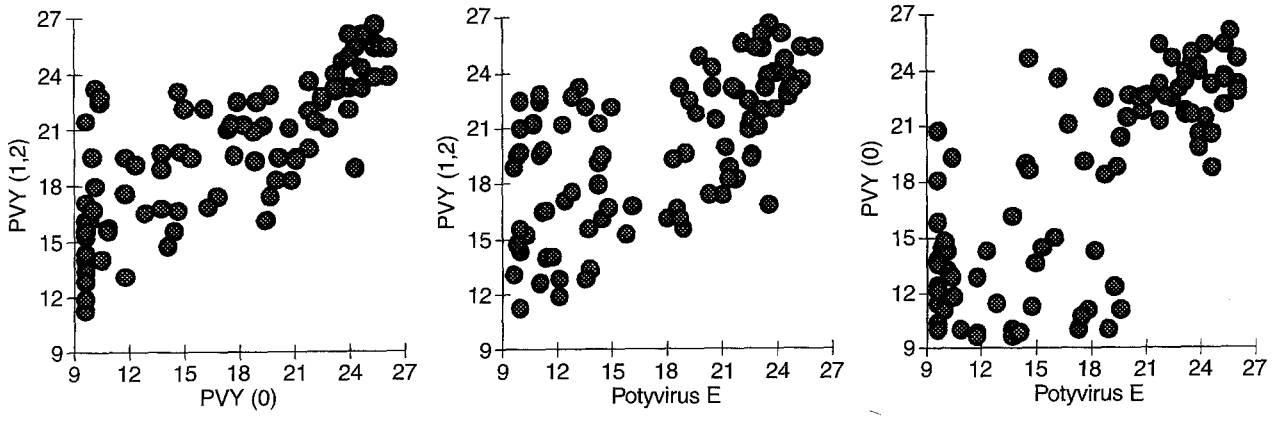
The relationships between the resistances to the different viruses were studied in the DH progeny. All of the traits were significantly genetically correlated ($P < 0.0001$), with a correlation coefficient of 0.51 between potyvirus E and PVY(1,2) resistance, 0.68 between PVY(0) and PVY(1,2) and 0.69 between PVY(0) and potyvirus E. The most resistant lines to the PVY(1,2) were also resistant to PVY(0) and potyvirus E (Fig. 2). However, this relationship was not reciprocal, and several lines with resistance to PVY(0) or potyvirus E were rather susceptible to PVY(1,2). Relationships between PVY(0) and potyvirus E resistances seem stronger since most lines show a similar behaviour towards both viruses.

The relationship between resistance to CVMV and resistances to the other viruses was tested by comparing the population of CVMV-resistant lines to the population of CVMV-susceptible lines for their mean AUSPC values obtained with the other viruses (t-test). No significant mean differences were detected between the two samples for resistance to PVY(0) ($P = 0.45$) or PVY(1, 2) ($P = 0.88$); however, CVMV-resistant lines appeared to be slightly more resistant to PVY(0) than CVMV-susceptible lines ($P = 0.055$).

Discussion

In many cases, resistance to plant viruses is under a simple genetic control involving a single dominant or recessive gene (Fraser 1990) and can be qualitatively evaluated by the presence/absence of the virus or by plant response to infection, i.e. symptoms polygenic. Resistance to viruses has rarely been analysed, perhaps because of the difficulties in accurately assaying the intermediate levels of resistant responses. In our study, qualitative differences were observed between the parents and between the segregating lines when they were all inoculated with CVMV. However, with respect to the other viruses, both symptom intensity and presence of the virus (checked by DAS-ELISA method) varied both as a function of time after inoculation and of the organs sampled. In order to more accurately evaluate the reaction of the whole plant and to include the intermediate segregating DH lines in the genetic analysis, we performed a quantitative evaluation using both the intensity of symptoms and incubation period (length

Fig. 2 Relationships between resistances to several potyviruses in the DH progeny. Susceptibility was measured by the AUSPC criterion



of time between inoculation and expression of symptoms). The AUSPC value calculated from these criteria probably takes different resistance mechanisms into account. However, the homogeneous behaviour of the plants from a single haplodiploid line and the reproducibility of the evaluation through three independent repeats for each virus confer a high heritability to this AUSPC index, indicating that the variations observed were mostly genetically controlled.

In the present study, the Indian line 'Perennial' was found to be totally resistant to PVY(0), potyvirus E and CVMV and highly resistant to PVY(1,2), confirming earlier results by Pochard et al. (1983). Resistance to CVMV appeared to be qualitatively controlled and two major genes, confering independantly the resistance, might account for the observed segregations. At least one of these genes was dominant. The quantitative segregation observed for the other potyviruses suggested that several recessive genes were segregating. The method of Choo and Reinbergs (1982 a) that was employed to estimate the minimum number of effective factors involved in the resistance assumes that linkage is absent, that gene effect is equal at each locus, that gene action is additive and that one extreme DH line contains all of the alleles segregating between the parents for resistance or susceptibility. Deviation from these assumptions leads to an underestimation of the number of genetic factors. However, relative values can be compared: fewer resistance factors were necessary to explain resistance to PVY(0) and potyvirus E (estimates of 2.7 and 3.1, respectively) than for PVY(1,2) resistance (at least seven). The skewing of the segregation observed for PVY(1,2) resistance also suggests that epistatic effects may be involved (Choo and Reinbergs 1982 b). Similarly, 19 DH lines among the 94 tested were found to be free of PVY(0), whereas only 5 lines were free of potyvirus E, and none of the 94 lines were found to be as resistant as 'Perennial' to PVY(1,2). This strongly confirms that more and more genes were necessary to confer resistance to PVY(0), potyvirus E and PVY(1,2), respectively. These results also indicate that the general resistance of 'Perennial' to potyviruses arises from the partial and complementary effects of several genes for PVY and potyvirus E plus major genes for resistance to CVMV.

The occurrence of common genetic factors being involved in the resistance to these different potyviruses was explored. According to genetic correlations and Fig. 2, most of genetic factors involved in potyvirus E and PVY(0) resistance appear to be the same, or related, and some of these genetic factors may also be involved in PVY(1,2) resistance, confering only a partial resistance to this virus. The presence of DH lines resistant to PVY(0) and potyvirus E but susceptible to PVY(1,2) indicates that additional specific factors for PVY(1,2) resistance are necessary. These results suggest that both common and specific resistance factors are involved in the resistances to potyviruses; these common genetic factors will be called polyvalent factors. According to Student's test, the genetic basis of CVMV resistance appears to be different from that of PVY resistance, however, because of the difference between CVMV-resistant and CVMV-susceptible lines for resistance to potyvirus E, we can hypothesize that one of the two CVMV resistance genes is also involved in resistance to potyvirus E. So, in this study, we have shown that multipotyvirus resistance in 'Perennial' is polygenically controlled and includes both polyvalent genetic factors and strain-specific genetic factors. This DH progeny was also used to construct a molecular intraspecific map of pepper (Lefebvre et al. 1995) that will allow us to more precisely define the number and position of genomic regions involved in potyvirus resistance and the individual effect of each resistance factor.

Cases in which associations between monogenic resistance to several potyviruses exist, and these have been described in pea (Provvidenti and Hampton 1993; Provvidenti and Niblett 1994), *Cucurbita moschata* (Gilbert-Albertini et al. 1993) and bean (Fisher and Kyle 1994). In these cases, the broad spectrum of viral resistance was explained either by the action of only one gene with pleiotropic effect or to a cluster of tightly linked genes. In pepper, recessive resistance to PVY strains and to TEV has been shown to be controlled by an allelic series at a single locus (Cook and Anderson 1959, Cook 1961), and dominant resistance to PVY and PeMV has also been shown to cosegregate at another locus (Dogimont et al. 1995). Pleiotropic effects of resistance genes or gene clusters were proposed by Kyle and Rybicki (1991) to result from the recognition by the host gene of a common determinant (sequences) shared by the different potyviruses. This hypothesis should indicate that the PVY strains, potyvirus E and, to a lesser extent, CVMV may share determinants of pathogenicity. Since the broad-spectrum genetic factors from 'Perennial' control quantitative variations in resistance, the correlations observed may also result from common defence mechanisms.

With respect to the pepper-PVY interaction, several distinct loci were characterized that confer complete resistance to PVY: resistance can be controlled by recessive alleles from the *vy* locus, by a dominant allele from 'Criollo de Morelos 334' or by polygenic resistance in 'Perennial'. Some of these resistance genes are strain-specific, others show a wide spectrum of action, indicating that these specific and non-specific resistance genes should be triggered by different determinants in the same virus. A similar variability among resistance factors was observed in the genetic components of the resistance of 'Perennial', showing that polygenic quantitative resistance and monogenic complete resistance could be related.

A large variability of resistance systems was developed by the host against these highly variable potyviruses. With such a variability of resistance sources, different gene combinations can be constructed (Palloix 1992), thereby associating high resistance to endemic viruses and partial resistance to other potyviruses.

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