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Inheritance of resistance to *Plum pox virus* in apricot (*Prunus armeniaca* L.)

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Abstract The inheritance of resistance to *Plum pox virus* (PPV) has been studied in 1,178 apricot hybrids. Seven hundred and eighteen F1 hybrids, obtained from controlled crosses between the susceptible Greek cultivar "Bebecou" and the resistant PPV cultivars of American origin ("Stark Early Orange," 'NJA2," 'Veecot," "Sunglo," "Harlayne," and "Orangered") were evaluated for resistance to the PPV-M (Marcus) strain, 8 years after artificial inoculation. The inheritance of resistance to PPV has been additionally studied for the first time in a BC_1 population of 95 apricot hybrids for four vegetative periods. Reaction of each hybrid to PPV-M was scored through visual symptoms, indexing onto GF-305 and double-antibody sandwich enzyme-linked immunosorbent assay tests. Segregation within the hybrids, determined by Chi-squared analysis, fits a 1:1 ratio ($P \leq 0.05$) of the resistant vs susceptible, indicating that resistance to PPV is controlled by a single dominant gene locus and that the above six resistant cultivars are heterozygous for the trait. Plants carrying this gene may initially develop disease symptoms on leaves but eventually recover and no virus can be detected in leaves. Susceptible plants show similar symptoms initially but remain symptomatic. Inheritance of resistance to PPV also has been studied in 365 F1 hybrids by crossing the resistant cultivar "Stella" with the susceptible "Bebecou" and the resistant cultivars "Sunglo" and "NJA2," for 8 years after inoculation. The segregation ratio was 1:0 (resistant/susceptible) suggesting that "Stella" is homozygous for the resistance trait. The purpose of this work was the enhancement of the knowledge of inheritance of resistance to PPV for the selection of new cultivars.

Keywords Resistance · Plum pox virus · Sharka · Breeding · RNA silencing · Recovery

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Introduction

Sharka disease, caused by *Plum pox virus* (PPV), is one of the most devastating and economically important diseases of stone fruits. The disease was first described in Bulgaria (Atanassoff 1932). It was detected in Greece in 1967 (Demetriades and Catsimbas 1968). The PPV-M (Marcus) is the prevalent local strain (Varveri et al. 2004). PPV is a member of the *Potyviridae* family, the largest and one of the most economically important groups of plant viruses. The virus is characterized by a single-stranded ribonucleic acid (RNA) genome, a simple infective molecule with a molecular weight of about 3.5×10^6. It spreads not only by infected propagating material but also by insect vectors in a nonpersistent manner. Apricots and plums are particularly susceptible to both D and M strains of the virus. Representatives of the M strain, used in experiments for comparison of apricot genotypes in Germany, did not prove to be more aggressive than isolates of the D strain (Fuchs et al. 2001). Nemeth 1986 described typical PPV-induced symptoms.

Although the virus is endemic to Eastern Europe, it has been spread throughout Europe, the Mediterranean region, and recently to several locations in the western hemisphere.

The first report of PPV in North America came from a peach orchard in Pennsylvania in 1999 (Levy et al. 2000), and in the summer of 2000, it was found in Ontario, Canada. PPV is still localized near the initial infection foci in these areas. Major stone fruit-producing states in the USA are threatened by the possibility of accidental movement of infected propagation material or introduction of infectious aphids. California, one of the largest peach, plum, and apricot producers in the world, grows very large commercial orchards in close proximity, under ideal environmental conditions for rapid aphid spread.

All apricot cultivars of European origin are susceptible to Sharka. Resistance has been found only in some North American cultivars (Syrgiannidis 1979; Karayiannis 1989; Dosba et al. 1992; Karayiannis and Mainou 1994; Karayiannis et al. 1999b). In a study of genetic diversity based on ten enzymatic systems, the North American PPV-resistant cultivars were found very distant from the rest of the cultivars, mainly because of the presence of rare alleles found in an Asian apricot-related species (Badenes et al. 1996).

Apricot breeding for resistance to PPV was initiated in Greece in 1982, and it was found that the resistance trait is inherited (Syrgiannidis and Mainou 1993). The program has been enlarged and is continued until now, to transfer resistance to the best commercial local cultivars (Karayiannis et al.1999a, 2006; Karayiannis 2003). The selection process, however, is slow because of the undesirable characteristics carried by the resistant cultivars, such as floral self-incompatibility, high chilling requirements, and undeveloped pistil.

Studies on the inheritance of resistance to PPV in apricot carried out previously present three different hypotheses. They suggest that resistance is controlled by one (Dicenta et al. 2000), two (Dosba et al. 1991; Moustafa et al. 2001; Vilanova et al. 2003; Rubio et al. 2004), or three genes (Guillet-Bellanger and Audergon 2001; Salava et al. 2005). More than 80% of the viral resistance reported to date is monogenically controlled, and the remainder shows oligogenic or polygenic control (Fraser 1990). Only slightly more than half of all reported monogenic resistance traits show dominant inheritance (Kang et al. 2005).

The objectives of the current study were to elucidate the inheritance of resistance to PPV in apricot for the selection of new cultivars and to evaluate the utility of different methods as criteria for selection of resistance.

Materials and methods

Plant material used

One thousand one hundred and seventy-eight apricot hybrids obtained from 12 different crosses were examined for resistance to Sharka. They included seven F_1 populations of 718 hybrids obtained from crosses of the local susceptible cultivar "Bebecou" with the PPV-resistant cultivars of American origin "NJA2," "Harlayne," "Stark Early Orange," "Veecot," "Sunglo," "Orangered," one BC_1 population of 95 hybrids obtained from the cross of the susceptible "Bebecou" with the resistant selection 165/92 (Veecot×Bebecou), and four F1 populations of 365 hybrids from crosses where the resistant cultivar "Stella" was one of the parents. All crosses were performed in 1991 except the "Bebecou×Orangered" and BC1 cross, which were completed in 2001. Seeds were stratified, and when germinated, they were planted in the nursery. They were transplanted in the field, in 1.5×3.0-m distances, 10 months later.

Inoculum used

The local PPV-M strain was used as inoculum. It was preserved in PPV-infected old trees of the very susceptible apricot cultivar "Proimo Tirynthos" grown in the field. Artificial inoculations by grafting were carried out in 1995 and 2002 for the hybrids of 1991 and 2001, respectively. Observations for disease symptoms on leaves and fruits of the hybrids were taken for a period of 4–8 years postinoculation. The PPV incidence was scored on each hybrid twice during each vegetative cycle as follows: 0=absence of symptoms, 1=symptoms in one or two leaves, 2=symptoms in three to five leaves, and 3=symptoms in more than five leaves. Malformation of leaves and the type, size, and number of discoloration were taken into account to adjust visual scoring of the symptoms. The mean of both scores was used.

Evaluation and genetic analysis

Natural inoculation

All hybrids were exposed under conditions of high inoculum pressure in the field where natural transmission of the virus is by aphids. Insecticidal sprays were suspended to allow aphids to transmit the virus from the trees of heavily infected neighboring apricot and peach orchards.

Artificial inoculation by grafting onto old PPV diseased apricot trees

Each hybrid was budded by two buds onto annual shoots of a very old apricot tree heavily infected by PPV (usually cultivar P. Tirynthos). Observations of PPV symptoms were made on leaves and fruits on the shoots coming from the buds of each hybrid, for eight vegetative periods after budding.

Artificial inoculation by grafting onto GF-305 rootstock in the field:

GF-305 peach rootstock, which is used as a plant index, is known to favor PPV multiplication. Each hybrid was budded onto two 1-year-old GF-305 seedlings. Inoculation of GF-305 plants was performed 2 weeks later by inserting two PPV-M-infected chips or buds below the point of the hybrid budding. The inoculations were done during August and September. Comprehensive observations of Sharka disease symptoms on the hybrids leaves were obtained at the end of April to the beginning of May for the next vegetative periods.

Indexing onto GF-305 peach seedlings in an insect-proof greenhouse

Hybrids not showing disease symptoms on leaves or fruits by the previous procedures were subjected to indexing, in an insect-proof house, by grafting them onto 6-month-old GF-305 seedlings. Two buds from each hybrid were grafted onto each of two GF-305 plants in August**.** One GF-305 plant was left as a control. Observations for disease symptoms on the hybrid's leaves and on the leaves of the plant index were made early in May of the following year.

Serological analyses

The double-antibody sandwich enzyme-linked immunosorbent assay (DAS-ELISA) procedure (Clark and Adams, 1977) was conducted for hybrids not showing symptoms after the indexing procedure**.** Three leaves from the top, middle, and bottom of each examined hybrid were collected in spring. Extracts were made by grinding samples (250 mg) in 5 ml phosphate-buffered saline buffer (containing 0.5 ml Tween 20; w/v), set to pH 7.2 and supplemented with 2% (w/v) polyvinylpyrrolidone and 0.2% (w/v) sodium diethyl dithiocarbamate. Samples were tested in duplicate wells in polystyrene microtiter plates using a commercially available polyclonal anti-PPV serum and alkaline phosphatase conjugate according to the protocol of the manufacturer (BIOREBA AG, Switzerland). The optical densities were read at 405 nm, in a Bio-Tek Instruments (Highland Park, Winooski, VT) automatic reader to detect the presence or absence of the virus within the leaves. At the end of the above procedures, only hybrids with no symptoms of the disease and ELISA negative were considered resistant.

Results

The results of the evaluation of 718 hybrids of crosses between susceptible and resistant cultivars, 8 years post-inoculation, are presented in Table 1. Approximately 50% of the segregants presented disease symptoms on leaves, fruits, and stones. The ratio of resistant to susceptible seedlings were 46: 60, 70:82, 38:50, 59:51, and 45:53 in susceptible×resistant and 62: 52 or 24:26 in resistant× susceptible crosses.

All hybrids from the cross "Stark Early Orange"× "Bebecou" produced Sharka disease symptoms on their leaves the first spring after artificial inoculation. However, only those hybrids that had inherited resistance recovered from symptoms by the second spring postinoculation and produced symptomless fruits thereafter, while the rest of the hybrids remained symptomatic. ELISA assays could not detect virus multiplication in leaves of the resistant hybrids.

Table 1 Genetic analysis of inheritance of resistance to PPV-M, in seven F_1 apricot hybrid populations created by crossing the susceptible cultivar "Bebecou" with the resistant cultivars "NJA2," "Harlayne," "S. Early Orange," "Veecot," "Sunglo," and "Orangered" and in a BC_1 population created by crossing "Bebecou" with the resistant hybrid "Veecot×Bebecou"

Cross combination $P_1 \times P_2$	Total Number of artificially inoculated hybrids F_1	Segregation observed r/s	Percent susceptible hybrids	χ^2	Ratio expect. r/s
Bebecou×NJA2	106	46: 60	56	1.85	1:1
Bebecou×Harlayne	152	70: 82	54	0.95	1: 1
S. Early Orange× Bebecou	114	62: 52	46	0.88	1:1
Veecot×Bebecou	50	24: 26	52	0.08	1:1
Bebecou×Veecot	88	38: 50	56	1.63	1:1
Bebecou×Sunglo	110	59: 51	46	0.58	1:1
Bebecou×Orangered	98	45: 53	54	0.65	1:1
Total	718	344: 374	52		1:1
Bebecou×(Veecot× Bebecou)	BC_1 95	42: 53	56	1.27	1:1

df=1, *P*=0.05, χ^2 =3.84, hypothesis tested 1:1
r Resistant, *s* susceptible

Ninety-eight hybrids of the cross "Bebecou"×"Orangered" segregated as 45 resistant/53 susceptible**.** In total, 46% of the hybrids expressed resistance to PPV, and 54% expressed susceptibility. Parent "Orangered" shows a reaction after artificial inoculation with leaf scorching up to 20 cm from the point of inoculation. Some shoots in resistant hybrids of this population began to show leaf necrosis, but the virus was localized, and the plants were PPV-free by ELISA test.

The study of inheritance of resistance to PPV in a BC1 population was carried out for the first time. The 95 members of the cross "Bebecou"×("Veecot"×"Bebecou") segregated as 42 resistant/53 susceptible, by the end of the fourth year postinoculation, following the 1:1 ratio (Table 1)**.** The same hybrids**,** grown on their own roots in the field, under conditions of natural aphid transmission of the virus, responded the same way after 5 years of high inoculum pressure, which is an additional proof of the mode of inheritance of resistance to PPV. In total, 44% of the hybrids were found to be resistant, and 56% were found to be susceptible.

The results of a comparative analysis of 58 members of the cross "Veecot"×"Bebecou" are presented in Table 2. Twenty-three trees among them expressed Sharka disease symptoms 10 years after growing in the field under conditions of natural aphid transmission of the virus, whereas 26 showed positive symptoms 8 years after artificial inoculation by grafting. The levels of relative concentration of PPV in artificially inoculated hybrids were determined by the DAS-ELISA test (Table 2). In two hybrids, A882/91 and A899/91, the ELISA value was low in contrast with the positive result of artificial inoculation. Two hybrids, A889/91 and A893/91, have apparently escaped infection in the field. In evaluating resistance, artificial inoculation is more important.

Evidence on the basis of the data (Tables 1 and 2) suggests that resistance to PPV is controlled by a single dominant gene locus. The ratio of inheritance observed was consistent with the expected after analyses in a χ^2 goodness-of-fit test at the P=0.05.

Inheritance of resistance to PPV proved to be completely different in crosses with cultivar "Stella" as one of the parents. One hundred and eighty-four hybrids from the cross and reciprocal cross of "Stella" with the susceptible cultivar "Bebecou" were found resistant to PPV. Moreover, 181 hybrids from the crosses of "Stella" with the resistant cultivars "Sunglo" and "NJA2" also were found to be resistant (Table 3).

Discussion

The information about the genetic control of resistance to Sharka disease in *Prunus armeniaca* L. has been controversial.

Table 2 Comparative analysis of leaf reaction to PPV-M of 58 F1 apricot hybrids "Veecot"×"Bebecou," after artificial inoculation by grafting, natural inoculation by the aphids in the field, and by the DAS-ELISA test

Hybrid number	[a]Natural inoculation	[b]Artificial inoculation	[c]DAS-ELISA optical density at 405 nm
A873/91	3	+	
A874/91	2	+	0.567
A875/91	3	+	0.318
A876/91	0	−	0.045
A877/91	3	+	0.527
Á878/91	0	−	0.049
A879/91	2	+	0.643
A880/91	0	−	0.038
A882/91	3	+	0.082
A884/91	0	−	0.044
A885/91	0	−	
A887/91	0	−	0.048
A888/91	3	+	
A889/91	0	+	0.740
A890/91	0	−	0.048
A891/91	0	−	0.041
A892/91	2	+	0.334
A893/91	0	+	0.731
A894/91	0	−	0.051
A895/91	0	−	0.054
A896/91	0	−	0.052
A897/91	3	+	0.573
A898/91	0	−	0.050
A899/91	2	+	0.049
A900/91	0	−	0.048
A901/91	0	−	0.044
A902/91	0	−	0.044
A903/91	3	+	0.471
A905/91	3	+	0.864
A906/91	0	−	0.061
A907/91	2	+	0.693
A908/91	0	+	0.130
A909/91	0	−	0.050
A910/91	0	−	0.051
A912/91	3	+	0.510
A913/91	3	+	0.838
A915/91	3	+	0.513
A916/91	0	−	0.047
A917/91	3	+	0.662
A920/91	0	−	0.046
A921/91	0	−	0.051
A922/91	0	−	0.047
A923/91	0	−	0.045
A924/91	3	+	
A925/91	0	−	
A926/91	0	−	0.043
A927/91	0	−	0.041
A928/91	0	−	0.045
A929/91	0	−	0.045
A930/91	0	−	0.050
A931/91	3	+	0.457
A932/91	3	+	0.666

Table 2 (continued)

Hybrid number	[a]Natural inoculation	[b]Artificial inoculation	[c]DAS-ELISA optical density at 405 nm
A933/91	2	+	0.652
A934/91	3	+	0.957
A935/91	2	+	0.787
A936/91	0	−	0.046
A937/91	0	−	0.052
A938/91	0	−	0.059
Positive control			0.921
Negative vcontrol			0.045

[a] Natural inoculation: degree of symptoms intensity (0–3); 0=absence of symptoms, 1=symptoms in one or two leaves, 2=symptoms in three to five leaves, and 3=symptoms in more than five leaves
[b] Artificial inoculation:+=presence of symptoms; −=absence of symptoms
[c] Optical density; average of paired wells

Published studies suggest that resistance to PPV in apricot may be controlled by one, two, or three genes. This controversy may be caused by the small number of descendants in the families studied, the short duration of time given in evaluating the trait, or the methodology used for inoculations.

This report is the first to investigate the mode of inheritance of resistance to PPV in many different large hybrid populations over an extended time frame. Our results suggest that resistance is inherited as a dominant monogenic trait and the resistant parents "NJA2," "Harlayne," "Veecot," "Sunglo," "Orangered," and "S. Early Orange" are heterozygous for the trait.

The PPV resistance for cultivars

"NJA2" and "Orangered" can be traced to a single source: *P. armeniaca mandshurica* "Scout" (Cociu 1993). "Scout" is a 1937 Canadian introduction, and it was obtained from seeds sent from Manchuria (China). The "Morden 604" selection was obtained from "Scout"×"McClure." Both "NJA2" ("Morden 604" open-pollinated seedling) and "Orangered" ("Lasgerdi Mashhad"×"NJA2") have been created at New Jersey (New Jersey Agricultural Experiment Station; Goffreda 1999). For cultivars "Harlayne" (Layne 1981) and "Veecot," both of Canadian origin, resistance can be traced to their ancestor "Reliable" ("Wenatchee Moorpark"×"Hewetson"; Cociu 1993).

In resistant hybrids of the cross "Veecot"×"Bebecou," PPV symptoms have not been observed near the point of inoculation. A host response apparent is that the virus cannot move beyond the initial focus of inoculation.

Cultivars "Orangered" and "Harlayne" are extremely resistant (Karayiannis et al. 1999b). They have been considered "immune" to PPV by Fuchs et al. 2001, after artificial inoculations and polymerase chain reaction tests, independently of the PPV isolates used. The mechanism of resistance inherited by their hybrids is probably related to resistance to virus movement. The response observed in "Orangered" and in some of its hybrids interferes with cell-to-cell movement. Dominant resistance is often associated with a hypersensitive reaction (HR; Fraser 1990). In the case of potyviruses, which do not encode a dedicated movement protein, the movement functions have been allocated to several proteins including the coat protein, helper component proteinase, the cylindrical inclusion protein, and the genome-linked protein (Revers et al. 1999).

In our experiments, it has been observed that resistance mechanisms do need some time to build up, as in the case of resistant hybrids "Stark Early Orange"×"Bebecou," which recovered the second spring postinoculation. It appears that the nature of resistance conferred by "Stark Early Orange" is not a hypersensitive reaction-related resistance or an immunity type but a recovery from PPV symptoms. The mechanism involved in this type of pathogenesis and resistance suggests a virus induced gene silencing (Baulcombe 2004), a major discovery of the past decade, which exploits an RNA-mediated antiviral defense mechanism. It has been shown that virus infection of nontransgenic plants induces a resistance mechanism similar to that of transgene-induced gene silencing, and leaves that develop after systemic infection of a plant by a virus contain lower concentrations of the virus and are

Table 3 Genetic analysis of inheritance of resistance to PPV-M in four F_1 apricot hybrid populations created by crossing the resistant apricot cultivar "Stella" with a susceptible or a resistant cultivar and in a F1 population from the cross of two resistant parents other than "Stella"

Parents	Total number of artificially inoculated hybrids F_1	Resistant hybrids	Susceptible hybrids	χ^2	Segregation ratio r vs s
Stella×susceptible					
Bebecou×Stella	108	108	0		1:0
Stella×Bebecou	76	76	0		1:0
Stella×resistant					
Stella×Sunglo	53	53	0		1: 0
Stella×NJA2	128	128	0		1:0
Total	365	365	0		1:0

df=1, P=0.05, χ^2=3.84
r Resistant, *s* susceptible

symptom-free and have essentially recovered (Ratcliff et al. 1997). When a plant virus infects a host cell, it activates an RNA-based defense that is transmitted systemically and is targeted against the viral genome (Ratcliff et al. 1999).

Parent cultivar Stella has been previously shown to be extremely resistant to PPV, and disease symptoms never developed on leaves or fruits since 40 years (Syrgiannidis 1979). The type of segregation followed in crosses with "Stella" was different. All the descendants from crosses of "Stella" with a susceptible or a resistant cultivar were found to be resistant to PPV, which suggests that "Stella" is homozygous for the dominant allele that induces resistance. Progeny from open pollination of "Stella" segregated by the same way (Dicenta and Audergon 1998).

Responses similar to our results have been observed on bean (*Phaseolus vulgaris* L.) infected by Bean common mosaic virus (BCMV), a member of the genus *Potyvirus*. A homozygote is immune to BCMV infection, while a heterozygote responds to infection with a HR, which may result in systemic necrosis (Collmer et al. 2000).

Our results indicated that there is evidence for monogenic inheritance of resistance to PPV in apricot but distinct resistance mechanisms, implying different resistance loci among the American parent cultivars. Apricot breeding in the future, for the incorporation of more and different genes for resistance, will increase the durability of resistance to Sharka disease.

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