Eur J Plant Pathol (2010) 126:241–247
DOI 10.1007/s10658-009-9536-x

Identification of resistance to bacterial canker (*Pseudomonas syringae* pv. *syringae*) disease on apricot genotypes grown in Turkey

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Received: 11 February 2009 / Accepted: 28 September 2009 / Published online: 11 October 2009

Abstract Bacterial canker caused by *Pseudomonas syringae* pv. *syrinage* (Pss) in apricot has widely spread in Turkey, especially in Malatya province, in recent years. The main objective of this study was to determine resistance of apricot cultivars to bacterial canker caused by Pss in apricot cultivars grown in Turkey. During the 2006–2007 growing period, bacterial isolations were taken from diseased apricot trees in Malatya and 53 *Pseudomonas syringae* isolates were obtained. Forty-two isolates were determined as *Pseudomonas syringae* pv. *syringae* and 11 isolates as pv. *morsprunorum*. In a pathogenicity test, leaves of cv. Hacihaliloğlu were used and five Pss isolates (K24, K25, K43, K47 and K51) were detected to be the most virulent and were used to test for cultivar resistance to Pss. Leaves of fifteen apricot cultivars (Alyanak, Çataloğlu, Çöloğlu, Erken Ağerik, Hacihaliloğlu, Hasanbey, İsmailağa, Kabaaşi, Karacabey, Sakit 2, Soğanci, Şam, Şekerpare, Tokaloğlu (Erzincan) and Turfanda Eski Malatya) were tested for resistance to Pss. Green shoots were spray-inoculated with a concentration of 10^8 cfu ml^{-1} Pss mixed culture. Sprayed shoots were covered with moist plastic bags for 3 days and maintained in the growth chamber and monitored for symptom development. Hasanbey, Çöloğlu, Soğanci and Şekerpare apricot cultivars were resistant and Şam, Tokaloğlu (Erzincan) and Erken Ağerik apricot cultivars were susceptible to Pss. This is the first report of a resistance source in apricot cultivars grown in Turkey against Pss.

Keywords *Prunus armeniaca* · Bacterial canker · Isolation · Identification · Cultivar response · Resistance

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Introduction

Apricot is the most important fruit crop grown in Anatolia; approximately 528,000 tons of fruit are produced annually, and Turkey is the biggest apricot-producing country in the world (FAO 2009). Malatya province is the main apricot-producing area not only in Turkey but also in the world. Although the main apricot cultivar grown in Malatya is Hacihaliloglu, a number of different cultivars such as Kabaasi, Hasanbey, Sekerpare, Soganci, Şam and Karacabey are grown in Malatya and Turkey.

Diseases of fruit trees caused by pathovars of *Pseudomonas syringae* are of major concern in fruit-

producing areas worldwide, are exceedingly difficult to control, and result in significant economic losses. Bacterial canker is caused by two closely related pathovars, *Pseudomonas syringae* pv. *syringae* (Pss) and pv. *morsprunorum*, (Psm), and is an important disease of apricot (*Prunus armeniaca*) and other stone fruits (Kennelly et al. 2007). Usually only one pathovar is found to cause the disease in most countries although the occurrence of both pathovars in orchards has been reported from different parts of the world (Jones 1971; Foulkes and Lloyd 1980; Wimalajeewa and Flett 1985). Disease symptoms include blossom blast and spur dieback, leaf and fruit lesions, cankers with associated gummosis of woody tissue, loss of scaffold limbs, and overall decreased fruit yields. The pathogen has the ability to kill both young and older trees. Systemic infection and death of young trees is a perennial problem in nurseries, and canker development leading to the girdling and death of scaffold limbs and entire trees are a common event that can lead to the rapid demise of older orchards (Kennelly et al. 2007).

Management of most fruit tree diseases caused by *Pseudomonas* spp. currently is almost unattainable, due to the lack of effective chemical or biological control measures, lack of host resistance, and the endophytic nature of the pathogen during some phases of the disease-cycle (Kennelly et al. 2007). Thus, use of apricot cultivars resistant to Pss is economically and technically the most practical method for effective management of bacterial canker (Bassi 1999).

Bacterial canker caused by Pss was determined in 20% of apricot trees in Malatya (Kavak and Citir 1995). Kotan and Sahin (2002) reported that a serious disease was observed on nearly 80% of apricot trees grown in commercial orchards and home gardens in provinces of Erzurum, Erzincan and Artvin in Turkey, and typical disease symptoms were especially shown in blossoms, shoots and trunk of trees. Unfortunately, no study on apricot resistance to Pss has yet been reported in Turkey. The objective of this study was to evaluate apricot cultivars commonly grown in Turkey for their response to Pss.

Materials and methods

Fifteen apricot cultivars (Alyanak, Çataloğlu, Çöloğlu, Kabaaşi, Karacabey, Şam, Erken Ağerik, Turfanda Eski Malatya, Sakit 2, Tokaloğlu (Erzincan), Hasanbey, İsmailağa, Hacihaliloğlu, Şekerpare and Soğanci) were tested in terms of resistance to Pss.

Isolation and characterisation of isolates

In 2006 and 2007, samples of diseased tissues from apricot fruit trees were collected from orchard sites in Malatya. Samples consisted of diseased dormant buds, twigs and branches. Bacteria were isolated from surface-disinfested (0.5% sodium hypochlorite for 1 min and rinsed in sterile water) stem cankers, symptomless buds and branches. Individual buds, tissue from margins of lesions and symptomless branches were removed aseptically, cut into small pieces, and transferred to screw-cap bottles containing sterile distilled water (SDW), then shaken vigorously. The liquid suspension was spread onto nutrient sucrose agar (NSA) containing 28.0 g of nutrient agar, 5.0 of sucrose, and 1,000 ml of distilled water. All cultures were incubated at 25–27°C. Bacteria were preserved at −80°C in sterile Lauryl Broth (LB) amended with 30% sterile glycerol (King et al. 1954; Klement et al. 1990).

Whole-cell fatty acids were extracted and methylated as described by Sasser (1990). Cells were streaked in a quadrant pattern and grown overnight on trypticase soy broth agar (TSBA). Approximately 50 mg of bacterial cells, harvested from the third and fourth quadrant streak of growth, were used for the extraction using standard extraction techniques. Fatty acid methyl esters (FAMEs) profiles were obtained by running samples on a Hewlett Packard Agilent GC 6890 GC fitted with a microprocessor containing the Sherlock Microbial Identification System (MIDI) Software (V.A. 06. 03). The FAME profiles were identified using the commercial TSBA database and these results confirmed with Biolog. Strains were tested for ability to metabolise carbon sources using Biolog GN2 plates (Biolog Inc., CA, USA). A single colony of each strain cultured on nutrient agar was inoculated onto TSBA and incubated for 24 h at 27°C. All following steps were performed according to the manufacturer's instructions. Results were read using the automated Biolog MicroStation reader and analysis was performed by using the MicroLog 3 Ver 4.20 software (Biolog). All 53 isolates of *Pseudomonas* spp. were tested for determination of biochemical characters (gram reaction, amylase test, catalase test, levan formation, arginine dihydrolase, nitrate produc-

Table 1 Fatty acid methyl ester (FAME) analysis results and some biochemical characters of *Pseudomonas* spp

Isolate no	MIS result	MIS similarity index (%)	Amylase production	Catalase production	Levan formation	Arginine dihydrolase	Nitrate production
K4	Pss	30	−	+	+	−	−
K9	Pss	89	−	+	+	−	−
K10	Pss	49	−	+	+	−	−
K13	Pss	88	−	+	+	−	−
K15	Pss	82	−	+	+	−	−
K17	Pss	77	−	+	+	−	−
K18	Pss	84	−	+	+	−	−
K20	Pss	75	−	+	+	−	−
K21	Pss	69	−	+	+	W^+	−
K22	Pss	55	−	+	+	−	−
K24	**Pss**	**75**	−	+	+	−	−
K25	**Pss**	**78**	−	+	+	−	−
K32	Pss	77	−	+	+	−	−
K33	Pss	54	−	+	+	−	−
K34	Pss	65	−	+	+	W^+	−
K36	Pss	55	−	+	+	−	−
K37	Pss	91	−	+	+	−	−
K38	Pss	74	−	+	+	−	−
K39	Pss	49	−	+	+	−	−
K40	Psm	80	−	+	+	−	−
K41	Psm	96	−	+	+	−	−
K42	Pss	79	−	+	+	−	−
K43	**Pss**	**76**	−	+	+	−	−
K44	Pss	63	−	+	+	−	−
K46	Psm	94	−	+	+	−	−
K47	**Pss**	**83**	−	+	+	−	−
K48	Pss	80	−	+	+	−	−
K49	Pss	73	−	+	+	−	−
K50	Pss	55	−	+	+	−	−
K51	**Pss**	**80**	−	+	+	−	−
K55	Pss	91	−	+	+	−	−
K56	Pss	37	−	+	+	−	−
K57	Psm	92	−	+	+	−	−
K58	Pss	92	−	+	+	−	−
K59	Pss	68	−	+	+	−	−
K60	Pss	54	−	+	+	−	−
K61	Psm	90	−	+	+	−	−
K62	Pss	92	−	+	+	−	−
K63	Psm	84	−	+	+	−	−
K64	Psm	83	−	+	+	−	−
K65	Psm	84	−	+	+	−	−
K68	Psm	94	−	+	+	−	−
K69	Pss	82	−	+	+	−	−
K70	Pss	54	−	+	+	−	−

Table 1 (continued)

Isolate no	MIS result	MIS similarity index (%)	Amylase production	Catalase production	Levan formation	Arginine dihydrolase	Nitrate production
K71	Psm	89	−	+	+	−	W^+
K72	Pss	75	−	+	+	−	−
K73	Pss	61	−	+	+	−	−
K74	Pss	67	−	+	+	−	−
K75	Pss	77	−	+	+	−	−
K77	Pss	76	−	+	+	−	−
K78	Pss	69	−	+	+	−	−
K96	Pss	72	−	+	+	−	−
K99	Pss	77	−	+	+	−	−

Pss: *Pseudomonas syringae* pv. *syringae*, Psm: *Pseudomonas syringae* pv. *morsprunorum*, − Negative, + Positive, W^+ Weakly positive

tion and oxidase reaction) as described by Lelliott and Stead (1987), Klement et al. (1990), Schaad (1994) and Narayanasamy (1997).

Hypersensitivity reaction (HR) and pathogenicity tests

The pathogenic potential of each isolate was estimated by its ability to induce a hypersensitivity reaction. Bacterial cells grown for 24–48 h on yeast dextrose calcium carbonate agar (YDC) at 27°C were suspended in SDW to a concentration of 10^8 cfu ml^{-1}. Bacterial suspensions were injected into the lower side of the leaves of tobacco (*Nicotiana tabacum* cv. Samsun) seedlings by using a 26-gauge needle. SDW was injected as a control. The plants were maintained in a greenhouse at 28°C and rated after 3 days for necrosis development in injected part of the leaves. Dead tissue in injected part of leaves or not was evaluated as HR positive (HR+) and negative (HR-), respectively (Klement et al. 1966; Lelliott and Stead 1987).

All *Pseudomonas* spp. isolates were tested for pathogenicity on leaves of apricot cv. Hacihaliloğlu (main apricot cultivar in Malatya). Green shoots of cv. Hacihaliloğlu were spray-inoculated with a concentration of 10^8 cfu ml^{-1} *Pseudomonas* spp. Sprayed shoots were covered with moist plastic bags for 3 days and maintained in the growth chamber with 25±2°C and 65–70% RH and monitored for symptom development. Bacterial strains causing necrosis in the leaves or not were evaluated as pathogenicity positive and negative, respectively. Reisolation from symptomatic leaves was performed by using the techniques described above (Scheck et al. 1997; Scortichini and Morone 1997).

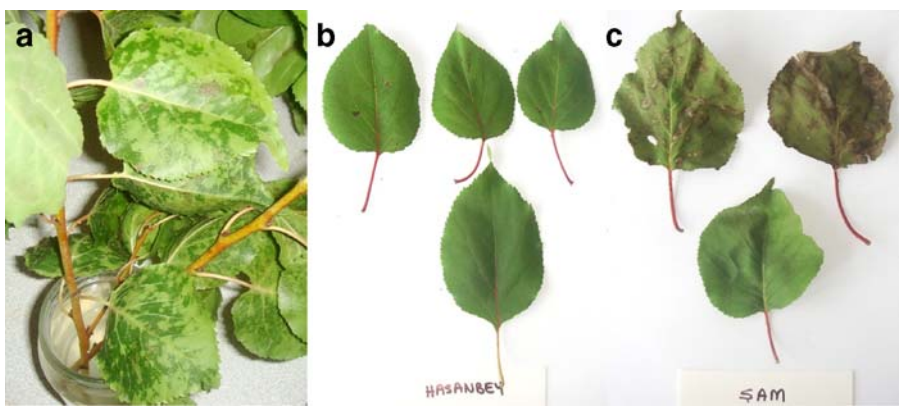

Fig. 1 Resistance of some apricot varieties to Pss. **a** Symptoms in cv. Tokaloğlu (Erzincan) four days after spraying; **b** in cv. Hasanbey, and **c** in cv. Şam 21 days after spraying

Table 2 Reaction of apricot cultivars to Pss

Cultivars	Mean[a]	Classes[b]
Alyanak	2.10 ef	MS
Çataloğlu	2.77 cd	MS
Çöloğlu	1.87 f	R
Erken Ağerik	3.00 bc	S
Hacıhaliloğlu	2.63 cd	MS
Hasanbey	1.73 f	R
İsmailağa	2.60 cd	MS
Kabaaşı	2.87 c	MS
Karacabey	2.73 cd	MS
Sakıt 2	2.63 cd	MS
Soğancı	1.87 f	R
Şam	3.40 a	S
Şekerpare	1.83 f	R
Tokaloğlu (Erzincan)	3.30 ab	S
Turfanda Eski Malatya	2.40 de	MS
LSD.01	0.37	

[a] Leaf disease rating scale: 1 = symptomless, 2 = a few necrotic symptoms, 3 = many spots, some coalescing, 4 = severe spots and leaf defoliation, and 5 = plant dead

[b] Classes: *R* resistant, *MS* moderately susceptible, *S* susceptible

Certain isolates were tested for apricot cultivar response to Pss. Mixed culture of Pss strains K24, K25, K43, K47 and K51 (the most virulent strains) were used for inoculation of leaves by a spray method. Control shoots were sprayed with SDW. The bacterial cultures were grown on YDC medium (Lelliott and Stead 1987) in Petri plates for 48 h at 27°C. Green shoots were spray-inoculated with a concentration of 10^8 cfu ml^{-1} Pss mixed culture. Sprayed shoots were covered with moist plastic bags for 3 days and maintained in the growth chamber with 25±2°C and 65–70% RH and monitored for symptom development. Disease severity was assessed 21 days after inoculation by using a leaf disease rating scale: 1 = symptomless; 2 = a few necrotic spots; 3 = more spots, some coalescing; 4 = severe spot and leaf defoliation; and 5 = plant dead. Green shoots were arranged separately on a growth chamber bench in a completely randomised design with three replicates and five shoots per replicate. Experiments were conducted twice for each cultivar. The data were evaluated by analysis of variance (ANOVA), and means were separated by Duncan's multiple range test.

Results and discussion

The bacterial canker disease caused by Pss and Psm was observed in several locations in Malatya province in 2006–2007. The disease was widely distributed in this province. Previously, bacterial canker caused by Pss was determined in 20% of apricot trees in Malatya (Kavak and Citir 1995). In our observations, the pathogen was mostly found on blossoms, shoots and trunks rather than leaves and fruits. The isolations from diseased tissue resulted in 90 bacterial isolates in total. According to MIS results, 53 isolates of 90 isolates were determined to be Pss (42 of 53) and Psm (11 of 53) (Table 1). All Pss and Psm isolates were rod-shaped, Gram-negative and produced florescent pigment. They showed positive responses in levan formation and the catalase test, and negative responses in amylase and oxidase tests. In addition, all isolates showed negative responses in nitrate production (except K71, Psm) and arginine dihydrolase test (except K21 and K34, Pss) (Table 1). Our results agree with previous studies (Luisetti et al. 1972; Young 1988; Schaad 1994; Ogawa et al. 1995; Ala 2004). In HR tests of these isolates, all were identified to be HR positive. In the pathogenicity test, all isolates of Pss and Psm produced necrotic lesions on leaves and K24, K25, K43, K47 and K51 (Pss) were determined to be the most virulent in apricot cv. Hacihaliloğlu. Crosse and Garrett (1966), Jones (1971) and Kennelly et al. (2007) reported previously that strains of Pss are more virulent on apricot and other stone fruit hosts except for sour cherry, although strains of both pathovars can cause bacterial canker symptoms and coexist on stone fruit trees. Latorre and Jones (1979) stated also that *P. syringae* pv. *morsprunorum* appears to be the more important pathogen in sour cherry.

Colour changes were observed in apricot cultivar leaves, especially susceptible cultivars such as Tokaloğlu (Erzincan), approximately 4 days after spraying (Fig. 1) and characteristic symptoms of Pss appeared on inoculated leaves 10 days after inoculation. Leaf infections appeared as water-soaked spots about 1–3 mm in diam. Later, the spots become brown, dry, and brittle and eventually fall out, giving the leaves a shot-hole or tattered appearance (Agrios 2005). Average Disease Severity Rating (ADSR) was determined to be between 1.73 (Hasanbey) and 3.40 (Şam). The lowest ADSR was found in cv. Hasanbey

followed by Şekerpare (1.83), Soğanci and Çöloğlu (1.87), and these cultivars were statistically different from other cultivars, except Alyanak. In contrast, the highest ADSR was determined in cv. Şam and this cultivar was found to be statistically higher than other cultivars, except Tokaloğlu (Erzincan) (3.30). Consequently, Şam, Tokaloğlu (Erzincan) and Erken Ağerik apricot cultivars were susceptible to Pss. Hasanbey, Şekerpare, Çöloğlu and Soğanci cultivars had a few visible necrotic lesions in each experiment and were thus considered as resistant to Pss (Fig. 1, Table 2). The remaining cultivars were assigned to a moderately susceptible group based on ADSR (Table 2). Of the 15 genotypes displaying Pss symptoms, Hasanbey, Şekerpare, Çöloğlu and Soğanci apricot cultivars were grouped as resistant, and had an ADSR of 1.73–1.87. They were significantly ($P \leq 0.01$) lower than ADSRs (2.40–3.40) of moderately susceptible (except Alyanak) and susceptible cultivars including Çataloğlu, Erken Ağerik, Hacihaliloğlu, İsmailağa, Kabaaşi, Karacabey, Sakit 2, Şam, Tokaloğlu (Erzincan) and Turfanda Eski Malatya (Table 2).

This is the first study to demonstrate the response of Turkish apricot cultivars to bacterial canker. In previous studies, only a few apricot cultivars such as Lasgerdi, Shahroodi, Orange Red, Ladakhi, Nari, Kaisi, Farming Dale, Shakarapara, Harcot, Harogem, Hargrand and Harlayne were reported as resistant to Pss (Layne 1978, 1979, 1981a, b; Gupta and Kashyap 1991; Jindal and Rana 1992; Jafarpour 1993; Scortichini et al. 1999; Singh et al. 2005). None of those apricot cultivars are grown in Turkey. Therefore, the Turkish apricot cultivars tested for the first time in this study resulted in cvs, Hasanbey, Çöloğlu, Soğanci and Şekerpare showing a new source of resistance against Pss. Two (Hasanbey and Şekerpare) of four cultivars determined resistant to Pss were evaluated as table consuming as fresh and the other two cultivars (Çöloğlu and Soğanci) were evaluated as dried. In general, pathogen populations previously develop on apparently healthy blossoms. Blossom blast symptoms can occur during periods of cool, wet weather or after frost events (Ram and Bhardwaj 2004; Agrios 2005). Nevertheless, spot symptoms are seen in especially susceptible cultivar leaves and fruits. These leaf symptoms are surrounded by chlorotic rings early in development, and the spots expand and eventually drop out of leaves, causing the shot-hole symptoms (Kennelly et al. 2007). In this respect, cultivars showing serious disease symptoms in leaves can develop serious symptoms in blossoms, shoots and trunks. Similarly, in cultivars developing lower leaf spot symptoms, pathogen efficiency can be lower in blossoms, shoots and trunk. In addition, Kennelly et al. (2007) reported that sources of resistance to Pss are not readily available, and studies concerning naturally occurring resistance have been sporadic. Therefore, these cultivars are important for both growing and breeding for resistance to Pss in apricot production. However, further studies need to be performed to determine the inheritance of resistance to Pss. It would be useful to 'tag' the resistant genotypes for resistance to Pss strains with molecular markers and to use these markers to pyramid resistant genes into susceptible apricot cultivars.

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