



# Disease and frost damage of woody plants caused by *Pseudomonas syringae*: Seeing the forest for the trees

Jay Ram Lamichhane, Leonardo Varvaro, Luciana Parisi, Jean Marc Audergon, Cindy E. Morris

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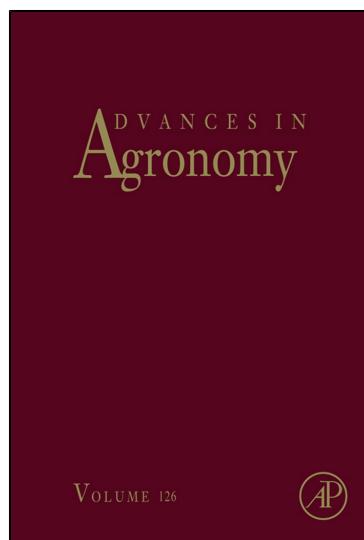
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# Disease and Frost Damage of Woody Plants Caused by *Pseudomonas syringae*: Seeing the Forest for the Trees

Jay Ram Lamichhane<sup>\*†</sup>, Leonardo Varvaro<sup>\*</sup>, Luciana Parisi<sup>†</sup>,  
Jean-Marc Audergon<sup>‡</sup> and Cindy E. Morris<sup>†,1</sup>

<sup>\*</sup>Department of Science and Technology for Agriculture, Forestry, Nature and Energy (DAFNE),  
Tuscia University, Viterbo, Italy

<sup>†</sup>INRA, Pathologie Végétale, Montfavet cedex, France

<sup>‡</sup>INRA, GAFL, Montfavet cedex, France

<sup>1</sup>Corresponding author; e-mail address: Cindy.Morris@avignon.inra.fr

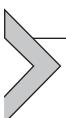
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## Abstract

*Pseudomonas syringae* is a phytopathogenic bacterium that causes diseases of monocots, herbaceous dicots, and woody dicots, worldwide. On woody plants, reports of disease due to *P. syringae* have markedly increased in the last years and the diseases have been recognized as a major threat to the primary products of agroforestry practices. Detection

in Italy of a new highly aggressive population of *P. syringae* in 2008 on kiwifruit, which caused severe epidemics in the following years throughout the kiwifruit-growing areas of Asia, Europe, Oceania, and South America, rendered the entire kiwifruit industry vulnerable to the disease. Similarly, occurrence of an aggressive population of *P. syringae* on horse chestnut in 2002 in the Netherlands has rapidly established itself as a major threat to horse chestnut throughout Northwest Europe. To better understand the origin of such disease epidemics, a thorough knowledge of the pathogen is needed in *sensu lato*. Here, we report the most important features of the pathogen and its hosts in an attempt to clarify some key aspects. In particular, the diseases and the economic losses they cause, disease epidemiology, pathogen diversity, and the possible means of disease control have been discussed throughout the manuscript. In addition to the ability to cause the disease, the damage caused to woody plants through the ice nucleation activity of this bacterium is discussed.



## 1. INTRODUCTION

The economic importance of a given plant species is an attribute of the quantity of services and products that it provides for humans. Edible components, firewood, and timber are usually considered to be the most important primary products obtained from plants. In addition, secondary products such as biofuels and plant-based medicines have a nonnegligible value for humans. Perennial plants are composed of the hard fibrous material consisting of secondary xylem tissue known as wood (Hickey and King, 2001; Plomion et al., 2001). Each year, most woody plants form new layers of woody tissue that are deposited on the inner side of the cambium, located immediately under the bark. However, in some monocotyledons, such as palms and dracaenas, the wood is formed in bundles scattered through the interior of the trunk (Chase, 2004). A few herbaceous perennial species such as *Uraria picta*, some other species belonging to the *Polygonaceae* family and herbaceous perennials from alpine and dry environments, that have a thickened and short hard perennial stem known as caudex (Welsh, 1981), develop wood-like stems. However, these species are not truly woody and they only develop hard densely packed stem tissue. Woody perennial plants, because of their long life cycle, ensure a regular income for growers and represent an important economic source for a large number of individuals. In addition to their primary products, the environmental services of trees are particularly significant to human life. Examples are prevention of soil erosion and land degradation (Wilkinson, 1999), carbon sequestration (Nowak and Crane, 2002), reduction of building energy consumption (Akbari, 2002), and of air pollution (Nowak et al., 2006). Among the numerous diseases of woody plants, those caused by *Pseudomonas syringae* have become markedly important in recent years. Since only the beginning of this century, 55 reports of disease outbreaks in 25 countries have been associated with *P. syringae* in 25 different woody hosts (Table 4.1). These

**Table 4.1** Disease Outbreaks on Woody Plants Caused by *Pseudomonas syringae* Reported Worldwide Since 2000

Continent	Country	Occurrence Year	Host	Incidence		Reference
				(%)	Pathogen	
Asia-Pacific	China	2006	Pear	NR	Pss	Xu et al. (2008)
		2008	Kiwifruit	NR	Psa	CABI/EPPO (2008)
	Iran	2001–2003	Almond	NR	Pss	Samavatian (2006)
		2003–2004	Chinaberry	NR	Pme	Taghavi and Hasani (2010)
		2005	Apricot, peach	NR	Pss	Karimi-Kurdistani and Harighi (2008)
		2008	Olive	NR	Pss	Ashporour et al. (2008)
		2007–2008	Jasmine	NR	Psav	Taghavi and Hasani (2012)
		2012	Hazelnut	NR	Ps	Mahdavian and Hasanzadeh (2012)
	Korea	1997–2000	Kiwifruit	NR	Psa	Koh et al. (2012)
	Nepal	2006–2007	Olive	NR	Pssav	Balestra et al. (2009a)
	Syria	2007	Olive	70	Pssav	Alabdalla et al. (2009)
	Turkey	1999–2001	Apricot	80	Pss	Kotan and Sahin (2002)
		2004	Orange, mandarin	100	Pss	Mirik et al. (2005)
		2008	Peach	10	Ps	Ozakatan et al. (2008)
		2009–2010	Kiwifruit	3	Psa	Bastas and Karakaya (2012)
		2000	Grapevine	NR	Ps	Hall et al. (2002)
	Australia	2001	Olive	NR	Ps	Hall et al. (2003)
		2003	Olive	NR	Pssav	Hall et al. (2004)
		2006–2007	Grapevine	60	Pss	Whitelaw-Weckert et al. (2011)
		2007	Mango	NR	Pss	Golzar and Cother (2008)
		2011	Kiwifruit	NR	Psa	EPPO (2011a)
	New Zealand	2010	Kiwifruit	NR	Psa	Everett et al. (2011)

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**Table 4.1** Disease Outbreaks on Woody Plants Caused by *Pseudomonas syringae* Reported Worldwide Since 2000—cont'd

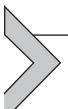
Continent	Country	Occurrence Year	Host	Incidence		Reference
				(%)	Pathogen	
Europe	Belgium	2007	Horse chestnut	NR	Psaes	Bultreys et al. (2008)
	Bulgaria	2004–2005	Firethorn	NR	Pss	Bobev et al. (2008)
		2004–2006	Apricot	85	Ps	Ivanova (2009)
	Czech Republic	2008–2010	Horse chestnut	NR	Psaes	Mertelik et al. (2013)
	France	2007	Horse chestnut	NR	Ps	Bardoux and Rousseau (2007)
		2008	Brazilian jasmine	NR	Psav	Eltlbany et al. (2012)
		2010	Kiwifruit	NR	Psa	Vanneste et al. (2011b)
	Germany	2006	Hazelnut	NR	Psc	Poschenrieder et al. (2006)
		2007	Horse chestnut	NR	Psaes	Schmidt et al. (2008)
	Ireland	2010	Horse chestnut	NR	Psaes	EPPO (2011c)
Italy	Italy	2003	Onondaga	NR	Psv	Garibaldi et al. (2005)
		2004	White bird of paradise	40	Psl	Polizzi et al. (2005)
		2005	Hazelnut	NR	Psc	Cirvilleri et al. (2007)
		2005	Nectarine	30–35	Pss	Scorticini and Janse (2008)
		2006	Apricot	30	Pss	Scorticini (2006)
		2007	Kiwifruit	30	Pv	Balestra et al. (2008)
		2007–2008	Kiwifruit	10–70	Psa	Balestra et al. (2009b)
		2013	Sweet olive	NR	Psav	Cinelli et al. (2013a)
	Lithuania	2007	Cherry and plum	NR	Pss, Psm1, Psm2	Vasinauskienė et al. (2008)
	Norway	2010	Horse chestnut	NR	Psaes	Talgø et al. (2012)
Portugal	2007	Kiwifruit	30	Ps	Balestra et al. (2009c)	
	2010	Kiwifruit	30	Psa	Balestra et al. (2010)	

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Spain	2011	Kiwifruit	80	Psa	Abelleira et al. (2011)	
	1999–2000	Kiwifruit	40	Ps	González and Ávila (2005)	
Switzerland	2011	Kiwifruit	NR	Psa	EPPO (2011d)	
The Netherlands	2002–2003	Horse chestnut	NR	Psaes	Dijkshoorn-Dekker (2005)	
	2009–2010	Plum	50	Pss, Psm	Wenneker et al. (2012)	
The UK	2000–2001	Wild cherry	50–100	Pss, Psm1, Psm2	Vicente et al. (2004)	
	2003	Horse chestnut	70	Psaes	Webber et al. (2008)	
	2006	Onondaga	NR	Psv	Stead et al. (2006)	
North America	The USA	2002	Sweet cherry	NR	Pss, Psm1	Renick et al. (2008)
South America	Brazil	2006	Coffee	1–2	Pstab	Destefano et al. (2010)
	Chile	2011	Kiwifruit	NR	Psa	EPPO (2011b)

Note: NR, not reported; Ps, *Pseudomonas syringae*; Pss, *P. syringae* pv. *syringae*; Psav, *P. savastanoi*; Pssav, *P. savastanoi* pv. *savastanoi*; Psm1 and Psm2, *P. syringae* pv. *morsprunorum* race 1 and 2; Psa, *P. syringae* pv. *actinidiae*; Psaes, *P. syringae* pv. *asculi*; Psc, *P. syringae* pv. *coryli*; Pme, *P. meliae*; Pstab, *P. syringae* pv. *tabaci*; Psv, *P. syringae* pv. *viburni*.

reports are also snapshots of recurring chronic problems, such as olive knot disease in Italy or apricot canker in France, that have been important over the past century. Hence, it can be assumed that this rate portends additional future outbreaks. To mitigate the consequences of such a disease emergence, it is important to understand the underlying causes. In this light, we have reviewed the literature in order to highlight the salient features of *P. syringae* as a pathogen of woody plant species and to underscore what remains unknown about the diseases it causes.



## 2. ECONOMIC IMPORTANCE

Reliable estimates of economic losses caused by plant pathogens are not only a prerequisite for optimal crop management at the farm level but also for basic decisions on broader issues such as research priorities and pesticide regulations. Although several methods have been proposed to evaluate economic losses caused by plant pathogens (Heaton et al., 1981; James, 1974), only few data are available in the literature. Insufficient information on the biological effects of the pathogens on their host is the major obstacle preventing more accurate crop-loss assessment in monetary terms. Crop-loss assessments have been made for major annual staple and cash crops (Oerke, 2005; Oerke et al., 1994). However, data inadequacies are particularly acute in the case of perennial crops, where intertemporal effects operate. For perennial plants, their health in any given season is likely to influence their health in future seasons (Heaton et al., 1981).

Bacterial cankers of perennial trees caused by *P. syringae* provoke serious economic losses around the world. Cankers are characterized by systemic wilting and dieback of twigs, branches or the entire tree resulting from systemic spread of the pathogen in the vessels. On hazelnut, bacterial canker was reported to be the most important factor limiting the cultivation and further expansion of hazelnut production in Greece (Psallidas, 1993) since its first outbreak (Psallidas and Panagopoulos, 1979). In Italy, *P. syringae* destroyed thousands of hectares of hazelnut, with an annual loss of approximately US\$ 1.5 million (Scorticini, 2002; Scorticini and Tropiano, 1994). Similarly, the recent bacterial canker epidemic of kiwifruit has decimated thousands of hectares around the world. In New Zealand alone, yield losses caused by the disease in 2012 were estimated to be 21% (90,820 ton less than in 2011) with economic losses of US\$ 76 million (<http://www.kvh.org.nz/newsroom>). However, the most dramatic consequences of the disease can be seen for the long term. A risk analysis, performed in 2012, estimated that over the next five years bacterial

canker of kiwifruit might cost between NZ\$ 310 million to 410 million to the New Zealand kiwifruit industry (Vanneste, 2012). It is estimated that over a 15-year period the cost might jump to NZ\$ 885 million (Greer and Saunders, 2012). In the Bay of Plenty area of New Zealand alone, 360 to 470 fulltime jobs might be lost between 2012 and 2016 due to the bacterial canker epidemic on kiwifruit (Greer and Saunders, 2012). Although no estimation has been made for Italy, the situation in that country might be even worse, given the intensity and diffusion of the bacterial canker epidemics (Scorticini et al., 2012; Vanneste, 2012). Due to the extent and serious impact of bacterial canker, it is thought that kiwifruit plantings in Italy will be significantly reduced over the next three to five years (<http://www.kvh.org.nz/newsroom>).

Bacterial canker also affects tree species in the genus *Prunus* (stone fruits and almond) (English et al., 1980; Kennelly et al., 2007; Vicente et al., 2004). In South Africa, annual damage caused by bacterial canker of stone fruit crops is estimated to be over US\$ 10 million (Hattingh et al., 1989). Damage caused by *P. syringae* is not limited to field production but also occurs in nurseries where sudden wilting is a major problem. For example, annual losses in woody plant nurseries in Oregon are estimated at US\$ 8 million (Scheck et al., 1996).

In Germany, bacterial canker causes annual tree mortality rates as high as 30% on plum (Hinrichs-Berger, 2004). In Oregon (the USA), bacterial canker is responsible for 17% annual tree deaths for sweet cherry (Spotts et al., 1990). The incidence of bacterial canker is reported to be 50–100% on wild cherry in the UK (Vicente et al., 2004), 50% on plum in the Netherlands (Wenneker et al., 2012), and 80% on apricot in Turkey (Kotan and Sahin, 2002). In addition to stone fruits, *P. syringae* diseases are economically important also on pome fruits (Mansvelt and Hattingh, 1986). The disease results in economic losses in pear production around the world (English et al., 1980; Fahy and Lloyd, 1983; Manceau et al., 1990; Montesinos and Vilardell, 1991). Another very recent epidemic caused by *P. syringae* is bleeding canker of horse chestnut. The disease is widespread in many European countries (Bultreys et al., 2008; Schmidt et al., 2008; Webber et al., 2008). However, no data are available on the potential losses.



### 3. TYPES OF DISEASES OF WOODY PLANTS CAUSED BY *P. SYRINGAE* AND THEIR IMPORTANCE

A broad range of crops is susceptible to diseases caused by the group of Gram-negative bacteria referred to as *P. syringae* (Bull et al., 2010). Concerning perennial plants, the literature suggests that a large number

of woody tree species are attacked by this pathogen. An exhaustive list of diseases of perennial plants caused by *P. syringae* is reported in Table 4.2. Deciduous fruit and nut trees (Kennelly et al., 2007; Mansvelt and Hattingh, 1986), ornamental trees and shrubs (Garibaldi et al., 2005; Temsah et al., 2007a,b), deciduous forest tree species (Ark, 1939; Green et al., 2009; Menard et al., 2003), and evergreen fruit trees (Young, 2004) are among the known hosts of *P. syringae*. Overall, phytopathogenic *P. syringae* causes two types of diseases on woody plants (Agrios, 2005), those whose symptoms are confined to parenchymatic tissue and those whose symptoms are manifested in vascular tissue.

### 3.1 Parenchymatic or Localized Diseases

One family of diseases of woody plants caused by *P. syringae* is characterized by the presence of localized symptoms affecting parenchymatic tissues. In this case, the pathogen does not move throughout the host vascular system. Typical symptoms are leaf spots (Destefano et al., 2010; Goto, 1983a, 1983b; Roberts, 1985a), fruit spots (Bedford et al., 1988; Burr and Hurwitz, 1981), apical necrosis (Cazorla et al., 1998; Golzar and Cother, 2008), blossom and bud blasts (Kennelly et al., 2007; Manceau et al., 1990; Mansvelt and Hattingh, 1986; Young, 1987), bud rot and necroses (Cazorla et al., 1998; Conn et al., 1993), shoot tip dieback (McKeen, 1955; Polizzi et al., 2005; Ramos and Kamidi, 1981; Tomihama et al., 2009), and gall or outgrowth on stems, leaves, and trunks (Kamiunten et al., 2000; Ogimi, 1977; Ogimi et al., 1990, 1992; Temsah et al., 2007a,b). The resulting localized infections rarely kill the plants and losses are due mainly to damage to flowers and photosynthetic parts (leaves).

### 3.2 Vascular or Systemic Diseases

Vascular diseases are characterized by the systemic movement of the disease throughout the plant and in particular the vessels (phloem and xylem). The vascular diseases caused by *P. syringae* are generally devastating. Sudden wilting and dieback (Scortichini, 2002), presence of extended cankers on stems, branches, and main trunks (Jones, 1971; Latorre and Jones, 1979a; Little et al., 1998; Sakamoto, 1999; Young, 1988a), and production of exudates and gummosis (Kennelly et al., 2007; Green et al., 2009; Scortichini et al., 2012) are common symptoms.

To attack stems, branches, and trunks, *P. syringae* infects the woody tissue of the plant. Of all the plant tissues, woody tissue has the highest content of lignin, a complex phenolic polymer common to vascular plants. Plant lignin

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**Table 4.2** Pathogens from the *Pseudomonas syringae* Complex Reported to be Virulent on Woody Plants

Organism	Phylogroup <sup>a</sup>	Disease	Host(s)	Plant Type	References <sup>b</sup>
<b><i>P. syringae</i> pv.</b>					
<i>actinidiae</i> <sup>f,h</sup>	1	Bacterial canker	Kiwifruit	Tree	Scorticini et al. (2012)
<i>avellanae</i> <sup>f,h</sup>	1	Bacterial canker	European hazelnut	Tree	Scorticini (2002)
<i>avir</i> <sup>f,h</sup>	1	Bacterial canker	Wild cherry	Tree	Menard et al. (2003)
<i>berberidis</i> <sup>g</sup>	1	Bacterial leaf spot	<i>Berberis</i> spp.	Woody shrub	Roberts (1985b)
<i>lachrymans</i> <sup>g</sup>	1	Bacterial blight	White bird of paradise	Tree	Polizzi et al. (2005)
<i>morsprunorum</i> <sup>f,h</sup>	1	Bacterial canker	Stone fruit	Tree	Crosse (1966)
<i>persicae</i> <sup>f,h</sup>	1	Bacterial dieback	Peach	Tree	Young (1988a)
<i>theae</i> <sup>g</sup>	1	Bacterial shoot blight	Tea	Tree	Tomihama et al. (2009)
<i>viburni</i> <sup>g</sup>	1	Bacterial blight	<i>Viburnum</i> spp.	Woody shrub	Thornberry and Anderson (1931a)
<i>aceris</i> <sup>g</sup>	2	Bacterial leaf spot	Maple	Tree	Ark (1939)
<i>dysoxyli</i> <sup>g</sup>	2	Bacterial leaf diseases	Kohekohe	Tree	Hutchinson (1949)
<i>papulans</i> <sup>g</sup>	2	Blister spot	Apple	Tree	Bedford et al. (1988)

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**Table 4.2** Pathogens from the *Pseudomonas syringae* Complex Reported to be Virulent on Woody Plants—cont'd

Organism	Phylogroup <sup>a</sup>	Disease	Host(s)	Plant Type	References <sup>b</sup>
<i>syringae</i>	2	Blossom blight	Lilac	Tree	Young (1991)
		Apical necrosis <sup>g</sup>	Mango	Tree	Cazorla et al. (1998)
		Influorescence rot	Grapevine	Liana	Whitelaw-Weckert et al. (2011)
		Bacterial canker <sup>f,h</sup>	Stone fruit	Tree	Jones (1971), Latorre and Jones (1979a)
		Bacterial canker <sup>f</sup>	Olive	Tree	Hall et al. (2003)
		Blister bark <sup>f,h</sup>	Apple	Tree	Mansvelt and Hattingh (1986)
		Blossom blight <sup>g</sup>	Pear	Tree	Moragrega et al. (2003)
		Bacterial blight and canker <sup>f</sup>	Hazelnut	Tree	Scorticchini et al. (2002)
		Bacterial blight <sup>g</sup>	Kiwifruit	Tree	Young (1988b)
		Bacterial canker <sup>f,h</sup>	Almond	Tree	Little et al. (1998)
		Citrus blast <sup>g</sup>	Orange and mandarin	Tree	Mirik et al. (2005)
		Fruit scab <sup>g</sup>	Nectarine	Tree	Scorticchini and Janse (2008)
		Bacterial canker <sup>f</sup>	Amurmaackia	Tree	Sakamoto (1999)
		Bacterial canker <sup>f,h</sup>	Blueberry	Woody shrub	Canfield et al. (1986)
		Leaf blight <sup>g</sup>	Cornelian cherry	Tree	Mmbaga and Nnodu (2006)
		Bacterial dieback	Willow	Tree	Ramstedt et al. (1994)
		Dieback and canker	Poplar	Tree	Whitebread (1967)
		Sucker and twig dieback	Black alder	Tree	Scorticchini (1997)

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<i>P. amygdali</i> <sup>c,f,h</sup>	3	Hyperplastic canker	Almond	Tree	Psallidas and Panagopoulos (1975)
<i>P. ficusrectae</i> <sup>g</sup>	3	Bacterial leaf spot	Japanese fig	Tree	Goto (1983b)
<i>P. meliae</i> <sup>f</sup>	3	Bacterial gall	Chinaberry	Tree	Ogimi (1977)
<i>P. tremae</i> <sup>f</sup>	3	Bacterial gall	Pigeon wood	Tree	Ogimi et al. (1988a)
<b><i>P. savastanoi</i> pv.</b>					
<i>savastanoi</i> <sup>f</sup>	3	Olive knot	European olive	Tree	Young (2004)
		Knot	Privet	Woody shrub	Janse (1982)
		Knot	Forsythia	Woody shrub	Janse (1982)
		Knot	Jasmine	Woody shrub	Janse (1982)
<i>nerii</i> <sup>f</sup>	3	Knot	Oleander	Woody shrub	Janse (1982)
<i>fraxini</i> <sup>f</sup>	3	Knot	Ash	Tree	Janse (1982)
<i>retacarpae</i> <sup>f</sup>	3	Knot	Spanish broom	Woody shrub	Garcia de los Rios (1999)
ND <sup>f</sup>		Knot	Buckthorn	Woody shrub	Temsah et al. (2007a,b)
ND <sup>f</sup>		Knot	Myrtle	Woody shrub	Temsah et al. (2007a,b)
ND <sup>f</sup>		Knot	Sweet olive	Woody shrub	Cinelli et al. (2013a)
ND <sup>f</sup>		Knot	Brazilian jasmine	Woody shrub	Eltlbany et al. (2012)

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**Table 4.2** Pathogens from the *Pseudomonas syringae* Complex Reported to be Virulent on Woody Plants—cont'd

Organism	Phylogroup <sup>a</sup>	Disease	Host(s)	Plant Type	References <sup>b</sup>
<b><i>P. syringae</i> pv.</b>					
<i>aesculi</i> <sup>f,h</sup>	3	Bleeding canker	Horse chestnut	Tree	<a href="#">Green et al. (2010)</a>
<i>castaneae</i> <sup>d,f</sup>	3	Bacterial canker	Chestnut	Tree	<a href="#">Takanashi and Shimizu (1989)</a>
<i>cerasicola</i> <sup>f</sup>	3	Bacterial gall	Cherry	Tree	<a href="#">Kamiunten et al. (2000)</a>
<i>ciccaronei</i> <sup>f</sup>	3	Bacterial spot	Carob	Tree	<a href="#">Ercolani and Caldarella (1972)</a>
<i>daphniphylli</i> <sup>f</sup>	3	Bacterial gall	Hime-yuzuriha	Tree	<a href="#">Ogimi et al. (1990)</a>
<i>dendropanacis</i> <sup>f</sup>	3	Bacterial gall disease	Kakuremino	Woody shrub	<a href="#">Ogimi et al. (1988b)</a>
<i>eriobotryae</i> <sup>f</sup>	3	Bacterial stem canker	Loquat	Woody shrub	<a href="#">McRae and Hale (1986)</a>
<i>moris</i> <sup>g</sup>	3	Bacterial blight	Mulberry	Tree	<a href="#">Takahashi (1980)</a>
<i>myricae</i> <sup>f</sup>	3	Bacterial knot disease	Yamamoto	Tree	<a href="#">Ogimi and Higuchi (1981)</a>
<i>photiniae</i> <sup>g</sup>	3	Leaf spots and shoot blight	Red-leaf photinia	Woody shrub	<a href="#">Goto (1983a)</a>
<i>rhiphiolepidis</i> <sup>f</sup>	3	Bacterial gall	Sharinbai	Tree	<a href="#">Ogimi et al. (1992)</a>
<i>tabaci</i> <sup>g</sup>	3	Bacterial leaf spot	Coffee	Woody shrub	<a href="#">Destefano et al. (2010)</a>
<i>ulmi</i> <sup>g</sup>	3	Bacterial spot/shoot blight	Elm	Tree	<a href="#">Sutic and Tesic (1958)</a>

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<i>garcae</i> <sup>g</sup>	4	Bacterial blight	Coffee	Woody shrub	Ramos and Kamidi (1981)
<i>philadelphii</i> <sup>g</sup>	5	Bacterial blight	Sweet mock-orange	Woody shrub	Roberts (1985a)
<i>ribicola</i> <sup>g</sup>	7	Leaf spot and defoliation	Golden currant	Woody shrub	Bohn and Maloit (1946)
<i>P. viridiflava</i> <sup>g</sup>	7	Blossom blight Tree apoplaxy	Kiwifruit Peach	Tree Tree	Young (1988b) Scorticini and Morone (1997)
<i>coryli</i> <sup>e,f</sup>	ND	Twig dieback and canker	European hazelnut	Tree	Scorticini et al. (2005)

ND: not determined.

<sup>a</sup>Phylogroups according to the study of [Parkinson et al. \(2011\)](#).

<sup>b</sup>Cited references are articles that provide general overviews of the disease and/or description of the pathogen.

<sup>c</sup>This pathogen is described as a forgotten pathogen in a recent work by [Janse \(2010\)](#).

<sup>d</sup>This pathogen is described as a forgotten pathogen in a recent work by [Janse \(2010\)](#).

<sup>e</sup>This pathogen is identified as *P. s. pv. syringae* belonging to group II as described by [O'Brien et al. \(2012\)](#).

<sup>f</sup>The pathogen attacks also woody tissues.

<sup>g</sup>The pathogen attacks only leaves and succulent parts of plant.

<sup>h</sup>The pathogen is vascular and highly aggressive.

can be broadly divided into three classes, softwood (gymnosperm), hardwood (angiosperm), and grass (graminaceous) lignin (Pearl, 1967). Lignin has a fundamental role in water transport, mechanical support, and biodefense (Robinson, 1990). Within a plant, lignin content varies greatly in different tissues. For example, lignin content is very low in young shoots and high in woody tissues (Novaes et al., 2010). The lignin content of woody perennial plants can vary from 15% to 40% (Sarkanen and Ludwig, 1971) whereas in annual plants it represents only from 4% to 10% of the oven-dry weight (Sullivan, 1955). How *P. syringae* can overcome such a robust barrier and cause a collapse of woody tissues is not known.

Interestingly, *P. syringae*-induced vascular diseases have been reported exclusively on deciduous trees (Green et al., 2009; Hattingh et al., 1989; Kennelly et al., 2007) while only parenchymatic diseases are caused on evergreen woody plants (Ramos and Kamidi, 1981; Young, 2004) (Table 4.2). Many aspects of the annual growth cycle differ significantly between deciduous and evergreen plants. Adaptation to a cold or dry season and to low nutrient levels has led to the differentiation of deciduous and evergreen woody plants (Monk, 1966). The annual phenology of deciduous trees is the process that begins with flower budbreak followed by leaf budbreak and sprouting in the spring and culminates in leaf fall in autumn followed by winter dormancy (Arora et al., 2003). These characteristics differentiate a deciduous tree from evergreens where such phenomena do not occur in synchrony for the whole tree. Accordingly, the risk of *P. syringae* infection might be strikingly higher on deciduous trees given the synchronized abundance of tender tissues and natural openings into the vascular system. Succulent swelling buds and shoots are fragile and highly sensitive to frost damage, thereby predisposing deciduous trees to frost damage induced by *P. syringae* (Gross et al., 1984; Nejad et al., 2004; Ramstedt et al., 1994). On the other hand, complete leaf fall that creates an enormous availability of leaf scars for a long period of time perfectly coincides with climatic conditions that are ideal for the survival and multiplication of *P. syringae* (Agrios, 2005; Scorticini, 2002). The presence of dormant buds, during the winter, represents an ideal overwintering site for *P. syringae* (Crosse, 1956; Roos and Hattingh, 1986; Sundin et al., 1988). On the contrary, evergreen species do not have autumn leaf shedding. However, leaf shed occurs very gradually over the years in evergreen plants (Aerts, 1995). Leaf spots caused by *P. syringae* are also almost exclusively reported on deciduous trees (Hattingh et al., 1989; Kennelly et al., 2007). It is not known why a ubiquitous epiphyte such as *P. syringae* (Hirano and Upper, 1990) causes foliar diseases only on a

very limited number of evergreen hosts, despite whole-year availability of leaf surfaces. In addition to the higher number of infection sites in deciduous trees, several studies have provided evidence of differences in protein content of bark between deciduous trees, including deciduous fruit trees (Kang and Titus, 1987; Kuroda et al., 1990; Lang and Tao, 1990; Mattheis and Ketchie, 1990), and evergreen woody plants (Craker et al., 1969; Hummel et al., 1990). For example, higher levels of polypeptide accumulation in bark tissues were observed in the deciduous peach compared with the evergreen one (Arora et al., 1992). Probably such polypeptides are involved in the process of vascularization of *P. syringae*. But this will need to be established by in-depth future studies. Species with both deciduous and evergreen traits, such as *Viburnum* spp., might provide some new insights in this regard.



## 4. EPIDEMIOLOGY

As for all plant diseases, biotic, abiotic and/or edaphic factors play an important role in epidemiology. Throughout the cultivation areas, these factors have been reported to weaken plant health and predispose them to pathogen attacks. In particular, soil texture, low soil pH, soil depth, tree nutrition, tree age, nematode parasitism, and environmental factors such as rain can influence *P. syringae* disease development (English et al., 1961, 1980; Scorticini, 2002, 2010; Scorticini et al., 2012; Vigouroux and Busi, 1994). In addition, cultural practices such as rootstock selection, height of grafting and early fall pruning have been reported to affect *P. syringae* disease susceptibility of apricot and peach (Dunquesne and Gall, 1975; Fratantuono et al., 1998; Lownsbury et al., 1977; Prunier et al., 1999; Vigouroux et al., 1987, 1997). Likewise, the correlation among tree water content, effect of exposure to freezing temperature, and necrosis caused by *P. syringae* has been reported for apricot (Klement et al., 1974; Vigouroux, 1989), cherry (Sobiczewski and Jones, 1992), and peach (Cao et al., 2013; Vigouroux, 1999; Weaver, 1978). There are few generalities that can be made about the impact of abiotic factors and the production practices to which they are linked. On the other hand, biotic factors concerning both the plant and the pathogen have been received in the most intense study and offer the opportunity to identify key factors critical in disease development as illustrated below.

### 4.1 Sources of Inoculum

Overall, epiphytic populations, latent infection, overwintering sites on the infected hosts, the presence of orchard groundcovers, weeds, and detached

plant parts (pollen and leaf litter) represent the inoculum reservoir of *P. syringae*. All these inoculum sources appear to be important in the epidemiology of diseases caused by *P. syringae*. Epiphytic populations of this pathogen on asymptomatic plants are the immediate source of inoculum for disease. Differences in epiphytic populations of *P. syringae* were observed on leaves of apple trees grown in two different orchards: one with a history of disease, the other where the disease had not been observed previously (Bedford et al., 1988). Larger pathogen population sizes were found in association with greater disease incidence in the orchard with a prior history of the disease. Similarly, epiphytic populations of *P. syringae* as the source of inoculum for disease have been demonstrated on olive (Young, 2004), maple and pear trees (Malwick and Moore, 1988), and stone fruits (Roos and Hattingh, 1986). Monitoring of *P. syringae* epiphytic populations associated with nursery trees in Oregon showed that the population increased rapidly and peaked during the first 2–3 weeks after budbreak (Baca et al., 1987; Moore and Malwick, 1987) increasing markedly the risk of bud infection.

Establishment of *P. syringae* populations inside symptomless tissues could represent a very important source of primary inoculum. For this reason, vegetative propagation of the apparently healthy but latently infected mother plants could be an important source of inoculum. Widespread disease dissemination with propagation material such as dormant cuttings or budwoods, and *in vitro*-propagated shoots can be therefore relevant. On hazelnut, a large-scale dissemination of *P. syringae* is thought to have occurred through distribution of latently infected suckers (Scorticini, 2002). In addition, infected nursery stock of olive has been reported as the main cause of *P. syringae* introduction into new regions (Young, 2004). The isolation of pathogenic *P. syringae* from the vascular tissues of symptomless cherry (Cameron, 1970) and pear trees (Whitesides and Spotts, 1991) clearly explains the latent presence of the pathogen also in deciduous woody hosts. Indeed, infected nursery materials have been reported as the main inoculum reservoir of *P. syringae* in *Prunus* spp. (Dowler and Petersen, 1967; Lyskanowska, 1976; Mansvelt and Hattingh, 1988).

Woody plants provide the unique overwintering sites compared to annual plants. The role of dormant buds in deciduous trees as an overwintering site for *P. syringae* has been demonstrated in South Africa (Roos and Hattingh, 1986a), in the United Kingdom (Crosse, 1956), and in the USA (Sundin et al., 1988). On apple, a consistent population of *P. syringae* has been recovered from the innermost bud tissues and the population was larger than that found in the external tissues (Bedford et al., 1988; Burr and Katz,

1984). Dormant buds in cherry are colonized via infection of leaf scars during autumn at leaf fall. Climatic conditions during leaf shedding typically include cooler temperatures and wind-driven rains during which *P. syringae* is transported from leaf surfaces to leaf scars where systemic colonization occurs (Crosse, 1956). Once *P. syringae* enters through leaf scars, it moves systematically throughout the plant and colonizes dormant buds where the pathogen overwinters. In the following spring, dormant and apparently healthy but infected buds provide the inoculum for blossom colonization.

The importance of groundcovers and weeds within and outside the orchard represent another potential source of inoculum reservoir of *P. syringae*. In particular, when no leaf surface is available on trees (autumn and winter) the pathogen can shift to the groundcovers where it multiplies thereby ensuring a constant source of inoculum. Work reported from California (Davis and English, 1969) was the first to implicate weeds as hosts for *P. syringae*. Successively, the association of *P. syringae* with stone fruit orchard weeds or grasses have been reported in the USA (Michigan (Latorre and Jones, 1979b), Oregon (Baca and Moore, 1987)), Poland (Lyskanowska, 1976), and South Africa (Roos and Hattingh, 1986b). Large populations of phytopathogenic *P. syringae* have been consistently isolated from perennial rye, red fescue, annual rye and broom grasses growing among ornamental trees in the maple nursery and on perennial rye grass in the pear orchard (Malwick and Moore, 1988).

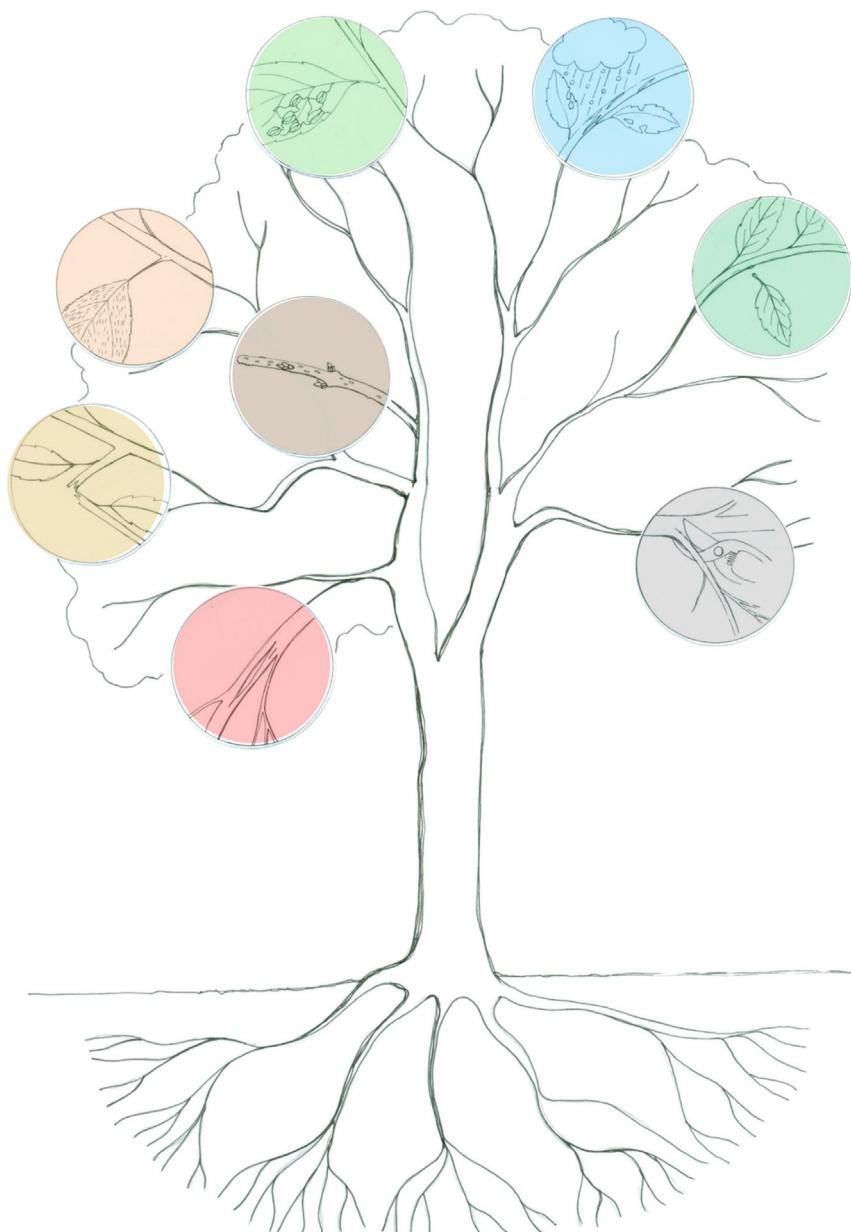
In some cases, pollen can be an important inoculum source of the pathogen. On kiwifruit, *P. syringae* has been found in pollen samples (Vanneste et al., 2011a) and it is believed that pollen has been a source of *P. syringae* introduction in New Zealand. In addition, *P. syringae* is able to survive in detached kiwifruit organs, such as leaf litter and twigs, until 45 days post-leaf fall (<http://www.kvh.org.nz>). Leaf litter across the alpine environment has been reported as an important reservoir of *P. syringae* populations (Monteil et al., 2012). Finally, it is thought that *P. syringae* survives poorly in soil or host debris, mostly throughout the regions with warmer summer (McCarter et al., 1983) although it is possible that the pathogen can survive for a long period in regions with cooler summer temperature, mostly under a constant soil moisture. Indeed, the ability of *P. syringae* to survive in the soil for a longer period has been demonstrated especially when the source of the inoculum was associated with plant debris and in the presence of constant soil moisture (Kritzman and Zutra, 1983). It is also worth noting that *P. syringae* has been isolated from the rhizosphere of crops and weeds (Knoche et al., 1987; Valleu et al., 1944). The fact that *P. syringae* can't be

found in agricultural soil all year round (mostly during the summer) might be because such lands are irrigated generally until the beginning of the summer and no water supply occurs afterward once the crops are harvested. Hence, warmer summer temperatures associated with a drastic reduction in soil moisture result in fatal conditions for the survival of *P. syringae*.

Snow is another important source of *P. syringae* inoculums, which may play an important role in a disease occurrence. An example is bacterial canker of blueberry (Canfield et al., 1986). This species grows naturally or in cultivated conditions across mountain areas where snowfall events are very common. At higher altitudes, spring frosts frequently occur where freezing and thawing are accentuated through the fluctuations in day/night temperatures. Such events cause lesions on plant parts exposing them to a high risk of infection. Snow per se can harbor populations of *P. syringae* as recently demonstrated (Morris et al., 2010). Besides snow, other environmental reservoirs of *P. syringae* including headwaters and epilithic biofilm have been described (Morris et al., 2007, 2010), and these eventually can come into contact with crops as irrigation water.

## 4.2 Ports of Entry for Infection of Plant Tissues

Unlike fungal pathogens, phytopathogenic bacteria are not able to make their own ports of entry (Agrios, 2005). Woody plants present a wide range of entry ports for bacteria including natural openings and wounds caused by natural phenomena and man-made practices. Natural openings (lenticels, hydathodes, stomata, trichomes) that serve as water and gas pores, and the wounds made by biotic (humans, insects, animals) and abiotic factors (hailstones, frost), are the main ports of entry (Agrios, 2005). For *P. syringae* pathogens of woody plants, the literature reports a large diversity of ports of entry as summarized in Figure 4.1. To gain access into plant tissues, the pathogen can use more than one port of entry. In general, foliar pathogens that cause localized symptoms enter mainly through the stomata, although some of them also enter via hydathodes and broken trichomes (Agrios, 2005; Hirano and Upper, 1990). Pathogens that cause woody parenchymatic diseases enter through wounds occurring on woody tissues (Garcia de los Rios, 1999; Kamiunten et al., 2000; Ogimi et al., 1990; Young, 2004). On the other hand, vascular pathogens use a vast number of ports including natural openings and lesions (Crosse, 1966; Kennelly et al., 2007; Hattingh et al., 1989). Lesions on woody tissues that allow a direct access to the vascular system are the most accessible port for vascular pathogens while natural openings are not always exploited by the different *P. syringae* pathogens of woody plants. There seems to be a



**Figure 4.1** Schematic representation of infection ports of phytopathogenic *Pseudomonas syringae*. Clockwise from lower left: tissue damage caused by frost, branch breaking due to wind or mechanical damage, lenticels, broken trichomes, stomata, wounds caused by hailstones, leaf scars and wounds caused by pruning.

relationship between the port of entry and the symptoms caused by vascular *P. syringae* pathogens. Interestingly, leaf symptoms do not occur on infected hazelnut (Psallidas, 1993; Psallidas and Panagopoulos, 1979) and horse chestnut (Green et al., 2009) while they occur on infected kiwifruit (Scorticchini et al., 2012; Young, 2012) and *Prunus* spp. (Bultreys and Kaluzna, 2010; Crosse, 1966; Freigoun and Crosse, 1975; Kennelly et al., 2007). On hazelnut, the only ports of entry used by *P. syringae* are leaf scars and lesions on woody tissues (Psallidas, 1993; Scorticchini, 2002) giving the pathogen direct access to the vascular system. For horse chestnut, in addition to lesions, the role of lenticels as ports of entry has been demonstrated (Green et al., 2009). Indeed, lesions in proximity to lenticels appeared when stems of horse chestnut were spray-inoculated (Green et al., 2009). By contrast, *Prunus* spp. and in particular apricot and cherry, contain an impressive number of lenticels (Guirguis et al., 1995) although no report is available about ingress of *P. syringae* through the lenticels on these hosts. It is worth exploring why an enormous availability of this port does not seem to be exploited by the pathogen. In *Prunus* spp. the role of infection seems to be quite different. For example, on cherry most canker symptoms are located on the branches since the pathogen enters through leaf scars while the main stem is affected on plum where the pathogen seems to gain access through small wounds, although of unknown origin (Crosse and Garrett, 1970). Although leaf scar is one of the main ports of entry for *P. syringae* that cause vascular disease, on cherry it is not always utilized as an avenue of infection at warmer temperatures in particular. In South Africa, it seems that *P. syringae* reaches buds through the systemic movement before the leaf fall (Hattingh et al., 1989). Because *P. syringae* is a psychrophilic bacterium, it is possible that epiphytic populations of the pathogen do not survive in warmer temperatures, thereby favoring the organisms that colonize systemically. In Oregon, where mass destruction of dormant buds is the most conspicuous feature of the disease, attempts to infect cherry leaf scars with *P. syringae* failed (Cameron, 1962). Here, the author concluded that the infection was a result of direct bud infection, through the outer scales after the leaf-fall period. A recent study, based on the artificial inoculation experiments, showed that cherry is more susceptible to *P. syringae* infection through leaf scars than peach and “French” prune (Cao et al., 2013).

### 4.3 Means of Dissemination

Several natural and man-made phenomena favor the long- and short-distance dissemination of *P. syringae*. This pathogen can be carried in aerosols and thereby transported by wind-driven rain (Crosse, 1966). In addition,

the discovery that the life history of *P. syringae* is linked to the water cycle (Morris et al., 2008) explains how easily the pathogen can be disseminated over a wide range of distances. *P. syringae* can also be transmitted through mechanical equipment and pruning tools although their role in pathogen dissemination is often overlooked. Furthermore, insects such as aphids have been demonstrated to be potential vectors of *P. syringae* (Stavrinides et al., 2009). On stone fruits, the role of insects as a potential carrier of *P. syringae* inoculum has been demonstrated (Wormland, 1931). However, the most important dissemination source of *P. syringae* remains the transportation of infested nursery stock (Dowler and Petersen, 1967; Lyskanowska, 1976; Mansvelt and Hattingh, 1986).

## 4.4 Variability in Host Genotype Susceptibility

The underlying genetic variability and intensity of production is also important in the epidemiology of diseases of woody plants caused by *P. syringae*. A recent example of kiwifruit bacterial canker epidemics reveals how the cultivation of a very narrow range of plant diversity threatens durability of plant resistance. The genus *Actinidia* consists of over 50 species native to China where all wild germplasm is naturally distributed (Liang, 1983). However, the breeding of kiwifruit in New Zealand has been founded on imported planting materials where only two commercial varieties (cv. Hayward and Hort 16A, belonging to *Actinidia deliciosa* and *Actinidia chinensis*, respectively) have been developed and commercialized. For decades, the commercial kiwifruit industry was dominated by the sole cv. Hayward, grown in more than 80% of the world's kiwifruit production areas. It was not until 1999 that commercial marketing of cv. Hort16A was initiated (Ferguson, 1999). Suddenly, the kiwifruit industry has become vulnerable to *P. syringae* infection where the pathogen has jeopardized the entire kiwifruit industry (Butler et al., 2013; Scortichini et al., 2012). Intensive kiwifruit cultivation with very low genetic diversity in areas that are far from its center of origin has been detrimental for the long-term durability of the crop. Bacterial canker of European hazelnut (*Corylus avellana* L.) illustrates a marked contrast to the case of kiwifruit canker (Scortichini, 2002). Unlike kiwifruit, European hazelnut has a wide natural geographical distribution ranging from the Mediterranean coast of North Africa northward to the British Islands and the Scandinavian Peninsula, and eastward to the Ural Mountains of Russia, the Caucasus Mountains, Iran, and Lebanon (Rushforth, 1999). Moreover, there are other wild *Corylus* species native to North American (*Corylus americana*, *Corylus cornuta*), European and western Asian countries (*Corylus*

*maxima*, *C. colurna*), China (*Corylus chinensis*, *Corylus fargesii*, *Corylus yunnanensis*, *Corylus wangii*, *Corylus tibetica*), Japan (*Corylus sieboldiana*), and Siberia (*Corylus heterophylla*) (Mehlenbacher, 1991). A large number of European hazelnut varieties are grown around the globe. In Turkey, the world's most important hazelnut producer, an assorted number of varieties are grown together often in the same field. Bacterial canker has not been reported under these conditions. Indeed, the most extensive cultivated collection of hazelnut genetic resources resides in Turkey, which preserves 739 selections with over 700 genotypes of *C. avellana* (Hummer, 1995; Thompson et al., 1996). In contrast, in Greece where a hazelnut cultivar imported from Turkey (cv. Palaz) was intensively grown in monoculture, bacterial canker caused devastating losses making the cultivation impossible in some areas (Psallidas and Panagopoulos, 1979). Similarly, bacterial canker of hazelnut is a serious problem in some areas of central Italy (Scorticini, 2002) where only one cultivar (cv. Tonda Gentile Romana) is grown in more than 85% of the areas. By contrast, the disease is not reported from other Italian hazelnut growing areas such as Campania, Piedmont, Sardinia, and Sicily where several local varieties are grown (Siscaro et al., 2006; Virdis, 2008).

Among other economically important woody crop species, grapevine (*Vitis vinifera*), Mango (*Mangifera indica*), and citrus (*Citrus* spp.) are the hosts of *P. syringae*, although attacks and economic losses on these species have been reported sporadically. A very large genetic diversity has been described in the cultivated grapevine (Arroyo-Garcia et al., 2006). The genus *Vitis* is native to Transcaucasia (McGovern, 2003), which comprises about 60 interfertile wild *Vitis* species. The natural distribution of such species ranges from Asia to Europe and North America (Terral et al., 2010). Species native to North America, such as *Vitis rupestris*, *Vitis riparia* or *Vitis berlandieri*, are widely used in breeding programs against different fungal pathogens that cause economically important diseases on grapevine. Several wild species of *Vitis* were found to grow along the river banks, and in alluvial and colluvial deciduous and semideciduous forests (Arnold et al., 1998). The distribution in the wild of these species ranges from Western Europe to the Trans-Caucasian zone and around the Mediterranean Basin except the most southern infra-Mediterranean and non-Mediterranean zones (Arnold et al., 1998). In Mango, in addition to commercial varieties, a large number of local and wild germplasm is reported to form a vast genetic resource to the mango breeder (Bally et al., 2008). In-depth lists of mango accessions in collections are also available (<http://www.ipgri.cgiar.org/germplasm/dbintro.htm>). Concerning *Citrus* spp., they originated in Southeast Asian countries. For

example, Key lime (*Corylus aurantifolia*) and citron (*Corylus medica*) are native to India while pomelo (*C. maxima*) is native to Malay Archipelago. Similarly, Mandarin orange (*Corylus reticulata*) is native to China while trifoliate orange (*Corylus trifoliata*) is native to Korea and adjacent China. Finger lime (*Corylus australasica*), Australian round lime (*Corylus australis*), and desert lime (*Citrus glauca*) are native to Australia (Bally et al., 2008). In *Citrus* spp., lower genetic diversity is reported among the cultivars of orange, grapefruit and lemon since they have originated from nucellar seedlings or budsports. Conversely, mandarins, pummelos, and citrons are reported to have a high level of genetic diversity since many of the cultivars have arisen through sexual hybridization (The Citrus and Date Crop Germplasm Committee, 2004). Only few reports of *P. syringae* disease on *Citrus* spp. seem to be linked with the high genetic diversity of this crop, throughout the range of cultivations, thereby enhancing its durability of resistance to the pathogen.



## 5. THE DIVERSITY OF *P. SYRINGAE* CAUSING DISEASE TO WOODY PLANT SPECIES

*Pseudomonas syringae* is a species complex that encompasses a wide variety of strains that are grouped into numerous phylogroups (Parkinson et al., 2011) based on phylogeny of housekeeping genes using the Multi Locus Sequence Typing method. Strains known to attack woody species are found in all phylogroups except phylogroup 6 that contains strains causing disease on papaya and on sunflower (Parkinson et al., 2011). An exhaustive list of existing *P. syringae* pathogens of woody plants is presented in Table 4.2. The names of these pathogens were attributed after instauration of the pathovar nomenclature (Dye et al., 1980; Young et al., 1978) and they were translated to pathovars according to this system for pathogens identified before 1978. Most of the taxonomic descriptions were performed at a time when it was impossible to classify bacteria precisely on the basis of phylogenetic analysis (Cai et al., 2011). Furthermore, in only very few cases the attribution of a pathovar name was accomplished via host range tests (Table 4.3). It is a matter of debate whether the use of the so-called pathovar without thorough host-range description is justified. Among the *P. syringae* pathogens of woody plants, there are 14 existing pathovars, which were originally proposed as *species novet* on the basis of a few phenotypic tests (Table 4.3) and are still poorly circumscribed. Often, simply a few differences in biochemical traits in comparison to the lilac pathogen *P. syringae* pv. *syringae* were the basis of the proposal that the pathogen be considered as *species novet*. Such

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**Table 4.3** Tests and Number of Strains Used to Describe Novel *Pseudomonas syringae* Pathovars

Pathovar	No. of Strains Used	Tests Used for Characterization	Host Range and Species Used	Original References	pv. Designation
<i>aceris</i> <sup>b</sup>	1	Biochemical and pathogenicity	No	Ark (1939)	Young et al. (1978)
<i>actinidiae</i>	a			Takikawa et al. (1989)	Takikawa et al. (1989)
<i>aesculi</i>	a			Durgopal and Singh (1980)	Durgopal and Singh (1980)
<i>avellanae</i>	18	Biochemical, serological, bacteriocin, phage typing, antibiotic resistance, pathogenicity and lesion tests	Yes (12 different species)	Psallidas (1993)	Psallidas (1993)
<i>avii</i> <sup>c</sup>	9	Biochemical tests, DNA–DNA hybridization, rep-PCR, cluster analysis, pathogenicity tests on different <i>Prunus</i> spp.	No	Menard et al. (2003)	Menard et al. (2003)
<i>berberidis</i> <sup>b</sup>	1	Biochemical and pathogenicity	No	Thornberry and Anderson (1931b)	Young et al. (1978)
<i>castaneae</i>	a			Takanashi and Shimizu (1989)	Takanashi and Shimizu (1989)
<i>cerasicola</i>	4	Biochemical and pathogenicity tests	Yes (66 species)	Kamiunten et al. (2000)	Kamiunten et al. (2000)
<i>ciccaronei</i> <sup>b</sup>	7	Biochemical and pathogenicity tests	No	Ercolani and Caldarola (1972)	Young et al. (1978)
<i>coryli</i> <sup>d</sup>	38	Fatty acid analysis, rep-PCR and genomic fingerprinting, 16S rDNA, <i>hrpL</i> gene, pathogenicity test	Yes (7 different species)	Scortichini et al. (2005)	Scortichini et al. (2005)
<i>daphniphylli</i>	a			Ogimi et al. (1990)	Ogimi et al. (1990)
<i>dysoxyli</i> <sup>b</sup>	NA			Hutchinson (1949)	Young et al. (1978)
<i>dendropanacis</i>	a			Ogimi et al. (1988b)	Ogimi et al. (1988b)

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<i>eribotryae</i> <sup>b</sup>	a			Takimoto (1931)	Young et al. (1978)
<i>garcae</i> <sup>b</sup>	a			Do Amaral et al. (1956)	Young et al. (1978)
<i>lachrymans</i> <sup>b</sup>	3	Biochemical, copper resistance, pathogenicity	No	Smith and Bryan (1915)	Young et al. (1978)
<i>mori</i> <sup>b</sup>	a			Boyer and Lambert (1993)	Young et al. (1978)
<i>morsprunorum</i> <sup>b</sup>	1	Biocheical and pathogenicity	No	Wormland (1931)	Young et al. (1978)
<i>myricae</i>	a			Ogimi and Higuchi (1981)	Ogimi and Higuchi (1981)
<i>papulans</i> <sup>c</sup>	9	Biochemical, serological and pathogenicity	Yes (2 species)	Rose (1917)	Dhanvantari (1977)
<i>persicae</i> <sup>b</sup>	57	Biochemical tests	No	Prunier et al. (1970)	Young et al. (1978)
<i>philadelphi</i>	20	Biochemical, phage typing and pathogenicity tests	Yes (plants of 10 different genera)	Roberts (1985a)	Roberts (1985a)
<i>photiniae</i>	a			Goto (1983a)	Goto (1983a)
<i>rhiphiolepidis</i>	a			Ogimi et al. (1992)	Ogimi et al. (1992)
<i>ribicola</i> <sup>b</sup>	3	Biochemical and pathogenicity	No	Bohn and Maloit (1946)	Young et al. (1978)
<i>syringae</i>	a			Van Hall (1902)	Young (1992)
<i>theae</i> <sup>b</sup>	a			Hori (1915)	Young et al. (1978)
<i>ulmi</i> <sup>b</sup>	a			Sutic and Tesic (1958)	Young et al. (1978)
<i>viburni</i> <sup>b</sup>	1	Biochemical and pathogenicity	No	Thornberry and Anderson (1931a)	Young et al. (1978)

<sup>a</sup>Published in different language than English and in local journal, NA: not available.

<sup>b</sup>Pathogen, originally described as a novel species, not included in the approved list, successively translated to pathovar without any description.

<sup>c</sup>In pathogenicity tests the strains caused symptoms also on sweet cherry.

<sup>d</sup>In host range test the strains caused mild symptoms also on some other hosts.

<sup>e</sup>In host range test the strains caused symptoms also on peach.

discriminate criteria for defining new pathovars are inconsistent with the common observation of high phenotypic and genetic diversity of *P. syringae* in a very restricted area and even in association with a single species of host plant (Table 4.4). Indeed, a high phenotypic and genetic variability is typical of *P. syringae*. For each disease, differences in phenotypes and genotypes among the strains incriminated in the disease have been reported in the literature (Table 4.4). In a very recent work, Cinelli et al. (2013c) demonstrated that even the disease symptoms caused by the strains isolated from the same woody plant markedly differ. Similarly, a high phenotypic variability has been reported among the *P. syringae* strains isolated from different plant organs of the same annual plant affected by the disease (Morris et al., 2000).

Although attempts to clarify the naming of pathovars were made after 1978, confusions still occur. According to the standards, in designating a novel pathovar, it is necessary to demonstrate, through a pathogenicity testing regime, that the pathogen has a distinct host range or causes a distinct disease when compared with previously described pathogens (Young et al., 1978). However, it seems that old habits persist. For example, *P. syringae* pv. *avii* is the name attributed to the causal agent of bacterial canker of wild cherry (Menard et al., 2003) without a host range test. Bacterial canker with identical symptoms is caused by *P. syringae* pv. *syringae* and *P. syringae* pv. *morsprunorum* races on wild cherry (Vicente et al., 2004). On *Prunus* spp., four distinct *P. syringae* pathovars (*avii*, *morsprunorum* race 1 and 2, *persicae* and *syringae*) have been described on the basis of some phenotypic differences. All of them cause bacterial canker on the same hosts and most of them are frequently associated with the same disease (Hattingh et al., 1989; Kennelly et al., 2007). Another important example concerns the pathogens of olive and oleander knots caused by *Pseudomonas savastanoi* pv. *savastanoi* and *P. savastanoi* pv. *nerii*, respectively. Previous studies demonstrated that there is an overlapping host range among these pathovars (Alvarez et al., 1998; Janse, 1982). Recently, Young (2008) questioned if they should be considered as separate pathovars (pv. *savastanoi* pathogenic to olive and pv. *nerii* pathogenic to oleander) or as a single pathovar. The debate about the naming of these pathovars can be set into the context of the studies of host range that have been conducted for single pathovars. In many cases, these pathovars infect hosts other than that of the original isolation. Dhanvantari (1977) reported that *P. syringae* pv. *papulans*, previously described as *species novum* from apple (Rose, 1917), also infects peach. Similarly, Scortichini et al. (2005) reported a so-called novel pathovar from European hazelnut, *P. syringae* pv. *coryli*, which also causes mild disease symptoms on pear, apricot, and peach shoots.

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**Table 4.4** Phenotypic and Genetic Diversity Observed among Strains of *Pseudomonas syringae* Pathovars Pathogenic on Woody Plants

Pathogen(s)	Isolated Host	No. of Strains Used	Analysis	Haplotypes	References
<b>Genetic diversity</b>					
Pss	Stone fruits	91	ERIC PCR	11	Little et al. (1998)
Psa	Kiwifruit	44	MLST	4	Chapman et al. (2012)
Pss	Stone fruit and almond	78	ERIC, REP, BOXA1R, IS50	9	Abbasi et al. (2012)
Pss, Psm	Stone fruit and hazelnut	33	MLST	5,3	Kaluzna et al. (2010)
Pss	Pome and stone fruits	280	BOX PCR	4	Gilbert et al. (2009)
Pss	Cherry and plum	87	rep-PCR	10	Vicente and Roberts (2007)
Pss	Pome and stone fruits	101	rep-PCR	17	Scorticchini et al. (2003)
Pss	Pear	90	BOX PCR	4	Natalini et al. (2006)
Psm1	Cherry and plum	19	Phage typing	6	Crosse and Garrett (1970)
<b>Phenotypic diversity</b>					
Pss	Apple	60	F, LOPAT, GATTa, INA, syringomycin	16	Mansvelt and Hattingh (1986)
Pss	Mixed hosts		LOPAT, syringomycin, other biochemical tests		Young (1991)
Psa	Kiwifruit	46	F, LOPAT, INA, syringomycin production, resistance to streptomycin and copper	3	Vanneste et al. (2013)
Psav	Olive	26	F, LOPAT	2	Marchi et al. (2005)
Psav	Myrtle	34	Pathogenicity	4	Cinelli et al. (2013b)
Pss	Pear	90	F, INA, LOPAT, GATTa, viru- lence tests on lemon fruits, pear and lilac leaves	4	Natalini et al. (2006)
Pss	Pome and stone fruits	70	F, LOPAT, GATTa, virulence tests	3	Gilbert et al. (2010)
Pss	Cherry and plum	54	Pathogenicity	4	Vicente et al. (2004)

Note: Pss, *Pseudomonas syringae* pv. *syringae*; Psav, *P. savastanoi*; Pssav, *P. savastanoi* pv. *savastanoi*; Psm1, *P. syringae* pv. *morsprunorum* race 1; Psa, *P. syringae* pv. *actinidiae*; Pav, *Pseudomonas avellanae*; F, fluorescence on KB, LOPAT, levan, oxidase, potato rot, arginine dihydrolase and tobacco hypersensitivity, respectively; GATTa, Gelatin hydrolysis, aesculin hydrolysis, tyrosinase activity and tartaric acid utilization, respectively; INA, ice nucleation activity.

Without comparative host range testing, it is difficult to know if apple is also a host of strains from peach or if hazelnut is a host of strains from pear and apricot cankers or if these are reports of truly novel pathotypes.

It is difficult to assess if the abundance of reports of different pathovars of *P. syringae* of woody species reflects a real diversification in host specialization or if it is a reflection of the wide range and inconsistency of tests used to characterize these pathogens. The literature reveals a surprising heterogeneity in tests used for the characterization of *P. syringae* from woody plants. Firstly, *in planta* pathogenicity tests on the original host of isolation, for the completion of Koch's postulate, is lacking in some cases (Gilbert et al., 2009; Ivanovic et al., 2012; Kaluzna et al., 2010). Secondly, host range tests have been performed only in a limited number of studies and on a very low number of host species (1–5) (Cirvilleri et al., 2007; Eltlbany et al., 2012; Golzar and Cother, 2008; Hall et al., 2004; Psallidas, 1993; Samavatian, 2006; Scorticini, 2006; Scorticini et al., 2005; Taghavi and Hasani, 2012; Vicente et al., 2004) making it difficult to know if the described pathovar is really different from previously described pathovars. On *Prunus* spp., where different pathovars cause the disease, cross-infection tests within the genus are lacking. There is also inconsistency in tests of ice nucleation activity (INA) of strains. INA has often been reported as a virulence factor of plant pathogenic *P. syringae* (Kennelly et al., 2007; Klement et al., 1984; Sule and Seemuller, 1987; Weaver, 1978) and in particular those attacking woody plants because flowering, leaf bud formation and surges of succulent growth can occur during periods with risk of frost. However, tests of ice nucleation activity are missing in many of the descriptions of *P. syringae* that are pathogenic to woody plants (Abbasi et al., 2012; Balestra et al., 2009c; Gilbert et al., 2009; Ivanovic et al., 2012; Kaluzna et al., 2010; Kamiunten et al., 2000; Mirik et al., 2005; Vicente et al., 2004). Biochemical tests, such as fluorescence on King's medium B, LOPAT (levan production, oxidase, potato rot, presence of arginine dihydrolase and induction of hypersensitivity on tobacco), GATTa (gelatin hydrolysis, aesculin hydrolysis, tyrosinase activity, and tartaric acid production), use of different carbohydrates as sole carbon sources, induction of lesions on detached plant organs, phytohormon production, resistance to copper compounds and antibiotics were among the most utilized for the characterization of strains (Table 4.4). While the identification of specific pathovars is the most common means for reporting new diseases, in some cases authors were reticent. Of the 55 outbreaks reported since 2000 (Table 4.1), 46 associated the causal agents to specific *P. syringae* pathovars, while only few reports described *P. syringae* "in sensu lato"

(Bardoux and Rousseau, 2007; González and Ávila, 2005; Hall et al., 2003; Ivanova, 2009; Mahdavian and Hasanzadeh, 2012; Ozakatan et al., 2008).



## 6. CONTROL OF DISEASES OF WOODY PLANTS CAUSED BY *P. SYRINGAE*

Strange and Scott (2005) described three categories of methods for minimizing plant disease: (1) exclusion, elimination or reduction of pathogen inoculum, (2) promotion of genetic diversity in the crop and (3) inhibition of pathogen virulence mechanisms. These measures are pertinent to limiting diseases of woody plants caused by *P. syringae*. In practice, these methods should not be used in exclusion but rather they are combined together through an integrated management approach.

### 6.1 Exclusion, Elimination, or Reduction of Pathogen Inoculum

In theory, hygiene and quarantine are effective control methods and they are currently being put to test for bacterial canker of kiwifruit. In 2012, *P. syringae* pv. *actinidiae* was declared a quarantine pathogen in the southern part of the EPPO region ([http://www.eppo.int/QUARANTINE/recent\\_additions.htm](http://www.eppo.int/QUARANTINE/recent_additions.htm)). Similarly, *P. syringae* pv. *persicae*, the causal agent of bacterial dieback of peach, is another EPPO quarantine pathogen (OEPP/EPPO, 2005). Whether the quarantine regulation applied to a specific pathovar can be effective in controlling the introduction of the pathogen into new areas is a matter of debate. In some cases, the pathogen can be latently present in plant tissues for long periods of time without causing diseases as those reported from *Prunus* spp. (Dowler and Petersen, 1967; Lyskanowska, 1976; Mansvelt and Hattingh, 1986). The development of early detection methods should be the first step to avoid the introduction of latently infected nursery materials to a given area. Several detection methods have been developed with the aim of early detection of *P. syringae* from asymptomatic plant parts (Bertolini et al., 2003; Biondi et al., 2013; Gallelli et al., 2011). However, the limit of such methods is based on the fact that they can detect only a narrow range of the diversity of the *P. syringae* population, usually targeting the so-called pathovar level. Only detection methods that are able to identify a broader *P. syringae* diversity can be effective in order to promptly detect the emerging pathogens from asymptomatic propagation materials, given the high phenotypic and genetic diversity of this pathogen.

Freedom from pathogens in planting material is not a static event and requires constant monitoring and maintenance during all stages of production, storage, and distribution (Janse and Wenneker, 2002). Hence, a clear understanding of the causal agent of a given disease might be a good starting point to develop sensitive and effective detection methods from asymptomatic propagation materials. Besides propagation materials, the pathogen can spread for long distance also through migrating birds, insects (honey bees), wind-driven slime and undetected infections on wild hosts as those reported for the quarantine pathogen *Erwinia amylovora*, which led to a complete failure of the eradication campaign of fire blight disease (Calzolari et al., 2000; Vanneste, 2000). There are several examples in the literature on the reintroduction of an eradicated bacterial disease of woody plants that clearly indicate the difficulty of effective application of quarantine measures. Citrus canker, caused by *Xanthomonas axonopodis* pv. *citri* on citrus, in Florida (Graham and Gottwald, 1991; Schubert and Miller, 1997; Schubert et al., 2001) and fire blight of pome fruits, caused by *E. amylovora*, in many countries (Calzolari et al., 2000; Vanneste, 2000) are examples. Overall, a complete eradication of plants is rarely accomplished and often the common practice in the field is to eliminate only heavily damaged plants that are no longer productive. Current epidemics of kiwifruit bacterial canker fully reflects this scenario in which no reports on plant eradication exist, except those reported from some areas of Spain (Abelleira et al., 2011). Furthermore, an accurate definition of the pathovar responsible for the disease is essential for quarantine. As mentioned earlier, it is possible that these definitions are too narrow at present, thereby limiting the scope of quarantine actions to a range of strains that is narrower than the full diversity truly responsible for disease.

The only way to reduce *P. syringae* pathogen inoculum is effective cultural practices (Scorticchini, 2002; Young, 2004, 2012). Treatments of propagative materials in nurseries with bactericides might be an effective solution but no efficient product is commercially available (see below). In fields, infected plants or plant parts may be either completely uprooted or pruned and burned. The nature of *P. syringae*, in that it lives, multiplies and overwinters on plant surfaces, makes its management extremely difficult compared to soil pathogens, for example. The reduction of inoculum of the latter is often successful through crop rotation and plowing (Summerell and Burgess, 1989), soil solarization (Katan, 1981), the addition of amendments (Lazarovits, 2001), and in some instances, by flooding (Thurston, 1990).

Overall, chemical control has been exclusively used for decades in the plant disease management. However, only a few effective and economical

bactericides have been developed to control bacterial pathogens. Plant pathogenic bacteria have been more recalcitrant to chemical treatments than their fungal counterparts (Jones et al., 2013). Unlike several products available for the control of phytopathogenic fungi (Oerke, 2005) (many of them systemic), only a limited number of products is commercially available for the control of phytopathogenic *P. syringae* (Agrios, 2005). The use of antibiotics is strictly forbidden in many countries including those in the European Union (Casewell et al., 2003), because of their implication for human health and the risk of resistance that the pathogen develops under selective pressure (Khachatourians, 1998; Lipsitch et al., 2002). Growers have to settle for only few commercially available chemicals. Among them, copper compounds are the standard bactericides, almost exclusively used, for the control of bacterial diseases (Agrios, 2005; Kennelly et al., 2007). Although these compounds often give satisfactory results on herbaceous plants, where *P. syringae* does not cause systemic disease, the success is limited on woody plants because they do not penetrate inside plant tissues to where the pathogen has invaded the vascular tissues (Alvarez, 2004; Kennelly et al., 2007). The only way to have success in controlling vascular pathogens is to intervene at the time of infection, by coinciding with periods when the host is susceptible, when the pathogen is accessible, and when conditions are favorable for disease (Kennelly et al., 2007). Previously, a predictive system for timing of chemical applications has been reported to improve chemical spray control of *P. syringae* (Jardine and Stephens, 1987). The authors used stagewise multiple linear regression techniques to identify meteorological and biological variables useful in predicting *P. syringae* disease development. However, it is worth noting that different hosts react differently to the same treatment. Stone fruits are examples of this differential reaction where preventive treatments, with copper-based compounds following leaf shedding, are effective to control *P. syringae* infection of peach and cherry but not of apricot. Knowledge of infection ports is useful for this reason. However, the phytotoxic effect of copper compounds on some species can be incompatible with such timings. For example, treatment during flower bloom is not possible for some species because flowers are highly sensitive to copper compounds (Lalancette and McFarland, 2007). Copper is one of the most used bactericides in agriculture, leading to the development of copper-resistant lines of *P. syringae* (Masami et al., 2004; Scheck et al., 1996; Sundin and Bender, 1993). Whatever the utility of copper, the environmental concerns provoked by its heavy use are leading to increasing restrictions in the European Union (Pietrzak and McPhail, 2004).

New directions in chemical control emerged recently to overcome the limitations of the common bactericides. Examples are elicitors such as harpins that activate plant defense and induce resistance, and polysaccharides such as chitosan (Dong et al., 1999; Reglinski and Elmer, 2011). Chitosan has attracted concern because of its strong antimicrobial activity toward a broad spectrum of pathogens, including common plant pathogenic bacteria and fungi. The major advantage of chitosan over conventional chemicals is its biocompatibility and biodegradability (Badawy and Rabea, 2011). In addition to chitosan, other compounds that have shown *in vitro* inhibitory effects on *P. syringae* include terpens (Ferrante and Scorticini, 2010) and antimicrobial peptides (Cameron and Sarojini, 2014). The latter are reported also to inhibit biofilms (De Zoysa et al., 2013), commonly formed by *P. syringae*. However, the use of these compounds has been limited to controlled environments and no information is available for open conditions. Other chemicals used in fields are Acibenzolar-S-methyl (ASM, BTH, Bion or Actigard), an inducer of systemic acquired resistance (SAR) (Scorticini and Ligouri, 2003) and prohexadione calcium (Costa et al., 2001; Norelli and Miller, 2004), a growth regulator that has been tested on several species. While inducers of SAR can be effective under controlled conditions, the host response can be highly variable in the field, raising questions about their potential for disease management. Although SAR inducers may reduce disease to a certain extent in some species such as apple and hazelnut (Maxson-Stein et al., 2002; Scorticini and Ligouri, 2003), in fields they may also have deleterious effects on certain other plant species and/or affect yield (Gent and Schwartz, 2005; Romero et al., 2001). Furthermore, it has been reported that plant inducers did not result in any effective disease control in some pathosystems such as citrus canker (Graham and Leite, 2004). Studies carried out in Italy and New Zealand showed that in glasshouse conditions SAR can reduce the disease incidence on young kiwifruit seedlings (Vanneste et al., 2012) although no information is available from the field. Since a large number of similarities have been reported among *P. syringae* diseases of woody plants (such as bacterial cankers of stone fruit and kiwifruit) and citrus canker (<http://www.kvh.org.nz/vdb/document/91507>), the effect of SAR on *P. syringae* disease control might not be effective in natural field conditions. However, in-depth future investigations are needed before making a conclusion.

During the last decades, there is a growing interest in adopting biological control measures. At present, only inundative methods of biological control have been developed for diseases of woody species whereby

nonpathogenic microorganisms are applied to foliar or root tissues resulting in disease suppression. Such strategies include the use of nonpathogenic or pathogenically attenuated strains of the pathogen species (Frey et al., 1994; Liu, 1998; Hert, 2007), saprophytic bacteria (Ji et al., 2006), nonpathogenic bacteriocin-producing *Agrobacterium radiobacter* strains that inhibit closely related pathogenic strains (Kerr, 1974; Kerr and Htay, 1974), and plant growth-promoting rhizobacteria (Ji et al., 2006). These strategies aim to suppress pathogen populations or induce SAR or a similar response in the plant that reduces the ability of the pathogen to colonize the plant and cause disease. Biological control approaches have been used to control bacterial pathogens of several woody plants such as fire blight diseases of pome fruits (Boulé et al., 2011; Vanneste et al., 1992), caused by *E. amylovora* and bacterial spot of peach (Biondi et al., 2009) caused by *Xanthomonas arboricola* pv. *pruni*. However, throughout the literature, there is only an example on the biocontrol of *P. syringae*, which involved *in vitro* efficacy of aqueous plants extracts (Bhardwaj, 2011).

The use of bacteriophages is another form of biological control and has been successful for managing several annual plant diseases (Jackson, 1989). Phages have been under evaluation for controlling several woody plant-pathogenic bacteria. Examples are fire blight on apple and pear (Schnabel and Jones, 2001) and bacterial canker and bacterial spot of citrus (Balogh, 2006; Balogh et al., 2008). To date, all the successful application of phages concerns foliar pathogens and there are virtually no reports for woody tissues. Only one study has shown a preventative and beneficial effect with phage treatment, on vascular disease system on citrus, caused by *X. axonopodis* pv. *citri* (Balogh et al., 2008). The disease symptoms and location of infection within or on the plant can pose challenges for phage biocontrol. For example, most of the economically important diseases on trees are caused by vascular *P. syringae* pathogens (Kennelly et al., 2007; Scorticini, 2002; Scorticini et al., 2012). The latter spend some of the disease cycle inside the host plant following infection through plant openings or wounds (Kennelly et al., 2007; Scorticini, 2002; Scorticini et al., 2012). High numbers of bacteria can accumulate within cankers in the plant and are protected from any control agent applied to the outside of the plant that cannot penetrate to deeper tissues. However, the ability of phage to act as a curative agent of cankers was not directly assessed. In a recent report, use of bacteriophages has been indicated among the biocontrol agents with potential to control or suppress bacterial canker of kiwifruit, caused by *P. syringae* pv. *actinidiae* (Stewart et al., 2011). In addition, there are some ongoing studies on the

bacteriophage of this pathogen (Qing, 2007). Despite several attempts to use of bacteriophage to control different phytopathogenic bacteria, little is known on the possibility of control of *P. syringae* pathogens of woody plants.

## 6.2 Enhancing Crop Genetic Diversity

As mentioned above, the diversity of the woody crop can have a marked impact on epidemics as illustrated by the contrasting cases of the cankers of kiwifruit and hazelnut. In the case of bacterial canker of kiwifruit, there has been a call to try to increase the diversity of the crop in an attempt to reduce disease pressure (McCann et al., 2013). This needs to be done with foresight, in order to not enhance the abundance of disease sensitive plants as in the case of yellow-fleshed kiwifruit, *A. chinensis*. A thorough knowledge of crop genetic resources and their diversity is a prerequisite for their preservation and further use in breeding programs. The centers of origin and diversification of the most important woody hosts of *P. syringae* are listed in Table 4.5. Wild genetic resources of most of the woody crops are present in more than one country and they have been domesticated for centuries or even millennia. However, their origins and the natural distribution of their progenitor species are usually matters of debate (Frankel and Bennett, 1970) mostly for *Citrus* spp., *Prunus* spp. and *Vitis* spp. By contrast, the domestication of kiwifruit has occurred only a century ago and almost all the diversity of the genus *Actinidia* is present in China (Ferguson and Huang, 2007). Aside from *Prunus* spp., almost all woody fruit crop species have Asia as the center of origin and domestication and this occurred several centuries ago. Domestication is reported as the main cause of reduction of genetic diversity in crops relative to their wild progenitors, due to human selection and genetic drift through bottleneck effects (Tanksley and McCouch, 1997).

For woody plant species characterized by a high genetic diversity, spontaneous variation in disease resistance can be selected and used in breeding programs. In *Prunus* spp., some successful control of *P. syringae* has been reported using plant germplasm resistant to this pathogen. Example is the rootstock F12-1 used in Oregon, which is found to be quite resistant to the pathogen (Moore, 1988). Similarly, some cultivars of sour cherry in Germany were resistant to bacterial canker caused by *P. syringae* (Schmidle, 1981).

Plantation of mixtures of varieties or cultivars versus only one variety within the same field might be useful to improve the efficacy of control methods. Because different plant genotypes present different resistance to pathogen, the overall yield loss in this way may be significantly reduced. An example is rice blast control in China (Zhu et al., 2000) where a significant

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**Table 4.5** Centers of Origin and Diversification of the Most Economically Important Woody Hosts of *Pseudomonas syringae*

Species	Origin	Area of Domestication	References
Apple	Central Asia	Central and East Asia	Janick (2005), Watkins (1995)
Pear	Central Asia	Central and East Asia	Janick (2005), Watkins (1995)
Citrus	Southeast Asia	Southeast Asia	Gmitter et al. (2009)
European hazelnut	Western Asia	Mediterranean areas, Turkey, Iran	Kasapligil (1964)
Grapevine	Transcaucasia	Transcaucasia and Mediterranean areas	McGovern (2003)
Kiwifruit	China	New Zealand	Ferguson and Bolland (1990)
Mango	South Asia	Tropical areas of Asia, Oceania and America	Bally et al. (2008)
Olive	Asia minor	Asia minor, Mediterranean basin	Zohary and Spiegel-Roy (1975)

***Prunus* spp.**

Almond	Southwest Asia	Central and East Asia	Janick (2005), Watkins (1995)
Apricot	Central and eastern Asia	Central and East Asia	Janick (2005), Watkins (1995)
Peach	China	Central and East Asia	Faust et al. (1998), Janick (2005), Watkins (1995)
Plum (Asian, European, American)	China, Europe and America	Central and East Asia, Europe, America	Janick (2005), Watkins (1995)
Sweet and tart cherry	Central Europe, western Asia	Central and East Asia, Europe	Janick (2005), Watkins (1995)

reduction of the disease has been observed through the interspaced cultivation of two rice cultivars, one resistant and the another susceptible to rice blast disease. Even more effective results might be obtained through intercropping whereby a mixture of different plant species is grown in close proximity. However, prior to intercropping, it is necessary to know whether two or more crop species that constitute the mixture are susceptible to the same pathogen. To date, the lack of comparative host range tests

limit our understanding of the full host range of *P. syringae* pathovars and make it difficult to predict the efficiency of intercropping as a means of disease control.

### 6.3 Inhibition of *P. syringae* Virulence Mechanisms

Exploitation of a pathogen's dependence on certain virulence mechanisms might be useful for their control. Table 4.6 presents a list of *P. syringae* virulence factors. It is well known that most of the virulence factors are induced by plant signal molecules (Ausubel, 2005; Tör et al., 2009). For example, the Type-Three Secretion System (TTSS) is the system in which bacteria can inject effector (virulence) proteins into the host cells Alfano and Collmer, 2004. These proteins, as well as the apparatus for the syringae, are coded by genes located in the exchangeable effector loci (EEL), *hrp/hrc* and conserved effector loci (CEL) (Block and Alfano, 2011). Type-III effectors are believed to contribute to pathogenesis in two ways: by eliciting the release of water and/or nutrients from the host cell in the apoplastic space; and by suppressing and/or evading plant host defense responses (Block and Alfano, 2011; Tampakaki et al., 2010). Recent studies have firmly established the concept that the suppression of various plant defenses, including basal defense, gene-for-gene resistance, and nonhost resistance (NHR), is a major virulence function of intracellular TTSS effectors (Nomura et al., 2005; Tampakaki et al., 2010). In particular, NHR is the most important form of resistance since it provides immunity to all members of plant species against all pathogens that cause disease to other plant species. Different plant genes have been reported to show NHR against plant pathogens. In arabidopsis, NONHOST1 was the first gene to confer NHR against *P. syringae* pv. *phaseolicola* (Kang et al., 2003). In tomato, host resistance to *P. syringae* disease is conferred by the Pto protein kinase, which acts in concert with the Prf nucleotide-binding lucine-rich repeat protein to recognize the pathogen expressing the TTSS effector genes *AvrPto* or *AvrPtoB* (Pedley and Martin, 2003). In tobacco and tomato, the successful use of a pattern recognition receptor gene, EFR, from Arabidopsis reduced the growth of *P. syringae* pv. *tabaci* and pv. *tomato*, respectively (Lacombe et al., 2010). In tomato, the R gene from pepper, Bs2, has been shown to impart resistance to bacterial pathogen *Xanthomonas campestris* pv. *vesicatoria* (Tai et al., 1999). The same pepper gene Bs2 confers resistance to lemon against the bacterial pathogen *X. axonopodis* pv. *citri*. In barley, penetration deficient genes (*PEN*) 1, 2 and 3 provide NHR against the pathogen *Blumeria graminis* f. sp. *hordei* at the prehaustorial level (Lipka et al., 2005). In addition, NHR Arabidopsis gene *PSS1* confers immunity

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**Table 4.6** Virulence Factor of *Pseudomonas syringae* Pathogen of Woody Plants

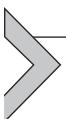
Virulence Factor	Pathogen	References
Functional T3SS	All	Cunnac et al. (2009), Jin et al. (2003)
Ice nucleation activity	Pss	Klement et al. (1984), Sule and Seemuller (1987), Weaver (1978)
Exopolysaccharides	All	Corsaro et al. (2001), Denny (1995), Yu et al. (1999)
Coronatin	Psa; Psm	Bender et al. (1999), Gross (1991), Han et al. (2003), Liang and Jones (1995)
Mangotoxin	Pss, Pav	Arrebolá et al. (2003), Carrión et al. (2013)
Persicomycin	Psp	Barzic and Guittet (1996)
Phaseolotoxin	Psa	Bender et al. (1999), Gross (1991)
Tabtoxin	Psg	Bender et al. (1999), Gross (1991), Mitchell (1984)
Tagetotoxin	Pst	Mitchell and Hart (1983)
Syringomycin	Pss	Bender et al. (1999), Gross (1991)
Yersiniabactin	Psm	Bender et al. (1999), Kaluzna et al. (2010)
Phytohormones	Pss, Psc, Psac, Psmy, Psph, Psav, Pa, Psr, Psac	Bultreys et al. (2008), Glickmann et al. (1998), MacDonald et al. (1986), Young (2004)

Note: T3SS, Effectors and type-three secretion system; Pss, *Pseudomonas syringae* pv. *syringae*; Psc, *P. syringae* pv. *ciceronei*; Psa, *P. syringae* pv. *actiidiæ*; Psm, *P. syringae* pv. *morsprunorum*; Psp, *P. syringae* pv. *persicae*; Pav, *P. syringae* pv. *avellanae*; Psav, *P. savastanoi* pathovars; Psg, *P. syringae* pv. *garcae*; Pst, *P. syringae* pv. *tagetis*; Psmy, *P. syringae* pv. *myricae*; Psph, *P. syringae* pv. *photinae*; Psr, *P. syringae* pv. *ribicola*; Psac, *P. syringae* pv. *aceris*; Pa, *P. amygdali*.

against *Phytophthora sojae* and *Fusarium virguliformae* (Sumit et al., 2012). Recognition of pathogen-associated molecular patterns (PAMPs) of nonadapted pathogens by PAMP recognition receptors (PRRs) triggers the PAMP-triggered immunity (PTI) in nonhost species (Dangl and Jones, 2001). PTI has been demonstrated to play a major role in NHR (Schwessinger and Zipfel, 2008). Both physical (callose deposition at the infection sites, waxy coating on leaves) and chemical (deposition of various reactive oxygen species such as H<sub>2</sub>O<sub>2</sub> and phenolic compounds at the infection sites) barriers induced by PTI restrict nonadapted pathogens from invading nonhost species (Bittel and Robatzek, 2007; Mittler et al., 1999). Hence, identification, characterization, and the use of different NHR genes from various plants may be a good strategy to develop *P. syringae* disease resistant woody crops, through breeding.

The virulence of numerous phytopathogenic bacteria has been correlated with their ability to produce exopolysaccharide polymers *in planta* (Denny, 1995; Kao et al., 1992; Katzen et al., 1998). Studies on the exopolysaccharide molecules produced by *P. syringae* *in planta* indicated that alginate was the major exopolysaccharide produced in water-soaked lesions (Fett et al., 1989; Rudolph et al., 1989). Alginate production by *P. syringae* has been associated with increased epiphytic fitness, resistance to desiccation and toxic molecules, and the induction of water-soaked lesions on infected leaves (Fett et al., 1989; Yu et al., 1999). Furthermore, a positive correlation between the virulence of *P. syringae* and the quantity of alginate produced *in planta* has been demonstrated (Osman et al., 1986; Gross and Rudolph, 1987; Yu et al., 1999). A recent study demonstrated the presence of phase variation in *P. syringae* where the same clonal line can give rise to two phenotypes, a mucoid and a transparent (Bartoli et al., 2014). Only the mucoid phenotype, where the production of exopolysaccharide occurs, is reported to have pectolytic activity and to induce tobacco hypersensitivity. In addition, the two phases of the pathogen can be fixed *in vitro* and are not revertible. In addition, the disease is caused only by the mucoid phase *in planta*. Hence, targeting the molecular switch between these different phases could be a means to manipulate the pathogen.

Phytotoxins are products of plant pathogens or of the host-pathogen interaction that directly injure plant cells and influence the course of disease development or symptoms (Bender et al., 1999). Although phytotoxins are not required for pathogenicity in *P. syringae*, they generally function as factors that enhance aggressiveness of the pathogen leading to increased disease severity. For example, *P. syringae* phytotoxins can contribute to systemic movement of bacteria *in planta* (Patil et al., 1974), to lesion size (Bender et al., 1987; Xu and Gross, 1988), and to multiplication of the pathogen in the host (Bender et al., 1987; Feys et al., 1994; Mittal and Davis, 1995). In nature, other microorganisms resistant to the toxins produced by *P. syringae* may exist thereby allowing their use as biocontrol agents, to detoxify the effect of *P. syringae* produced toxins in plant. An example is the use of a strain of *Pantoea dispersa* as a biocontrol agent (Zhang and Birch, 1997) which is resistant to the toxin albicidin produced by *Xanthomonas albilineans*, the causal agent of sugarcane leaf scald.



## 7. THE CASE OF FROST DAMAGE DUE TO ICE NUCLEATION-ACTIVE *P. SYRINGAE*

In addition to its pathogenicity *sensu stricto*, *P. syringae* causes significant crop losses through frost damage. The frost sensitivity of plants is augmented

when they harbor large population of INA bacteria (Hirano and Upper, 2000). The amount of frost damage at a given temperature increases directly with increasing numbers of INA bacteria on that plant (Hirano and Upper, 1990; Lindow, 1980). In the presence of INA populations of *P. syringae*, the freezing point of plant tissue is raised compared to when INA bacteria are absent. Overall, plants can resist temperatures below freezing by supercooling. At temperatures below 0 °C, a process known as ice nucleation can occur whereby a solid particle (dust, bacteria etc.) serves as a catalyst for ice formation. As discussed previously, *P. syringae* can survive and multiply both as an endophyte and epiphyte, even in association to symptomless plants thereby increasing the risk of frost injury.

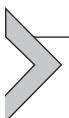
Frost injury in association to *P. syringae* has been reported as a predisposing factor for blossom blight, bud and twig diebacks in numerous woody plants. Blossom blights are reported on kiwifruit (Balestra et al., 2009c; Young, 1987, 1988b), pear (Panagopoulos and Crosse, 1964), magnolia (Goto et al., 1988), pistachio (Rostami, 2012), and rhododendron (Baca et al., 1987). Similarly, twig dieback has been reported in tea (Goto et al., 1988) and coffee (Ramos and Kamidi, 1981). *P. syringae* is reported to affect ice nucleation of grapevine buds (Luisetti et al., 1991). In addition, the predisposition of plants to *P. syringae* leaf infection was increased in the presence of readily available inoculum, within 20 min of thawing. However, after 30 min, no effect of thawing has been observed in the formation of leaf lesions (Sule and Seemuller, 1987).

In contrast to its effect on tender and succulent plant parts, the contribution of *P. syringae* INA population to frost injury of lignified tissues is limited as demonstrated previously (Gross et al., 1983). Indeed, the woody stem tissue of these plants has a source of INA material that by itself can promote ice formation at -2 to -4 °C (Andrews et al., 1983; Ashworth et al., 1985; Proebsting et al., 1982). Freezing and thawing events, irrespective of INA bacteria, have been reported to predispose woody plants to infection by phytopathogenic bacteria in orchards (Ferrante and Scorticini, 2014; Lamichhane et al., 2013). In nurseries, frost damage is reported to predispose a large number of woody plants to *P. syringae* infection (Baca et al., 1987). Moreover, in short-rotation forestry, twig dieback of poplar (Ramstedt et al., 1994) willow (Nejad et al., 2004; Ramstedt et al., 1994) and black alder (Scorticini, 1997) has been attributed to *P. syringae* in combination with freezing stress.

Preventive measures can be applied to reduce the propensity of ice nucleation. Prediction of critical conditions that favor frost damage is the basis to avoid crop losses. More specifically, prediction of INA *P. syringae* population sizes maybe compared to weather forecasting. Classical

methods of preventing frost damage include sprinkler irrigation and heating of orchards (Lindow, 1983a). In addition, bacterial ice nucleation can be inhibited by agents that kill whole cells such as bactericides (Kawahara et al., 2000; Menkissoglu-Spiroudi et al., 2001), antibiotics (Hirano et al., 1985; Lindow, 1983a, 1983b), various heavy metal ions in a soluble state (Hirano et al., 1985; Lindow, 1983a, 1983b; Menkissoglu and Lindow, 1991), surfactants (Himelrick et al., 1991), organic solvents (Turner et al., 1990), and phospholipases (Govindarajan and Lindow, 1988; Turner et al., 1991). These compounds inactivate the nucleus produced by the INA bacteria. Copper compounds were used successfully to reduce frost injury induced by INA *P. syringae* (Lindow, 1983b). A combination of copper-streptomycin sprays was also used to control pear blossom blast in California (Bethell et al., 1977).

Efforts at biological control have been directed almost entirely at frost control using bacterial antagonists to prevent buildup of *P. syringae* INA populations (Lindemann and Suslow, 1987; Lindow, 1983b). Lindow and Staskawicz (1981), by using recombinant DNA technology, used an INA positive *P. syringae* strain from which the gene responsible for ice nucleation had been removed. Other tests for biocontrol of frost damage concerned the use of chemically mutated *P. syringae* INA<sup>-</sup> strains and of naturally occurring INA<sup>-</sup> bacterial antagonists, which reduced significantly the population sizes of naturally occurring INA<sup>+</sup> *P. syringae* (Lindow, 1990, 1995). Application of these antagonists prior to budbreak (woody plants) or at seedling (annual plants) allowed the antagonist to obtain a dominant competitive position in the phylloplane. Examples are reduction of frost injury to strawberry (Lindemann and Suslow, 1987), almond (Lindow and Connell, 1984), and potato (Lindow and Panagopoulous, 1988). The use of bacterial ice nucleation inhibitors appears to offer a “day-before” type of immediate prevention of INA-bacteria-induced frost, which is not provided by bactericides or antagonistic bacteria (Lindow, 1980) and hence is reliant on accurate prediction of frost events. However, frost control was not achieved by using such antagonists or chemical bactericides in field trials on apple and pear in Washington (Gross et al., 1984). It is believed that, in these hosts, there are intrinsic INA molecules within plant tissue independent from the *P. syringae* INA populations.



## 8. CONCLUSIONS AND PERSPECTIVES

Within *P. syringae*, the designation of the so-called pathovars has complicated the understanding of disease epidemiology and the establishment of management practices. Such pathovar definitions are too narrow at present

thereby strongly limiting the objective of quarantine actions to a range of strains that is narrower than the full diversity involved in disease development. Do the pathovar names truly suggest that the strains within a pathovar are specialized to a host exclusive from other pathovars? If so how can we then explain the variability that can be observed among the strains of the same pathovar? Without comparative host range testing, it is difficult to know whether peach is also a host of strains from kiwifruit or if hazelnut is a host of strains from apricot canker or if these are reports of truly novel pathotypes. Such aspects are critical and should be addressed for the management of inoculum reservoirs, breeding for resistance and control targets.

Other criteria for grouping hosts, beyond that of shared sensitivity to *P. syringae*, might be informative. Groupings based on the availability of ports of entry may provide a clear picture on the gradient of sensitivity to *P. syringae* infection. To this aim, two major groups of woody plants (deciduous and evergreen) can be classified in different groups. For example, plants with a very high, moderate and low stomatal and lenticellular density on leaves and lignified tissues, respectively. Likewise, on the basis of the time required for suberization of leaf scar followed by leaf fall, a further subgrouping in plants with a high, moderate, and low rate of suberization is needed. Such groupings of woody plants might allow us to investigate whether there is a correlation between the ports of entry and the rate of infection thereby allowing breeders to find further solutions.

Genetic diversity can be deployed at spatial scales down to the level of orchards. In the field, large-scale intensive cultivation of vegetatively propagated clonal plants leads to an increased vulnerability of plants to pathogen attacks. Only the promotion of genetic mixtures within a field can ensure a greater stability of plant resistance in the long term. With this mind, the introduction of either different plant cultivars of the same species or different species within the same field (intercropping) may significantly reduce the risk of severe infection. The cultivation of different landraces of the same species might present some disadvantages such as unsynchronized phenological phases among the genotypes with different flowering, fruiting, and ripening time. However, this technique may be economically advantageous in the long term if it reduces the severity of infections that could otherwise lead to drastic yield losses.

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