



Review of *Pseudomonas* species causing bacterial canker of *Prunus* species with emphasis on sweet cherry (*Prunus avium*) in New Zealand

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Abstract Bacterial canker of cherry is a major constraint to stone fruit production worldwide, including New Zealand. Six pathovars of the bacterium *Pseudomonas syringae* are known to cause bacterial canker on *Prunus* species. From those six pathovars, *P. s. pv. syringae* (Pss), *P. s. pv. morsprunorum* race 1 (Psm1) and *P. s. pv. persicae* have been reported as pathogens of *Prunus* species in New Zealand, and Pss and Psm1 on sweet cherry (*Pr. avium*). On sweet cherry, extensive development of cankers and gummosis is usually observed, particularly during late winter and spring, with the progressive decline of trees resulting from the death of branches or death of the plant. In young orchards in New Zealand, losses of 20–50%, and sometimes the removal of entire cherry blocks have been observed. This review reports on the current knowledge of *P. syringae*

pathovars causing bacterial canker of *Prunus* species, with specific focus on sweet cherry in New Zealand, and covers their identification, the infection process, virulence associated factors, epidemiology, symptoms, and management strategies.

Keywords Bacterial blast · Cherry canker · *Prunus* · Cherry · *Pseudomonas*

Introduction

The genus *Prunus*, in the *Rosaceae* family contains approximately 430 species and includes some well-known commercially grown species such as cherry (*P. avium*), sour cherry (*P. cerasus*), peach (*P. persica*), apricot (*P. armeniaca*) and plum (*P. domestica*), which are cultivated for their fruit and ornamental value (Niklas, 1997). Within the genus *Prunus*, cherries are members of the subfamily *Prunoideae* and belong to the *Cerasus* subgenus (Webster & Looney, 1996). The sweet cherry is *Pr. avium* and the sour or tart cherry is *P. cerasus* L. The sweet cherry is a deciduous tree, vigorous and with strong apical dominance. It is thought to have originated close to the Caspian and Black Seas of Asia Minor and later spread throughout Europe and other regions (Kappel et al., 2012).

The New Zealand summer fruit (the term used in New Zealand to collectively describe cherries, apricots, peaches, nectarines and plums) industry comprises

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approximately 2350 ha of orchards corresponding to an estimated 280 growers (Horticulture New Zealand, 2019; New Zealand Horticulture Export Authority, 2021). Central Otago and Hawke's Bay are the main growing areas, with 59.0 and 31.0% of the planted area, respectively. Other regions, which include, Marlborough/Nelson, Auckland and Canterbury, account for the remaining 10.0% area (Summerfruit New Zealand, 2021). Cherry orchards comprise 38.2% of the summer fruit planted area, while 19.4% correspond to apricots, 16.3% to peaches, 13.3% to nectarines and 12.6% to plums. Central Otago is the main growing region for cherries and apricots, with 83.3 and 67.8% of the total area, respectively. Marlborough/Nelson contributes 6.7% of the total cherry area. Hawke's Bay is the main region for plum production, while only a small proportion of cherries are cultivated there. Seventy percent of all summer fruit produced in New Zealand is consumed within the domestic market. The remainder is exported, with Taiwan, Australia and the USA being the predominant destinations and cherries and apricots being the main export crops (New Zealand Horticulture Export Authority, 2021). Among the factors limiting the production of cherries are pest and diseases, with bacterial canker caused by *P. syringae* considered as one of the main issues for the stone fruit industry in New Zealand and worldwide.

This review provides an overview of the current understanding of *P. syringae* pathovars that cause bacterial canker in *Prunus* species, particularly in sweet cherry in New Zealand. The review includes aspects such as pathogen identification, infection mechanisms, virulence factors, disease epidemiology, symptomatology and strategies for disease management. This review significantly adds to the literature by elucidating recent insights gained from the New Zealand's context and therefore contributing valuable information to the wider scientific community and fostering knowledge exchange.

The *Pseudomonas syringae* species complex

Pseudomonas syringae, a Gram-negative gammaproteobacterium, is a ubiquitous group of bacteria able to colonise a variety of environments to live as saprophytes or parasites. It was first reported as a pathogen of lilac in 1902 (Van Hall, 1902) and subsequently shown to cause disease on more than 180

hosts (Berge et al., 2014). *Pseudomonas syringae* is a phytopathogenic bacterium that is currently divided into more than 60 pathovars known as the '*P. syringae* species complex' (Bull et al., 2010; Gardan et al., 1997, 1999; Xin et al., 2018).

Classification and identification of *Pseudomonas syringae*

The classification of strains within *P. syringae* was initially based on phenotypic characteristics, revealed through physiological and biochemical tests (Lelliott et al., 1966). Subsequent studies showed that tests based on phenotypic characteristics, including the LOPAT tests, were insufficient to differentiate closely related pathogens within the *P. syringae* taxonomic group (Janse et al., 1996; Sands et al., 1970; Young & Triggs, 1994). This gave rise to the concept of *P. syringae* as a phylogenetic complex of strains, the '*P. syringae* species complex' (*P. syringae* sensu lato), which consisted of a single species comprising pathogens capable of infecting a limited range of hosts (Young, 2010). Given the limited ability to separate strains based on phenotypic characteristics, sub-populations of strains within the *P. syringae* species complex were classified as pathovars on the basis of pathogenicity to one or more plant hosts. For example, *P. s.* pv. *pisi* is a pathogen restricted to peas (Hollaway et al., 2007), while *P. s.* pv. *persicae* affects peach, nectarine and Japanese plum (Young, 1988). Currently, the *P. syringae* species complex is divided into more than 60 pathovars (Baltus et al., 2017; Bull et al., 2010; Gomila et al., 2017; Young, 2010) that cause disease to different host plants, including important cultivated plant species (Agrios, 2005; Green et al., 2009; Vanneste et al., 2013; Young, 2010). The relevance of the pathovar approach, however, has been questioned because it is based on limited cross-pathogenicity tests. Most tests have been on cultivated plants, which does not capture the diversity of *P. syringae* associated with non-cultivated plants or from the environment (Hirano & Upper, 1990; Morris et al., 2008, 2019; Tyson et al., 2012).

Molecular typing of microorganisms offers higher discriminatory power than conventional phenotypical approaches and allows the differentiation of strains below the species or subspecies level, down to strain (Ruppitsch, 2016). Molecular typing methods are based on the differences between genomes,

particularly single nucleotide polymorphisms (SNP) and insertions and deletions (INDELS). PCR-based typing methods include the targeting of SNPs in conserved genes (such as the 16S rRNA gene), accessory genes that encode for production of toxins, virulence or antibiotic resistance (Bender & Cooksey, 1986; Bultreys & Gheysen, 1999; Gilbert et al., 2009; Van Neste & Voyle, 2003), or repetitive element palindromic regions (rep-PCR), with the resolution of the molecular typing depending on the method (Ruppitsch, 2016). DNA-sequence based typing methods are considered to provide a higher resolution than PCR-based typing methods when studying the diversity of the *P. syringae* species complex. For example, DNA-DNA hybridization defined nine genomospecies within the *P. syringae* species complex (Gardan et al. (1999), which were subsequently confirmed by genome sequencing studies (Berge et al., 2014; Bull et al., 2011). The DNA hybridization approach is limited by the expertise required, the inherently high associated costs and the time to undertake the test. As a result, Multilocus Sequence Analysis (MLSA) and Multilocus Sequence Typing (MLST) have increasingly been used to identify and classify bacterial strains and to establish the relatedness among bacterial strains, including *Pseudomonas* strains (Almeida et al., 2010; Gomila et al., 2017; Maiden et al., 1998; Sarkar & Guttman, 2004). MLSA and MLST use the DNA sequences of selected housekeeping genes (genes highly conserved in the bacterial core genome associated with basic metabolic function) from the bacterial genomes to compare related bacteria (Sarkar & Guttman, 2004; Urwin & Maiden, 2003). The number of loci selected for MLST/MLSA studies depends on the resolution required to provide a reliable identification of organisms within the target group and requires the selected loci to be sufficiently diverse in nucleotide composition (unlike the 16S rRNA gene) to successfully identify variants of closely related bacteria (e.g. below the species level) (Maiden 2006). The first studies of this type used approximately seven loci of approximately 400 to 600 base pairs (bp). In MLST, allele sequences at each locus were assigned unique numbers. The allele number combination resulted in a sequence type (ST), however, to conduct MLSA, only the number of nucleotide differences between alleles is considered. Using MLST of seven housekeeping genes, Sarkar and Guttman (2004)

divided the *P. syringae* species complex into four major clades. Parkinson et al. (2011) described seven phylogroups (PG) based on the phylogeny using the sequence of the *rpoD* housekeeping gene. More recently, using MLST, Berge et al. (2014) grouped strains within the *P. syringae* species complex into 13 phylogroups and 23 subgroups, including strains obtained from crops and those isolated from non-cropping areas. With the inception of whole-genome sequencing and the increasing number of bacterial genomes available from public databases, the *P. syringae* species complex has been most recently divided into 19 phylogenomic groups (Gomila et al., 2017). Comparisons of whole-genome sequences of between 12 and 18 strains have been used to examine the diversity of *Pseudomonas* from specific hosts such as *Prunus* (Hulin et al., 2018; Ruinelli et al., 2019). These studies have shown good correlation with the previously described phylogroups (Berge et al., 2014).

Epiphytic and endophytic phases of *Pseudomonas syringae*

Pseudomonas syringae has been extensively studied and has served as a model system to study pathogenicity, ecology and epidemiology of bacteria (Hirano & Upper, 2000; Mansfield et al., 2012; Hulin et al., 2018). An epiphytic and endophytic phase both occur on the host. During the epiphytic phase the bacteria live on the plant surface. On leaves, the cuticle and epidermis provide structural barriers against the entry of bacterial pathogens. To gain access into the foliar/herbaceous host tissues, *P. syringae* uses natural avenues such as stomata, hydathodes, lenticels and trichomes or wounds (e.g. those caused by frost, hail, insects or pruning) (Lindow & Brandl, 2003; Agrios, 2005). Woody tissue offers additional barriers to entry. In this case, wounds (e.g., caused by frost, hail, insect damage or pruning) are readily utilised for entry, while natural openings such as lenticels are not always exploited by *P. syringae* pathogens of woody plants (Lamichhane et al., 2014). Once the pathogen has gained entry into the plant tissue it colonises the intercellular apoplast (the space between the mesophyll cells). Disease occurs once bacteria have entered and multiplied within the apoplast.

Virulence factors influencing colonisation and infection by *Pseudomonas syringae*

Before and after entry into the plant tissue, *P. syringae* needs to overcome the plant defense system, including constitutive preformed and induced defense responses (Doughari, 2015). For example, the plant surface poses a constitutive preformed physical barrier limiting pathogen invasion. In addition, the first layer of plant defense involves pattern-recognition receptors (PRRs), which recognise specific conserved microbial molecules called pathogen-associated molecular patterns (PAMPs), such as flagellin (Arnold & Preston, 2019; Jones & Dangl, 2006). Perception of PAMPs by the plant results in pattern triggered immunity (PTI), leading, for example, to the reinforcement of the plant cell walls by production of callose (Nürnberg et al., 2004; Tsuda & Katagiri, 2010). A number of virulence-associated factors are known in *P. syringae* to suppress plant defenses, some of them being necessary for infection and others increasing the severity of symptoms. It is recognised that *P. syringae* uses three major virulence-associated systems during the establishment of host–pathogen interactions: secretion of effector proteins through the type III secretion system (T3SS), production of phytotoxins and ice nucleation activity (INA) (Kennelly et al., 2007). Within the different classification schemes (e.g. into phylogroups), these virulence factors have been used to further characterise *P. syringae* strains and to relate to the symptoms they induce in host tissue (Gilbert et al., 2010; Giovanardi et al., 2018; Parisi et al., 2019; Ruinelli et al., 2019).

Ice nucleation activity

Ice nucleation activity (INA) is a property exhibited by gram-negative proteobacteria such as *P. syringae*, *Xanthomonas* spp., *Erwinia* spp. and *Pantoea* spp. This property is the result of the production of a protein, InaZ, that can act as an ice nucleus, catalysing ice formation within plant tissues at temperatures just below 0°C (Lindow, 1983b). Ice nucleation active (Ice+) bacteria are able to reduce the supercooling ability of plants resulting in frost-injury. Ice formed in, or on, frost-sensitive plants causes mechanical disruption of cell membranes, resulting in frost damage (Burke et al., 1976) and predisposing frost-sensitive plants to infection by *P. syringae* (Kennelly et al., 2007; Lindow,

1983b; Renick et al., 2008). Ice formation results in tissue damage and subsequent nutrient and water leakage from the host cells that can be used by the pathogen and facilitates its entry into the host tissue. Frost alone would contribute to increased disease severity by providing wounds that can then be colonised by *Pseudomonas* strains. Ice+Pss strains may play a more active role and contribute to frost damage by limiting the ability of the host tissue to supercool and avoid ice formation at temperatures just below 0°C, resulting in tissue damage (Lindow, 1983b; Montesinos & Vilardell, 1991). In a study of 150 *P. syringae* strains in New Zealand, 97% (145/150) were Ice+ (Marroñi, 2021). In Central Otago orchards, frequent frosts between autumn and spring provide numerous opportunities for *P. s. pv. syringae* (Pss) and *P. s. pv. morsprunorum* (Psm) race 1 (Psm1) colonisation of damaged host tissue, but the presence of Ice+Pss strains and their prevalence in Central Otago orchards (Marroñi, 2021) may help to exacerbate the damage and subsequent disease development.

Type III secretion system

The T3SS is a protein delivery organelle found in gram-negative bacterial pathogens that injects Type III effectors (T3E) directly into the host cell cytoplasm to suppress PTI (Coburn et al., 2007; Galán & Wolf-Watz, 2006; Jin et al., 2003). Effectors can be directly or indirectly recognised (through plant immune receptors such as nucleotide-binding domain and leucine-rich repeat region (NLR-containing proteins) by proteins encoded by resistance (*R*) genes triggering effector-triggered immunity (ETI) (Arnold & Preston, 2019; Doughari, 2015; Naveed et al., 2020). Successful ETI usually translate into disease resistance and programmed cell death (hypersensitive response, HR) (Jones & Dangl, 2006). If PTI is suppressed by one or more pathogen effectors, pathogen infection of the host occurs and in the absence of effective *R* genes, ETI is overcome, ultimately leading to effector triggered susceptibility (ETS) (Arnold & Preston, 2019; Naveed et al., 2020). The T3SS is essential for pathogenicity and its disruption (e.g. by effector gene deletion) renders pathogenic *Pseudomonas* strains avirulent (Lindeberg et al., 2009; Xin et al., 2018). The particular range of effectors produced by a specific *P. syringae* pathovar (or even strain) is thought to influence its host range or host

specificity (Fouts et al., 2003). In New Zealand, for both Pss and Psm1 strains, a functional T3SS in these strains was inferred (Xin et al., 2018) given the positive hypersensitivity reaction on a study using inoculated tobacco leaves and by the symptoms on cherry tissue (Marroni, 2021).

Phytotoxins

Some *P. syringae* strains can produce phytotoxins to overcome the plant defence system. Unlike T3SS, phytotoxins are not essential for pathogenicity, but they may contribute to increased disease severity, *in planta* spread or habitat colonisation (Bender et al., 1999; Volksch & Weingart, 1998). Coronatine is a chlorosis-inducing toxin produced by strains of Psm, while syringomycins are produced by *P. syringae* strains (Alarcon-Chaidez et al., 1999; Bender et al., 1991; Gilbert et al., 2010). The detection by the host of bacterial PAMPs stimulates immune signalling in stomatal guard cells, which leads to salicylic acid (SA) signalling and stomatal closure (Xin et al., 2018). Coronatine mimics the plant hormone jasmonic acid, and through antagonism with the SA pathway, can inhibit its accumulation in stomatal guard cells, suppressing stomatal closure and facilitating *P. syringae* entry into the apoplast (Melotto et al., 2008). Syringomycin is a necrosis-inducing toxin that induces pore formation on plant membranes leading to leaking of nutrients, and acts as a biosurfactant and increased wetness of the plant surface, which enhance bacterial movement (Xin et al., 2018). It has been established that Psm1 relies on a larger set of effectors and less on production of toxins to infect and colonise their host. In contrast, Pss is reported to possess a small set of effectors that is compensated for by the production of phytotoxins (Baltrus et al., 2011; Xin et al., 2018). In a study in commercial orchards in New Zealand (Central Otago), production of syringomycin was common (88%) among Pss strains, while the vast majority of Psm1 strains studied did not produce coronatine (Marroni, 2021). In the same study, Pss was the prevalent taxa compared with Psm1. Lack of coronatine may render Psm1 strains less competitive in plant tissue (Bender et al., 1987) in the presence of Pss syringomycin-producing strains, which may occur in the natural environment.

Pseudomonas syringae pathovars that cause diseases on *Prunus* species

Six pathovars of *P. syringae* and three other *P. spp.*, within phylogroups (PG) one, PG2, PG3 and PG7, as defined by Berge et al. (2014), are known to cause disease on *Prunus* species (Table 1). In peach, nectarines and Japanese plum, *P. s. pv. persicae* causes decline symptoms and dieback (Young, 1987a). Bacterial canker in wild cherries is caused by *P. s. pv. avii* in Europe (Ménard et al., 2003) while *P. s. pv. cesasicola* causes bacterial gall on sweet cherry in East Asia (Kamunten et al., 2000). Three pathovars of *P. syringae* can cause bacterial canker of cherry: Pss, Psm1 and Psm race 2 (Psm2). From the six pathovars of *P. syringae* known to cause disease on *Prunus* species (Table 1), only Pss, Psm1 and *P. s. pv. persicae* have been reported as pathogens of *Prunus* species in New Zealand, and only Pss and Psm1 on cherry (Wilkie et al. 1973; Young, 1988; Vanneste, 2011; Marroni 2014; Visnovsky et al., 2019).

Pathovars causing bacterial canker of cherry

Of the three pathovars that cause bacterial canker of cherry, Pss is a genetically heterogeneous pathovar that can infect more than 180 host species including herbaceous and fruit crops (Bradbury, 1986; Hulin et al., 2020). Pss has been reported from sweet cherry grown in most cherry growing regions worldwide, including Europe, the Americas, South Africa, Asia and Australasia (Freigoun & Crosse, 1975; Gilbert et al., 2009; Giovanardi et al., 2018; Hulin et al., 2020). Within the *morsprunorum* pathovar, two highly homogeneous races, race 1 (Psm1) and race 2 (Psm2), were initially described based on their differing physiological and pathological characteristics (Freigoun & Crosse, 1975; Ménard et al., 2003; Vicente & Roberts, 2007; Vicente et al., 2004). Later genetic studies showed that Psm1 and Psm2 were distantly related, and they should not be considered as different races of the same pathovar (Bultreys & Kaluzna, 2010). More recently it has been proposed that Psm1 and Psm2 are reclassified as members of the species *P. amygdali* and *P. avellanae*, respectively (Bull et al., 2010; Gomila et al., 2017; Hulin et al., 2018).

Psm1 has been reported from Europe, Central America, South Africa, North America and Australasia

Table 1 The classification of *Pseudomonas* spp. known to cause disease on *Prunus* species and the disease they cause in the context of the different typing schemes and their occurrence in New Zealand

Pseudomonad	<i>Prunus</i> host	Disease caused	LOPAT	G	PG	In NZ	Reference
<i>Pseudomonas syringae</i> pv. <i>syringae</i>	Sweet cherry, sour cherry, European plum, peach, apricot and almond	bacterial canker/blast	1A	1	2b 2d	Yes	Wilson (1939)
<i>P. s.</i> pv. <i>morsprunorum</i> race 1	Sweet cherry, sour cherry, European plum, peach, apricot and almond	bacterial canker/blast	1A	2	3	Yes	Wormald (1932)
<i>P. s.</i> pv. <i>morsprunorum</i> race 2	Sweet cherry, sour cherry, European plum, apricot	bacterial canker/blast	1A	3	1b	No	Freigoun and Crosse (1975)
<i>P.s.</i> pv. <i>persicae</i>	Peach, nectarine and European and Japanese plum	bacterial decline/bacterial canker/dieback	1A	3	1a	Yes	Young (1987a)
<i>P. s.</i> pv. <i>avii</i>	Cherry for wood production	bacterial canker	1A	3	1a	No	Ménard et al. (2003)
<i>P. s.</i> pv. <i>cerasicola</i>	Sweet cherry, apricot, wild Himalayan cherry, Japanese cherry, North Japanese hill cherry, Yoshino cherry, <i>Prunus leveilleana</i>	bacterial gall	2	2	3	No	Kamiunten et al. (2000)
<i>P. amygdali</i>	Almond	hyperplastic canker	N/A	2	3	No	Psallidas (1997)
<i>P. cerasi</i>	Sweet cherry and sour cherry		N/A	N/A	2a	No	Kaluźna et al. (2016b)
<i>P. viridiflava</i>	Sweet cherry, sour cherry, European plum apricot and peach	apoplexy/canker/shoot necrosis	1A, 1B or 2	6	7	Yes*	Scortichini and Morone (1997); Harzallah et al. (2004); Parisi et al. (2019); Bophela et al. (2020)

Genomospecies (G) according to (Gardan et al., 1999); Phylogroup (PG) according to (Berge et al., 2014); NA, not applicable; *, *P. viridiflava* is present in New Zealand but has not been reported as a pathogen of *Prunus*

(Bultreys & Gheysen, 2003; Crosse & Garrett, 1966; Gilbert et al., 2009; Giovanardi et al., 2018; Kaluzna et al., 2010; Latorre & Jones, 1979b; Visnovsky et al., 2019). Psm2 has been reported from Europe and South Africa (Freigoun & Crosse, 1975; Giovanardi et al., 2018; Hulin et al., 2020; Roos & Hattingh, 1986b). Strains of Pss are reported to be the more virulent on sweet cherry, peach and apricots (Crosse & Garrett, 1966; Jones, 1971; Kennelly et al., 2007) while Psm2 seems to be more important on sour cherry and European plum (Kennelly et al., 2007; Latorre & Jones, 1979b). In the UK, bacterial canker of sweet cherry is considered to be caused mainly by Psm1 (Burkowicz

& Rudolph, 1994; Crosse, 1955; Garrett et al., 1966) while in other European countries, South Africa, the USA and New Zealand the disease on sweet and sour cherry has been associated with Pss and Psm1 (Latorre & Jones, 1979b; Renick et al., 2008; Roos & Hattingh, 1986b; Vanneste, 2011).

In New Zealand, *P. syringae* was initially recognized in 1954 as a pathogen causing losses to apricot, cherry, nectarine, peach, and European and Japanese plum and reported as occurring in both the South Island (Central Otago) and North Island (Auckland and Hawke's Bay)(Dye, 1954). However, “die-back, gummosis and sour sap”, which are symptoms now

linked to *P. syringae* in *Prunus* species, had been recognized as conditions in New Zealand prior to this report (Cunningham, 1925). Currently, national bacterial culture collections hold *Pseudomonas syringae* strains isolated from *Prunus* hosts from North and South Islands of New Zealand. However, because sweet cherry production is concentrated in Central Otago, most historical and current cherry isolates have been collected from this region. In 2009, bacteria were isolated from sweet cherry trees displaying gummosis or collapse symptoms across four orchards in Central Otago and one nursery in the North Island. Using biochemical and genetic (BOX-PCR) tests, the primary pathogens linked to these symptoms were identified as Pss and Psm1 (Vanneste & Yu, 2009). Subsequent MLST of two housekeeping genes (*gltA* and *gyrB*) confirmed that isolates recovered from cherry trees experiencing sudden decline were associated with Psm1. Furthermore, analysis of strains isolated in 1970 from Central Otago's sweet cherry trees exhibiting similar symptoms confirmed that Psm1 had been present in New Zealand at least several decades earlier (Vanneste, 2011). In a recent study of the *P. syringae* species complex in cherry orchards in New Zealand, 250 putative *Pseudomonas* isolates were obtained from symptomatic and asymptomatic cherry tissue across 23 commercial orchards (Marroni et al., 2023). By analyzing the *gltA* gene sequence and performing MLSA of four housekeeping genes, the isolates were classified into three taxonomic groups. The two main taxonomic groups were Pss and Psm1, PG2 and PG3, respectively (Berge et al., 2014). The third group comprised nonpathogenic strains classified as *Pseudomonas* spp. Strains of Psm1 formed a monophyletic group, representing an almost clonal population. There was more variation detected within strains of Pss, although they were restricted to group PG2d. Nonpathogenic *Pseudomonas* spp. and pathogenic Pss and Psm1 strains coexisted in the same orchard. It was concluded that Pss is the predominant pathovar in Central Otago. (Marroni et al., 2023).

Bacterial canker of cherry

Disease economic importance

Bacterial canker of sweet cherry is one of the most important bacterial diseases affecting cherry orchards

worldwide (Bophela et al., 2020; Hulin et al., 2020; Kennelly et al., 2007; Ruinelli et al., 2019; Vicente et al., 2004). The disease causes direct losses by reducing plant vigour and growth as well as the number of buds, blossoms, branches and plants that are lost, which in turn reduces yields from orchards.

Severity of the disease seems to be greatest in young orchards (Latorre et al., 1985; Puławska et al., 2017; Roos & Hattingh, 1986b). Tree losses of up to 75% have been reported in young orchards in USA where conditions have been favourable for disease development compared to between 10 and 20% under normal conditions (Spotts et al., 1990, 2010b). In young orchards in New Zealand, losses of 20–50% have been observed, and sometimes the removal of entire cherry blocks has been observed (Marroni et al., 2023).

Disease symptoms

Symptoms of bacterial canker occur on all above-ground organs of the tree and include bud necrosis, blossom blast, cankers in branches and the trunk, gummosis, death of main branches and total tree collapse, particularly during the first 1–3 years after planting (Kennelly et al., 2007; Lamichhane et al., 2014) (Fig. 1). In spring, dormant buds fail to break and there is production of gum exudates at their base. When the bark is removed, the underlying tissue appears brown. Infection in spring can also cause blossom blast. Leaf symptoms can occur early in the leaf development as reddish circular spots surrounded by chlorotic rings. These spots expand and eventually drop out of leaves causing the 'shot hole' symptom. On sweet cherry, leaf spot also occurs on the margins of leaves, resulting in a curling of the leaves. During the growing season, fruit spot symptoms can occur on susceptible varieties and appear as water-soaked lesions that develop a chocolate-brown colour. Cankers develop in the trunk and main branches, which may exude a substantial amount of gum. The trunk or main branches may subsequently die leading to loss of tree structure and whole trees within the orchard (Kennelly et al., 2007).

Disease cycle

The proposed disease cycles of Pss and Psm1 on cherry are similar. The primary inoculum that

Fig. 1 Symptoms of bacterial canker on sweet cherry. **A** buds that failed to break in spring with production of gum exudates and browning of underlying tissue; **B** blossom blast can also occur in spring particularly after a frost or following periods of wet and cold weather; **C** bacterial leaf spot; **D** necrotic lesions on fruits are sometimes observed in spring; **E** cankers on leader branch of a mature tree (red arrow); **F** loss of the leader branch due to bacterial canker on a mature tree; **G** whole trees are sometimes removed in mature orchards due to bacterial canker; **H** systemic invasion and collapse of a 2-year old tree; **I** removal of trees in a newly established block due to bacterial canker. Photo credits: **A** and **B**, Ian Harvey; **C** to **I**, Virginia Marroni



initiates the disease cycle in spring originates in cankers and dormant buds (Kennelly et al., 2007; Sundin et al., 1988). An epiphytic phase occurs throughout the growing season, with the bacteria growing on the surface of apparently healthy blossoms and leaves (Puławska et al., 2017; Sundin et al., 1988). Blossom blast can occur in spring, particularly during periods of cool, wet weather or after frost events, with small cankers subsequently forming at the base of the bud or fruit spur (Kennelly et al., 2007; Puławska et al., 2017). Leaf spotting can also occur in susceptible cultivars in spring. In summer, epiphytic populations often decline to undetectable levels and increase again in autumn with a drop in temperature and an increase in rainfall (Crosse, 1957, 1966; Sundin et al., 1988). Infection of trees is reported to occur through tissue damaged by frost, pruning cuts, insects, or at the site of the leaf scar (Dye, 1954; Latorre & Jones, 1979a; Spotts et al., 2010b; Sundin et al., 1988), with systemic invasion of plant tissue demonstrated (Roos & Hattingh, 1987). During the growing season, bacteria can enter the host tissue from leaves (through

stomata) and petioles, and establish an important source of inoculum inside asymptomatic tissue. Once the bacterium has entered the plant (e.g. through the leaf scar, pruning wounds, stomata), it moves through the intercellular spaces and progresses into the bark and the medullary rays of the xylem and phloem. The bacteria can break down the parenchyma cells resulting in the development of lysogenic cavities filled with bacteria (Hattingh et al., 1989; Roos & Hattingh, 1987) and the development of cankers. Affected areas are slightly sunken and usually show a darker brown colour compared with the rest of the bark (Puławska et al., 2017). Cankers develop faster in autumn, before trees are in dormancy and prior to the occurrence of low winter temperatures. Canker development is slow in winter but increases rapidly between the end of winter and the beginning of spring (Agrios, 2005; Crosse & Garrett, 1966).

Dormant buds are considered a major overwintering site for Pss and Psm1 (Crosse, 1956; Puławska et al., 2017; Sundin et al., 1988). The leaf scar is considered a major route for infection of buds in

autumn, particularly for Psm1 (Crosse, 1956; Sundin et al., 1988). In South Africa, Pss and Psm1 are considered to reach axillary buds by systemic spread well before leaf fall occurs (Hattingh et al., 1989). In cherry orchards in Oregon (the USA), infection of buds is thought to originate at the base of the outer bud scales and to then spread throughout the base of the bud (Cameron, 1962) rather than through the leaf scar. In New Zealand, the colonisation of cherry buds by Pss and Psm1 in Central Otago orchards occurs mainly through external sources soon after buds form (December, Southern Hemisphere) and long before leaf fall (Marroni et al., 2022).

Numerous studies have shown that *P. syringae* can live as an epiphyte on weeds, grasses and other plants and can be isolated during the growing season (Baca & Moore, 1987; Latorre & Jones, 1979a; Ross & Hattingh, 1986). Although the significance of weeds as overwintering sites for *Pseudomonas* pathovars is unknown, some authors have speculated that pathogenic *Pseudomonas* may spread from weeds by rain splash and infect stone fruit trees as a resident population (Roos & Hattingh, 1986c). *Pseudomonas syringae* pathovars can move from site to site and from cankers or weeds to the new tree foliage by wind or rain (Crosse, 1966; Malvick & Moore, 1988). Additionally, long distance dispersal can occur through movement of nursery stock. Roos and Hattingh (1986a) showed that a significant proportion of asymptomatic budwood from stone fruit trees carried pathogenic *Pseudomonas*. Indeed, nursery trees grafted with asymptomatic budwood may not show symptoms until conducive field conditions occur.

Host predisposing factors

Bacterial canker development on *Prunus* species is affected by predisposing factors that affect host susceptibility. All wounds or injuries to trees can provide avenues for infection. Frost injury is a well-known predisposing factor for the development of bacterial canker in *Prunus* species (Sobiczewski, 1992; Weaver, 1978), with the association between *P. syringae* and frost damage, in the initiation of infection, being well documented (Siile & Seemüller, 1987; Kennelly et al., 2007). Frost events may cause severe damage and result in systemic infection of trees by *P. syringae* followed by canker formation (Kennelly et al., 2007). Sandy and very clayey soils as well as soils

characterised by low calcium content, can enhance the sensitivity of apricot and peach trees to Pss (Scortichini, 2010). Overseas, plant pathogenic nematodes have been associated with increased disease severity, however this association has not been proven in New Zealand (Young, 1987b).

Management of bacterial canker in orchards

Chemical control

Pseudomonas diseases of fruit trees are difficult to control. The bacteria can live within plant tissue, making it challenging for chemical products to reach their target. Chemical options for control of bacterial canker are limited. For the control of bacterial canker of cherry in New Zealand and overseas, copper-based compounds are generally applied in orchards at leaf fall in autumn to reduce leaf scar infection, and during winter dormancy before bud break (Kennelly et al., 2007; Puławska et al., 2017; Young, 1987b). The main copper-based compounds applied in orchards are copper oxide, copper oxychloride and copper hydroxide (Puławska et al., 2017). The usefulness of copper is limited by its lack of systemic activity and the emergence of copper-resistant strains, phytotoxicity and environmental concerns (Kennelly et al., 2007). Recently, the use of copper-based products has been restricted across Europe (Hulin et al., 2018). Resistance to streptomycin, which is occasionally used in New Zealand cherry orchards, has been reported in New Zealand (Vanneste et al., 2005) and overseas (Sundin & Bender, 1993). Due to the limitations regarding the commonly used bactericides, other options for control for *Pseudomonas* pathogens are being explored or developed. Fosetyl-Al, an organophosphorous compound, is registered as a bactericide in Belgium and other countries. Bultreys et al. (2018) investigated its potential for the control of flower blast in pear, thought to be associated with *P. syringae*, and determined that Fosetyl-Al applications after flowering prevented the colonisation by *P. syringae* on the new season's buds (to open the following spring). Fosetyl-Al has also been used for control of *P. s. pv. actinidiae* in kiwifruit (Monchiero et al., 2015).

Biological control

Biological control of *P. syringae* on woody or annual plants has focused on the use of bacterial antagonists to prevent or replace the increase of *P. syringae* Ice+ populations (Lindemann & Suslow, 1987; Lindow, 1983a, 1995). Approaches included the use of chemically mutated *P. syringae* Ice[−] strains and of naturally occurring Ice[−] bacterial antagonists, which significantly reduced the populations of Ice+ *P. syringae* on the crop. However, it is thought that in some of the studied hosts (such as *Prunus* species) there are intrinsic INA factors within the plant tissue independent from *P. syringae* Ice+ populations, which limits the use of this approach (Lindow, 1990, 1995). The use of Double Nickel 55 (*Bacillus amyloliquefaciens* D747) and Serenade (*Bacillus subtilis* QST 413) have been proposed but their effectiveness is at best moderate (Puławska et al., 2017). Bacteriophages have also been investigated as potential natural and sustainable tools against *P. syringae* on cherry and in other hosts (Di Lallo et al., 2014; James, 2015; Rabiey et al., 2020).

Disease resistance

There are currently no cherry cultivars fully resistant to *P. syringae* pathovars. Breeding efforts are slowed down by the diversity of bacteria causing the disease (Hulin et al., 2020; Kappel et al., 2012). Cultivars ‘Merla’, ‘Mermat’, ‘Merpet’, ‘Vittoria’, ‘Rigikirsche’, ‘Heidegger’, and ‘Schauenburger’ are reported to have moderate to high resistance to Psm (Kappel et al., 2012). Cherry cultivars do differ in their susceptibility to bacterial canker. The cultivars ‘Rainier’ and ‘Regina’ were reported as being more resistant than ‘Sweetheart’ and ‘Bing’ (Spotts et al., 2010a). Cultivar ‘Merton Glory’ exhibited resistance to all strains of Pss, Psm1 and Psm2, compared with ‘Napoleon’, ‘Roundel’ and ‘Van’ (Hulin et al., 2018).

The choice of rootstock can affect cultivar susceptibility to bacterial canker. The main cherry rootstocks used in commercial cherry cultivation worldwide are seedlings or clonal selections of *P. avium* known as Mazzard and Mahaleb (Kappel et al., 2012; Ogawa et al., 1995; Webster, 1980; Webster & Looney, 1996). Mahaleb is considered to be tolerant, while Mazzard is considered susceptible. The rootstock F/12-1 is a clonal selection of *P. avium* released by East Malling

Experimental Station and is reported as moderately tolerant to Pss, while the semi-dwarfing rootstock ‘Colt’, a hybrid of *P. avium* x *P. pseudocerasus* is considered resistant to Psm (Cameron, 1971; Webster, 1980, 1992). In general, the semi-dwarfing rootstock ‘Colt’ has replaced the canker-tolerant but very vigorous F/12-1 rootstock (Garrett, 1986). ‘Colt’ is the main rootstock used in commercial cherry orchards in New Zealand, where both Pss and Psm1 can cause bacterial canker. Tolerance of this rootstock, which was tested in the UK, has only been established for Psm1 as it is the main pathovar of concern in the UK.

The mechanisms underlying *Pseudomonas* resistance in *Prunus* species are not fully understood, but progress is being made in this regard. A study by Omrani et al. (2019) identified quantitative trait loci (QTLs) associated with partial resistance in apricot. The loci contained genes involved in the abscisic acid pathway which may play a role in plant-pathogen interactions. Similar strategies could be applied to sweet cherry. Genetic markers identified in the apricot study could serve as a starting point for sweet cherry breeding programs, contributing to the process of creating more resistant cultivars. A recent study by Hulin et al. (2022), identified several *Prunus* accessions that exhibited partial resistance including to 16 *P. syringae* strains pathogenic to sweet cherry and plum. Progeny from a cross between resistant ornamental *Prunus incisa* and susceptible sweet cherry (*P. avium*) displayed inherited resistance. A resistant wild cherry and *Prunus* accessions from this study have been integrated into breeding efforts to introduce resistance into commercial sweet cherry cultivars.

Cultural practices

Management practices in the orchard can play an important role in reducing disease incidence and severity. The selection of healthy nursery plant material is crucial to prevent the disease in the field. Plants should be free of disease and infected trees should be rejected or removed from the orchard as soon as they show bacterial canker symptoms (Puławska et al., 2017). High importance should be placed on site selection, with orchards located in areas less likely to be affected by frost and slow drying conditions and with optimal soil conditions for growing cherries (Spotts et al., 2010a; Young,

1987b). Pruning time can considerably influence the penetration of the host tissue by the pathogen, particularly when pruning is conducted in autumn or winter (Chandler & Daniell, 1976; Otta & English, 1970). Branches and stems are susceptible to infection between late autumn and early spring, but are highly resistant at all other times of the year (Crosse, 1966). The resistant period overlaps the growing season and is thought to be related to meristematic activity in the host.

Tree diseases caused by *Pseudomonas* spp. are most prevalent in regions with cool climates such as that encountered in Central Otago in New Zealand, with the occurrence of frequent autumn and spring frosts (Hattingh et al., 1989; Kennelly et al., 2007; Scortichini, 2002). The high incidence of the disease observed in New Zealand, with tree losses of up to 50% on newly established orchards, agrees with this observation (Marroni et al., 2021a, 2021b; Young, 1987b). Wounds, such as those originating from frost damage, are a major predisposing factor to the development of bacterial canker through offering entry points for infection. Frost protection is generally achieved in New Zealand through the use of overhead or orchard floor sprinklers or the use of wind machines. One of the main limitations, particularly of overhead sprinklers, is the further dissemination and spread of the bacteria within the orchard canopy. Both overhead and orchard floor sprinklers contribute to the excessive volume of water applied to orchard soil (estimated as 20–30 mm per night for frost control). Serious waterlogging and associated root damage to trees can occur after several heavy consecutive frosts during spring (Young, 1987b). Studies by Marroni et al., (2021a, 2021b), using thermocouples inserted under the bark of cherry trees, showed that the occurrence of cycles of freezing and thawing of the cambium tissue was associated with the development of lesions on the tree trunk. Protection of trunks of young cherry trees with different wrapping materials or with white interior latex paint reduced the incidence of cankers by up to 79% compared with the unprotected control (Marroni et al., 2021a, 2021b). Irrigation and fertilisation at the end of the summer promote late-season growth that is susceptible to low-temperature injury in early winter and may be infected by *Pseudomonas* pathovars. In addition, these practices can reduce cold hardiness, increasing frost damage and disease severity.

Future research

Knowledge of the identity, prevalence and host interaction of *Pseudomonas* strains present in orchards is important to understand the disease epidemiology, which in turn may contribute to the development of better disease control strategies. Research into the co-existence of pathogenic and non-pathogenic strains within orchards/plants/organs and understanding of how *P. spp.* strains from the different PGs interact with the host may help to elucidate their ability to cause symptoms and affect niche colonisation. In addition, studies on how the microbial community as a whole (i.e., the microbiome) influences Pss and Psm infection and development would inform innovative approaches for controlling these pathogens in cherry orchards.

Most of the research worldwide into the epidemiology of bacterial canker of cherry has been based on conventional culturing methods and inoculation studies. These require either biochemical or complicated and time-consuming identification methods (Crosse & Garrett, 1966; Freigoun & Crosse, 1975; Gilbert et al., 2009; Kaluzna et al., 2010; Roos & Hattingh, 1986b, 1988; Vicente & Roberts, 2007). Cost-effective, culture-independent molecular methods to detect and quantify *Pseudomonas* strains for large numbers of field samples for epidemiology studies are desirable. The availability of such methods is particularly important due to the presence of mixed taxa and low population levels of the target species in natural environments. For *Pseudomonas* strains associated with *Prunus* species, molecular tools, such as MLSA and MLST, have mainly been used for identification of host strains, determination of their host associations and establishment of their genetic relatedness (Kaluzna et al., 2010; Kałużna et al., 2016b; Ménard et al., 2003; Visnovsky et al., 2019). Due to reduction in costs, whole-genome sequencing is increasingly being used for the identification of *Pseudomonas* strains. However, for epidemiological studies these methods are laborious. Specific *P. syringae* pathovars primers and qPCR protocols have been developed (Kałużna et al., 2016a) but have not been used for field epidemiological studies. Further development and optimisation of specific qPCR tools to detect and track Pss and Psm1 strains in orchards will allow for such studies in a cost-effective manner.

Summary

Research on bacterial canker of *Prunus* commenced more than 100 years ago (Hulin et al., 2020), yet, this pathogen continues to be a major constraint to the production of cherry worldwide. In New Zealand, bacterial canker threatens the long-term sustainability of the summerfruit industry. Knowledge of the identity, prevalence and host interaction of *Pseudomonas* strains present in orchards is important to understand the disease epidemiology and elucidate control strategies. Novel sequencing and molecular methods have been used in the last 15 years to improve the identification of strains and their tissue association. Differences in disease expression among strains has been observed worldwide and in New Zealand, and may reflect the differential repertoire of virulence associated factors, which in turn can affect niche colonisation, persistence and severity of symptoms (Hulin et al., 2018; Mo & Gross, 1991; Ruinelli et al., 2019; Scholz-Schroeder et al., 2001). Further progress in understanding the epidemiology of bacterial canker in orchards will benefit from the development of new molecular tools, such as specific qPCR, that will allow for cost-effective studies particularly in the presence of mixed taxa and low population levels of the target species in natural environments. Bacterial canker of cherry is difficult to control, and there are limited chemical or biological options available. The use of bacteriophages has been explored but their effectiveness is at best moderate (Puławska et al., 2017; Rabiey et al., 2020). Progress has been made to understand the mechanisms underlying bacterial canker resistance in *Prunus* and identifying sources of resistance on sweet cherry. As the identified resistant accessions are integrated into breeding programmes, partially resistant commercial sweet cherry cultivars should become available. Studies by Marroni et al., (2021a, 2021b) showed that the occurrence of cycles of freezing and thawing of the cambium tissue was associated with the development of lesions on the tree trunk and subsequent colonisation by Pss and Psm1 in New Zealand. Protection of trunks of young cherry trees with different wrapping materials or with white interior latex paint reduced the incidence of cankers (Marroni et al., 2021a, 2021b). Finally, continued research on bacterial canker, aided with new molecular identification and monitoring tools will contribute to a better understanding of the pathosystem and

hopefully lead to the elucidation of much needed management tools.

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Data Availability The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declarations

Conflict of interest The authors declare no conflict of interest.

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References

- Agrios, G. (2005). *Plant Pathology* (5th ed.). Academic Press.
- Alarcon-Chaidez, F. J., Penaloza-Vazquez, A., Ullrich, M., & Bender, C. L. (1999). Characterization of plasmids encoding the phytotoxin coronatine in *Pseudomonas syringae*. *Plasmid*, 42, 210–220.
- Almeida, N. F., Yan, S., Cai, R., Clarke, C. R., Morris, C. E., Schaad, N. W., Schuenzel, E. L., Lacy, G. H., Sun, X., & Jones, J. B. (2010). PAMDB, a multilocus sequence typing and analysis database and website for plant-associated microbes. *Phytopathology*, 100, 208–215.
- Arnold, D. L., & Preston, G. M. (2019). *Pseudomonas syringae*: Enterprising epiphyte and stealthy parasite. *Microbiology*, 165, 251–253.
- Baca, S., & Moore, L. (1987). Variations in *Pseudomonas syringae* isolated from grass species occurring in woody plant nurseries in the Pacific Northwest. *Plant Disease*, 71, 724–726.
- Baltrus, D. A., McCann, H. C., & Guttman, D. S. (2017). Evolution, genomics and epidemiology of *Pseudomonas*

- syringae*: Challenges in bacterial molecular plant pathology. *Molecular Plant Pathology*, 18, 152–168.
- Baltrus, D. A., Nishimura, M. T., Romanchuk, A., Chang, J. H., Mukhtar, M. S., Cherkis, K., Roach, J., Grant, S. R., Jones, C. D., & Dangl, J. L. (2011). Dynamic evolution of pathogenicity revealed by sequencing and comparative genomics of 19 *Pseudomonas syringae* isolates. *PLoS Pathogens*, 7, e1002132.
- Bender, C., Stone, H., Sims, J., & Cooksey, D. (1987). Reduced pathogen fitness of *Pseudomonas syringae* pv. *tomato* Tn5 mutants defective in coronatine production. *Physiological and Molecular Plant Pathology*, 30, 273–283.
- Bender, C. L., Alarcón-Chaidez, F., & Gross, D. C. (1999). *Pseudomonas syringae* phytotoxins: Mode of action, regulation, and biosynthesis by peptide and polyketide synthetases. *Microbiology and Molecular Biology Reviews*, 63, 266–292.
- Bender, C. L., & Cooksey, D. A. (1986). Indigenous plasmids in *Pseudomonas syringae* pv. *tomato*: Conjugative transfer and role in copper resistance. *Journal of Bacteriology*, 165, 534–541.
- Bender, C. L., Young, S. A., & Mitchell, R. E. (1991). Conservation of plasmid DNA-sequences in coronatine-producing pathovars of *Pseudomonas syringae*. *Applied and Environmental Microbiology*, 57, 993–999.
- Berge, O., Monteil, C. L., Bartoli, C., Chandeysson, C., Guilbaud, C., Sands, D. C., & Morris, C. E. (2014). A user's guide to a data base of the diversity of *Pseudomonas syringae* and its application to classifying strains in this phylogenetic complex. *PLoS ONE*, 9, e105547.
- Bophela, K. N., Petersen, Y., Bull, C. T., & Coutinho, T. A. (2020). Identification of *Pseudomonas* isolates associated with bacterial canker of stone fruit trees in the Western Cape, South Africa. *Plant Disease*, 104, 882–892.
- Bradbury, J. (1986). *Pseudomonas syringae* pv. *syringae*. *Guide to plant pathogenic bacteria*, (pp. 175–177) CAB International Mycological Institute, Kew, England.
- Bull, C. T., Clarke, C. R., Cai, R., Vinatzer, B. A., Jardini, T. M., & Koike, S. T. (2011). Multilocus sequence typing of *Pseudomonas syringae* sensu lato confirms previously described genomospecies and permits rapid identification of *P. syringae* pv. *coriandricola* and *P. syringae* pv. *apii* causing bacterial leaf spot on parsley. *Phytopathology*, 101, 847–858.
- Bull, C. T., De Boer, S., Denny T., Firrao, G., Saux, M. F.-L., Saddler, G., Scortichini, M., Stead, D., & Takikawa, Y. (2010). Comprehensive list of names of plant pathogenic bacteria, 1980–2007. *Journal of Plant Pathology*, 92, 551–592.
- Bultreys, A., & Gheysen, I. (1999). Biological and molecular detection of toxic lipopeptide-producing *Pseudomonas syringae* strains and PCR identification in plants. *Applied and Environmental Microbiology*, 65, 1904–1909.
- Bultreys, A., & Gheysen, I. (2003). Diversity among *Pseudomonas syringae* strains from Belgian orchards using secondary metabolite-based tests. (In N. S. Iacobellis, A. Collmer, S. W. Hutcheson, J. W. Mansfield, C. E. Morris, J. Murillo, N. W. Schaad, D. E. Stead & G. Surico (Eds.), *Pseudomonas syringae and related pathogens: Biology and Genetics* (pp. 69–77). Springer: Dordrecht.)
- Bultreys, A., Gheysen, I., Rousseau, G., Pitchugina, E., Planchon, V., & Magein, H. (2018). Antibacterial activity of fosetyl-Al, ethyl-phosphite and phosphite against *Pseudomonas syringae* on plant surfaces and *in vitro*. *Plant Pathology*, 67, 1955–1966.
- Bultreys, A., & Kaluzna, M. (2010). Bacterial cankers caused by *Pseudomonas syringae* on stone fruit species with special emphasis on the pathovars *syringae* and *morsprunorum* Race 1 and Race 2. *Journal of Plant Pathology*, 92, S21–S33.
- Burke, M., Gusta, L., Quamme, H., Weiser, C., & Li, P. (1976). Freezing and injury in plants. *Annual Review of Plant Physiology*, 27, 507–528.
- Burkowicz, A., & Rudolph, K. (1994). Evaluation of pathogenicity and of cultural and biochemical tests for identification of *Pseudomonas syringae* pathovars *syringae*, *morsprunorum* and *persicae* from fruit trees. *Journal of Phytopathology (Berlin)*, 141, 59–76.
- Cameron, H. (1971). Effect of root or trunk stock on susceptibility of orchard trees to *Pseudomonas syringae*. *Plant Disease Reporter*, 55, 421–423.
- Cameron, H. R. (1962). Mode of infection of sweet cherry by *Pseudomonas syringae*. *Phytopathology*, 52, 917–921.
- Chandler, W. A., & Daniell, J. W. (1976). Relation of pruning time and inoculation with *Pseudomonas syringae* van Hall to short life of peach trees growing on old peach land. *HortScience*, 11, 103–104.
- Coburn, B., Sekirov, I., & Finlay, B. B. (2007). Type III secretion systems and disease. *Clinical Microbiology Reviews*, 20, 535–549.
- Crosse, J. (1955). Bacterial canker of stone-fruits. I. Field observations on the avenues of autumnal infection of cherry. *Journal of Horticultural Science*, 30, 1–142.
- Crosse, J. E. (1956). Bacterial canker of stone fruits. II. Leaf scar infection of cherry. *Journal of Horticultural Science*, 31, 212–224.
- Crosse, J. E. (1957). Bacterial canker of stone fruits. III. Inoculum concentration and time of inoculation in relation to leaf scar infection of cherry. *Annals of Applied Biology*, 45, 19–35.
- Crosse, J. E. (1966). Epidemiological relations of the *Pseudomonad* pathogens of deciduous fruit trees. *Annual Review of Phytopathology*, 14, 291–310.
- Crosse, J. E., & Garrett, C. M. E. (1966). Bacterial canker of stone fruits. VII. Infection experiments with *Pseudomonas morsprunorum* and *P.s. pv. syringae*. *Annals of Applied Biology*, 58, 31–41.
- Cunningham, G. H. (1925). Physiological diseases and diseases of unknown cause. *Fungous diseases of fruit trees in New Zealand and their remedial treatment*. (pp. 312–316) Auckland : New Zealand Fruitgrowers' Federation, Ltd.
- Di Lallo, G., Evangelisti, M., Mancuso, F., Ferrante, P., Marcelletti, S., Tinari, A., Superti, F., Migliore, L., D'Addabbo, P., & Frezza, D. (2014). Isolation and partial characterization of bacteriophages infecting *Pseudomonas syringae* pv. *actinidiae*, causal agent of kiwi-fruit bacterial canker. *Journal of Basic Microbiology*, 54, 1210–1221.

- Doughari, J. (2015). An overview of plant immunity. *J Plant Pathol Microbiol*, 6(10), 4172.
- Dye, D. W. (1954). Blast of stone-fruit in New Zealand. *New Zealand Journal of Science and Technology*, 35, 451–461.
- Fouts, D. E., Badel, J. L., Ramos, A. R., Rapp, R. A., & Collmer, A. (2003). A *Pseudomonas syringae* pv. *tomato* DC3000 Hrp (type III secretion) deletion mutant expressing the Hrp system of bean pathogen *P. syringae* pv. *syringae* 61 retains normal host specificity for tomato. *Molecular Plant-Microbe Interactions*, 16, 43–52.
- Freigoun, S., & Crosse, J. (1975). Host relations and distribution of a physiological and pathological variant of *Pseudomonas morsprunorum*. *Annals of Applied Biology*, 81, 317–330.
- Galán, J. E., & Wolf-Watz, H. (2006). Protein delivery into eukaryotic cells by type III secretion machines. *Nature*, 444, 567–573.
- Gardan, L., Shafik, H., & Grimont, P. A. (1997). DNA relatedness among pathovars of *P. syringae* and related bacteria. *Pseudomonas syringae* pathovars and related pathogens (pp. 445–448). Springer.
- Gardan, L., Shafik, H., Belouin, S., Broch, R., Grimont, F., & Grimont, P. (1999). DNA relatedness among the pathovars of *Pseudomonas syringae* and description of *Pseudomonas tremiae* sp. nov. and *Pseudomonas cannabina* sp. nov. (ex Suty & Dowson 1959). *International Journal of Systematic and Evolutionary Microbiology*, 49, 469–478.
- Garrett, C. M., Panagopoulos, C., & Crosse, J. (1966). Comparison of plant pathogenic *Pseudomonads* from fruit trees. *Journal of Applied Bacteriology*, 29, 342–356.
- Garrett, C. M. E. (1986). Influence of rootstock on the susceptibility of sweet cherry scions to bacterial canker caused by *Pseudomonas syringae* pv. *morsprunorum* and *syringae*. *Plant Pathology*, 35, 114–119.
- Gilbert, V., Legros, F., Maraite, H., & Bultreys, A. (2009). Genetic analyses of *Pseudomonas syringae* isolates from Belgian fruit orchards reveal genetic variability and isolate-host relationships within the pathovar *syringae*, and help identify both races of the pathovar *morsprunorum*. *European Journal of Plant Pathology*, 124, 199–218. <https://doi.org/10.1007/s10658-008-9406-y>
- Gilbert, V., Planchon, V., Legros, F., Maraite, H., & Bultreys, A. (2010). Pathogenicity and aggressiveness in populations of *Pseudomonas syringae* from Belgian fruit orchards. *European Journal of Plant Pathology*, 126, 263–277. <https://doi.org/10.1007/s10658-009-9538-8>
- Giovanardi, D., Ferrante, P., Scortichini, M., & Stefani, E. (2018). Characterisation of *Pseudomonas syringae* isolates from apricot orchards in north-eastern Italy. *European Journal of Plant Pathology*, 151, 901–917.
- Gomila, M., Busquets, A., Mulet, M., García-Valdés, E., & Lalucat, J. (2017). Clarification of taxonomic status within the *Pseudomonas syringae* species group based on a phylogenomic analysis. *Frontiers in Microbiology*, 8, 2422.
- Green, S., Laue, B., Fossdal, C. G., A'Hara, S. W., & Cottrell, J. E. (2009). Infection of horse chestnut (*Aesculus hippocastanum*) by *Pseudomonas syringae* pv. *aesculi* and its detection by quantitative real-time PCR. *Plant Pathology*, 58, 731–744.
- Harzallah, D., Sadallah, S., & Larous, L. (2004). Characterization of *Pseudomonas* pathovars isolated from rosaceous fruit trees in East Algeria. *Communications in Agricultural and Applied Biological Sciences*, 69, 443–447.
- Hattingh, M. J., Roos, I. M., & Mansvelt, E. L. (1989). Infection and systemic invasion of deciduous fruit trees by *Pseudomonas syringae* in South Africa. *Plant Disease*, 73, 784–789.
- Hirano, S. S., & Upper, C. D. (1990). Population biology and epidemiology of *Pseudomonas syringae*. *Annual Review of Phytopathology*, 28, 155–177.
- Hirano, S. S., & Upper, C. D. (2000). Bacteria in the leaf ecosystem with emphasis on *Pseudomonas syringae*—a pathogen, ice nucleus, and epiphyte. *Microbiology and Molecular Biology Reviews*, 64, 624–653.
- Hollaway, G., Bretag, T., & Price, T. (2007). The epidemiology and management of bacterial blight (*Pseudomonas syringae* pv. *psii*) of field pea (*Pisum sativum*) in Australia: A review. *Australian Journal of Agricultural Research*, 58, 1086–1099.
- Horticulture New Zealand. (2019). *FreshFacts*. Retrieved May, 2019, from <https://www.freshfacts.co.nz/> Retrieved July 2020.
- Hulin, M. T., Jackson, R. W., Harrison, R. J., & Mansfield, J. W. (2020). Cherry picking by pseudomonads: After a century of research on canker, genomics provides insights into the evolution of pathogenicity towards stone fruits. *Plant Pathology*, 69, 962–978.
- Hulin, M. T., Mansfield, J. W., Brain, P., Xu, X., Jackson, R. W., & Harrison, R. (2018). Characterization of the pathogenicity of strains of *Pseudomonas syringae* towards cherry and plum. *Plant Pathology*, 67, 1177–1193.
- Hulin, M. T., Vadillo, D. A., Cossu, F., Lynn, S., Russell, K., Neale, H. C., Jackson, R. W., Arnold, D. L., Mansfield, J. W., & Harrison, R. J. (2022). Identifying resistance in wild and ornamental cherry towards bacterial canker caused by *Pseudomonas syringae*. *Plant Pathology*, 71, 949–965.
- James, S. L. (2015). *Using experimental evolution to develop a phage biological control agent to target the horse chestnut bleeding canker pathogen Pseudomonas syringae* pv. *aesculi*. Unpublished PhD thesis, University of Reading, United Kingdom.
- Janse, J., Rossi, P., Angelucci, L., Scortichini, M., Derks, J., Akkermans, A., De Vrijer, R., & Psallidas, P. (1996). Reclassification of *Pseudomonas syringae* pv. *avellanae* as *Pseudomonas avellanae* (spec. nov), the bacterium causing canker of hazelnut (*Corylus avellana* L.). *Systematic and Applied Microbiology*, 19, 589–595.
- Jin, Q., Thilmony, R., Zwiesler-Vollick, J., & He, S.-Y. (2003). Type III protein secretion in *Pseudomonas syringae*. *Microbes and Infection*, 5, 301–310.
- Jones, A. L. (1971). Bacterial canker of sweet cherry in Michigan. *Plant Disease Reporter*, 55, 961–965.
- Jones, J. D., & Dangl, J. L. (2006). The plant immune system. *Nature*, 444, 323–329.
- Kaluzna, M., Ferrante, P., Sobiczewski, P., & Scortichini, M. (2010). Characterization and genetic diversity of *Pseudomonas syringae* from stone fruits and hazelnut using

- repetitive-PCR and MLST. *Journal of Plant Pathology*, 92, 781–787.
- Kałużna, M., Albuquerque, P., Tavares, F., Sobiczewski, P., & Puławska, J. (2016a). Development of SCAR markers for rapid and specific detection of *Pseudomonas syringae* pv. *morsprunorum* races 1 and 2, using conventional and real-time PCR. *Applied Microbiology and Biotechnology*, 100, 3693–3711.
- Kałużna, M., Willems, A., Pothier, J. F., Ruinelli, M., Sobiczewski, P., & Puławska, J. (2016b). *Pseudomonas cerasi* sp. nov. (non Griffin, 1911) isolated from diseased tissue of cherry. *Systematic and Applied Microbiology*, 39, 370–377.
- Kamiuntun, H., Nakao, T., & Oshida, S. (2000). *Pseudomonas syringae* pv. *cerasicola*, pv. nov., the causal agent of bacterial gall of cherry tree. *Journal of General Plant Pathology*, 66, 219–224.
- Kappel, F., Granger, A., Hrotkó, K., & Schuster, M. (2012). Cherry. *Fruit Breeding* (pp. 459–504). Springer.
- Kennelly, M. M., Cazorla, F. M., de Vicente, A., Ramos, C., & Sundin, G. W. (2007). *Pseudomonas syringae* diseases of fruit trees: Progress toward understanding and control. *Plant Disease*, 91, 4–17.
- Lamichhane, J. R., Varvaro, L., Parisi, L., Audergon, J.-M., & Morris, C. E. (2014). Disease and frost damage of woody plants caused by *Pseudomonas syringae*: Seeing the forest for the trees. *Advances in Agronomy*, 126, 235–295.
- Latorre, B. A., Gonzalez, J. A., Cox, J. E., & Vial, F. (1985). Isolation of *Pseudomonas syringae* pv. *syringae* from cankers and effect of free moisture on its epiphytic populations on Sweet Cherry trees. *Plant Disease*, 69, 409–412. <https://doi.org/10.1094/pd-69-409>
- Latorre, B. A., & Jones, A. L. (1979a). Evaluation of weeds and plant refuse as potential sources of inoculum of *Pseudomonas syringae* in bacterial canker of cherry. *Phytopathology*, 69, 1122–1125. <https://doi.org/10.1094/Phyto-69-1122>
- Latorre, B. A., & Jones, A. L. (1979b). *Pseudomonas morsprunorum*, the cause of bacterial canker of sour cherry in Michigan, and its epiphytic association with *P. syringae*. *Phytopathology*, 69, 335–339.
- Lelliott, R. A., Billing, E., & Hayward, A. C. (1966). A determinative scheme for the fluorescent plant pathogenic *Pseudomonads*. *Journal of Applied Bacteriology*, 29, 470–489. <https://doi.org/10.1111/j.1365-2672.1966.tb03499.x>
- Lindeberg, M., Cunnac, S., & Collmer, A. (2009). The evolution of *Pseudomonas syringae* host specificity and type III effector repertoires. *Molecular Plant Pathology*, 10, 767–775.
- Lindemann, J., & Suslow, T. V. (1987). Competition between ice nucleation-active wild type and ice nucleation-deficient deletion mutant strains of *Pseudomonas syringae* and *P. fluorescens* biovar I and biological control of frost injury on strawberry blossoms. *Phytopathology*, 77, 882–886.
- Lindow, S. (1990) Design and results of field tests of recombinant Ice-*Pseudomonas syringae* strains. In: *Risk assessment in agricultural biotechnology: proceedings of the International Conference, Davis, California, USA, August, 1988*.
- Lindow, S. (1995). Control of epiphytic ice nucleation-active bacteria for management of plant frost injury. In: Lee, R.E., at al. (Ed.), *Biological ice nucleation and its applications* (pp. 239–256). American Phytopathological Society, St. Paul, Minn.
- Lindow, S. E. (1983a). Methods of preventing frost injury caused by epiphytic ice nucleation-active bacteria. *Plant Disease*, 67, 327.
- Lindow, S. E. (1983b). The role of bacterial ice nucleation in frost injury to plants. *Annual Review of Phytopathology*, 21, 363–384. <https://doi.org/10.1146/annurev.py.21.090183.002051>
- Lindow, S. E., & Brandl, M. T. (2003). Microbiology of the phyllosphere. *Applied and Environmental Microbiology*, 69, 1875–1883.
- Maiden, M. C. (2006). Multilocus sequence typing of bacteria. *Annual Review of Microbiology*, 60, 561–588.
- Maiden, M. C., Bygraves, J. A., Feil, E., Morelli, G., Russell, J. E., Urwin, R., Zhang, Q., Zhou, J., Zurth, K., & Caugant, D. A. (1998). Multilocus sequence typing: A portable approach to the identification of clones within populations of pathogenic microorganisms. *Proceedings of the National Academy of Sciences*, 95, 3140–3145.
- Malvick, D., & Moore, L. (1988). Survival and dispersal of a marked strain of *Pseudomonas syringae* in a maple nursery. *Plant Pathology*, 37, 573–580.
- Mansfield, J., Genin, S., Magori, S., Citovsky, V., Sriariyanum, M., Ronald, P., Dow, M., Verdier, V., Beer, S. V., & Machado, M. A. (2012). Top 10 plant pathogenic bacteria in molecular plant pathology. *Molecular Plant Pathology*, 13, 614–629.
- Marroni, M., Casonato, S., Pitman, A., Beresford, R., Visnovsky, S., & Jones, E. (2022) Colonization of sweet cherry buds (*Prunus avium*) by *Pseudomonas syringae* pathovars in commercial orchards in Central Otago. 75th New Zealand Plant Protection Society Conference, 9–11 August, Christchurch (p. 18). New Zealand Plant Protection Society.
- Marroni, M., Colhoun, K., Attfield, B., Visnovsky, S., & Butler, R. (2021a) Factors contributing to bacterial canker on newly established cherry orchards in Central Otago. 73rd New Zealand Plant Protection Society Conference, 10–12 August, Napier (p. 8). New Zealand Plant Protection Society.
- Marroni, M., Colhoun, K., Attfield, B., Visnovsky, S., & Butler, R. (2021b) Trunk protection reduces bacterial canker on young cherry trees. 73rd New Zealand Plant Protection Society Conference, 10–12 August, Napier (p. 10). New Zealand Plant Protection Society.
- Marroni, M. V. (2021). Genetics and ecology of *Pseudomonas syringae* pathovars in New Zealand cherry orchards: 247. (Unpublished PhD thesis), Lincoln University.
- Marroni, M. V., Casonato, S., Visnovsky, S. B., Pitman, A. R., Beresford, R. M., & Jones, E. E. (2023). Genetic characterization and prevalence of *Pseudomonas syringae* strains from sweet cherry orchards in New Zealand. *Plant Pathology*.
- Marroni, V. (2014). Characterization of strains of *Pseudomonas* pathovars from cherry orchards in Central Otago Plant & Food Research, Auckland. Client's report 10170.

- Melotto, M., Underwood, W., & He, S. Y. (2008). Role of stomata in plant innate immunity and foliar bacterial diseases. *Annual Review of Phytopathology*, 46, 101–122.
- Ménard, M., Sutra, L., Luisetti, J., Prunier, J., & Gardan, L. (2003). *Pseudomonas syringae* pv. *avii* (pv. nov.), the causal agent of bacterial canker of wild cherries (*Prunus avium*) in France. *European Journal of Plant Pathology*, 109, 565–576.
- Mo, Y.-Y., & Gross, D. C. (1991). Expression *in vitro* and during plant pathogenesis of the *syxB* gene required for syringomycin production by *Pseudomonas syringae* pv. *syringae*. *Molecular Reproduction & Development*, 4, 28–36.
- Monchiero, M., Gullino, M. L., Pugliese, M., Spadaro, D., & Garibaldi, A. (2015). Efficacy of different chemical and biological products in the control of *Pseudomonas syringae* pv. *actinidiae* on kiwifruit. *Australasian Plant Pathology*, 44, 13–23.
- Montesinos, E., & Vilardell, P. (1991). Relationships among population-levels of *Pseudomonas syringae*, amount of ice nuclei, and incidence of blast of dormant flower buds in commercial pear orchards in Catalunya, Spain. *Phytopathology*, 81, 113–119. <https://doi.org/10.1094/Phyto-81-113>
- Morris, C. E., Lamichhane, J. R., Nikolić, I., Stanković, S., & Moury, B. (2019). The overlapping continuum of host range among strains in the *Pseudomonas syringae* complex. *Phytopathology Research*, 1, 4.
- Morris, C. E., Sands, D. C., Vinatzer, B. A., Glaux, C., Guilbaud, C., Buffiere, A., Yan, S., Dominguez, H., & Thompson, B. M. (2008). The life history of the plant pathogen *Pseudomonas syringae* is linked to the water cycle. *ISME Journal*, 2, 321–334.
- Naveed, Z. A., Wei, X., Chen, J., Mubeen, H., & Ali, G. S. (2020). The PTI to ETI Continuum in Phytophthora-Plant Interactions. *Frontiers in Plant Science*, 11, 2030.
- New Zealand Horticulture Export Authority. (2021). *Summerfruit (cherries, apricot, peaches, nectarines, plums). Summerfruit industry profile*. Retrieved 19 July, 2021, from <https://www.hea.co.nz/2012-05-11-03-05-28/summerfruit-trade>. Retrieved December 2021.
- Niklas, K. (1997). *The Evolutionary Biology of Plants*. Chicago: Univ. (Chicago Press).
- Nürnberg, T., Brunner, F., Kemmerling, B., & Piater, L. (2004). Innate immunity in plants and animals: Striking similarities and obvious differences. *Immunological Reviews*, 198, 249–266.
- Ogawa, J. M., Zehr, E. I., Bird, G. W., Ritchie, D. F., Uriu, K., & Uyemoto, J. K. (1995). *Compendium of stone fruit diseases*. American Phytopathological Society.
- Omran, M., Roth, M., Roch, G., Blanc, A., Morris, C. E., & Audergon, J.-M. (2019). Genome-wide association multi-locus and multi-variate linear mixed models reveal two linked loci with major effects on partial resistance of apricot to bacterial canker. *BMC Plant Biology*, 19, 1–18.
- Otta, J., & English, H. (1970). Epidemiology of the bacterial canker disease of French Prune. *Plant Disease Reporter*, 54, 332–336.
- Parisi, L., Morgaint, B., Blanco-García, J., Guilbaud, C., Chan-deysson, C., Bourgeay, J.-F., Moronvalle, A., Brun, L., Brachet, M., & Morris, C. E. (2019). Bacteria from four phylogroups of the *Pseudomonas syringae* complex can cause bacterial canker of apricot. *Plant Pathology*, 68, 1249–1258.
- Parkinson, N., Bryant, R., Bew, J., & Elphinstone, J. (2011). Rapid phylogenetic identification of members of the *Pseudomonas syringae* species complex using the *rpoD* locus. *Plant Pathology*, 60, 338–344.
- Psallidas, P. (1997). Hyperplastic canker—a perennial disease of almond caused by *Pseudomonas amygdali*. *EPPO Bulletin*, 27, 511–517.
- Puławska, J., Gétaz, M., Kałuzna, M., Kuzmanović, N., Obradović, A., Pothier, J. F., Ruinelli, M., Boscia, D., Saponari, M., & Végh, A. (2017). Bacterial Diseases. (In J. Quero-García, A. Lezzoni, J. Pulawska & G. A. Lang (Eds.), *Cherries: botany, production and uses* (pp. 365–385). CABI International.
- Rabiey, M., Roy, S. R., Holtappels, D., Franceschetti, L., Quilty, B. J., Creeth, R., Sundin, G. W., Wagemans, J., Lavigne, R., & Jackson, R. W. (2020). Phage biocontrol to combat *Pseudomonas syringae* pathogens causing disease in cherry. *Microbial Biotechnology*, 13, 1428–1445.
- Renick, L. J., Cogal, A. G., & Sundin, G. W. (2008). Phenotypic and genetic analysis of epiphytic *Pseudomonas syringae* populations from sweet cherry in Michigan. *Plant Disease*, 92, 372–378. <https://doi.org/10.1094/pdis-92-3-0372>
- Ross, I., & Hattingh, M. (1986). Weeds in orchards as potential source of inoculum for bacterial canker of stone fruits. *Phytophylactica*, 18, 5–7.
- Roos, I., & Hattingh, M. (1986a). Pathogenic *Pseudomonas* spp. in stone fruit buds. *Phytophylactica*, 18, 7–9.
- Roos, I. M. M., & Hattingh, M. J. (1986b). Bacterial canker of sweet cherry in South Africa. *Phytophylactica*, 18, 1–4.
- Roos, I. M. M., & Hattingh, M. J. (1986c). Resident populations of *Pseudomonas syringae* on stone fruit tree leaves in South Africa. *Phytophylactica*, 18, 55–58.
- Roos, I. M. M., & Hattingh, M. J. (1987). Systemic invasion of cherry leaves and petioles by *Pseudomonas syringae* pv. *morsprunorum*. *Phytopathology*, 77, 1246–1252.
- Roos, I. M. M., & Hattingh, M. J. (1988). Systemic invasion of immature sweet cherry fruit by *Pseudomonas syringae* pv. *morsprunorum* through blossoms. *Journal of Phytopathology (berlin)*, 121, 26–32.
- Ruinelli, M., Blom, J., Smits, T. H., & Pothier, J. F. (2019). Comparative genomics and pathogenicity potential of members of the *Pseudomonas syringae* species complex on *Prunus* spp. *BMC Genomics*, 20, 172.
- Ruppitsch, W. (2016). Molecular typing of bacteria for epidemiological surveillance and outbreak investigation/ Molekulare Typisierung von Bakterien für die epidemiologische Überwachung und Ausbruchsabklärung. *Die Bodenkultur: Journal of Land Management, Food and Environment*, 67, 199–224.
- Sands, D., Schroth, M., & Hildebrand, D. (1970). Taxonomy of phytopathogenic pseudomonads. *Journal of Bacteriology*, 101, 9–23.
- Sarkar, S. F., & Guttman, D. S. (2004). Evolution of the core genome of *Pseudomonas syringae*, a highly clonal, endemic plant pathogen. *Applied and Environmental Microbiology*, 70, 1999–2012.

- Scholz-Schroeder, B. K., Hutchison, M. L., Grgurina, I., & Gross, D. C. (2001). The contribution of syringopeptin and syringomycin to virulence of *Pseudomonas syringae* pv. *syringae* strain B301D on the basis of sypA and syrB1 biosynthesis mutant analysis. *Molecular Plant-Microbe Interactions*, 14, 336–348.
- Scortichini, M. (2002). Bacterial canker and decline of European hazelnut. *Plant Disease*, 86, 704–709.
- Scortichini, M. (2010). Epidemiology and predisposing factors of some major bacterial diseases of stone and nut fruit trees species. *Journal of Plant Pathology*, 92, S73–S78.
- Scortichini, M., & Morone, C. (1997). Apoplexy of peach trees caused by *Pseudomonas viridiflava*. *Journal of Phytopathology*, 145, 397–399.
- Siile, S., & Seemüller, E. (1987). The role of ice formation in the infection of sour cherry leaves by *Pseudomonas syringae* pv. *syringae*. *Phytopathology*, 77, 173–177.
- Sobiczewski, P. (1992). Effect of exposure to freezing temperatures on necrosis in sweet cherry shoots inoculated with *Pseudomonas syringae* pv. *syringae* or *P. s. pv. morsprunorum*. *Plant Disease*, 76, 447–451.
- Spotts, R. A., Facticeau, T. J., Cervantes, L. A., & Chestnut, N. E. (1990). Incidence and control of Cytospora canker and bacterial canker in a Young Sweet Cherry Orchard in Oregon. *Plant Disease*, 74, 577–580. <https://doi.org/10.1094/pd-74-0577>
- Spotts, R. A., Olsen, J. L., Long, L. E., & Pscheidt, J. W. (2010a). *Bacterial canker of sweet cherry in Oregon: disease symptoms, cycle and management*. Retrieved Sept, 2015, from http://extension.oregonstate.edu/wasco/sites/default/files/bacterial_canker_of_sweet_cherry_in_oregon_may10.pdf.
- Spotts, R. A., Wallis, K. M., Serdani, M., & Azarenko, A. N. (2010b). Bacterial canker of sweet cherry in Oregon. Infection of horticultural and natural wounds and resistance of cultivar and rootstock combinations. *Plant Disease*, 94, 345–350. <https://doi.org/10.1094/pdis-94-3-0345>
- Summerfruit New Zealand. (2021). *Commercial growing areas*. Retrieved May, 2021, from <https://www.summerfruitnz.co.nz/industry/#:~:text=Overview,Central%20North%20Island%20and%20Canterbury>.
- Sundin, G., & Bender, C. (1993). Ecological and genetic analysis of copper and streptomycin resistance in *Pseudomonas syringae* pv. *syringae*. *Applied and Environmental Microbiology*, 59, 1018–1024.
- Sundin, G. W., Jones, A. L., & Olson, B. D. (1988). Overwintering and population dynamics of *Pseudomonas syringae* pv. *syringae* and *P. s. pv. morsprunorum* on sweet and sour cherry trees. *Canadian Journal of Plant Pathology*, 10, 281–288.
- Tsuda, K., & Katagiri, F. (2010). Comparing signaling mechanisms engaged in pattern-triggered and effector-triggered immunity. *Current Opinion in Plant Biology*, 13, 459–465.
- Tyson, J., Rees-George, J., Curtis, C., Manning, M., & Fullerton, R. (2012). Survival of *Pseudomonas syringae* pv. *actinidiae* on the orchard floor over winter. *New Zealand Plant Protection*, 65, 25–28.
- Urwin, R., & Maiden, M. C. (2003). Multi-locus sequence typing: A tool for global epidemiology. *Trends in Microbiology*, 11, 479–487.
- Van Hall, C. (1902). *Bijdragen tot de kennis der bacteriële plantenziekten*. (University of Amsterdam, The Netherlands.
- Vanneste, J. (2011). *Characterisation of the causal organisms responsible for sweet cherry collapse in Central Otago*. (Plant & food Research Ltd. Client report No: 5801.
- Vanneste, J., McLaren, G., Yu, J., Cornish, D., & Boyd, R. (2005). Copper and streptomycin resistance in bacterial strains isolated from stone fruit orchards in New Zealand. *New Zealand Plant Protection*, 58, 101–105.
- Vanneste, J., & Voyle, M. (2003). Genetic basis of copper resistance in New Zealand strains of *Pseudomonas syringae*. *New Zealand Plant Protection*, 56, 109–112.
- Vanneste, J., & Yu, J. (2009). *Identification of the causal agents of the collapse of sweet cherry in Central Otago*. Plant & Food Research Ltd. Auckland, New Zealand. Client report No: 30746.
- Vanneste, J., Yu, J., Cornish, D., Tanner, D., Windner, R., Chapman, J., Taylor, R., Mackay, J., & Dowlut, S. (2013). Identification, virulence, and distribution of two biovars of *Pseudomonas syringae* pv. *actinidiae* in New Zealand. *Plant Disease*, 97, 708–719.
- Vicente, J. G., Alves, J. P., Russell, K., & Roberts, S. J. (2004). Identification and discrimination of *Pseudomonas syringae* isolates from wild cherry in England. *European Journal of Plant Pathology*, 110, 337–351.
- Vicente, J. G., & Roberts, S. J. (2007). Discrimination of *Pseudomonas syringae* isolates from sweet and wild cherry using rep-PCR. *European Journal of Plant Pathology*, 117, 383–392. <https://doi.org/10.1007/s10658-007-9107-y>
- Visnovsky, S. B., Marroni, M. V., Pushparajah, S., Everett, K. R., Taylor, R. K., Vinatzer, B. A., & Pitman, A. R. (2019). Using multilocus sequence analysis to distinguish pathogenic from saprotrophic strains of *Pseudomonas* from stone fruit and kiwifruit. *European Journal of Plant Pathology*, 155, 643–658.
- Volksch, B., & Weingart, H. (1998). Toxin production by pathovars of *Pseudomonas syringae* and their antagonistic activities against epiphytic microorganisms. *Journal of Basic Microbiology*, 38, 135–145.
- Weaver, D. J. (1978). Interaction of *Pseudomonas syringae* and freezing in bacterial canker on excised peach twigs. *Phytopathology*, 68, 1460–1463.
- Webster, A. (1980). Dwarfing rootstocks for plums and cherries. *Acta Horticulturae*, 114, 201–207.
- Webster, A. (1992). New dwarfing rootstocks for apple, pear, plum and sweet cherry - A brief review. *Acta Horticulturae*, 349, 145–153.
- Webster, A. D., & Looney, N. E. (1996). *Cherries: crop physiology, production and uses*. CAB INTERNATIONAL.
- Wilson, E. E. (1939). Factors affecting development of the bacterial canker of stone fruits. *Hilgardia*, 12, 259–298.
- Wormald, H. (1932). Bacterial diseases of stone-fruit trees in Britain: IV. The organism causing bacterial canker of plum trees. *Transactions of the British Mycological Society*, 17, 157–IN152.
- Xin, X.-F., Kvitko, B., & He, S. Y. (2018). *Pseudomonas syringae*: What it takes to be a pathogen. *Nature Reviews Microbiology*, 16, 316.
- Young, J. (1987a). New plant disease record in New Zealand: *Pseudomonas syringae* pv. *persicae* from nectarine,

- peach, and Japanese plum. *New Zealand Journal of Agricultural Research*, 30, 235–247.
- Young, J. (1987b). Orchard management and bacterial diseases of stone fruit. *New Zealand Journal of Experimental Agriculture*, 15, 257–266.
- Young, J. (1988). *Pseudomonas syringae* pv. *persicae* from nectarine, peach, and Japanese plum in New Zealand. *EPPO Bulletin*, 18, 141–151.
- Young, J. (2010). Taxonomy of *Pseudomonas syringae*. *Journal of Plant Pathology*, 92, S1.5–S1.14.
- Young, J. M., & Triggs, C. M. (1994). Evaluation of determinative tests for pathovars of *Pseudomonas syringae* Van-Hall 1902. *Journal of Applied Bacteriology*, 77, 195–207.