PSEUDOMONAS SYRINGAE AS IMPORTANT PATHOGEN OF FRUIT TREES WITH EMPHASIS ON PLUM AND CHERRY

Dmitrijs Konavko[1,2], Inga Moročko-Bičevska[2], Biruta Bankina[1]
[1]Latvia University of Agriculture
[2]Latvia State Institute of Fruit-Growing
dmitrijs.konavko@lvai.lv

Abstract
The aim of this article was to provide an overview on the current status of fruit tree diseases caused by *Pseudomonas syringae*, their importance and distribution, epidemiology and control possibilities with emphasis on plums and cherry. The plant diseases caused by *Pseudomonas syringae* are economically important and occur worldwide on large diversity of plants. On stone fruits, diseases caused by different pathovars of *Pseudomonas syringae* are economically important in major fruit growing regions. The severity of damages and economic importance depends on the geographical region, host plant species and pathovar of *P. syringae* involved in the disease. Bacterial canker induced by *P. syringae* pv. *syringae* on all commercially grown stone fruit species and by pv. *morsprunorum* predominantly on cherries and plums is considered to be the most serious one. Bacterial decline caused by *P. syringae* pv. *persica*e is severe on nectarine and peach. Despite the wide spread and economic importance in the most stone fruit growing areas the diseases caused by *Pseudomonas syringae* in some areas, e.g. Baltic States, are poorly studied, and the data on distribution and pathovars involved in the diseases observed are still missing.
Key words: *Pseudomonas syringae* pv. *syringae*, pv. *morsprunorum*, stone fruits, pathovars, canker.

Introduction

The plant diseases caused by *Pseudomonas* bacteria are economically important and occur worldwide on large diversity of plants. Among plant pathogenic *Pseudomonas*, *P. syringae* can cause diseases in more than 180 plant species including annual and perennial plants, fruit trees, ornamentals and vegetables (Little et al., 1998; Agrios, 2005). The phytopatogenic pseudomonads cause plant diseases with different symptoms, including cankers, diebacks, blossom, twig, leaf or kernel blights, leaf spots (*P. syringae* different pathovars), soft and brown rots (*P. viridiflava*, *P. marginalis* different pathovars), tumors or galls (*P. savastanoi* pathovars), mushroom blights (*P. tolasii*, *P. agarici* pathovars) (Braun-Kiewnick and Sands, 2001). Among plant pathogenic *Pseudomonas* 14 species are well known, of which *Pseudomonas syringae* is the most economically important with many pathovars (Braun-Kiewnick and Sands, 2001). According to J.M. Young et al. (2001) definition, the term *pathovar* is used to refer to a strain or set of strains with the same or similar characteristics, differentiated at infrasubspecific level from other strains of the same species or subspecies on the basis of distinctive pathogenicity to one or more plant hosts whereas in other sources it is pointed out that pathotype or pathovar is a subdivision of a species distinguished by common characters of pathogenicity, particularly in relation to host range. In bacteriology, where pathovar is the preferred and more used term, pathotype is used to describe the type (or reference) culture of a pathovar (Waller et al., 2002). Further in this article we use the term 'pathovar'.

An exact number of the *Pseudomonas syringae* pathovars is not defined. In 1994, about 40 pathovars were recognized (Young and Triggs, 1994). Later, the number of defined pathovars was increased to more than 50 (Braun-Kiewnick and Sands, 2001; Höfte and De Vos, 2006; Young, 2010). Among fluorescent *Pseudomonas* species, *P. marginalis*, *P. savastanoi* and *P. syringae*, contain several pathovars, which are defined based on pathogenicity to host plant species and biochemical properties (Young et al., 1996; Braun-Kiewnick and Sands, 2001).

The aim of this article is to provide an overview on current status of fruit tree diseases caused by *Pseudomonas syringae*, their importance and distribution, epidemiology and control possibilities with emphasis on cherry and plums.

Materials and Methods

Monographic method has been used for this research. As the research on *Pseudomonas* pathogens and present situation in Latvia is quite new, available scientific literature from other countries, basically from Europe has been used.

Results and Discussion

The pathogens and pathovars

The studies on bacterial canker started Brzezinski (Bultreys and Kaluzna, 2010) in Poland. He determined that the gummosis and dieback of peach, plum, apricot and sweet cherry trees were of bacterial origin. At the same time in Holland, van Hall (1902) described the causal agent of lilac bacterial blight as *Pseudomonas syringae*. Several years later, Aderhold and Ruhland described *Bacillus spongiosus* as the causal agent of death of sweet cherry in Germany. Later Griffin proved that gummosis and cankers on sweet cherries in Oregon (USA) were caused by *Pseudomonas cerasi*

and scientist Barrs confirmed the pathogenicity of this organism on various organs. In England, H. Wormald described the bacterium *Pseudomonas morsprunorum* as the causal agent of bacterial canker of plum trees. Also, he found out that *Pseudomonas prunicola* was associated with bacterial canker of stone fruits and blossom blight of pear (Bultreys and Kaluzna, 2010).

As summarized by Schaad et al. (2000), the term 'pathovar' or 'pathotype' emerged to conserve the name of the pathogens and proposals to rename several pathovars of *Pseudomonas* and *Xanthomonas* as species have caused great confusion in the literature. Pathovars of *P. syringae* vary in their capacity for epiphytic survival, the nature and severity of the symptoms they cause and their host range (Kerkoud et al., 2000; Kerkoud et al., 2002; Lindeberg et al., 2006). *P. syringae* at the beginning was isolated from diseased lilac (*Syringa vulgaris* L.) and characterized by M. Beijerinck in 1899 (Hirano and Upper, 2000). Bacteria with similar characteristics were isolated from a variety of tissues and a vast number of host plants, and following this involved pathogens were classified as separate species (Hirano and Upper, 2000). When count of the hosts and bacteria became more than 40, all these bacteria were classified as one species *P. syringae* (Hirano and Upper, 2000).

Bacterial canker of stone fruits is caused by two *Pseudomonas syringae* pathovars *P. syringae* pv. *syringae* van Hall and *P. syringae* pv. *morsprunorum* (Wormald) Young. *P. syringae* pv. *syringae* can cause canker on any commercially grown fruit species while *P. syringae* pv. *morsprunorum* (Wormald) Young infect predominantly sweet and sour cherries and plums. The behaviour of both pathovars in host tissue is similar.

Some authors highlight that bacterial canker of stone fruits is caused mostly by *P. syringae* pv. *syringae* (predominantly on sour cherries) and *P. syringae* pv. *morsprunorum* (predominantly on sweet cherries and its subspecies) (Young, 1991; Gardan et al., 1999; Kałużna et al., 2009). In the fruit growing *P. syringae* is an important pathogen also on pears and causes pear blast that limit pear production throughout the world (Moragrega et al., 1998; Moragrega et al., 2003).

Diversity of the *P. syringae* pathovars is rather complicated. *P. syringae* pv. *morsprunorum* race 1 is characterized and separated in 4 strains, *P. syringae* pv. *morsprunorum* race 2 is characterized and separated in 7 strains (Endert and Ritchie, 1984; Gardan et al., 1999). Most of the *P. syringae* pv. *syringae* strains produce the toxic lipodepsipeptides syringomycins and syringopeptins and molecular tests based on this characteristic can be used for the diagnosis of the pathovar (Bereswill, 1994; Gilbert et al., 2009). *P. syringae* pv. *syringae* is genetically heterogeneous pathovar with a wide host range. This pathovar shows different virulence on the lilacs, sweet cherries and plums (Gilbert et al., 2009). Phytotoxins of the pathogen can be host-specific, but usually phytotoxins produced by *P. syringae* are nonhost specific and cause symptoms on many plant species, which cannot be infected by the toxin-producing pathogen (Bender, 1999).

P. syringae pv. *persicae* was proved to be pathogenic on nectarine and peach, and also as weak pathogen on Japanese and European plum, but it is not pathogenic on apricots and cherries (Young, 1987). Pathovar *P. syringae* pv. *persicae* (Prunier et al., 1985) is a quarantine organism in Europe (EPPO) and causes leaf spots, cankers, gummosis of fruits on peach in France and bacterial decline of peach, nectarine and Japanese plum in New Zealand (Hattingsh and Roos, 1995).

In France, 2003, it was found that causal agents of bacterial canker of stone fruits are not limited only to *P. syringae* pv. *morsprunorum* and *P. syringae* pv. *syringae*, and one more pathovar *P. syringae* pv. *avii* (pv. nov. Menard) should be distinguished, which causes bacterial canker on sweet cherries (Menard et al., 2003). However, this pathovar is not officially confirmed. As described by M. Menard et al. (2003) pv. *avii* is characterized by slow growth on King`s B medium producing colonies 1.0 – 1.5 mm in diameter after three days of incubation and not producing fluorescent pigment. Japanese scientists have also proposed to recognize one more pathovar of *P. syringae* – pv. *cerasicola* (pv. nov. Kamiunten), which induces galls on cherry and apricots, but it is not pathogenic on other stone fruit species (Kamiunten et al., 2000). So far this pathovar is not recorded outside Japan.

Each *P. syringae* pathovar usually has a strong biochemical, immunological and DNA relatedness with several other pathovars, including *P. syringae* pv. *syringae*, which can be isolated from many species of plants, e.g. apples, even if these are not pathogens for these plants (Kerkoud et al., 2002).

Distribution and importance

Bacterial canker of stone fruit occurs in all fruit growing areas in the world (Hattingsh and Roos, 1995). On sweet cherries, bacterial canker is widespread, and it is economically important disease in all regions in the world where sweet cherries are grown (Prunier and Cotta, 1985; Prunier et al., 1985; Bradbury, 1986). On the West coast of the USA, only *P. syringae* pv. *syringae* was detected on sweet cherry while in Michigan *P. syringae* pv. *syringae*, *P. syringae morsprunorum* race 1 and probably a third pathovar were responsible for disease outbreaks in sweet and sour cherries (Jones, 1971).

Bacterial canker on sweet cherries can be caused by *P. syringae* pv. *morsprunorum*, *P. viridiflava* or *P. syringae* pv. *syringae* (Prunier and Cotta, 1985; Menard et al., 2003). In the United Kingdom predominantly *P. syringae* pv. *morsprunorum* is considered to be a causal agent of bacterial canker while in the other European countries, South Africa and the USA both pathovars are considered to be a causal agent of canker on sweet and sour cherries (Lattore and Jones, 1979; Vicente and Roberts, 2003; Vicente et al., 2004). Only one of these pathovars is usually found as a causal agent of bacterial canker in the most of the countries, although distribution of both pathovars is reported in the some parts of the world (Wimalajeewa and Flett, 1985). *P. syringae* pv. *morsprunorum* has been recorded as the cause of bacterial canker in Europe, the USA, Canada, but in some countries, e.g., Chile, it has not been identified (Latorre et al., 2002).

Damages caused by pathovars of *P. syringae* vary depending on stone fruit growing region and host plants. Since 2005, a disease similar to bacterial canker of stone fruit trees was observed only in some areas of Iran while in several provinces in Turkey severe bacterial canker was observed on almost 80% of apricot trees in commercial orchards and home gardens (Kotan and Sahin, 2002; Karimi-Kurdistani and Harighi, 2008). Reports from France, the UK, Germany, Poland, New Zealand, Lithuania and other countries indicate bacterial canker occurrence over many years, including wild cherry plantations grown for wood production (Menard et al., 2003; Vasinauskiene et al., 2008). In Poland, due to the favourable climate, bacterial canker is observed every year. The last serious outbreak occurred in 2007, resulting in substantial economic losses, especially in sour cherry. Both pathovars and both races of *P. syringae* pv. *morsprunorum* have been isolated from infected trees (Bultreys and Kaluzna, 2010).

The available data on fruit tree bacterial diseases caused by *P. syringae* in Baltic States are limited. In Lithuania and similarly in Latvia, mostly fungal diseases have been considered important in orchards for a long time. Only recently surveys of stone fruit orchards for possible infection of *P. syringae* were started in Lithuania (Vasinauskiene et al., 2008). The research was focused on the occurrence of bacterial canker in stone fruit orchards, evaluation of disease symptoms and preliminary diagnostics of the causal agents. The disease was recorded only on single trees and did not have economic importance. Pathogenicity tests with strains isolated from stone fruits and identification of pathovars and studies of the genetic diversity are still in progress (Vasinauskiene et al., 2008). Records on bacterial canker and its occurrence in Estonia were not found. In Latvia, commercially grown stone fruit species are plums, sweet and sour cherry. Peaches and apricots are grown only in home gardens or in varietal collections. Research on bacterial diseases of stone fruits, their distribution and causal agents in Latvia started in 2008 within the frame of COST Action Nr.873 and national research programs. The bacterial canker was not widely spread in stone fruit orchards, and *P. syringae* pv. *syringae* was detected as a cause of bacterial canker in sweet cherry and plums (Moročko-Bičevska et al., 2010; Konavko, 2011).

Symptoms caused by P. syringae and epidemiology

Depending on the symptoms and pathogens involved, diseases caused by *P.syringae* are known under various names, such as bacterial canker, bacterial decline, gummosis, blossom blast, dieback, spur blight, twig blight, bud bacteriosis (Braun-Kiewnick and Sands, 2001; Bultreys and Kaluzna, 2010). The studies show that the most important stone fruit diseases caused by *P. syringae* are bacterial canker and bacterial decline or dieback (Hattingsh and Roos, 1995). Symptoms of bacterial canker on trunks and branches occur predominantly on pears and sweet cherries, but also sour cherries, apricots, peaches, plums and in some years also apple trees suffer from bacterial canker (Prunier and Cotta, 1985; Prunier et al., 1985; Bradbury, 1986). As summarized by A. Bultreys and M. Kaluzna (2010), *P. syringae* basically can damage all areal parts of stone fruits. It causes cankers and necroses on trunks, branches, around spurs and in branch junctions. On leaves symptoms occur as small, rounded, light brown spots in various sizes, which become necrotic and drop off resembling 'shot hole' symptom. Blossoms turn brown and often fall before full blooming. On immature fruits, predominantly in cherry, symptoms are either regular or irregular, brown to black in colour, necrotic spots. *P. syringae* can cause canker also on sea buckthorn, but this disease is poorly studied (Janick and Paul, 2008).

Bacterial decline or dieback caused by *P. syringae* pv. *persicae* is a quarantine disease and affects nectarine, peaches, Japanese plums and Myrobalan plum (Young, 1995). In nectarine and peaches, symptoms include shoot dieback, limb and root injury, tree death, leaf spots and fruit lesions (Luisetti et al., 1976; Young, 1995). Distinctive characteristics of decline are staining of wood in branches above necrosis and the absence of obvious boundary between morbid and healthy bark in the lower tree parts (Luisetti et al., 1976; Young, 1995).

Besides causing cankers on trunks and branches of stone fruit trees *P. syringae* pv. *syringae* and pv. *morsprunorum* also infects leaves, shoots, flowers and fruits. In Europe, the disease is more common

on cherries and plums (Ephinstone et al., 2008). As summarized by J. Ephistone et al. (2008), cankers, which are not perennial, are formed in the late autumn and winter, but do not increase much in size till next spring, when they enlarge rapidly and kill large areas of green bark. When blossom fall occurs, the progress of cankers is stopped, and populations of these bacteria within the canker decline and often die out. During this time leaf infection phase occurs. Leaves of the spurs tend to be resistant as they mature, whereas young leaves on extension shoots are infected. Bacteria as epiphytes on all leaves in the summer reside till the leaf fall, being the main source of new infection (Ephinstone et al., 2008).

The diseases caused by *P. syringae* pathovars have a different epidemiology. As summarized by J. Ephistone et al. (2008), for *P. syringae* pv. *syringae* and pv. *morsprunorum*, cankers caused by these pathovars may be perennial and the bacteria as small populations overwinter in them. Fast multiplications of bacterial populations occur in the spring, bacterial ooze is often produced, and bacteria spread to leaves by the splash of the rain. Unlike *P. syringae* pv. *morsprunorum*, *P. syringae* pv. *syringae* gain to woody tissue only via wounds in scars of leaf and bark, from which new cankers arise further.

Symptoms vary between different host species. Cherry trees of all ages are susceptible, and most of the cankers are found at sites of leaf scars on fruiting spurs. This results in dieback of spur, but it can occasionally spread to form a canker in the parent branches. Cankers may also be located on the branches, mostly on the crotch and angles between branches. On younger branches with thin bark cankers at the beginning they are visible in the spring as shallow, discoloured sunken lesions often showing the presence of gummy exudates. On plums cankers occur mostly on trunks frequently leading to death of trees. Cankers can extend in length on the trunk and often appear like dark and linear depressions in the bark. Gumming on plums is less common and less obvious as on cherry. Spots on the leaves are caused by both pathovars and usually are reddish brown, rounded, sometimes angular, often coalescing to form big, irregular necrotic spots, which can drop out as a 'shot hole' effect (Ephinstone et al., 2008).

In spring, the first symptoms appear after late frosts, and this period is the most dangerous. Young leaves are the most susceptible. When they mature, infected areas become dry and fall out; the leaves then have a shot hole appearance and when they become mature, these leaves are not infected anymore (Süle and Seemüller, 1987). As summarized by M. J. Hattingsh and I. M. M. Roos (1995), terminal shoots or twigs of cankered trees can die back. If girdled by a canker, infected lateral branch or trunk dies during several weeks. The root system of diseased trees usually remains healthy, and suckers grow in the crown area. Pathogens can be present in dormant leaves and buds of flowers. Infected dormant buds are often killed, but some of the invaded buds normally open in the spring and then collapse in early summer. Leaves from buds like this wilt and fruits tend to dry out. Leaves and flowers arising from other diseased buds may remain symptomless. Leaf infections, mostly on cherry appear as water-soaked spots and those after that become brown, dry. After that, shot holes may be seen. Symptoms on the leaves occur sporadically and are not always typical of the disease. Flat, superficial, dark brown spots develop on the infected fruits, but lesions can be depressed in the fruit flesh, especially if cherry fruits are infected (Hattingsh and Roos, 1995). Bacterial blossom blight is attributed to *P. syringae* pv. *syringae* and pv. *morsprunorum*, and in most countries this is regarded as the cause of bacterial canker (Crosse, 1966).

Pathogenic bacteria may survive in the buds of infected trees (Roos and Hattingh, 1986). Dangerous source of infection are branches with infection, where from *Pseudomonas* bacteria spread further by a wind and rain (Hirano and Upper, 2000). *P. syringae* does not survive in the soil for a long time (Hirano and Upper, 2000). Most of the *P. syringae* pathovars are known to be epiphytes on their hosts. According to C. Leben (Leben, 1965, cited by Luisetti, 1996), these pathogenic bacteria are able to survive on the aerial parts of the plants even if conditions are not favourable, like high temperature or low humidity. In these unfavourable conditions, they can multiply and survive until environmental conditions become favourable again (e.g. moisture after rain, dew) (Luisetti, 1996).

Control possibilities

When fruit trees are already infected with *P. syringae* pathovars and express disease symptoms, it is impossible to treat them. Therefore, important control strategy is the use of preventive measures. The use of canker-free nursery stock is good and effective practice to reduce disease occurrence (Young, 1995). Selection of a suitable site and soil for the establishment of new orchards is an important step to prevent disease spread. Establishment of orchards under marginal soil and climatic conditions create a risk for development of the disease (Hattingsh and Roos, 1995; Young, 1995).

Other measures include selection of cultivars with partial resistance, avoidance from early winter pruning, application of fixed-copper sprays in autumn to reduce inoculum (Sundin et al., 1989; Young, 1995). Breeding for resistance is a slow process with woody trees, because of the time involved for tree growth

and the threat of *Pseudomonas syringae* to adapt genetically and infect the new germplasm. However, some successful control of *Pseudomonas syringae* has been realized using plant germplasm resistant to this pathogen (Moore, 1988). The cultivars of sweet cherry are not resistant (Moore, 1988). Cultivars differ in susceptibility, and some exhibit partial resistance, but some of them are immune (Luisetti et al., 1976; Young, 1995).

Copper sprays are aimed at reducing epiphytic populations of bacteria and are timed to correspond with 20% and 80% leaf drop and can be up to three late dormant applications (Wimalajeewa et al., 1991). Copper compounds are commonly used to minimize the distribution of disease in sweet cherry orchards, but these compounds have limited efficacy and may have also phytotoxic effects (Hibberd, 1988; cit. by Vicente et al., 2004). As noted by J. G. Vicente et al. (2004), the control of diseases caused by *P. syringae* pathovars is problematic in woodlands because it is not economical and practical to make sprays in woodland plantations. Therefore, the only practical approaches to control canker in woodlands are disease avoidance and use of resistant plants. However, both of these approaches are limited because of lack of sufficient amount of knowledge and understanding of the pathogens involved as well as lack of consistent methods for their detection and discrimination as also pointed by J. G. Vicente et al. (2004).

Conclusions

1. Bacterial diseases of stone fruits caused by different pathovars of *Pseudomonas syringae* are economically important in major fruit growing regions. The severity of damages and economic importance depends of the growing region, host species and pathovar involved.
2. Among stone fruit diseases caused by different *Pseudomonas syringae* pathovars as the most serious is considered bacterial canker caused by *P. syringae* pv. *syringae* on any stone fruit species and *P. syringae* pv. *morsprunorum* on predominantly sour and sweet cherries and plums, and bacterial decline caused by *P. syringae* pv. *persica*e on nectarine and peach.
3. Despite a wide spread and economic importance of diseases caused by *Pseudomonas syringae* in several stone fruit growing areas, in some areas, e.g. Baltic States, the data on distribution and *P. syringae* pathovars involved in the diseases observed are still missing.

Acknowledgements

Participation in the conference and preparation of the manuscript was supported by the Latvian Council of Science, Grant. No.672/2014.

References

1. Agrios G.N. (2005) *Plant Pathology, Fifth Edition*. Elsevier Academic Press, University of Florida. 922 p.
2. Bender C.L. (1999) Chlorosis-inducing phytotoxins produced by *Pseudomonas syringae*. *European Journal of Plant Pathology*, Vol. 105. pp. 1 – 12.
3. Bereswill S., Bugert P., Volksch B., Ullrich M., Bender C.L., Geider K. (1994) Identification and relatedness of coronatine-producing *Pseudomonas syringae* pathovars by PCR analysis and sequence determination of the amplification products. *Applied Environmental Microbiology*, Vol. 60. pp. 2924 – 2930.
4. Bradbury J.F. (1986) *Pseudomonas*. *Guide to Plant Pathogenic Bacteria*, CAB International Mycological Institute, Key, UK. pp. 110 – 185.
5. Braun-Kiewnick A., Sands D.C. (2001) Pseudomonas. In: Schaad N.W., Jones J.B., Chun W. (eds) *Laboratory Guide for identification of plant pathogenic bacteria*. Third Edition. St. Paul, Minesota, USA, pp. 84 – 120.
6. 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 (1, Supplement), S1. pp. 21 – 33.
7. Crosse J.E. (1966) Epidemiological relations of the Pseudomonads pathogens of deciduous trees. *Annual Review of Phytopathology*, 4; pp. 291 – 310.
8. Endert E., Ritchie D.F. (1984) Detection of pathogenicity, measurement of virulence, and determination of strain variation in *Pseudomonas syringae* pv. *syringae*. *Plant Disease*, 68 pp. 677 – 680.
9. Ephinstone J., Stead D., Boonham N., Tomlison N., Thwaites R., Parkinson N., Stanford H., Steel E. (2008) *Short term Training Mission – Plant Bacteriology MANUAL*. COST 873 "StoneFruitNutHealth". 3. – 7. March, Central Science Laboratory, York, UK. 59. p.
10. Gardan L., Shafik H., Belouin S., Broch H., Grimont F., Grimont P.A.D. (1999) DNA relatedness among the pathovars of *Pseudomonas syringae* and description of *Pseudomonas tremae* sp. nov. and *Pseudomonas cannabina* sp. nov. (ex Sutic and Dowson 1959). *International Journal of Systematic Bacteriology* 49, pp. 469 – 478.

11. Gilbert V., Planchon V., Legros F., Maraite H., Bultreys A. (2009) Pathogenicity and aggressiveness in populations of *Pseudomonas syringae* from Belgian fruit orchards. *European Journal of Plant Pathology*. Vol. 126. pp. 263 – 277.
12. Hattingsh M.J., Roos I.M.M. (1995) Bacterial canker. *In: Compendium of stone fruit diseases.* Ogawa J. M. (eds.) *The American Phytopathological Society St. Paul*: APS Press. pp. 48 – 50.
13. Hirano S.S., Upper C.D. (2000) Bacteria in the Leaf Ecosystem with Emphasis on *Pseudomonas syringae* – a Pathogen, Ice Nucleus, and Epiphyte. *Microbiol. Mol. Biol. Rev.* 64, pp. 624 – 653.
14. Höfte M., De Vos P. (2006) Plant pathogenic *Pseudomonas* species. In: *Plant-Associated Bacteria*. Part 3. pp. 507 – 533.
15. Janick J., Paul R.E. (2008) *The encyclopedia of fruit and nuts*. CABI Publishing, 954 p.
16. Jones A.L. (1971) Bacterial canker of sweet cherry in Michigan. *Plant Disease Reporter*. Vol. 55. pp. 961 – 965.
17. Kałużna M., Puławska J., Sobiczewski P. (2009) The use of PCR melting profile for typing of *Pseudomonas syringae* isolates from stone fruit trees. *European Journal of Plant Pathology*. Vol. 126, pp. 437 – 443.
18. Kamiunten 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*. Vol. 66. pp. 219 – 224.
19. Karimi-Kurdistani G., Harighi B. (2008) Phenotypic and molecular properties of *Pseudomonas syringae* pv. *syringae* the causal agent of bacterial canker of stone fruit trees in Kurdistan province. *Journal of Plant Pathology* 90, pp. 81 – 86.
20. Kerkoud M., Manceau C., Paulin J.P. (2002) Rapid diagnosis of *Pseudomonas syringae* pv. *papulans*, the causal agent of blister spot of apple, by polymerase chain reaction using specifically designed *hrpL* gene primers. *Phytopathology*, Vol. 92, pp. 1077 – 1083.
21. Kerkoud M., Manceau C., Gardan L., Samson R., Paulin J.P. (2000) Epiphytic occurrence of *Pseudomonas syringae* pv. *Papulans* (Rose) in France, where blister spot has never been seen. *European Journal of Plant Pathology*. 106, pp. 481 – 485.
22. Konavko D. (2011) Master Thesis Paper Kauleņkoku bakteriālā vēža izplatība Latvijā un tā ierosinātāju identifikācija (Occurence of bacterial canker of stone fruits in Latvia and identification of causal agents). Latvia University of Agriculture. Jelgava, LLU. 63 lpp. (in Latvian).
23. Kotan R., Sahin F. (2002) First record of bacterial canker caused by *Pseudomonas syringae* pv. *syringae* on apricot trees in Turkey. *Plant Pathology*, 51, 798 p.
24. Latorre B.A., Lillo C., Rioja M.E. (2002) Effects of temperature, free moisture duration and inoculum concentration on infection of sweet cherry by *Pseudomonas syringae pv. syringae*. *European Journal of Plant Pathology*. Vol. 30, pp. 410 – 419.
25. Lattore B.A., Jones A.L. (1979) *Pseudomonas morsprunorum*, the cause of bacterial canker of sour cherry in Michigan and its epiphytic association with *P. syringae*. *Phytopathology*. Vol. 69. pp. 335 – 339.
26. Lindeberg M., Cartinhour S., Myers C.R., Schechter L.M., Schneider D.J., Collmer A. (2006) Closing the Circle on the Discovery of Genes Encoding Hrp Regulon Members and Type III Secretion System Effectors in the Genomes of Three Model *Pseudomonas syringae* Strains. *Molecular Plant-Microbe Interactions*. Vol. 19. pp. 1151 – 1158.
27. Little E.L., Bostock R.M., Kirkpatrick B.C. (1998) Genetic Characterization of *Pseudomonas syringae* pv. syringae Strains from Stone Fruits in California. *Applied Environmental Microbiology*. Vol. 64. pp. 3818 – 3823.
28. Luisetti J., Prunier J.P., Gardan L., Gaignard J.L., Vigouroux A. (1976) *Le dépérissement bactérien du pêcher* (*Pseudomonas syringae* f. sp. *persicae*). *Institut national de vulgarization pour les fruits, legumes et champignons* (Invuflec), Paris, France. 60 p.
29. Luisetti J.L. (1996) Spatial and Temporal Variations in Size and Phenotypic Structure of Populations of *Pseudomonas Syringae* on Fruit Trees. *Aerial Plant Surface Microbiology*. Part V. pp. 249 – 264.
30. Menard M., Sutra L., Luisetti J., Prunier J.P., Gardan L. (2003) *Pseudomonas syringae pv. avii* (pv. *nov.*), the Causal Agent of Bacterial Canker of Wild Cherries in France. *European Journal of Plant Pathology*. Vol. 109. pp. 565 – 576.
31. Moore L.W. (1988) *Pseudomonas syringae*: disease and Ice Nucleation Activity. *Ornamentals Northwest Archive*. Vol. 12, Issue2, pp. 3 – 16.
32. Moragrega C., Manceau Ch., Montesinos E. (1998) Evaluation of drench treatments with phosphonate derivatives against *Pseudomonas syringae* pv. *syringae* on pear under controlled environment conditions. *European Journal of Plant Pathology*. Vol. 104. pp. 171 – 180.

33. Moragrega C., Llorente I., Manceau Ch. (2003) Susceptibility of European pear cultivars to *Pseudomonas syringae* pv. *syringae* using immature fruit and detached leaf assays. *European Journal of Plant Pathology.* Kluwer Academic Publishers. Vol. 109. pp. 319 – 326.
34. Moročko-Bičevska I., Sokolova O., Konavko D., Stalažs A. (2010) Characterization of bacterial diseases of stone fruits in Latvia (abstract). In: COST Action 873 "*Bacterial Diseases of Stone Fruits and Nuts*" September 13 – 15, 2010 Jūrmala, Latvia. p. 18.
35. Prunier J.P., Pelissier A., Bonnal R., Cotta J. (1985) Le chancre bactérien du cerisier en France. Sensibiliéde l'arbre et cycle biologique d'une des bactéries responsables. *L'Arboriculture fruitiére* 374. pp. 55 – 58.
36. Prunier J.P., Cotta J. (1985) Le chancre bactérien du cerisier en France, un risque sérieux. Description de la maladie. *L'Arboriculture fruitiére*, 372. pp. 39 – 42.
37. Roos I.M.M., Hattingh M.J. (1986) Pathogenic *Pseudomonas* in stone fruit buds. *Phytophylactica*: Vol. 18. pp. 7 – 9.
38. Schaad N.W., Vidaver A.K., Lacy G.H., Rudolph K., Jones J.B. (2000) Evaluation of proposed amended names of several pseudomonads and xanthomonads and recommendations. *Phytopathology*, 90, pp. 208 – 213.
39. Süle S., Seemüller E. (1987) The Role of Ice Formation in the Infection of Sour Cherry Leaves by *Pseudomonas syringae* pv. *syringae*. *Phytopathology*, Vol. 77. pp. 173 – 177.
40. Sundin G.W., Jones A.L., Fulbright D.W. (1989) Copper resistance in *Pseudomonas syringae* pv. *syringae* and its associated transfer in vitro and in planta with a plasmid. *Phytopathology* 79, pp. 861 – 865.
41. Vasinauskiene M., Baranauskaite L., Burokiene D. (2008) Search for P*seudomonas syringae* on stone fruits in Lithuania. *Proceedings of STF Meeting on "Determination of the incidence of the different pathovars of Pseudomonas syringae in stone fruits" COST Action 873 "Bacterial diseases of stone fruits and nuts"* 9 p.
42. Vicente J.G., Alves J.P., Russell K. (2004) Identification and Discrimination of *Pseudomonas syringae* Isolates from Wild Cherry in England. *European Journal of Plant Pathology*. Vol. 110. pp. 337 – 351.
43. Vicente J.G., Roberts S.J. (2003) Screening wild cherry micropropagated plantlets for resistance to bacterial canker. In: Iacobellis N.S. *Developments in Plant Pathology: Pseudomonas syringae pathovars and related pathogens,* Dordrecht: *Klewer Academic Publishers*. pp. 23 – 27.
44. Waller J.M., Lenné J.M., Waller S.J. (2002) *Plant Pathologist's Pocketbook*, 3rd edn. CABI Publishing, Wallingford, Oxon, 528 p.
45. Wimalajeewa D.L.S., Flett J.D. (1985) A study of populations of *Pseudomonas syringae* pv. *syringae* on stone fruits in Victoria. *Plant Pathology*. Vol. 34. pp. 248 – 254.
46. Wimalajeewa D.L.S., Cahill R., Hepworth G., Schneider H.G., Washbourne J.W. (1991) Chemical control of bacterial canker (*Pseudomonas syringae* pv. *syringae*) of apricot and cherry in Victoria. *Australian Journal of Experimental Agriculture*. 31, pp. 705 – 708.
47. Young J.M. (1987) New plant disease record in New Zealand: *Pseudomonas syringae* pv. *Persicae* from nectarine, peach, and Japanese plum. *New Zealand Journal of Agricultural Research. Vol.* 30, pp. 235 – 247.
48. Young J.M. (1991) Pathogenicity and identification of the lilac pathogen, *Pseudomonas syringae* pv. *syringae* van Hall 1902. *Annual of Applied Biology*, 118. pp. 283 – 298.
49. Young J.M., Triggs C.M. (1994) Evaluation of determinative tests for pathovars of *Pseudomonas syringae* van Hall 1902. *Journal of Applied Bacteriology*. Vol. 77. pp. 195 – 207.
50. Young J.M. (1995) Bacterial decline. In: *Compendium of stone fruit diseases.* Ogawa J. M. (eds.) *The American Phytopathological Society St. Paul*: APS Press. 50 p.
51. Young J.M., Saddler G.S., Takikawa Y., De Boer S.H., Vauterin L., Gardan L., Gvozdyak R.I., Stead D. (1996) Names of plant pathogenic bacteria 1864 –1995. *Review of Plant Pathology.* 75, pp. 721 – 763.
52. Young J.M. (2010) Taxonomy of *Pseudomonas syringae*. Landcare Research, Private Bag 92170, Auckland, New Zealand. *Journal of Plant Pathology,* 2010, 92 p.
53. Young J.M., Bull C.T., De Boer S.H., Firrao G., Gardan L., Saddler G.E., Stead D.E., Takikawa Y. (2001) International Standards for Naming Pathovars of Phytopathogenic Bacteria. Available at: http://www.isppweb.org/about_tppb_naming.asp, 06 April 2014.