Risks to New Zealand's primary industries from *Xylella*

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Summary

Background

The bacterium *Xylella fastidiosa* is a plant pathogen of worldwide biosecurity concern. This review aims to describe the potential biosecurity threat of *X. fastidiosa* to New Zealand's valued plant systems. The work seeks to build awareness of the threat posed by *X. fastidiosa* across industry, government, research providers and tangata whenua in order to prioritise readiness activities that would minimise its impact upon incursion. Key findings from the review are:

Pathogen
- *X. fastidiosa* is an obligate inhabitant of plant xylem vessels that also colonises the foregut of xylem-sap feeding insect vectors.
- Genomic research shows that *X. fastidiosa* can be divided into genetically distinct subspecies: ssp. *fastidiosa*, ssp. *multiplex*, ssp. *pauca*, ssp. *morus*, ssp. *sandyi* which have evolved in different regions of the Americas.
- Plant diseases are associated with specific subspecies and host combinations.

Hosts
- *X. fastidiosa* has a very wide host range, with most infections remaining asymptomatic.
- It seems possible that almost any plant can be a host, if associated with a xylem-sap feeding vector.
- Specific combinations of hosts and *X. fastidiosa* genotypes result in plant disease.
- Most hosts showing disease are woody perennials; fewer annual plants show symptoms.
- It is currently impossible to predict the development of new diseases caused by *X. fastidiosa*.
- Many native plant species in New Zealand can act as *X. fastidiosa* hosts and several are regarded as symptomatic in response to infection.
- Amenity, ornamental and weed plants known to host *X. fastidiosa* overseas are common and widespread in New Zealand.

Insect vectors
- *X. fastidiosa* is transmitted between plants by xylem-sap feeding insects, especially sharpshooter leafhoppers and spittlebugs.
- There are no known sharpshooters in New Zealand but there are several spittlebug species.
- The most invasive vector of *X. fastidiosa*, the glassy-winged sharpshooter, is now found in several islands in the Pacific. It has never been intercepted at the New Zealand border.
- *Philaenus spumarius*, the meadow spittlebug, which is known to transmit *X. fastidiosa* in Europe, is widespread and common in New Zealand.
- The role of cicadas in *X. fastidiosa* transmission has been little researched.

Environment
- *X. fastidiosa* survival is limited by cold temperatures, in a genotype-specific manner.

- Strains of *X. fastidiosa* may be eliminated from plants exposed to freezing temperatures
- Predictions of climate suitability for *X. fastidiosa* are highly variable and precise prediction is not yet possible.
- Average minimum temperatures in winter are considered a useful predictor of climate suitability for *X. fastidiosa*; using this measure, North Island would be highly suitable while parts of South Island would be partially suitable.
- Some genotypes of *X. fastidiosa* may be less sensitive to cold, and capable of invading a wider area of New Zealand.

Economic risk

- Only two recent economic evaluations have been carried out into the economic consequences of an *X. fastidiosa* incursion. The current costs in California and the expected costs of establishment in Australia have been reported as very high, but it is not possible to precisely extrapolate these to the New Zealand situation.
- The economic effects in New Zealand will be highly dependent on climate-determined pathogen establishment and on the financial strength of affected enterprises, which will determine their ability to replant with resistant cultivars (if available).
- The costs associated with establishment could be substantially reduced if it were possible to confine the spread of an *X. fastidiosa* incursion.
- Horticultural production worth between $301 million per year and $1.7 billion per year would be at risk if *X. fastidiosa* became established in New Zealand.

Introduction — Historical overview of *Xylella fastidiosa* spread

Xylella fastidiosa is a bacterium responsible for plant disease epidemics worldwide. For a long period, *X. fastidiosa* was considered to be of significance only in North America, but it has recently become of worldwide concern due to its global spread and association with new diseases (Purcell 2013). The first known outbreak of a *X. fastidiosa* disease was in the grape industry of southern California which was devastated by an epidemic of Pierce's disease in 1892 (Pierce 1892), followed by a re-appearance in 1930–1940. Another severe epidemic occurred in the 1990s upon the invasion of a new vector, the glassy-winged sharpshooter (GWSS; *Homalodisca vitripennis*). At the same time as Pierce's disease was affecting the grape industry, diseases with similar characteristics were observed in North America including almond scorch (Davis et al. 1980), phony peach disease (Wells et al. 1983) and plum leaf scald (Davis et al. 1981).

In 1987, *X. fastidiosa* became a concern outside of North America when it was associated with citrus variegated chlorosis (CVC) in Brazil, causing substantial losses from entire orchards (Chang et al. 1993; Hartung et al. 1994). Strains of *X. fastidiosa* related to the CVC strains (Rosato et al. 1998) were also discovered causing coffee leaf scorch in Sao Paulo state in 1997 (Paradela Filho et al. 1997; Badel and Zambolim 2019). The discovery of CVC spurred a burst of new research on *X. fastidiosa*. *X. fastidiosa* became of even greater international interest with the development of the first major epidemic outside of the Americas. Olives in the Apulia region of Italy suffered olive quick decline syndrome (OQDS) characterised by severe branch desiccation and rapid death that was first attributed to *X. fastidiosa* in 2013 (Saponari et al. 2013). OQDS has now spread to cover an area of c. 7000 km^2 on the Salento Peninsula (Saponari et al. 2019). Lethal infections were also observed in

ornamental and shelter belt hosts such as oleander (*Nerium oleander*), acacia (*Acacia saligna*) and myrtle leaf milkwort (*Polygala myrtifolia*). *X. fastidiosa* was also found to be widespread in parts of France and Spain, showing the potential for this bacterium to accompany plant material across international borders (Denance et al. 2017).

1.   Pathogen

1.1.   Lifecycle overview

X. fastidiosa is a gram-negative rod-shaped (0.25–0.35 x 0.9 x 3.5 µm) bacterium in the family Xanthomonadaceae (Gammaproteobacteria) (Sicard et al. 2018). It does not have flagella but is mobile by means of Type IV pili-mediated twitching (Rapicavoli et al. 2018). *X. fastidiosa* grows only in the xylem of infected plants.

X. fastidiosa is moved from region to region by insect vectors carrying the bacterium or by human assisted movement of infected plant material (Sicard et al. 2018). The bacterium colonises the foregut of insects that feed from xylem sap. Known vectors are sharpshooter leaf-hoppers (Ciadellidae subfamily Cicadellinae) and spittlebugs (Cercopoidea, families Aphrophoridae, Cercopidae, and Clastopteridae). *X. fastidiosa* is also graft-transmissible (Pierce 1892). *X. fastidiosa* multiplies and spreads in the xylem of the host plant, inducing blockages of vessels and pit membranes, resulting in water stress symptoms such as die back of leaf tips (scorch), defoliation, desiccation of berries ('raisining') and wilting (Rapicavoli et al. 2018; Sicard et al. 2018).

1.2.   Genetic diversity of *X. fastidiosa* strains and worldwide distribution.

The bacterium responsible for Pierce's disease was first cultured in vitro in the 1970s (Davis et al. 1978). Genetic analyses showed differences between strains that allowed their categorisation into subspecies. The first comprehensive phylogenetic organisation of *X. fastidiosa* strains was provided by a multi locus sequence typing (MLST) framework based on seven loci (Scally et al. 2005). The MLST database currently contains 147 sequences and 87 profiles (https://pubmlst.org/X.fastidiosa/). As strains were organised using this tool, considerable host specialisation by sequence types was observed. *X. fastidiosa* ssp. *fastidiosa* causes Pierce's disease of grapevines but also has an extremely wide host range including almonds and lucerne (Purcell 1980b). A high percentage (>75% of those tested) of crops, wild plants and weed species can carry Pierce's disease strains without symptoms (Raju et al. 1983; Hopkins and Adlerz 1988; Hill and Purcell 1995). *X. fastidiosa* ssp. *multiplex* causes diseases of peach, plum, almond, maple, elms, sycamore and blueberry in North America. *X. fastidiosa* ssp. *pauca* is pathogenic on citrus and coffee in South America, and olives in Italy and southeast Brazil. *X. fastidiosa* ssp. *pauca* ST53 has been isolated from several other hosts associated with Italian olive orchards including *Catharanthus*, *Myrtus* and *Prunus* species. *X. fastidiosa* ssp. *morus* causes a disease of mulberry (*Morus* spp.) in the United States of America (USA) (Nunney et al. 2014b) and *X. fastidiosa* ssp. *sandyi* affects oleander in the southern California regions of USA (Nunney et al. 2013). *X. fastidiosa* ssp. *morus* was shown to be the result of recombination between *X. fastidiosa* ssp. *fastidiosa* and *X. fastidiosa* ssp. *multiplex* (Nunney et al.

2014b). The origin of *X. fastidiosa* ssp. *sandyi* is yet to be determined (Vanhove et al. 2019). A sixth subspecies, *tashke*, has recently been isolated from *Chitalpa tashkenensis* in southwestern USA (Randall et al. 2009; Mendonca et al. 2017), although this identification is controversial (Rodrigo Almeida, pers. comm).

X. fastidiosa was the first plant pathogen bacterium to have a fully sequenced genome (Simpson et al. 2000) and strains are increasingly characterised by whole genome sequencing. Based on a phylogenetic analysis of 72 genome sequences, the separation of five *X. fastidiosa* subspecies was confirmed (Vanhove et al. 2019), and the timings of *X. fastidiosa* movements into different geographical regions was estimated. The introduction of *X. fastidiosa* ssp. *fastidiosa* into USA was estimated as c. 1904, close to the recorded introduction into the USA in 1892 (Vanhove et al. 2019). Strains found on the eastern and western US coasts were separated from a common ancestor by a similar amount of time, suggesting human assisted spread.

X. fastidiosa ssp. *pauca* was estimated to have originated in Brazil in 1597, and the separation of the strains associated with CVC and coffee leaf scorch (CLS) was estimated to have occurred in 1254. *X. fastidiosa* ssp. *pauca* isolates from Costa Rica were dated to 1943, and their introduction to southern Italy to 2008 (Vanhove et al. 2019).

1.3. Genetics and genomics of *X. fastidiosa*

The average size of 72 *X. fastidiosa* genomes was 2.5Mbp (Vanhove et al. 2019). Some 622 genes were shared by 99% of isolates (the "core" genome), and 291 genes were shared by 95–99% of isolates. *X. fastidiosa* ssp. *fastidiosa* genomes were the most clonal, and *X. fastidiosa* ssp. *pauca* the most genetically diverse (Vanhove et al. 2019). Fifty-nine unique genes were found in ssp. *fastidiosa*, 457 in ssp. *multiplex*, 96 in ssp. *pauca* and four in ssp. *sandyi.* Amongst these genes were those responsible for pathogenicity, hemagglutinin, polysaccharide deacetylase, regulation of cell shape and adhesion to surfaces (Vanhove et al. 2019).

Analysis showed that the ssp. *fastidiosa* and ssp. *multiplex* genomes had strong signs of adaptation. The number of genes possibly experiencing selection was highest for ssp. *pauca* (90), followed by ssp. *fastidiosa* (76) and ssp. *multiplex* (43). The functions of these genes differed; for ssp. *fastidiosa* these genes were associated with pilus assembly, N-terminal protein amino acid acetylation, response to oxidative stress, and manganese ion transmembrane transport. For ssp. *multiplex*, genes were associated with lipoprotein transport or response to antibiotics, and for ssp. *pauca* responses to nitroactive stress, biofilm formation or oxygen transport (Vanhove et al. 2019).

Analysis showed that ssp. *multiplex* had the highest number of recombination events (221 genes) of which 206 (93.2%) originated from ssp. *fastidiosa* (Vanhove et al. 2019). Recombining regions were found in 40% of the ssp. *fastidiosa* strains, 73.3% of the *multiplex* strains, and 45% of the ssp. *pauca* strains (Vanhove et al. 2019).

X. fastidiosa ssp. *pauca* is the most widely disseminated subspecies and results from there analyses showed it is the most genetically diverse subspecies and also showed the strongest signs of

adaptation. *X. fastidiosa* ssp. *pauca* did not have the most unique genes despite being the most ancient lineage, perhaps because it has exchanged genetic material with other subspecies, notably *X. fastidiosa* ssp. *fastidiosa*.

X. fastidiosa ssp. *fastidiosa* strains were the most clonal. Of the 20 strains sequenced, 18 were from US grapevines. It also showed strong signs of adaptation, consistent with a relatively recent introduction to a new environment, and possibly a new host.

X. fastidiosa ssp. *multiplex* has the highest number of unique genes, and also showed strong signs of adaptation, probably because of the relatively recent introduction of a large number of new hosts (cultivated crops and ornamental plants) into North America following European colonisation.

1.4.   Pathogenicity and host interactions

The ability of *X. fastidiosa* to form biofilms (a robust surface-attached population) is integral for pathogenicity, and is required for colonising the mouth parts of insect vectors and the wall of xylem cells (Rapicavoli et al. 2018). When *X. fastidiosa* cells reach the plant xylem, they can migrate basipetally against the flow of the plant sap by using Type IV pili, despite the strong currents generated during transpiration. Calcium signalling enhances twitching motility. Acropetal transport to the tips of the plant is probably passive in the transpiration stream. Movement through the plant is facilitated by cell wall active enzymes that degrade the pit membranes between xylem vessels (Rapicavoli et al. 2018).

X. fastidiosa cell concentration in the xylem is regulated, with only 10–15% of vessels heavily colonised and most vessels containing just a few cells. At low densities the bacterium uses biochemical pathways associated with colonisation and movement inside the plant, but at high densities alternate pathways are activated that facilitate insect acquisition (Newman et al. 2003; Baccari and Lindow 2011). The production of adhesins increases cell 'stickiness' and thus the chances of acquisition by an insect vector. *X. fastidiosa* may regulate movement within the plant using outer membrane vesicle (OMVs) production to adjust its adhesiveness. Studies have shown that insects are deterred from feeding on water stressed plants (Andersen et al. 1992; McElrone et al. 2001), and that the bacterial population is greatest just before disease expression (Daugherty et al. 2017). The switch in pathways from multiplication and spread inside the plant host to insect acquisition is probably an adaptation to maximise spread of the bacterium by reducing population expansion to just below symptomatic, thus encouraging vector feeding (Sicard et al. 2018).

Early colonisation of insect vectors probably occurs in two stages; during the initial stage cells must adhere to the cuticular surface of the vector foregut via adhesins. The next phase of colonisation, which results in long-term persistence observed for several months, is not well understood, but may involve the use chitin as a food source (Killiny et al. 2010; Labroussaa et al. 2017). While attached to the foregut, cells still need to be released in order to infect plants (Killiny and Almeida 2014). Release can be almost immediately after acquisition. There is no latent period required for transmission (Purcell and Finlay 1979) and vectors with undetectable *X. fastidiosa* titres can transmit the bacterium at substantial rates (Hill and Purcell 1995).

In contrast to most other plant pathogenic bacteria, *X. fastidiosa* has Type I, II, IV and V secretion systems, but lacks a Type III secretion system. These systems are used to secrete protein effectors that enhance virulence and/or suppress host defences. The Type I system secretes proteases, hydrolases and toxins, as well as having a pathway responsible for multidrug resistance (Rapicavoli et al. 2018). The Type II secretion system (T2SS) is closely related to those found in *Xanthomonas* spp. Proteins transported by this system are often proteases and plant CWDEs, and the majority of those found in *X. fastidiosa* are produced by the T2SS (Rapicavoli et al. 2018). The function of the Type IV secretion system is unknown (Rapicavoli et al. 2018). The Type V system consists of a family of autotransporter proteins that may have a function in adhesion (Rapicavoli et al. 2018).

1.5 Pathogen — Summary points

- *X. fastidiosa* is an obligate inhabitant of plant xylem cells. It colonises the foregut of insect vectors.
- Genomic research shows that *X. fastidiosa* can be divided into genetically distinct subspecies ssp. *fastidiosa*, *multiplex*, *pauca*, *morus*, *sandyi*.
- Plant diseases are associated with specific genotypes and host combinations.

2. Hosts

2.1 Which plants are infected by *X. fastidiosa*?

X. fastidiosa infects a wide range plants including many hardwood species and some herbaceous hosts, crops and ornamentals (Lopes et al. 2003). A comprehensive and regularly updated host list is maintained at the European Food Safety Authority (EFSA) (2018). The most recent update of *X. fastidiosa* hosts was released on 11 April 2019 12th update EFSA. The "xylella spp. host plants database 2019" (https://zenodo.org/record/2536025 released in Jan 2019) served as the major host record reference for this review. This document collates information on detections from a wide array of sources, although the majority are observations in the field. Few records have been supported by rigorous infection experiments under controlled conditions (Sicard et al. 2018) although the EFSA records do include artificial infections made by mechanical inoculation (budding, grafting, needle, root uptake, stem absorption, syringe) or experimental vector transmission. Recorded detection methods for *X. fastidiosa* span bacterial isolation, microscopy, immunological techniques (e.g. Enzyme-linked immunosorbent assay (ELISA)), the polymerase chain-reaction (PCR), and DNA sequencing.

More than 350 species are recognised as hosts of *X. fastidiosa* (Sicard et al. 2018), with the current list consisting of 359 plant species (including hybrids) from 204 genera and 75 families (European Food Safety Authority 2018). A high percentage of experimentally-tested plant species have proven to be hosts. Information on *X. fastidiosa* host status for a list of crop plants in New Zealand is shown in Table 1. This information was summarised from the EFSA records (https://zenodo.org/record/2536025) and shows the number of samples examined and the number of positive detections. Because of the complexity of the database, and because many crop plants of

interest have not been examined, the records shown in Table 1 are filtered at the plant genus level. For example, five positive detections have been made from *Asparagus*, but all are from the evergreen wild asparagus plant, *Asparagus acutifolius*. Ten positive records for *Helianthus* are from the sunflower, *Helianthus annuus*. We have chosen this approach because we think that infection within a genus increases the likelihood that closely related plant species will also be susceptible to infection, but this remains to be confirmed. In addition to these crop plants, the infection status of a small number of New Zealand native plants is shown in Table 1.

2.2.   Host infection process and symptom development

Many of the plant-*Xylella* relationships are commensal, with only a small number of genotype—host relationships resulting in plant diseases (Almeida and Nunney 2015; Sicard et al. 2018). Indeed, it has been suggested that *X. fastidiosa* is possibly an endophyte (Chatterjee et al. 2008). In many plants, *X. fastidiosa* persists at insect feeding or mechanical inoculation sites but it appears that many such asymptomatic infections decrease over time (Purcell and Saunders 1999). This raises the possibility that some asymptomatic hosts are infected only temporarily (Almeida and Nunney 2015). In symptomless hosts, *X. fastidiosa* likely multiplies to lower populations and does not move systemically through the plant. Hosts can be classified as propagative or non-propagative, systemic or non-systemic, and symptomatic or asymptomatic (Purcell and Saunders 1999). For example, blackberry (*Rubus* spp.) is a systemic host, whereas *X. fastidiosa* multiplies in mugwort (*Artemisia douglasiana*) without becoming systemic (Hill and Purcell 1995).

Colonisation of the plant by *X. fastidiosa* is not well understood. Xylem vessels are connected by 5–20 nm diameter pit membranes that allow transport of water and small molecules, but impede pathogens (Rapicavoli et al. 2018). Nevertheless, *X. fastidiosa* moves through pit membranes, possibly facilitated by extracellular enzymes (Baccari and Lindow 2011; Almeida and Nunney 2015). Infected plants respond to *X. fastidiosa* invasion by producing gels and tyloses. At this time it is unclear if these may inhibit infection or in fact contribute, along with pathogen cells, to vessel obstruction, i.e. the vascular occlusion hypothesis (Goodwin et al. 1988). Symptoms of *X. fastidiosa* diseases are similar to the physiological response to water deficit (Sun et al. 2013). However, some gene alterations induced by *X. fastidiosa* infection are considered responsible for symptoms not associated with water stress (Rapicavoli et al. 2018).

Few annual plants are known to display disease symptoms, possibly because disease symptoms result from the gradual development of *X. fastidiosa* titre in the plant and blocking of xylem cells, by which time most annual plants will have been harvested or have senesced. Supporting this view are experiments showing maximum development of symptoms in those lucerne plants with the highest levels infection and after multiple/later harvests (Lopes et al. 2010). Inoculation of ragweed (*Ambrosia artemisiifolia*) with *X. fastidiosa* produced stunt symptoms after 11 months (Timmer et al. 1983). In *Catharanthus roseus* (Madagaskar periwinkle) first symptoms were observed as soon as 2 months after inoculation with CVC strains of *X. fastidiosa* (Monteiro et al. 2001), but took 6 months until infected periwinkle plants showed symptoms of stunting and wilting, and had deformed leaves (Monteiro et al. 2001). Other examples of annuals showing disease include sweet clover (*Melilothus* spp.) (Freitag 1951) and stunting of radish (*Raphinus sativus*) (Lopes et al, unpublished).

2.3   Disease in hosts plants found in New Zealand

In Table 1, New Zealand plant hosts known to show disease symptoms in response to *X. fastidiosa* infection are noted. In this section, further details on the diseases occurring in these plants is provided. Details are also provided in some instances where reports of disease can be considered uncertain or controversial. **For plants where no further details are provided below (e.g. kiwifruit and most annuals), we have been unable to identify evidence of disease.**

111 plant species are listed by the European and Mediterranean Plant Protection Organisation (EPPO) as displaying minor or major disease symptoms (https://gd.eppo.int/taxon/XYLEFA/hosts). Some of the more well-known diseases include: alfalfa dwarf, almond leaf scorch, bacterial leaf scorch, blueberry bacterial leaf scorch, CVC, coffee leaf scorch, crespera, elm leaf scorch, leaf scorch disease, mulberry leaf scorch, oleander leaf scorch, OQDS, pear leaf scorch, pecan bacterial leaf scorch, periwinkle wilt, phony peach disease, Pierce's disease, plum leaf scald, potato purple top disease, ragweed stunt, sweetgum dieback, and sycamore leaf scorch (European Food Safety Authority 2018).

Grapevines (*Vitis*)

The most characteristic symptom of Pierce's disease is leaf scorching — drying of part, and often eventually all, of the leaf. Diseased stems may mature irregularly, with patches of brown and green tissue. Leaves are small and distorted with interveinal chlorosis. Shoots have shortened internodes. Susceptible cultivars rarely survive more than 2–3 years. Young vines succumb more quickly than do older vines. More tolerant cultivars may survive chronic infection for more than 5 years (Hewitt et al. 1942; Goodwin and Purcell 1992). There is limited efficacy of heavy pruning for reducing Pierce's disease in grapevines (Daugherty et al. 2018). No *Vitis vinifera* are known to be immune to Pierce's disease strains of *X. fastidiosa*, but American *Vitis* species may be resistant (Goheen and Hopkins 1988).

Prunus

X. fastidiosa causes peach phony disease (*Prunus persica*) (Wells et al. 1983), which is associated with ongoing losses in the southern US peach industry since the 1890s; in 1973 phony disease was confirmed to share characteristics of Pierce's disease and the bacterium was cultured from diseased peaches in 1983 (Davis et al. 1980; Sicard et al. 2018). All peach cultivars are susceptible. Leaf scald is also seen in plums (*Prunus domestica*), almonds (*P. dulcis*), and apricots (*P. armeniaca*). Plum leaf scald was first reported in Argentina, then Brazil, Paraguay and southeastern USA (Davis et al. 1981). Trees are compact with dark green, stunted new growth due to shorter internodes (Hopkins and Purcell 2002). Leaves and flowers appear early with reduced size and number of fruit until after 3–5 years they become economically worthless. Trees are not generally killed, but suffer fruit yield losses and are susceptible to attack from insects and other diseases. The South American plum leaf scald strains are the result of a single introduction from North America approximately 80 years ago (Coletta-Filho et al. 2017). The plum leaf scald strains in Brazil may have wide plant host ranges (Leite et al. 1997). The plant host range of *Prunus* strains from the south eastern USA has not been investigated extensively.

Citrus

X. fastidiosa causes citrus variegated chlorosis (CVC) in Brazil and Argentina (Brlansky et al. 1993; Chang et al. 1993). Affected trees show foliar chlorosis followed by development of brown lesions on the underside of mature leaves. Fruit size is greatly reduced, with more fruit remaining on the tree. The sugar content of affected fruit is higher than in non-affected fruit, and the fruit has a hard rind. Affected trees show stunting and slow growth rate; twigs and branches die back and the canopy thins, but affected trees do not die (Lee et al. 1991; Chang et al. 1993). Trees of all ages can show symptoms of variegated chlorosis but trees of 1–3 years age become completely colonised by *X. fastidiosa* much faster than do older trees. Symptoms in trees of > 8–10 years old are usually confined to the extremities of branches.

CVC control has been achieved by removing inoculum, production of disease-free nursery trees, and aggressive control of insects (including through the extensive use of pesticides for control of psyllids causing citrus greening). Symptomatic branches are pruned from trees older than 3 years while younger trees are completely removed from orchards. Clean material is propagated from tested material in sharpshooter-free conditions. The disease affects mostly sweet oranges (*Citrus sinensis*).

Olive (*Olea europaea*)

In 2013, *X. fastidiosa* spp. *pauca* was associated with OQDS in the Apulia region of southern Italy (Saponari et al. 2019). Experimental evidence (Saponari et al. 2016) has confirmed *X. fastidiosa* as the causal agent of OQDS in Italy (European Food Safety Authority 2016). OQDS is characterised by leaf scorching and desiccation of small twigs, and eventually, desiccation of the whole canopy and tree collapse (Almeida and Nunney 2015).

The Apulia strain of *X. fastidiosa* was able to reproduce symptoms and systemic infection in olives, oleanders and *Polygala myrtifolia* but not citrus, grapes and *Quercus ilex* (Loconsole et al. 2016). *X. fastidiosa* ssp. *pauca* has been implicated in olive plant dieback and leaf desiccation in Argentina (Haelterman et al. 2015) and Brazil (Coletta-Filho et al. 2016).

Lucerne (*Medicago*)

Widespread stunting of alfalfa plants was observed throughout California in the 1930–40s in parallel with a Pierce's disease epidemic (Lopes et al. 2010). Later, it was confirmed that *X. fastidiosa* could be isolated from these plants and that the same species of vector insects could be found on both lucerne and grapes. At that time, there was a significant research on alfalfa dwarf but problems have declined in California and it is no longer considered a major threat to production. A resistant lucerne variety was released in 1950, and this may have contributed to the decline, although inoculations indicate that some current cultivars are susceptible to disease (Daugherty et al. 2010). Changes in rotation time from 7–8 years (1930s) to 3-4 years may have reduced the time for *X. fastidiosa* to cause disease symptoms, and reduce the amount of *X. fastidiosa* inoculum in the field.

Recent sampling of Californian lucerne fields showed that infection levels were low. The green sharpshooter (*Draeculacephala minerva*) was the most abundant *X. fastidiosa* vector, but showed a preference for weeds over lucerne. Cool winter temperatures reduced *X. fastidiosa* to undetectable levels but did not remove infection from lucerne (Daugherty et al. 2010).

Blueberry (*Vaccinium*)

Bacterial leaf scorch is a disease of southern highbush blueberry cultivars (*Vaccinium corymbosum* interspecific hybrids). Symptoms include leaf scorching, stem yellowing, dieback, drought-like

symptoms and eventual plant death. GWSS is the prevalent vector in blueberry plantings in southern Georgia. Management is limited to the use of resistant cultivars and insecticides for vector control; pruning and mowing are unlikely to be effective in curing affected plants of bacterial leaf scorch (Holland et al. 2014). Both ssp. *multiplex* and ssp. *fastidiosa* isolates caused symptoms in blueberry; two ssp. *multiplex* isolates from blueberry caused more severe symptoms when re-inoculated into blueberry (Oliver et al. 2015).

Avocado (*Persea*)

The first reports of putative *Xylella* infection of avocado were made in Costa Rica (Montero-Astúa et al. 2008), in mountains to the north and south of the Central Valley (Alajuela and San José provinces). Disease symptoms included chlorotic mottling, leaf scorching and deformation, defoliation, shortening of internodes and branch dieback. The pathogen in Costa Rica was not isolated from avocado and the vector is unknown. Subsequent records of avocado infection are very limited; GWSS are known to infest avocado orchards when other suitable hosts are not available or when insect populations are very high. Tests for *X. fastidiosa* have been positive on avocado in some areas of the USA, but no symptoms have been identified or linked to positive *X. fastidiosa* test results (California Avocado Commission and California Minor Crops Council 2003). An industry discussion document suggests *X. fastidiosa* is a concern for Australian growers, although this presents no new evidence of disease (Geering and Parkinson 2019). EPPO lists *Persea americana* as a host with both minor and incidental symptoms (https://gd.eppo.int/taxon/XYLEFA/hosts).

Solanum

A study from Mexico has previously linked *X. fastidiosa* infection to potato purple top disease (Gutiérrez-Ibáñez et al. 2009). However, this report is now considered unconfirmed/dubious for *Solanum tuberosum* (European Food Safety Authority 2018). There has been no further association of potato purple top disease or 'zebra chip' diseases with *X. fastidiosa*. Potato purple top disease has since been linked with other pathogens such as *Candidatus* Phytoplasma americanum (Lee et al. 2006) and *Candidatus* Liberibacter solanacearum (Liefting et al. 2009). There are no clear-cut reports of *X. fastidiosa* disease in other *Solanum* spp. such as tomato (*Solanum lycopersicum*).

Pears (*Pyrus*)

Leaf scorching was seen on Taiwanese pear trees (*Pyrus pyrifolia*) in 1990 (Leu and Su 1993). Scorching was followed by twig dieback and even tree death within a few years. Genomic sequences from the xylem-limited bacterium associated with this disease showed that it was a different *Xylella* spp. (Su et al. 2016).

Native New Zealand plants

Screening of New Zealand native plants has been carried out on plants growing oversees in *X. fastidiosa* infested regions, primarily in California where the dominant vector-pathogen combination is GWSS and ssp. *fastidiosa* (Groenteman et al. 2015). A small number of other samples have been screened during surveys in the Northern hemisphere (European Food Safety Authority 2018); knowledge of infection in New Zealand natives is biased towards those plants common in landscape or ornamental plantings overseas. The native plants listed in Table 1 are those considered to be clearly hosts (European Food Safety Authority 2018) but the selection of native plants capable of being hosts is almost certainly higher (Groenteman et al. 2015).

Amenity plants and weeds

X. fastidiosa infects and causes disease in a wide range of hosts that serve primarily as ornamental or amenity plants in New Zealand. Many woody hosts such as maples (*Acer rubrum*), oaks (*Quercus rubra*) and elms (*Ulmus americana*) suffer from such disease in USA (Hearon et al. 1980; Sherald et al. 1987; Chang and Walker 1988). One of the most high profile diseases of ornamentals is oleander leaf scorch of *Nerium oleander*, which was first observed in southern California in the 1990s. Caused by ssp. *fastidiosa*, oleander leaf scorch is now widespread in the southern USA. It is a highly damaging disease; plants decline and die within 3–5 years of first symptoms. Another major *X. fastidiosa* disease is found in myrtle-leaf milkwort (*Polygala myrtifolia*), an ornamental plant that is a relatively common weed in parts of Auckland and Northland (Rebecca Stanley, pers. comm.).

2.4   Hosts — Summary points

- *X. fastidiosa* has a very wide host range, with most infections remaining asymptomatic.
- It seems possible that almost any plant can be a host, given the presence of a xylem-sap feeding vector.
- Specific combinations of hosts and *X. fastidiosa* genotypes result in plant disease.
- Most hosts showing disease are woody perennials; fewer annual plants show symptoms.
- It is currently impossible to predict the development of new diseases caused by *X. fastidiosa*.
- Many native plant species in New Zealand can act as *X. fastidiosa* hosts and several are regarded as showing symptoms in response to infection.
- Amenity, ornamental and weed plants known to host *X. fastidiosa* are common and widespread in New Zealand.

Table 1. Infection status for plants grown in New Zealand. The list of crop plants was developed in consultation with a range of New Zealand agriculture/horticulture industry groups. The number of screened plant sample records from the European Food Safety Authority (EFSA) and number of samples with positive *Xylella fastidiosa* infection were filtered at the genus level. 'Disease symptoms' summarises records from European and Mediterranean Plant Protection Organisation (EPPO). NP = no records present for these hosts in the EFSA data. ND = not assessed in this work because there a multiple minor plant species is these categories. * = a named plant disease associated with *Xylella* spp. but not listed in these EPPO records.

Organisation	Common name	Family	Genus	Species	Positive detections in plants tested (EFSA)	Number of plants tested (EFSA)	Disease symptoms
New Zealand Winegrowers	Grapevines	Vitaceae	*Vitis*	spp.	1278	1568	Major
Summerfruit New Zealand	Cherries	Rosaceae	*Prunus*	*avium*	942	1110	Minor
	Peaches	Rosaceae	*Prunus*	*persica*	942	1110	Minor
	Apricots	Rosaceae	*Prunus*	*armeniaca*	942	1110	Minor
	Nectarines	Rosaceae	*Prunus*	*persica*	942	1110	Minor
Dairy NZ	Ryegrass	Poaceae	*Lolium*	*perenne*	5	5	-
	Clover	Fabaceae	*Trifolium*	spp.	10	12	-
	Plantain	Plantaginaceae	*Plantago*	spp.	1	8	-
	Maize, sweetcorn	Poaceae	*Zea*	*mays*	NP	NP	-
	Barley	Poaceae	*Hordeum*	*vulgare*	4	7	-
	Wheat	Poaceae	*Triticum*	*aestivum*	NP	NP	-
	Oats	Poaceae	*Avena*	*sativa*	3	4	-
	Fodder beet, beetroot, silverbeet	Amaranthaceae	*Beta*	*vulgaris (ssp. vulgaris)*	0	1	-
	Brassicas	Brassicaceae	*Brassica*	spp.	4	5	-
	Lucerne (Alfalfa)	Fabaceae	*Medicago*	*sativa*	113	124	Minor
New Zealand Apple and Pears	Apples	Rosaceae	*Malus*	spp.	1	3	-
	Pears	Rosaceae	*Pyrus*	spp.	81	84	-
Kiwifruit Vine Health	Kiwifruit, kiwiberry	Actinidiaceae	*Actinidia*	spp.	NP	NP	-

New Zealand Avocado	Avocado	Lauraceae	*Persea*	*americana*	2	3	Minor
New Zealand Citrus Growers Incorporate	Orange	Rutaceae	*Citrus*	x sinensis	1212	1522	Major
	Lemon	Rutaceae	*Citrus*	x limon	1212	1522	Minor
Vegetables	Artichokes (Globe)	Asteraceae	*Cynara*	*cardunculus* var. *scolymus*	NP	NP	-
Horticulture New Zealand	Artichokes (Jerusalem)	Asteraceae	*Helianthus*	*tuberosus*	10	16	-
Vegetables New Zealand Incorporate	Asian Vegetables				ND	ND	-
Process Vegetables New Zealand	Beans	Fabaceae	*Phaseolus/Vicia*	spp.	5	6	-
Tomatoes New Zealand	Box Thorn	Solanaceae	*Lycium*	chinense	NP	NP	-
	Burdock	Asteraceae	*Arctium*	spp.	NP	NP	-
	Capsicums, Chilli peppers	Solanaceae	*Capsicum*	spp.	NP	NP	-
	Carrots	Apiaceae	*Daucus*	carota	1	2	-
	Celery, Celeriac	Apiaceae	*Apium*	*graveolens* (var. *rapaceum*)	0	1	-
	Chokos	Cucurbitaceae	*Sechium*	*edule*	NP	NP	-
	Courgettes, Scallopini, Zucchini, Marrows, Pumpkin	Cucurbitaceae	*Cucurbita*	*pepo*	0	1	-
	Cucumbers, Gherkins	Cucurbitaceae	*Cucumis*	*sativus*	NP	NP	-
	Egg Plant (or aubergine)	Solanaceae	*Solanum*	*melongena*	1	1	-
	Florence Fennel	Apiaceae	*Foeniculum*	*vulgare*	0	2	-
	Garland chrysanthemum	Asteraceae	*Glebionis*	*coronaria*	NP	NP	-

	Herbs				ND	ND	-
	Indian Vegetables		Variety		ND	ND	-
	Kumara	Convolvulaceae	*Ipomoea*	*batatas*	4	6	-
	Leeks	Amaryllidaceae	*Allium*	*ampeloprasum* 'Leek Group'	NP	NP	-
	Lettuce	Asteraceae	*Lactuca*	*sativa*	5	8	-
	Melons	Cucurbitaceae	*Cucumis/Citrullus*	spp.	NP	NP	-
	Okra	Malvaceae	*Abelmoschus*	*esculentus*	NP	NP	-
	Parsnips	Apiaceae	*Pastinaca*	*sativa*	NP	NP	-
	Peas	Fabaceae	*Pisum*	*sativum*	NP	NP	-
	Puha	Asteraceae	*Sonchus*	*kirkii*	5	9	-
	Purslane	Portulacaceae	*Portulaca*	*oleracea*	5	9	-
	Radishes (including Chinese radish & daikon)	Brassicaceae	*Raphanus*	*raphanistrum* ssp. *sativus*	0	2	-
	Rhubarb	Polygonaceae	*Rheum*	spp.	1	1	-
	Salad Leaves		Multiple		ND	ND	-
	Salsify	Asteraceae	*Tragopogon*	*porrifolius*	NP	NP	-
	Scorzonera	Asteraceae	*Scorzonera*	*hispanica*	NP	NP	-
	Shallots, Spring onions, Garlic, Rakkyo, onion	Amaryllidaceae	*Allium*	*cepa* (var. *aggregatum/sativum*)	NP	NP	-
	Spinach	Amaranthaceae	*Spinacia*	*oleracea*	NP	NP	-
	Sprouted Beans and Seeds	Fabaceae	*Vigna*	*radiata*	NP	NP	-
	Squash	Cucurbitaceae	*Cucurbita*	*moschata*(?)	0	1	-
	Taro	Araceae	*Colocasia*	*esculenta*	NP	NP	-
	Tomato	Solanaceae	*Lycopersicon*	*esculentum*	NP	NP	-
	Ulluco	Basellaceae	*Ullucus*	*tuberosus*	NP	NP	-

	Watercress	Brassicaceae	*Nasturtium*	*officinale*	0	2	-
	Witloof (or chicory or endive)	Asteraceae	*Cichorium*	*intybus*	NP	NP	-
	Yakon	Asteraceae	*Smallanthus*	*sonchifolius*	NP	NP	-
	Yams	Oxalidaceae	*Oxalis*	*tuberosa*	0	1	-
HortNZ - umbrella	Aparagus	Asparagaceae	*Asparagus*	*officinalis*	5	6	Minor
	Blueberry	Ericaceae	*Vaccinium*	sect. *Cyanococcus*	62	76	Minor
	Blackcurrant	Grossulariaceae	*Ribes*	*nigrum*	NP	NP	-
	Boysenberry	Rosaceae	*Rubus*	*ursinus × R. idaeus*	28	35	-
	Feijoa	Myrtaceae	*Acca*	*sellowiana*	NP	NP	-
	Passionfruit	Passifloraceae	*Passiflora*	*edulis*	2	2	-
	Persimmon	Ebenaceae	*Diospyros*	spp.	NP	NP	-
	Potatoes	Solanaceae	*Solanum*	*tuberosum*	11	18	-
	Strawberry	Rosaceae	*Fragaria*	spp.	2	3	-
	Tamarillo	Solanaceae	*Solanum*	*betaceum*	11	18	-
New Zealand natives	Ngaio	Scrophulariaceae	*Myoporum*	*laetum*	4	9	Minor
	Taupata	Rubiaceae	*Coprosma*	*repens*	6	9	Minor
	Koromiko	Plantaginaceae	*Hebe*	spp.	8	8	Minor
	Pohutukawa	Myrtaceae	*Metrosideros*	*excelsa*	10	16	Minor
	Akeake	Sapindaceae	*Dodonaea*	*viscosa*	2	2	Minor

# 3.	Vectors

## 3.1	Types of insect vectors for *X. fastidiosa*

X. fastidiosa is transmitted to plants by sharpshooter leafhoppers (order Hemiptera) that feed exclusively or almost exclusively from the plant xylem vessels. Xylem "specialists" belong to the taxonomic families Cicadellidae (subfamily Cicadellinae; sharpshooter leafhoppers), Cercopidae (spittlebugs), Machaerotidae (tube-building spittlebugs), and Cicadidae (cicadas) (Almeida and Nunney 2015). Sharpshooters and spittlebugs are the most prominent vectors of *X. fastidiosa* (Purcell 1990). All insects from these two groups have so far been found capable of transmitting this bacterium (Frazier 1965; Sicard et al. 2018), and transmission is not strain specific.
Species that occasionally feed from xylem, but are not xylem-feeding specialists, have never been shown to be capable of transmitting *X. fastidiosa* (Purcell 1980b). In olive groves, 16 of 46 phloem-feeding *Euscelis lineolatus* (Cicadellidae, Deltocephalinae) leafhoppers were positive for *X. fastidiosa* (Elbeaino et al. 2014). However, there was no evidence for transmission of the bacterium. This is consistent with the observation that other sap-sucking insects may acquire plant pathogens but not act as vectors (Purcell and Almeida 2005). Plants where *X. fastidiosa* infection becomes propagative and systemic hosts are the best hosts for vector acquisition, but *X. fastidiosa* can still be acquired from non-systemic hosts. Acquisition efficiency is proportional to the populations of live bacterial cells within plant tissues (Hill and Purcell 1997).

## 3.2	Insect colonisation by *X. fastidiosa*

X. fastidiosa cannot be transmitted by transovarial means; that is, it cannot be passed from mother to offspring (Freitag 1951). Nymphs and adults must gain *X. fastidiosa* from infected plants. Once acquired, *X. fastidiosa* propagates within the insect foregut but does not cross the insect gut wall to circulate in the haemolymph. *X. fastidiosa* is unique among vector-borne plant pathogens, since it is persistent and propagative but not circulative within its insect vector (Hill and Purcell 1995). Adults can transmit throughout their lifespan (up to 122 days tested in *Graphocephala atropunctata*) (Severin 1949, 1950; Turner and Pollard 1959). Infectivity is lost from nymphs during moulting (Turner and Pollard 1959; Almeida and Purcell 2003). Studies indicate that transmission efficiency is not affected by insect gender (Severin 1950; Krugner et al. 2019). All insects in these groups appear to be capable of vectoring all *X. fastidiosa*, but there are differences in efficiency in plant-vector-pathogen combinations (Redak et al. 2004; Almeida and Nunney 2015).
X. fastidiosa colonises and forms a biofilm on the insect foregut; bacterial cells are introduced into the plant during stylet probing. For all insect vectors studied there is no detectable latent period for transmission. Transmission efficiency increases with the time that a vector feeds from an infected host and with the time on a new host (Almeida and Nunney 2015).

## 3.3	Known vectors of *X. fastidiosa*

In the Americas, where *X. fastidiosa* is thought to have originated (Nunney et al. 2014a), efficient insect vectors of *X. fastidiosa* can be found in several families (above), however the Cicadellidae

contains the largest number of vectors (Redak et al. 2004). A complete list of insect vectors of *X. fastidiosa* in the southern US can be found in Overall and Rebek (2017). There are over 60 known vector species of *X. fastidiosa* in North America affecting a wide variety of agricultural and natural ecosystems (Redak et al. 2004; Krell et al. 2007; Myers et al. 2007; Zhang et al. 2011; Overall and Rebek 2015, 2017). The Cicadellidae species, GWSS, *Homalodisca vitripennis* (Germar), is an important vector *X. fastidiosa* in some geographical locations (Overall and Rebek 2017) due to its broad host range, frequent host switching, abundance in crops and strong flying ability. GWSS, like a number of other vectors, overwinters as an adult; insects that do so are more important in disease prevalence as they are able to infect plants earlier in the new season (Purcell 1997). For regions with freezing winter temperatures, most infections after the early growing season do not survive the subsequent winter (Purcell 1981; Feil et al. 2003). As winter approaches, adult GWSS migrate into forest areas and undergo incomplete hibernation in the spring.

While GWSS is not present in New Zealand and has never been intercepted at the New Zealand border (D. Gunawardana, Biosecurity New Zealand, personal communication, 22 July 2019) its importance as a vector and its spread across the Pacific makes this insect a vector of special interest to New Zealand. The isolated location of New Zealand has been considered an advantage due to the extended travel time a 'hitchhiker GWSS' would have to survive without feeding (Rathé et al. 2015). However, GWSS invaded French Polynesia in 1999 and has spread across the Pacific to Hawaii (2004), Easter Island (2005) and the Cook Islands (2007) (Hoddle and Van Driesche 2009). New Zealand and Australia are at risk because of regular air flights to these countries (Hoddle and Van Driesche 2009).

Scenarios for new incursions of *X. fastidiosa* and/or GWSS have been outlined by Rathé et al. (2012). These included the possibility that *X. fastidiosa* may be in a country in a symptomless form (e.g. *X. fastidiosa* appears to have been present in Corsica and the Balearic Islands for some time without being detected or causing pronounced disease symptoms (Denance et al. 2017)), or that *X. fastidiosa* is present but not being moved around by a suitable vector. To investigate this latter possibility, over 100 hosts have been surveyed in Australia without finding *X. fastidiosa* (see citations in (Rathé et al. 2012)). In the absence of GWSS, an incursion of *X. fastidiosa* in New Zealand might be limited in spread, as it was in California before GWSS arrival. In coastal California the incidence of Pierce's Disease is highest near riparian habitats, where the most abundant xylem fluid-feeder is the blue-green sharpshooter*, G. atropunctata* (Redak et al. 2004). The pattern of Pierce's disease in these vineyards (Purcell 1974) suggests that transmission of the pathogen to grapes originates from riparian habitats of the vector (Hewitt et al. 1942). Numerous riparian plant species are hosts of *X. fastidiosa* (Freitag 1951). In the absence of GWSS, spread of *X. fastidiosa* in New Zealand would require other vectors, including native vectors (see below). Transmission of newly introduced strains of *X. fastidiosa* by native vector species is considered likely (Almeida et al. 2005).

X. fastidiosa has been reported in several Asian countries, however some of the detections were unconfirmed or later refuted (see Amanifar et al. 2014; Cornara et al. 2019). Data on Asian vectors are currently available only from Taiwan, where the sharpshooters *Kolla paulula* (Walker) and *Bothrogonia ferruginea* (Hemiptera: Cicadellidae) have been identified as vectors of pear leaf scorch disease (Su et al. 2016; Tuan et al. 2016; Cornara et al. 2019). *K. paulula* is frequently found on weeds around pear orchards and grape vineyards (Chang et al. 2014; Tuan et al. 2016) and might be more effective than *B. ferruginea* as a vector of *X. fastidiosa* (Tuan et al. 2016). Both species have been shown capable of transmitting *X. fastidiosa* to grapevines (Chang et al. 2014; Tuan et al. 2016). Additionally, *Xyphon* sp. (Hemiptera: Cicadellidae), *Anatkina horishana* Matsumura (Hemiptera:

Cicadellidae), and *Poophilus costalis* Walker (Hemiptera: Aphrophoridae), collected in the shrubs and weeds near vineyards, were found to be carrying *X. fastidiosa*, but no transmission experiments were conducted (O'Connell et al. 2012).

Following the recent introduction of *X. fastidiosa* to Italy, a thorough analysis of potential vectors was undertaken. Of 119 potential vectors that feed on xylem sap (Chauvel et al. 2015), only the meadow spittlebug (*Philaenus spumarius*) has been identified as an effective vector of *X. fastidiosa* in southern Italy (Saponari et al. 2014; Cornara et al. 2016). The meadow spittlebug is common and abundant in diverse European ecosystems, feeding on nearly 400 recorded hosts including mono- and dicotyledonous grasses, trees and shrubs (Cornara et al. 2018b). In Italy, *P. spumarius* completes one generation per year, and develops as a low mobility nymph on weeds during the spring (Morente et al. 2018). The nymphs are commonly found on herbaceous plants belonging to the Asteraceae, Fabaceae, Apiaceae and Geraniaceae families. (Wiegert 1964; Mangan and Wutz 1983). It moves beneath olive trees and then to olive canopy during the dry period (Cornara et al. 2017). *P. spumarius* has the potential to live under different environmental conditions as long as the host plants are actively growing and not subjected to severe water stress (Cornara et al. 2018a). Although able to both jump and fly, the travel distance of adults seems to be limited to short-distance flights (Cornara et al. 2018a). Mark-release-recapture experiments in Italy showed that *P. spumarius* had a mean dispersal of 60–70 m over 15 days (Plazio et al. 2017). Two other species of spittlebugs, *Philaenus italosignus* and *Neophilaenus campestris,* have been shown to transmit *X. fastidiosa* to olive trees under experimental conditions, although their role in field epidemiology is unknown (Cavalieri et al. 2018).

There are striking differences in the fauna of xylem-sap feeding insects between the Americas and Europe (Bosco et al. 2014). Unlike North and South America, there are a few species of sharpshooters present in Europe, and these have a limited distribution (Wilson et al. 2009). Spittlebugs and cicadas are considered the most likely vectors in Europe (European Food Safety Authority 2015). Spittlebugs have no close relatives, but are distantly related to both leafhoppers and cicadas. They feed on a wide variety of plants, including grasses, herbs, and trees. Thirty-six spittlebug species (families Aphrophoridae and Cercopidae) are present in Europe and many species are common and widespread (Bosco et al. 2014).

3.4   Potential *X. fastidiosa* vectors in New Zealand

In New Zealand, no species of the subfamily Cicadellinae (i.e. sharpshooter leafhopper) have been recorded. Sixteen species of spittlebug in four genera are recorded from New Zealand (Table 2). All but the recently introduced *Philaenus spumarius* (Linnaeus) and *Bathyllus albicinctus* (Erichson) are endemic to New Zealand. The great majority of the species occur in the North Island. Most of the endemic spittlebugs in New Zealand are closely related, suggesting recent speciation events. In regard to *P. spumarius,* the particular subspecies now found in New Zealand is the same as that common in North America, and in the Europe represented mainly in central Europe and southern England (Hamilton 1979). The New Zealand population appears to be genetically rather homogeneous, suggesting a single introduction with subsequent dispersal from one site (Hamilton & Morales 1992). Native spittlebugs in New Zealand seem to favour woody plants, notably manuka (*Leptospermum scoparium*), rata (*Metrosideros umbellata*) and *Coprosma* spp. (Hamilton and Morales 1992). *Carystoterpa fingens* and *Carystoterpa vagans* are both widespread and found on numerous host plants. *P. spumarius* is established (Table 2) and widely spread in New Zealand where it prefers mostly herbaceous plants (Hamilton and Morales 1992); it is common in New Zealand

grape growing regions (Nielsen unpublished data). *P. spumarius* is ubiquitous and highly polyphagous; the nymphs can feed on huge varieties of herbaceous plants (from grasses and vegetables to herbs and cereals), while the adults disperse to an even higher number of plant species, including many trees and shrubs (Delong and Severin 1950; Weaver and King 1954). A comprehensive list of host plants of *P. spumarius* nymphs can be found in Weaver and King (1954).

Cicadas are obligate xylem-sap feeders (Cheung and Marshall 1973), and are thus theoretically capable of acting as vectors of *X. fastidiosa*. Nevertheless, previous studies reported only a small percentage of cicadas managed to successfully acquire and transmit the bacteria. In addition, their biology and seasonality may also influence the vector status of cicadas (Purcell 1997). Studies of *Diceroprocta apache* in grapes and *Dorisiana viride* in *Coffea* spp. report that cicadas are capable of *X. fastidiosa* transmission (Paião et al. 2002; Krell et al. 2007). Paião et al. (2002) tested an additional Cicadidae species on coffee plants but did not identify it.

New Zealand has a large cicada fauna, all of which are endemic. This consists of 42 species and subspecies in five genera, with additional species yet to be formally described. New Zealand cicadas are found in a variety of habitats, including tall forest, scrub, grasslands, swamps, riverbeds and sand dunes. Most of the forest species are found in North Island, while South Island is characterised by a cicada fauna that mostly live in rocky open spaces (Larivière 2019). Chorus cicadas (*Amphipsalta zelandica*) and, in some areas, clapping cicadas (*Amphipsalta cingulata*), occur in kiwifruit orchards in both North and South Islands. The red-tailed cicada *Rhodopsalta cruentata* is an open habitat species and is more likely to feed and lay eggs in grapevines than chorus cicadas which tend to be forest species. Cicadas cause some feeding damage apple orchards in Motueka, and presumably elsewhere in New Zealand (information on cicadas from Shona Seymour, Rebecca Campbell, David Logan pers. comm.). With their presence in New Zealand orchards, the role of cicadas as vectors of *X. fastidiosa* requires more research; their vector status overseas is unconfirmed (Cornara et al. 2019).

Table 2: Species of spittlebugs (Cercopidae: Aphrophorinae) recorded from New Zealand. The information is sourced from Hamilton and Morales (1992) and Larivière et al. (2010). A = adventive; E = endemic; N = native but not endemic to New Zealand.

Species	Collected from[1]	Potential host plant (New Zealand)[2]
Basilioterpa bullata (E)	Tree Kings Islands	Puka/pukanui (*Meryta sinclairii*) *Myrsine* *Pittosporum*
Bathyllus albicinctus (A)	Northland	Shore bindweed *(Calystegia soldanella)* Purple ragwort *(Senecio elegans)* Ivy (*Senecio mikanioides*) White Arctotis (*Arctotis stoechadifolia*) Cocksfoot (*Dactylis glomerata*) Peavine (*Lathyrus latifolius*)
Carystoterpa aurata (E)	Northland	Host unknown
C. chelyon (E)	Northland	Manuka *(Leptospermum scoparium)*

C. minor (E)	Northland Bay of Plenty Coromandel Wanganui	Host unknown
C. subtacta (E)	Macauley Island (Kermadecs)	None recorded
C. subvirescens (E)	Northlands Auckland Poor Knights Islands	Tauhinu (*Ozothamnus leptophyllus*) *Coprosma* Toetoe (*Cortaderia toetoe*) Prickly Mingimingi *(Cyathodes juniperina)* *Metrosideros* *Hebe* Houpapa *(Pseudopanax lessonii)* Rushes Ferns Grasses Coastal vegetation
C. tristic (E)	Chatham Island	*Coprosma* Pohuehue (*Muehlenbeckia australis*) *Plagianthus* Pasture Ferns Coastal vegetation
C. trimaculata (E)	Three Kings Islands	None recorded
C. fingens (E)	Three Kings Islands Northland Auckland Bay of Plenty Wanganui. Taranaki Wellington Marlborough Sounds Nelson	*Astelia* Cabbage trees *(Cordyline australis)* Shiny Karamu (*Coprosma lucida*) Macrocarpa *(Coprosma macrocarpa)* New Zealand Laurel (*Coprosma repens*) *Geniostoma* *Hebe* Hollyhock *Hymenanthera* Manuka *(Leptospermum scoparium)* *Melicytus* Pohutukawa *(Metrosideros excelsa)* *Muehlenbeckia* Ngaio *(Myoporum laetum)*, *Olearia* *Pseudopanax* *Solanum nodifloum* *Tecoma* Coastal shrubs
C. ikana (E)	Northland Waikato Wellington	Host unknown
C. maori (E)	Auckland Wellington Wairarapa	*Coprosma* Grass (genus/species not specified) *Nothofagus*

	Marlborough Sounds Nelson Buller North Canterbury Westland	*Sphagnum*
C. minima (E)	Northland	None recorded
C. vagans (E)	Auckland Bay of Plenty Coromandel Gisborne Hawke's Bay Northland Rangitikei Taranaki Taupo Wairarapa Wanganui Wellington Waikato Buller Dunedin Fiordland Kaikoura Canterbury Nelson Marlborough Sounds Southland Westland	Leafy coprosma (*Coprosma parviflora*) Parataniwha (*Elatostema rugosum*) Ferns *Geniostoma* *Griselinia* Kokomuka (*Hebe elliptica*) *Leptocarpus* Manuka (*Leptospermum scoparium*) Mahoe (*Melicytus ramiflorus*) Pohutukawa (*Metrosideros excelsa*) Red Beech (*Nothofagus fusca*) Tree Daisy (*Olearia arborescens*) White poplar (*Populus alba*) Raukawa (*Pseudopanax edgerleyi*) New Zealand groundsel (*Senecio minimus*) Lilac (*Syringa vulgaris*) Broad bean (*Vicia faba*) Various coastal shrubs
Philaenus spumarius (A)	Hawke's Bay Taranaki Taupo Wanganui Welling Central Otago Dunedin Mid Canterbury MackeNew Zealandie North Canterbury Otago Lakes South Canterbury	Found on a wide variety of plants, including: Californian thistle (*Cirsium arvense*) *Coriaria* Wild carrot *(Daucus carota)* *Hieracium* spp. Introduced grasses *Lavandula* Alfalfa *(Medicago sativa)* *Sonchus* *Clover (Trifolium)* *Aciphylla* *Celmisia* *Hebe* Ornamentals
Pseudaphronella jactator (E)	Bay of Plenty Coromandel Gisborne Hawke's Bay Rangitikei	*Blechnum* *Coprosma* *Dracophyllum* *Nothofagus* Mountain Holly (*Olearia ilicifolia*)

	Taranaki Taupo Wellington Waikato	Subalpine scrub vegetation

[1] Collecting location (area codes) based on the Crosby et al. (1998) specimen localities recording maps in the New Zealand sub region

[2] The records are based around potential true hosts where breeding occurs. However, the literature does not always specify and some may be collection hosts only.

3.5   Insect vectors — Summary points

- *X. fastidiosa* is transmitted between plants by xylem-sap feeding insects, especially sharpshooters and spittlebugs.
- There are no known sharpshooters in New Zealand, but several spittlebugs.
- The invasive vector of *X. fastidiosa*, the GWSS, is now found in several islands in the Pacific. It has never been intercepted at the New Zealand border.
- *P. spumarius*, the meadow spittlebug, which is known to transmit *X. fastidiosa* in Europe, is widespread and common in New Zealand.
- Understanding the role of cicadas in *X. fastidiosa* transmission requires further research.

4.   Environment

4.1   Optimum growth temperatures of *X. fastidiosa*

The optimum growth rate for one isolate of *X. fastidiosa* in vitro was determined to be approximately 28°C (Davis et al. 1978). *In vitro* and *in planta* experiments by Feil et al. (2001) showed that temperatures between 25 and 32°C may be critical for the epidemiology of Pierce's disease because *X. fastidiosa* has a rapid growth rate at these temperatures, whereas below 12°C and above 34°C the survival of *X. fastidiosa* may be affected.

4.2   Effect of climate on geographic range of *X. fastidiosa* and symptom expression

Winter temperature is a key factor in delimiting *X. fastidiosa* persistence between seasons. The bacterium is rare in areas where average minimum winter (January) temperatures fall below 1.1°C (Feil and Purcell 2001). Diseases caused by *X. fastidiosa* occur in areas with mild winters, presumably in relation to survival of vectors and the bacterium in dormant plants (Purcell 1980b; Hopkins and Purcell 2002). However, *X. fastidiosa* was detected in oak in Ontario, Canada, in 2012, a region which experiences temperatures well below 1.1°C (Goodwin and Zhang 1997). The extent of *X. fastidiosa* distribution in southern Ontario, and its importance in causing leaf scorching of landscape trees there, is uncertain.

In regions with freezing winter temperatures, infected plants can recover in winter, curing previously infected and symptomatic grapevines (Purcell 1997). The biological mechanism behind this cold-curing or winter recovery is not fully understood. Experimental cold therapies, such as -8°C for several hours, show that freezing temperatures can eliminate the bacterium from diseased

grapevines (Purcell 1980a). However, freezing winter temperatures that reduce *X. fastidiosa* to undetectable levels in lucerne did not permanently remove the infection (Daugherty et al. 2010). In studies by Lieth et al. (2011), 195 h at 6°C achieved 100% curing of Pinot noir grapes while 20 h at 6°C resulted in 10% curing. Like grapevines, almond shows significant levels of overwinter recovery but it is not dependent on date of infection (Cao et al. 2011).

Temperature also has an effect on the outcome of infections, depending on the seasonal timing. Pierce's disease symptoms appear earlier in the growing season in warmer climates while the severity of the disease decreases following colder winters. Purcell (1981) showed that infections of grapevines in spring lead to chronic disease (i.e. the infection survives into subsequent years) whereas a high proportion of vines lack symptoms in the year following summer and autumn infections (Feil et al. 2003). Experiments showed that vines were as easily infected in summer as in spring (Purcell 1981; Feil et al. 2003).

4.3   Predictions of *X. fastidiosa* climate suitability — worldwide

The current worldwide distribution of *X. fastidiosa* is presented in Figure 1 (EPPO — geographical distribution). *X. fastidiosa* ssp. *pauca* has been detected in Central and South America (Argentina, Brazil, Costa Rica, Ecuador) and in Europe (France, Italy, Balearic Islands) (Daugherty et al. 2018). *X. fastidiosa* ssp. *multiplex* was detected in South America (Argentina, Brazil, Paraguay) and in the Balearic Islands, France (including Corsica) as well as its origin in North America (Sabella et al. 2018). Likewise *X. fastidiosa* ssp. *fastidiosa* has been detected in Germany, the Balearic Islands, Taiwan, North America, and Costa Rica (2018). Although present in Iran (Amanifar et al. 2014; Amanifar et al. 2016) and Canada, the subspecies present in these countries are unknown.

A recently updated list of countries (European and Mediterranean Plant Protection Organization 2019) states that *X. fastidiosa* is absent from Africa and India, and is not present in most countries in Europe. In Asia, only Iran, Israel and Taiwan have confirmed reports of *X. fastidiosa*.
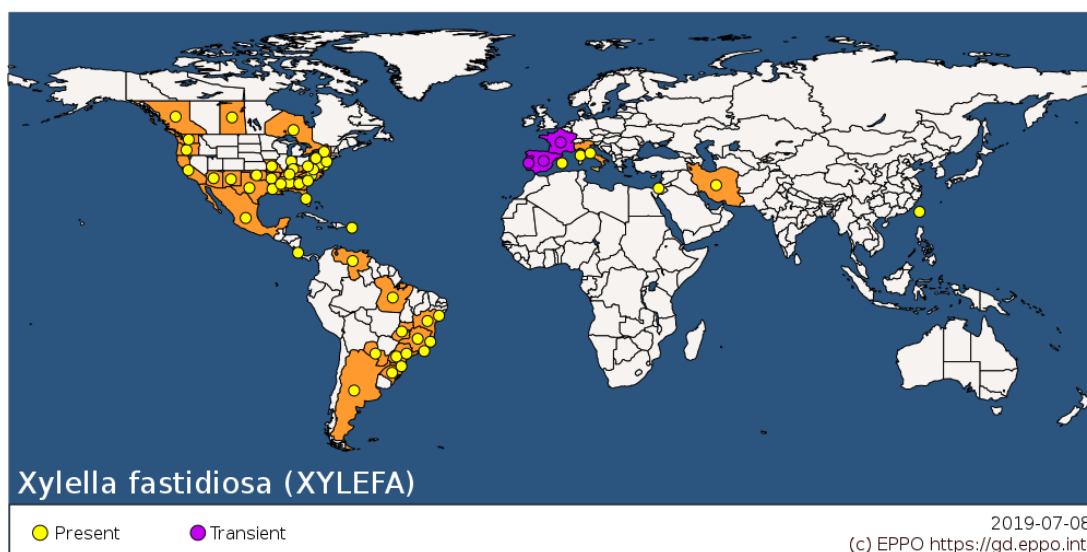

Xylella fastidiosa (XYLEFA)
○ Present   ● Transient
2019-07-08
(c) EPPO https://gd.eppo.int

Figure 1. Current worldwide distribution of *Xylella fastidiosa*. Source (European and Mediterranean Plant Protection Organization 2019) EPPO — geographical distribution**.**

Beyond one-off low temperatures, the accumulated average minimum temperature seems likely to be the most important determinants of *X. fastidiosa* geographical range. The suitability of a region for the establishment of *X. fastidiosa* has been determined using different climatic models such as CLIMEX, CLIMATCH, or an average minimum winter temperature cut-off model (Hafi et al. 2017) and bioclimatic species distribution models (SDMs) (Godefroid et al. 2019). CLIMEX is a predictive model that is used to determine an organism's potential abundance and distribution using biological data and observations of its known geographical ranges (Sutherst and Maywald 1985). For Europe, CLIMEX predictions by Hoddle (2004) suggested that most Mediterranean areas were suitable for *X. fastidiosa*, but cold winters would presumably hinder *X. fastidiosa* expansion into the most economically important wine producing regions of France, Spain and Italy. The same model indicated that the Balkans were unsuitable for the *X. fastidiosa*, yet Pierce's disease has been report in Kosovo (Berisha et al. 1998), although this report is considered to be unconfirmed/dubious by EPPO (European Food Safety Authority 2018).

Bioclimatic SDMs use climate data alone, allowing historical distributions to be modelled against climate records (Hijmans and Graham 2006; Beaumont et al. 2007). Using such a model, Godefroid et al. (2019) estimated that the known distribution of *X. fastidiosa* in Europe is small compared to the climatically suitable areas. Regions predicted to be at highest risk included large areas of Spain, Greece, Italy and France, and the Atlantic coasts of France, Portugal and Spain. In this study, distributions for ssp. *fastidiosa*, ssp. *pauca* and ssp. *multiplex* were individually modelled, with substantial differences for the three subspecies. *X. fastidiosa* ssp. *multiplex* was predicted to have a wide potential distribution in large areas of Spain, France, Italy and even the British Isles. *X. fastidiosa* ssp. *fastidiosa* was predicted to have a slightly narrower distribution, whereas ssp. *pauca* was much more limited to the Mediterranean and south Atlantic areas of Portugal and Spain (Godefroid et al. 2019).

For Australia, Hoddle (2004) predicted that cold stress would exclude *X. fastidiosa* from most of the wine production areas in Tasmania and Victoria. Predictions for *X. fastidiosa* climate suitability in Australia have also been calculated using the average minimum temperature of the coldest month (temperature cut-off model). According to this model, *X. fastidiosa* is rare if the average minimum temperature in the coldest month falls below 1.7°C, whereas conditions will be partially suitable between 1.7–4.5°C (occasional occurrence of Pierce's disease) and highly suitable above 4.5°C (Luck et al. 2001; Hafi et al. 2017). Based on this model, the majority of Australian grape-growing regions (that experience minimums July temperatures of 3–6°C according to the Australian Bureau of Meteorology 2009) are suitable for establishment of *X. fastidiosa*. These regions include much of Queensland, coastal Northern Territory, New South Wales, South Australia and southern Western Australia.

4.3   Predictions of *X. fastidiosa* climate suitability — New Zealand

For New Zealand, CLIMEX predictions (Hoddle 2004) indicated that cold stress would exclude *X. fastidiosa*/Pierce's disease from most of New Zealand's wine production areas. However, CLIMEX

predictions (Biosecurity New Zealand 2018) for bacterial leaf scorch of peach indicated that all of North Island is highly suitable for the establishment of *X. fastidiosa*, while the South Island is overall less suitable (Figure 2).
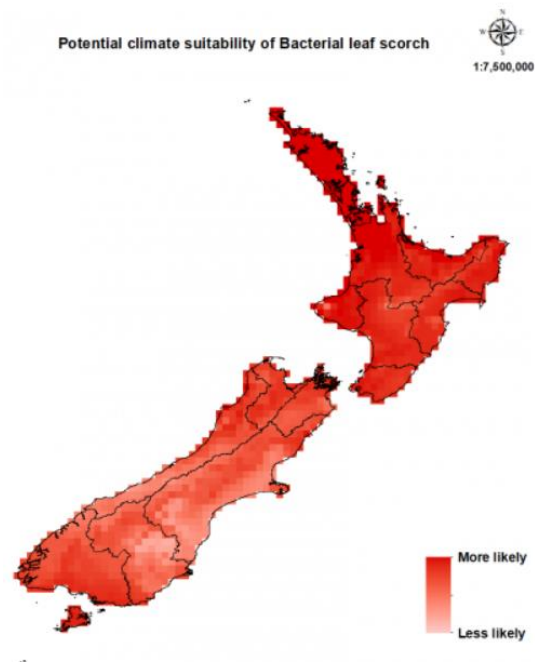

Figure 2. Climatic suitability for bacterial leaf scorch of peach in New Zealand. Source = Biosecurity New Zealand website; Protection and response, Bacterial leaf scorch (Biosecurity New Zealand 2018).

The suitability of New Zealand's climate to support the establishment of *X. fastidiosa,* determined by the average minimum July temperatures cut-off model was calculated for this report (Marroni unpublished) (Figure 3), indicated that most regions in North Island would be highly suitable habitats for *X. fastidiosa* (average minimum July temperatures greater than 4.5°C), whereas regions in the South Island, such as Blenheim and Nelson, would be partially suitable (average July temperatures between 1.7 to 4.5°C). According to this model, frost prone regions in the South Island, such as Alexandra and Queenstown, would have a low risk *of X. fastidiosa* establishment and development of related diseases.
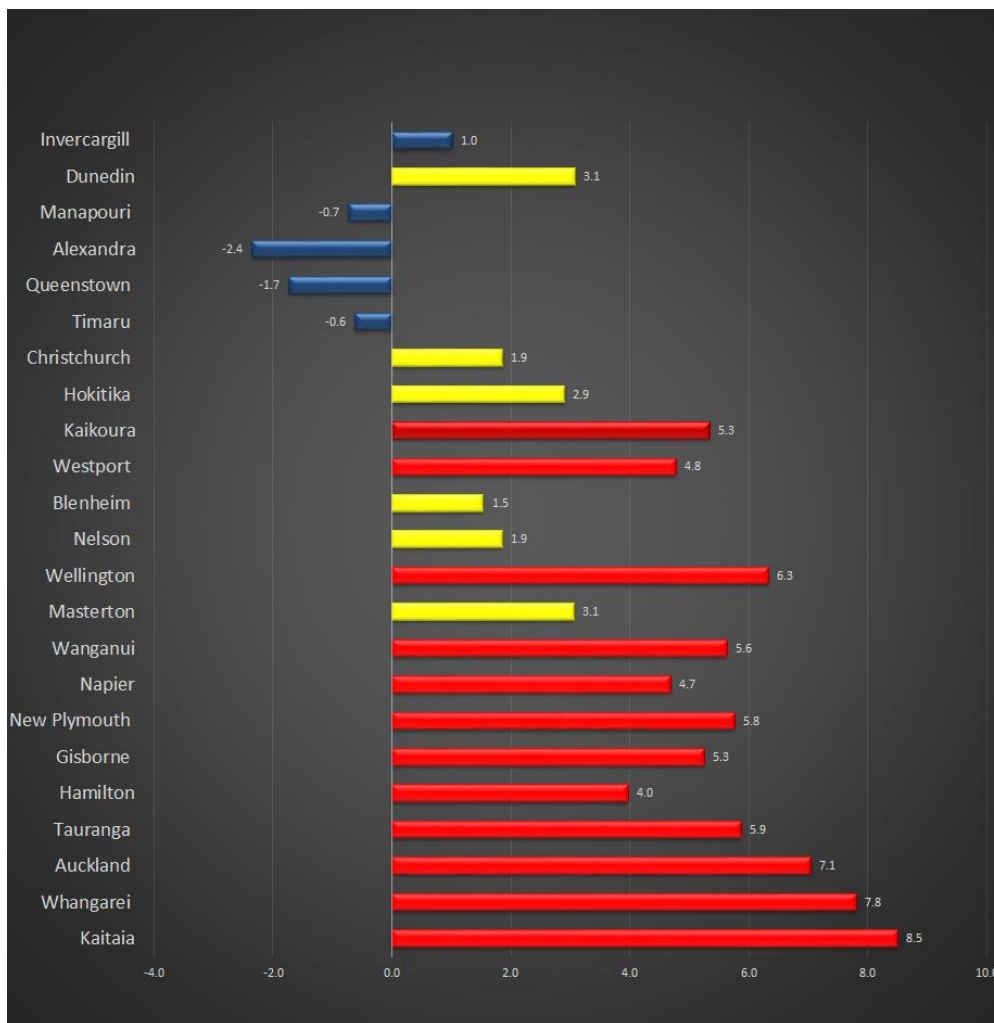
Figure 3. Suitability of New Zealand regions for establishment of *Xylella fastidiosa* calculatedaccording to the average minimum July temperature cut-off model as in Luck et al. (2001) (Marroni, unpublished, this project). Blue bars indicate lack of suitability (T<1.7°C), yellow bars partially suitable (1.7–4.0°C), and red bars highly suitable (>4.0°C). Data presented are from weather stations with at least 5 years of data.

4.4. Environment — Summary points

- *X. fastidiosa* survival is limited by cold temperatures.
- Plants exposed to cold winter temperatures have been observed to be cured of disease.
- Average minimum temperatures in winter are considered a useful measure of climate suitability for *X. fastidiosa*; using this measure, North Island would be highly suitable while parts of South Island would be partially suitable.
- Predictions of climate suitability for *X. fastidiosa* are highly variable and precise prediction is not yet possible.
- Some subspecies of *X. fastidiosa* may be less sensitive to cold, and capable of invading a wider area of New Zealand. Cold-curing efficiency may be variable between different host plant species.

5. Economic risk assessment

5.1. International economic assessments

There have been few economic evaluations of the effects of *X. fastidiosa* on horticultural industries in countries in which it has established, and only one ex-ante evaluation of the potential consequences if biosecurity measures fail to prevent pest incursion and establishment.

The orange industry in Brazil has been severely affected by CVC. By 2001 it was estimated that approximately 40% of citrus plants were infected resulting in annual losses of $US110 million, approximately 6% of the total industry value in that year (Ayres 2001). However, the 2018 Fundecitrus CVC Disease Survey reports that the level of CVC infection has declined from 37.6% to 1.3% during the 6 years from 2012 to 2018 (Barros 2018). This reflects the success of mandatory requirements for certified nursery trees, introduced in 2003, and a programme of aggressive large-scale management practices (Almeida et al. 2014). The total costs of this programme were not reported, although it was considered to be labour-intensive and costly, so possibly not feasible in the US.

In 2010, the total cost of Pierce's disease in Californian grapevines was estimated to be $US104.4 million per year (Tumber et al. 2014) across California's 346,000 ha of grapes, of which 236,000 are planted in winegrapes (California Department of Food and Agriculture 2018). The grape industry, of which the winegrapes sector comprised 66% by value, sustained losses of $US56.1 million a year in lost production and vine replacement. Additional costs of $US48.3 million were incurred for research and containment activities funded by the federal and state governments, and in compliance costs imposed on the nursery and citrus industries to protect the grape industry. Related research by the same authors (Alston et al. 2013) used a simulation model to estimate that if the current Pierce's Disease Control Programme ended, allowing GWSS to spread throughout California, the total annual cost of the disease to growers, and ultimately consumers, would increase by $US185 million per year.

In 2015, Australia's biosecurity authorities introduced emergency measures to reduce the risk of an *X. fastidiosa* incursion, and requested the Australian Bureau of Agricultural Resource Economics and Sciences to assess the economic costs to the wine sector of a potential disease outbreak (Hafi et al. 2017). Ecological and economic models were used to evaluate three maximum-extent scenarios defined in terms of habitat suitability for the pest including:

- Scenario 1 — all existing wine grape growing areas (132,390 hectares)(Plant Health Australia 2019).
- Scenario 2 — all existing wine grape growing areas with an average minimum winter temperature above 1.7°C.
- Scenario 3 — all existing wine grape growing areas with an average minimum winter temperature above 1.7°C that are in close proximity to riparian vegetation.

The scenarios were evaluated under two price levels; the 2014/15 actual prices and a 25% increase in price. The costs included in the analysis were those of adjustment, largely replanting, and the reductions in gross margins associated with the reduction in vine numbers only.

The evaluation showed that the extent to which conditions suit the disease and its vectors is a major determinant of economic cost, which ranged from $AU2.8 to $AU7.6 billion over 50 years in net present value (NPV) terms. It also demonstrated that the financial position of vineyards before the

outbreak will determine whether their response will be to replant with resistant varieties, provided these are available, or exit the industry. Under actual 2014/15 prices, approximately 75% of cost was borne by the wine-making industry, as growers unable to afford to replant affected vines exited the industry and wine production declined. At higher prices growers would carry a higher proportion of cost as more replanting was assumed (Hafi et al. 2017).

The research also examined the economic costs if the pest is detected early and able to be confined to a single region, either the Murray Darling or Lower Murray regions for example and, perhaps unsurprisingly, found these to be very much lower at between $AU0.2 billion and $AU2 billion. Whether a containment programme would be justified depends on the cost of the programme relative to the benefits of containment.

5.2   The economic value of production at risk in New Zealand

Much of New Zealand's horticultural production takes place in the areas defined as highly or partially suitable for the establishment of *X. fastidiosa* by the average minimum July temperatures cut-off model (Figure 3). If the MPI model (Figure 2) is used, potential losses would be much higher.

In addition to winegrapes, other crops such as olives, citrus and summerfruit would be at high risk. The proposed large-scale expansion of the blueberry industry in the North Island may also be at risk. Table 2 shows the areas at risk by industry, and the total export and domestic value of those industries in 2018. The at-risk value of production has been estimated by applying the proportion of area at risk to the total industry value which may introduce some distortion, since yields and returns differ among regions.

Table 2: New Zealand winegrape, olive, citrus, summerfruit and blueberry production areas and returns at risk from *Xylella fastidiosa* establishment.

Region[1]	Wine grapes ha[2]	Olives ha	Citrus ha	Summer-fruit ha	Blue-berries ha
Northland	S	118	313	8	29
Auckland	836	128	121	31	27
Waikato	15	34	13	50	316
Bay of Plenty	75	26	62	7	24
Gisborne	1,245	S	1,136	12	1
Hawke's Bay	3,616	120	41	633	99
Taranaki	S	S	2	6	1
Manawatu-Wanganui	88	34	1	13	12
Wellington	832	181	1	15	8
Tasman	980	72	7	34	27
Nelson	24	0	0	0	0
Marlborough	23,051	50	0	45	2
West Coast	S	0	0	11	6
Canterbury	1,769	133	0	81	11
Otago (except DN)	1,173	19	0	1,144	0
Southland	S	0	2	31	63
Total New Zealand area (ha)	33,981	921	1,700	2,140	624
Highly suitable area (ha)	5,876	459	1,689	769	514
Partially suitable area (ha)	26,656	436	9	174	47
Industry value (M)[3]	$1,705	$3	$9	$238	$91
Industry value in highly suitable areas (M)	$295	$1	$9	$86	$75
Industry value in highly/partially suitable areas (M)	$1,632	$3	$9	$105	$82

1   Colours indicate climatic suitability as shown in Figure 3. DN = Dunedin.

2   Source: Statistics New Zealand. 2018. Agricultural production statistics June 2017 (final)-additional tables. https://www.stats.govt.nz/information-releases/agricultural-production-statistics-june-2017-final. S = "some", possibly commercially sensitive, not specifically reported.

3   The New Zealand Institute for Plant and Food Research Limited (PFR) 2018. Fresh Facts 2018. 20th ed. PFR, Auckland and Horticulture NZ.

The effects of a *X. fastidiosa* incursion into New Zealand would be dependent on the particular subspecies involved. The establishment of *X. fastidiosa* in New Zealand would impose the greatest losses on the wine industry since wine grapes are highly susceptible and 96% of wine production occurs in areas deemed highly or partially suitable for disease establishment. The summerfruit industry, which plans large-scale expansion during the next 15 years (Summerfruit New Zealand 2018), currently has 44% of area in these areas, but this is likely to increase as the industry

development plan is implemented. Almost 100% of both citrus and olive production also happens in these areas of greatest suitability, although the total value of production from those industries comprises only a small production of New Zealand's total horticultural output. In total, $NZ391 million per year of output from the four industries identified as being most at risk is produced from areas estimated to be highly suitable for establishment of the disease, and $NZ1.7 billion from areas considered highly or partially suitable for establishment. If the disease affects the blueberry industry, which is based almost entirely in areas highly suitable for establishment, the costs are likely to be very much higher.

It is impossible to determine the likely cost of *X. fastidiosa* establishment in New Zealand on the basis of the three economic evaluations reported in the literature. Only two, both evaluating the effects of the disease on the grape and wine industries, report the costs in any detail. The Californian costs estimated (Tumber et al. 2014) are equivalent to $US301/ha under the current control programme and $US535/ha if the programme were to be discontinued (Alston et al. 2013). The equivalent annual return (EAR) of the NPVs reported for the range of outcomes for the Australian wine industry (Hafi et al. 2017) have been calculated as $AU431 to $AU1,169/ha per annum. However, the differences in the structure and profitability of the Californian, Australian and New Zealand wine industries make extrapolation from these to the New Zealand situation of limited value. New Zealand wines attract an average premium on world markets of 20%, so the loss of production will result, on average, in a greater loss of value than losses experienced elsewhere. However, too many factors will determine the outcome of a disease outbreak to be able to say more than that *X. fastidiosa* would likely impose very high costs on the horticultural industry if it were to establish in New Zealand.

5.3   Economic effects — Summary points

- Only two economic evaluations of the economic effects of the establishment of *X. fastidiosa* have been reported in recent years. The current costs in California and the expected costs of establishment in Australia have been shown to be very high, but it is not possible to extrapolate these to the New Zealand situation.
- The economic costs to New Zealand will be highly dependent on pathogen spread under the influence of climate. These will also be dependent on the financial strength of individual industries, including their ability to replant with resistant cultivars as well as the availability of those cultivars.
- The effects of establishment could be substantially reduced if it were possible to confine the spread of an *X. fastidiosa* incursion.
- Horticultural production worth between $301 million per year and $1.7 billion per year would be at risk if *X. fastidiosa* became established in New Zealand.

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