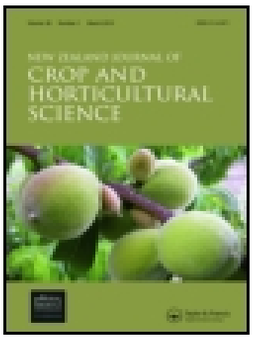

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History of Boysenberry and Youngberry in New Zealand in relation to their problems with Boysenberry decline, the association of a fungal pathogen, and possibly a phytoplasma, with this disease

G. A. Wood, M. T. Andersen, R. L. S. Forster, M. Braithwaite & H. K. Hall

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History of Boysenberry and Youngberry in New Zealand in relation to their problems with Boysenberry decline, the association of a fungal pathogen, and possibly a phytoplasma, with this disease

G. A. WOOD
M. T. ANDERSEN
R. L. S. FORSTER
The Horticulture and Food Research Institute of New Zealand
Mt Albert Research Centre
Private Bag 92 169
Auckland, New Zealand

M. BRAITHWAITE
Ministry of Agriculture and Forestry
National Plant Pest Reference Laboratory
P. O. Box 24
Lincoln, New Zealand

H. K. HALL
The Horticulture and Food Research Institute of New Zealand
Nelson Research Centre
P. O. Box 220
Motueka, New Zealand

Abstract The history of Boysenberry (*Rubus ursinus* Chamisso & Schlenhtendal) and Youngberry (*R. ursinus* derivative) were traced from their origins in the United States, to their importation into New Zealand, and subsequent use as the major *Rubus* crops. The investigation into possible causes of Boysenberry decline disease are described, and the probable association of the recently detected *Cercosporella rubi* fungus with this decline is discussed, as is the presence of *Phormium* yellow leaf phytoplasma in some Boysenberry decline affected plants. A graft-transmissible agent in Boysenberries, which may be *C. rubi*, was shown to take several seasons to induce visible symptoms following graft inoculation. The means by which *C. rubi* may have gained entry to New Zealand by the importation of infected blackberry (*Rubus* spp.) cultivars from the southern United States is discussed, as is the means by which *C. rubi* may have been spread within New Zealand.

Keywords *Rubus*; Boysenberry; Youngberry; Boysenberry decline disease; *Cercosporella rubi* fungus; *Phormium* yellow leaf phytoplasma; disease introduction; disease spread

INTRODUCTION

In New Zealand, Boysenberry (*Rubus ursinus* Chamisso & Schlenhtendal) has achieved status as the most important and widely grown *Rubus* crop (Scott et al. 1993), and its recent problems with Boysenberry decline disease have been of major concern to small fruit growers. The exact parentage of the Boysenberry is unknown. One parent was originally thought to be the Himalaya trailing blackberry (*R. procerus* P.H. Muell.) (Morris 1938; Shoemaker 1950) but this was later discounted (Thompson 1961). It was also suggested that native trailing blackberries of the Pacific Coast of the United States may have been involved in its parentage (Waldo 1950, 1968). Boysenberry is now believed to have arisen from a cross between Loganberry (*R. loganobaccus* Bailey) and a trailing blackberry (*R. baileyanus* Britt.) cultivar such as 'Lucretia' or 'Austin Mayes' (Jennings 1988), and is considered to have originated on the farm of John Lubben in Napa County, California, United States, in 1921 (Thompson 1961) or 1923 (Steller 1937). Rudolph Boysen, who was farming the Lubben property during the 1920s was also experimenting with the crossing of *Rubus* cultivars (Knott 1935; Morris 1938; Havis 1941). When he later moved to Anaheim, California, and became Superintendent of Parks, he took with him some plants of a large-fruited, luxurious growing berry (Anon. 1954). It has been suggested that this berry may originally have been one of plant breeder Luther Burbank's

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seedlings, and known as Lubbenberry, had been taken by John Lubben from his home in Alameda, San Francisco, to the Napa County farm (Butterfield 1938). It may also have been a seedling of Lubbenberry, but its exact origin may never be known. Presence of the new berry became known to George Darrow, Berryfruit Specialist of the United States Department of Agriculture, based in Beltsville, Maryland, who travelled to California primarily to see it. Impressed by the new berry, George Darrow contacted small fruit grower and nurseryman Walter Knott at his Buena Park, California, Berry Farm (or Berry Place as it was known then) (Steller 1937), and in 1932 Walter Knott trialled the new berry under field conditions (Knott 1935), where it out performed every anticipation (Steller 1937). As the new berry had no name, Walter Knott, in consultation with the United States Government Bureau of Plant Industries at Beltsville, decided that the new berry should be named Boysenberry (Anon. 1954; Bailey & Bailey 1978). By 1935, Boysenberry plants were being made available for sale commercially (Knott 1935; Anon. 1936) and by 1937, Boysenberry was being promoted in the United States as a promising new trailing blackberry type suitable for commercial use (Darrow 1937). By 1949, Boysenberry was being grown more extensively than any of the other blackberries in California (Baker 1949), and is still widely grown on the Pacific Coast (Anon. 1997).

The Boysenberry was introduced to New Zealand directly from Knott's Berry Farm c. 1935–37 (Anon. 1954). Early correspondence between the Horticulture Division of the then New Zealand Department of Agriculture, and the Plant Diseases Division of the then New Zealand Department of Scientific and Industrial Research (DSIR), indicates that at least two importations of Boysenberry plants from the United States were probably made. Imported plants were recorded as being grown at Masterton in Wairarapa district in February 1938 by the orchard instructor M. Davey, and at Kerikeri in Northland district in September 1939 by the orchard instructor P. Everitt (archive correspondence). Boysenberries were also reported to be present at Mapua, Nelson in January 1939 by the orchard instructor A. Grainger (archive correspondence). However, it is apparent that in the late 1930s, Boysenberry plants were available commercially, and being more widely grown in Auckland district than elsewhere, as plants were being advertised for sale by a large Auckland commercial nursery by 1939 (Anon. 1939), and 20 ha were recorded in cultivation in Auckland in a 1939 survey made by the then New Zealand Department of Agriculture (Woodhead & Chamberlain 1940a). No other New Zealand districts were recorded as growing Boysenberries in the survey, but small trial areas were being planted out in Hawkes Bay in early 1938 (Sharp 1939) and it was thought likely they would supersede the Loganberry. By 1948 Boysenberries were well established in Nelson (Hogg 1948). Archive correspondence indicates that a Department of Agriculture bulletin on the culture of both Boysenberry and Loganberry was in preparation in the early 1940s, but this was apparently not finished, probably in deference to the Second World War which was in progress at the time. Data on the culture of Boysenberries in New Zealand were made available to potential growers shortly after the war in a trade journal article (Davey 1947).

From that time on, the planting of Boysenberries in New Zealand steadily increased as its suitability as a processed fruit was recognised. By 1980, the main districts growing Boysenberries were Nelson/Marlborough with 227 ha, Hawkes Bay/Poverty Bay/Wairarapa with 160 ha, and Waikato/Bay of Plenty with 101 ha (Langford & Mavromatis 1981). Smaller areas were in production in Auckland, Manawatu, and Canterbury. Subsequently, the Boysenberry industry fluctuated considerably, and by 1993 yields were considerably reduced compared with those 10 years previously. By 1993, 161 growers were recorded as having 254 ha in production in New Zealand (Scott et al. 1993).

Often grown in conjunction with Boysenberry in New Zealand, the Youngberry (*R. ursinus* derivative) was selected in 1905 by B. M. Young of Morgan City, Louisiana, United States, from a seedling progeny resulting from hand pollination of the Phenomenal berry (a type very similar to the Loganberry and sometimes known as 'Burbank's Logan' (Jennings 1988)) with 'Austin Mayes' (Thompson 1961). The Youngberry was not made available commercially to north American growers until 1926 (Darrow 1937; Shoemaker 1950; Jennings 1988). An importation of Youngberry was made by DSIR in the 1930s (Cunningham 1940), though this may not have been its only introduction to New Zealand. As Knott's Berry Farm introduced the Youngberry to California growers in 1926 (Anon. 1936), New Zealand Youngberries may also have been obtained from this source. When later evaluated with other hybrid berries, it was the only one to merit further study (Anon. 1942). Youngberry was listed for sale in 1937, for the first time, by the same Auckland commercial nursery which later listed

Boysenberry (Anon. 1937). The survey of New Zealand small fruit made in November–December of 1939 recorded occasional commercial plantings of Youngberry (Woodhead & Chamberlain 1940a).

In comparative assessments made in 1981 (Langford & Mavromatis 1981) the leaves of Youngberry were found to have a more pointed terminal leaflet and to be slightly lighter in colour than those of Boysenberry. Youngberry canes were dark red in winter compared to the orange-red colour of Boysenberry. The fruit was similar to Boysenberry, but smaller, and ripened to black, compared to the deep wine red of Boysenberry. Youngberry fruit was sweeter and quite shiny when ripe, usually ripened 7–10 days earlier than Boysenberry, and was often called "Early Boysenberry" by growers. Because of their similarity, considerable confusion occurred as to the identity of both types on growers properties, particularly as both are prone to producing sports which could vary in cropping potential, fruit size, vigour of cane, and thorniness (Langford & Mavromatis 1981). Most growers today have a predominance of Boysenberries in cultivation, but commonly have a smaller area producing Youngberries. Youngberries and Boysenberries were also established in New South Wales, Australia (Ballantyne 1947), probably about the same time that they were introduced to New Zealand, but as a crop, did not achieve the importance that they did in New Zealand.

In the late 1970s and in the early 1980s, two collections of 60 Boysenberry and Youngberry selections were established from reselection of New Zealand grower sources at the DSIR (now HortResearch) research station at Riwaka, Motueka, in an endeavour to select the most suitable clones for the various growing districts of New Zealand. From this collection a high yielding Boysenberry selection known as 'Riwaka's Choice' was made (Hall 1992), as was a similar Youngberry known as 'RS4'. A breeding programme, initiated at the same time, has produced a number of *Rubus* types with Boysenberry characteristics, and several of these are now undergoing commercial evaluation.

At the time the 1939 survey was made, Boysenberries appeared to be comparatively free from diseases (Woodhead & Chamberlain 1940b), but later dryberry disease caused by the downy mildrew fungus (*Peronospora sparsa*) became a limiting factor in Boysenberry production (Newhook & Brown 1963). The first indication of a further serious problem emerged in the early 1980s when initially one, and later several growers in the Kumeu district north-west of Auckland City noticed abnormalities in the growth of their Boysenberries. Following preliminary investigations of the problem by the Ministry of Agriculture and Fisheries and DSIR (Wood & Mossop 1984), it became evident that a plant pathogen was probably involved, and a warning to this effect was given to New Zealand growers (Eden & Wood 1986). The disease was given the name "Boysenberry decline", and by the late 1980s infection had been found in more properties in the Auckland district, and by 1990 caused the demise of Boysenberry growing in most of the district. The 'Riwaka's Choice' selection of Boysenberry appeared to be particularly susceptible. In a Ministry of Agriculture and Fisheries evaluation trial of several clones of Boysenberries, including 'Riwaka's Choice', planted on a commercial property at Huapai where Boysenberry decline was present, pronounced symptoms of the disease showed on many of the fruiting cane flowers within 15 months of planting (Michael Eden pers. comm.). It was thought that if it could be confined to Auckland, other Boysenberry growing districts would escape infection. However, in the 1980s Boysenberry decline appeared on a property in the Bay of Plenty. In the 1990s infection in the Bay of Plenty became more serious, with the disease occurring on several properties, and as well as occurring on Boysenberry, plants of Loganberry, Youngberry, and 'Aurora', 'Kotata', and 'Marion' hybrid berries were also affected (Wood & Langford 1996). Subsequently, infection was found in Boysenberries in Tauranga in the Bay of Plenty, Cambridge and Te Awamutu in the Waikato, and in New Plymouth. Until 1995, the disease had been confined to the North Island and it was hoped that warnings to growers of the dangers of transferring Boysenberry material from the North to the South Island would prevent the disease from becoming established in the South Island, where the main growing areas of the crop were located. Unfortunately, at this time, Boysenberry decline was identified in a block of 'Marion' on a property in Motueka. In 1996 Boysenberry decline was discovered in Boysenberries on one property in Motueka and two in Nelson.

From the time that Boysenberry decline was first found in the Kumeu district, efforts have been made to identify the causal agent and its vector, and to try to implement methods of control (Langford et al. 1995). Boysenberry plants affected with decline were examined for diseases of both a fungal and bacterial nature in the 1980s (Eden & Wood 1986) and again in the early 1990s, but no evidence for their presence could be found. Early investigations also

considered herbicides or soil nematodes as the cause, but there was no evidence of their involvement (Wood et al. 1992). In 1982, the chrysanthemum foliar nematode (*Aphelenchoides ritzemabosi*) was found in the leaves of some of the affected Boysenberries on two Auckland properties (Chris Barber pers. comm.). It was later concluded that this foliar nemotode was not associated with the decline symptom. Tests for presence of *Rubus* viruses were also negative (Wood 1991). The disease was shown to be transmissible by grafting from diseased to healthy Boysenberry (Wood 1991), and the possibility of it being caused by a mycoplasma-like organism (or phytoplasma as these organisms are now known) was then considered. The disease had some similarities (but also differences) to the phytoplasma disease *Rubus* stunt, which occurs in Europe (Murant & Roberts 1971; van der Meer 1987). However, when the cultivar 'Malling Landmark', a *Rubus* stunt susceptible red raspberry (*R. idaeus* L.) was grafted with Boysenberry decline affected material, no symptoms of stunt developed (Wood 1991). Grafting of Boysenberry decline affected material to cultivars sensitive to the blackberry dwarf disease of the western United States (Zeller 1927; Wilhelm 1951; Wagnon & Williams 1970) gave inconclusive results (Wood 1991). The latter disease had been found to be graft-transmissible to Boysenberry and Youngberry in the United States, though the plants were not severely affected (Zeller & Milbrath 1940), and neither were found naturally affected under field conditions (Wilhelm 1951). Another phytoplasma disease causing a witches' broom symptom on *Rubus* was reported from Oregon, United States, and as it had a lethal effect on growth and appeared to be confined to black raspberries (*R. occidentalis* L.) (Converse et al. 1982), it seemed unlikely to have any relationship to Boysenberry decline. DNA tests in New Zealand of Boysenberry plants using Polymerase Chain Reaction (PCR) provided evidence of the presence of *Phormium* yellow leaf phytoplasma (Andersen et al. 1998a), but this could not be specifically related to the presence of Boysenberry decline (Forster & Andersen 1998). In early spring of 1995, fungal mycelium was observed on some flowers of 'Marion' affected with Boysenberry decline at Motueka (Warren Thomas pers. comm.), though flowers examined later in the spring did not give any indication of the presence of a fungal pathogen. However, further investigations of decline-affected *Rubus* cultivars in the spring of 1998 disclosed the presence of a fungal pathogen, previously recorded only from the United States, and first described in detail in 1911 (Cook 1911). In the United States it was particularly severe on 'Lucretia' dewberry and 'Rathbone' blackberry. The disease was commonly referred to as "double blossom", as the petals were usually wrinkled to varying degrees giving the flowers the appearance of being doubled. Foliage growth was stunted and shoots massed to give a witches' broom affect. Cook (1911) attributed the disease to *Fusarium rubi* G. Winter. A subsequent investigation in Louisiana of a rosette disease of blackberries and dewberries showed that it was identical with the double blossom disease (Plakidas 1934), and that the causal agent was an undescribed *Cercosporella.* As rosette was more descriptive of the symptoms, rosette is now used as the common name of the disease, and it is now considered to be incited by the fungus *Cercosporella rubi* (G. Winter) Plakidas (Buckley 1996).

This paper therefore: (1) describes the detection of the fungal pathogen which appears to be associated with Boysenberry decline; (2) outlines the detection of *Phormium* yellow leaf phytoplasma in Boysenberry using PCR; (3) describes further graft-transmission trials with Boysenberry decline; (4) suggests how the fungal pathogen could have entered New Zealand; and (5) suggests the means by which the pathogen may have been spread within New Zealand.

MATERIALS AND METHODS

Detection of a fungal pathogen in Boysenberry decline affected flowers

Boysenberry decline samples of 'Marion', exhibiting symptoms of shoot rosette and fungal growth on the anthers of flowers, were collected from Whakatane district of the North Island of New Zealand. Affected shoots and flowers were placed in a humidity chamber for up to 48 h to induce growth and sporulation of the fungus. Affected anthers were then directly mounted into lactophenol cotton blue (0.1%) on a glass slide for microscopic examination. Morphological characteristics of the spores were compared to those described for *Cercosporella rubi* by Smith & Fox (1991). Spore masses were transferred with a sterile needle, to prune extract agar. Plates were incubated at 25 ± 0.5°C for up to 4 weeks and morphological characteristics of the culture were compared with those described by Plakidas (1937).

Detection of a phytoplasma in Boysenberry using Polymerase Chain Reaction (PCR)

Twenty plants showing advanced symptoms of Boysenberry decline were sampled on three occasions during the mid summer of 1996–97 (mid December–late January). DNA was extracted from symptomatic tissues, usually rosettes of proliferation indicative of the disease, and then tested using PCR. The PCR-competence was established using universal prokaryotic primers Gd1 and Berg54, and the presence of phytoplasma was detected using primers R16F2, R16R2, NGF, and NGR in nested-PCR. All primers used are based on the 16S rRNA gene, and the latter four are considered universal to all phytoplasmas. The procedures used were essentially as described previously (Andersen et al. 1998a). PCR products obtained were sequenced on an ABI Prism 377 DNA Sequencer according to manufacturer's protocols.

Graft transmission trials with Boysenberry decline

Previously, Wood (1991) reported the successful transmission of Boysenberry decline from an infected Kumeu field plant of Boysenberry to a clone of Boysenberry obtained from North America and named 'Boysen-72'. In this trial symptoms had shown on the 'Boysen-72' in the second season after graft inoculation, and possible, but not conclusive transmission had also obtained to several blackberry cultivars.

During the early 1990s, with concerns growing over the increasing incidence of Boysenberry decline in New Zealand, further graft transmission work was initiated. A series of small trials was commenced to try to determine the sensitivity to Boysenberry decline of new hybrid berries obtained from the Riwaka *Rubus* breeding programme, or Boysenberry types from other sources selected as possible suitable alternatives to standard Boysenberry. The trials commenced in the 1993/94 season, and graft inoculations continued as material became available, until the 1996/97 season. As only a limited amount of material was available, testing was usually limited to two plants of each cultivar, one being grafted with a floricane showing symptoms of Boysenberry decline, and the other being retained as an uninoculated control. The cultivars were grown under glass in containers during the early part of the season, with graft inoculation carried out in the late summer using the inarch bottle grafting technique (Garner 1958). Generally two infected shoots were grafted to each plant. Following successful graft unions the plants were placed outdoors to induce winter dormancy, and encourage subsequent disease symptom expression. During each winter when the plants were dormant they were replaced in larger containers with fresh soil, to promote growth. In the season following graft inoculation the plants were examined several times for symptoms of Boysenberry decline.

Cultivars used to test for sensitivity to Boysenberry decline were 'Boysen-72', 'Marion', and 'Waldo', imported from the United States (HortResearch plant quarantine records); 'Logan L654' and 'Logan LY59' imported from East Malling Research Station, England (HortResearch plant quarantine records); 'Riwaka's Choice' Boysenberry from the Riwaka collection, 'Karaka Black', 'Kaiteri', 'Mahana', 'Ranui', 'Riwaka Tahi', 'Taranaki', 'Waimate', and '8443W2' from the Riwaka breeding programme, and the clonal reselections 'Mapua', 'McNicol's Choice', 'RS4' Youngberry, and 'Tasman' from growers selections. Sources of Boysenberry decline infection used as inoculum were Boysenberry from Kumeu, Nelson, and Whakatane, 'Kotata' from Whakatane, Loganberry from Whakatane, and 'Marion' from Motueka. Graft inoculations, and the seasons in which these were done, are shown in Table 1. The Boysenberry decline source from Nelson was subsequently found to also be infected with raspberry bushy dwarf virus, but this was not thought to influence the expression of Boysenberry decline symptoms.

A further attempt was also made to infect several cultivars of red raspberry with Boysenberry decline. Cultivars used were 'Autumn Bliss' imported from England, 'Malling Landmark' and 'Norfolk Giant' from Scotland, 'Clutha' 'Selwyn', 'Waiau', and 'Waimea' from the Riwaka breeding programme. Graft inoculations, as shown in Table 1, used Boysenberry decline sources from Kumeu, Motueka, and Whakatane.

Investigating the means by which the involved fungal pathogen became established in New Zealand

Investigations in the United States have shown that *C. rubi* is confined to the genus *Rubus*, affecting most cultivars of erect blackberries, and also trailing blackberries, and dewberries (which are trailing blackberries of eastern North America (Jennings 1988)), but only rarely affecting red and black raspberries (Smith & Fox 1991). A number of species of blackberry growing wild have been present in New Zealand since the mid 19th Century, several of these originating in North America (Pennycook 1998). The possibility that *C. rubi* could have been

Table 1 Boysenberry and hybrid berry selections, and cultivars of red raspberry graft-inoculated with Boysenberry decline. (+ = general symptoms of decline showing, but where asterisk appended, symptoms observed only as small down-curled, purple-bronzed leaves developing on floricanes in spring; – = no decline symptoms apparent.)

Cultivar graft inoculated	Source of infection	Growing season when graft-inoculated	Subsequent numbers of growing seasons for symptoms to develop				
			1 season	2 seasons	3 seasons	4 seasons	5 seasons
Boysenberry and hybrid berry selections							
Boysen-72	Kotata, Whakatane	1993/94	–	–	–	+*	+*
Kaiteri	Boysenberry, Kumeu	1993/94	–	–	–	+	+
Mahana	Boysenberry, Kumeu	1993/94	–	–	–	–	–
Ranui	Boysenberry, Kumeu	1993/94	–	–	–	–	–
Riwaka Tahi	Boysenberry, Kumeu	1993/94	–	–	+	+	+
Taranaki	Boysenberry, Kumeu	1993/94	–	–	–	–	–
Karaka Black	Boysenberry, Kumeu	1994/95	–	–	+*	+*	
Waimate	Boysenberry, Kumeu	1994/95	–	–	–	+*	
Boysen-72	Marion, Motueka	1995/96	–	–	+*		
Karaka Black	Boysenberry, Kumeu	1995/96	–	–	+*		
Logan L654	Loganberry, Whakatane	1995/96	–	–	+*		
Logan LY59	Loganberry, Whakatane	1995/96	–	–	+*		
Marion	Kotata, Whatakane	1995/96	–	–	–		
Marion	Marion, Motueka	1995/96	–	–	–		
Riwaka's Choice	Boysenberry, Whakatane	1995/96	–	–	+*		
Riwaka's Choice	Kotata, Whakatane	1995/96	–	–	–		
Riwaka's Choice	Loganberry, Whakatane	1995/96	–	–	+*		
Waimate	Boysenberry, Kumeu	1995/96	–	–	+*		
8443W2	Boysenberry, Kumeu	1995/96	–	+*	+*		
Logan L654	Loganberry, Whakatane	1996/97	–	+*			
Logan LY59	Loganberry, Whakatane	1996/97	–	+*			
Mapua	Boysenberry, Nelson	1996/97	–	–			
McNichol's Choice	Boysenberry, Nelson	1996/97	–	+*			
RS4 Youngberry	Boysenberry, Nelson	1996/97	–	+			
Tasman	Boysenberry, Nelson	1996/97	–	–			
Waldo	Boysenberry, Nelson	1996/97	–	+*			
Cultivars of red raspberry							
Selwyn	Boysenberry, Kumeu	1994/95	–	–	–	–	
Autumn Bliss	Boysenberry, Kumeu	1995/96	–	–	–		
Clutha	Boysenberry, Kumeu	1995/96	–	–	–		
Malling Landmark	Kotata, Whakatane	1995/96	–	–	–		
Malling Landmark	Marion, Motueka	1995/96	–	–	–		
Norfolk Giant	Kotata, Whakatane	1995/96	–	–	–		
Selwyn	Boysenberry, Kumeu	1995/96	–	–	–		
Waiau	Boysenberry, Kumeu	1995/96	–	–	–		
Waimea	Boysenberry, Kumeu	1995/96	–	–	–		

introduced in any of them was considered, but it was thought that if so, although not easy to identify, the fungus would have been detected in them in the early part of this century. As a number of rust fungi have reached New Zealand from Australia via their presumed flight path on the prevailing trans-Tasman sea westerly winds (Pennycook 1998), the possibility that *C. rubi* could have reached New Zealand in this way was also considered. However, there have been no reports of *C. rubi* in Australia, and even if it was present there, it is unlikely that spore releases would have been of a sufficient quantity to achieve infection in New Zealand over such a long distance.

Thus to try to determine how *C. rubi* became established in New Zealand, an investigation was made of the history of blackberry and hybrid berry cultivars which had been imported in the 20 years prior to 1982, the date when symptoms of Boysenberry decline were first observed. During this time all *Rubus* imports passed through a New Zealand plant quarantine station (unless there were any illegally imported, although no such cultivars are known to have become established here). During this 20-year period, there were 20 blackberry or hybrid berry cultivar imports, all but two from northern hemisphere countries (Table 2). An assessment was therefore made as to the origin of the 20 cultivars to see if any could be linked with the introduction of *C. rubi*.

Investigating the means by which *C. rubi* may have been spread within New Zealand

In an attempt to determine how *C. rubi* was spread through the various Boysenberry growing districts of New Zealand, an investigation was made of the means by which this could have occurred. This is with the assumption that *C. rubi* was present in one or more blackberry cultivars imported from the United States, and that this infection was later associated with symptoms of Boysenberry decline in New Zealand. To follow the theoretical trail, plant quarantine records from the 1970s were examined, as were research reports on small fruit from that time and into the 1980s, and data obtained from the spread of Boysenberry decline in the Auckland district during the 1980s.

RESULTS

Detection of a fungal pathogen

Fungal sporulation, exhibited as a white mass, was observed on the anthers of flowers and examination of prepared slides (400×) showed that spore shape and size matched those of *C. rubi* (Smith & Fox 1991). This was confirmed by Dr E. McKenzie, Landcare Research, Auckland, New Zealand. The fungus was only detected on the anthers of the

Table 2 Cultivars of blackberry and hybrid berry introduced to New Zealand from 1962 and released by 1982, their origin, and time period in plant quarantine.

Cultivar	Origin	Imported	Released
Darrow	New York State, United States	1962	1962
Logan LY59	England	1962	1962
Bramble (cv. unknown)	Maryland, United States	1963	1964
Aurora	Oregon, United States	1964	1965
Marion	Oregon, United States	1964	1965
Evergreen Thornless	Oregon, United States	1965	1967
Smoothstem	Maryland, United States	1966	1967
Thornfree	Maryland, United States	1966	1967
Olallie	California, United States	1967	1968
Logan L654	England	1968	1970
Thornless Boysen	England	1970	1972
Bedford Giant	Scotland	1970	1972
Himalaya Giant	Scotland	1970	1972
Scoresby Selection	Australia	1972	1973
Black Satin	Maryland, United States	1975	1976
Cherokee	Arkansas, United States	1975	1976
Comanche	Arkansas, United States	1975	1976
Dirksen Thornless	Maryland, United States	1975	1976
Georgia Thornless	Australia	1976	1977
Cheyenne	Arkansas, United States	1980	1982

affected flowers, and not from non-affected flowers from plants either exhibiting or not exhibiting foliar symptoms of Boysenberry decline. Similar mycelial growth was later induced in affected flowers of 'Riwaka's Choice' Boysenberry from a Boysenberry decline-affected planting at Albany, Auckland, by removing floricane growth and enclosing the flowers in plastic bags for 36 h to produce a high humidity. In some instances, by close examination, the whitish mycelium was visible in affected flowers of 'Riwaka's Choice' on this property.

Detection of a phytoplasma

From 20 plants affected with Boysenberry decline, four tested positive by nested PCR for the presence of a phytoplasma. All four plants were from the same row, although not immediately adjacent to each other. DNA sequence of the PCR fragments obtained from the symptomatic Boysenberry plants was identical to that of the phytoplasma associated with a disease of New Zealand flax (*Phormium tenax* J.R. & G. Forst.), *Phormium* yellow leaf (PYL).

Graft transmission trials

Sources of Boysenberry decline infection had been bottle grafted to primocanes of Boysenberry and hybrid berry selections in the season of graft inoculation. By spring of the following season these primocanes had become fruiting canes (floricanes). The infected grafted shoots grew readily on these floricanes, having shown little or no winter growth dormancy. In contrast, the primocanes of the test cultivars had become fully dormant by winter. As subsequent floricanes they did not recommence growth until spring. In spring and early summer the attached grafts developed conspicuous shoot and foliage stunting symptoms of Boysenberry decline, and sometimes produced long, wavy shoots with down-curled, reddened leaves. With the red raspberry cultivars, the shoots which had been grafted to them in most instances only remained alive for the season of graft inoculation, and by the following season usually were dead.

In the first season after graft-inoculation no symptoms of Boysenberry decline were seen on any of the Boysenberry and hybrid berry selections. In the second season after graft-inoculation symptoms appeared on six of the hybrid berry selections. Five of these, '8443W2', Logan 'L654', Logan 'LY59', 'McNichol's Choice', and 'Waldo' developed leaf down-curling, and purple-bronze colouring only on the earliest spring growth, recovering to make normal growth later in spring. Symptoms on the sixth, 'RS4' Youngberry were similar, but one of the floricane shoots had, in addition, a multiplicity of shoots, flowers with shortened stamens and leaf-like extensions on the sepals, and initially green, and later brown, hairy fruit. Some of the affected flowers on this plant were enclosed in plastic bags for 36 h to produce a high humidity, and a whitish, powdery mycelium growth and spores, typical of *C. rubi*, appeared on the styles and stigmas. In the third season after graft-inoculation, a further nine hybrid berry selections showed foliage symptoms (Table 1). All of these showed the down-curling and purple-bronzing of early spring growth, and in addition 'Riwaka Tahi' developed stunted shoot and foliage growth and flower abnormalities. Symptoms did not appear on three of the graft-inoculated hybrid berries until the fourth season (Table 1). 'Boysen 72', 'Kaiteri', and 'Waimate' all developed the down-curling and purple-bronzing on early spring growth and, in addition, 'Kaiteri' produced stunted shoot and foliage growth and flower abnormalities which became more pronounced in the fifth season. No symptoms which could be attributed to Boysenberry decline were observed on the remaining graft-inoculated cultivars. Symptoms were not seen on any of the red raspberry cultivars which had been graft-inoculated or on any of the non-graft inoculated control plants.

Establishment of *C. rubi* in New Zealand

Of the 20 blackberry and hybrid berry cultivars imported during the 20-year period, 2 were received from Australia, 3 from England, 2 from Scotland, and the remaining 13 from the United States (Table 2). As *C. rubi* has not been reported from Australia, England, or Scotland, it is most unlikely that *C. rubi* infection came on imports from those countries. Four of the United States imports came from western states (California and Oregon), areas where *C. rubi* has not been reported (Smith & Fox 1991). Five came from the eastern state of Maryland, and one, 'Darrow' from New York State. *C. rubi* was reported to be a very destructive disease on dewberries on the Delaware–Maryland peninsular early this century (Cook 1911), but was subsequently said to occur less commonly in the more northern states (Dodge & Wilcox 1941). Of the five cultivars introduced from Maryland, four of them, 'Black Satin', 'Dirksen Thornless', 'Smoothstem', and 'Thornfree' all have 'Merton Thornless' in their ancestry (Jennings 1988). 'Merton Thornless' may have contained the resistance to *C. rubi* that has been found in 'Arapaho,' one of the recent progeny of 'Thornfree'

(Buckley 1996). These four cultivars may thus have one parent resistant to *C. rubi*, which provides some resistance to infection. 'Darrow' and the unnamed bramble from Maryland were both released from plant quarantine in 1962, but did not have any subsequent use. The remaining imported cultivars 'Cherokee' and 'Comanche' (imported 1975), and 'Cheyenne' (imported 1980), were all from the southern state of Arkansas, an area where *C. rubi* commonly occurs (Plakidas 1937). As a source of the New Zealand *C. rubi* infection, 'Cheyenne' can be discounted, as it was not released from plant quarantine until May 1982, by which time Boysenberry decline symptoms had already been observed in New Zealand. It therefore seems possible that either the 'Cherokee' import, or the 'Comanche' import, or both, could have been infected with *C. rubi* on importation, and have been the means by which the fungus became established in New Zealand.

Spread within New Zealand

Plant quarantine records from 1975 comment that the cultivars 'Cherokee' and 'Comanche' on arrival from the United States as dormant rooted plants were "very dirty, with much soil adhering", suggesting that they may have been obtained from a neglected area in which *C. rubi* could have flourished. No apparent abnormalities were observed in the growth of the two cultivars during the year they were in plant quarantine and no diseases found, and both were released the following year to their importer at the former Levin Horticultural Research Centre of the Ministry of Agriculture and Fisheries (LHRC). In 1977 a field trial commenced at the LHRC to evaluate 'Cherokee' and 'Comanche' in comparison with Boysenberry (Porter et al. 1979). This continued until 1980. It was noted that there was some out-of-season flowering, and that abundant vegetative laterals from floricanes developed in the fruiting season (Anon. 1980). It is possible that both of these conditions could have been symptoms indicating presence of *C. rubi*. Plants of both cultivars were also grown at the Mt Albert Research Centre of DSIR as part of an investigation into raspberry bushy dwarf virus, without any growth abnormalities being observed. A further planting of 'Cherokee' and 'Comanche' was made in 1978 at the Kumeu Research Orchard of DSIR, as part of a collection of *Rubus* cultivars used to determine if any genetic resistance to dryberry (*P. sparsa*) could be located within the genus as a basis for breeding work (Hammett 1978), and this continued for several years. No growth abnormalities were reported on during this time. It is not known how closely this Kumeu trial was monitored, but as observation for dryberry in the fruit was the primary objective, symptoms of *C. rubi* on the flowers in spring may not have been noticed. Three of the four blackberry cultivars from Maryland ('Black Satin', 'Dirksen Thornless', and 'Thornfree') were also planted both for evaluation at LHRC, and in the Kumeu Research Orchard dryberry trial, similarly without reports of any growth abnormalities occurring. The first observation of Boysenberry decline occurred in mid summer (February) of 1982 (Table 3) on a commercial Boysenberry property c. 1 km from the planting of the *Rubus* cultivars on the Research Orchard, downwind of the prevailing spring time north-westerly to south-westerly winds.

Thus, if *C. rubi* was present in the *Rubus* collection, spores could have been blown downwind or carried by pollinating honey bees or other insects into the commercial Boysenberry planting. From its initial detection in the Kumeu district (Wood & Mossop 1984), Boysenberry decline rapidly spread to other properties in the Kumeu district (Eden & Wood 1986), to other areas north or north-west of Auckland city, and also to Drury and Thames (Table 3). This early spread was traced primarily to the Kumeu property where the disease was originally found, and which had sold Boysenberry plants to other properties including those at Drury and Thames. It is now believed that Boysenberry plants were also sold to growers in the Bay of Plenty explaining how the disease became established there. In 1982, Boysenberries on a commercial property at Te Puke were reported with bud proliferation and short flowering laterals. This may have been an early instance of Boysenberry decline, as presence of the disease was confirmed there in 1989. Boysenberry plants were also thought to have been sent to Motueka in the 1980s and planted within a short distance and upwind of where Boysenberry decline infected 'Marion' were found in the 1990s. In 1998, Boysenberry decline was found for the first time in several cultivars of red raspberry in Tauranga.

DISCUSSION

Rosette (sometimes referred to as witches' broom or double blossom), caused by *C. rubi* is still considered a limiting factor of blackberry production in the southern states of the United States (Gupton & Smith 1997). It was first reported there affecting

Youngberry in 1934 (Plakidas 1934) and Boysenberry in 1938 (Morris 1938). The detection in New Zealand of *C. rubi* in the flowers of some Boysenberry decline-affected *Rubus* plants has provided the best evidence yet of a possible cause of this disease. However, until Koch's postulates have been demonstrated, with previously healthy plants of Boysenberry inoculated with *C. rubi*, and producing typical Boysenberry decline symptoms, it will not be conclusively proven if this fungus is responsible for all of the symptoms of Boysenberry decline. There are some distinct differences in the symptoms described on blackberries affected with *C. rubi* in the United States from those occurring on Boysenberry in New Zealand. Symptoms resembling the "double blossom" symptom occurring on affected flowers in the United States, have been observed on 'Kotata' in New Zealand (Wood 1998), but this symptom is not typical of those occurring on affected Boysenberry and Youngberry flowers in New Zealand (Wood 1991). On New Zealand Boysenberry, the flower symptoms (shortened stamens, enlarged styles and stigmas forming hard, hairy, green-coloured cones of growth), and stem and foliage symptoms (large, compacted balls of growth on the floricanes, and long, wavy, proliferated shoots extending a metre or more in length above the Boysenberry row) (Wood 1998) do not seem to occur in the United States. These differences could be due to differences in climate, or other environmental conditions occurring between the two countries, or it may be that *C. rubi* is not entirely responsible for all of the symptoms seen in Boysenberry in New Zealand.

Although *Phormium* yellow leaf phytoplasma was detected in four plants showing symptoms of Boysenberry decline, this phytoplasma was not detected in the other 16 diseased Boysenberry plants tested. This may be because phytoplasmas are often sporadically distributed in affected plants, making detection unreliable. Alternatively, the phytoplasma

Table 3 Year and locations of Boysenberry decline observed in New Zealand, and the blackberry and hybrid berry cultivars affected.

Year infection first found	Location	District	Number of properties affected (new infections)	Cultivars affected
1982	Kumeu	Auckland	1	Boysenberry
1982	Te Puke	Bay of Plenty	1	Boysenberry
1984	Coatesville	Auckland	1	Boysenberry
1984	Puriri Valley	Thames	1	Boysenberry
1985	Drury	South Auckland	1	Boysenberry
1985	Kumeu	Auckland	1	Boysenberry
1986	Albany	Auckland	1	Boysenberry
1986	Huapai	Auckland	2	Boysenberry
1986	Kumeu	Auckland	1	Boysenberry
1986	Whenuapai	Auckland	2	Boysenberry
1989	Te Puke	Bay of Plenty	1	Boysenberry
1992	Whakatane	Bay of Plenty	3	Boysenberry
1993	Whakatane	Bay of Plenty	1	Kotata
1994	Cambridge	Waikato	1	Boysenberry
1994	Whakatane	Bay of Plenty	1	Aurora
1994	Whakatane	Bay of Plenty	1	Loganberry
1994	Whakatane	Bay of Plenty	1	Marion
1994	Whakatane	Bay of Plenty	1	Youngberry
1995	Motueka	Motueka	1	Marion
1995	New Plymouth	Taranaki	1	Boysenberry
1996	Motueka	Motueka	1	Boysenberry
1996	Nelson	Nelson	2	Boysenberry
1996	Te Awamutu	Waikato	1	Boysenberry
1997	Albany	Auckland	1	Youngberry
1997	Whakatane	Bay of Plenty	1	Karaka Black
1998	Tauranga	Bay of Plenty	1	Red raspberry
1998	Whakatane	Bay of Plenty	1	Olallie
1998	Whakatane	Bay of Plenty	1	Tayberry

may affect plant health without directly contributing to the symptoms of Boysenberry decline. However, the results could also, indicate that Boysenberry decline is a complex disease involving both a fungal pathogen and a phytoplasma. As PYL occurs in a wide range of plant hosts, including strawberry (*Fragaria x ananassa* Duch.) (Andersen et al. 1998b), it is not surprising that it has also been detected in Boysenberry. Although the role of PYL in Boysenberry decline is yet to be determined, it may be that the commonly occurring Boysenberry leaf hopper *Ribautiana tenerrima* (Herrick-Schauffer) is a vector (Wood & Charles 1989). Leaf hopper control in New Zealand commercial Boysenberry plantings, through the use of insecticidal sprays, was implemented during the 1990s (Wood & Langford 1996). It has subsequently been observed that Boysenberry decline has not spread rapidly in the areas where leaf hopper control has been practiced (Motueka and Nelson), compared with the rapid spread in areas where there was no leaf hopper control practiced (Auckland, and in the early years at Whakatane). There may be other reasons for this, for example differences in climate between the districts, but it does raise the possibility that PYL phytoplasma, a leaf hopper transmitted pathogen, could be involved in Boysenberry decline.

Until recently it was not known what infectious agent was being transmitted in grafting experiments. It now seems probable that *C. rubi*, present in the decline affected floricanes, infected the primocanes to which they were grafted. This infection could then have moved into, and then become established in the crown of the plant. Rosette type growth has been observed in New Zealand in the crown of naturally infected 'Marion'. Overseas research has shown that in blackberry cultivars which, for propagation purposes have been tip rooted, the fungus can become established in the crown of new plants (Plakidas 1937; Smith & Fox 1991). The grafting trials showed that when such infection does take place through graft inoculation, it will take at least two, and possibly up to four seasons before symptoms become apparent (Table 2), and that in many instances only a leaf reddening and epinasty will be apparent on the spring floricane growth, and that the flower, stem, and long extension growth symptoms may not occur. Thus, if infection does become established in the crown of susceptible *Rubus* types, it may be several years before plants begin to show symptoms, and this could explain the inability of growers to keep ahead of the infection even though they remove plants as soon as symptoms are seen.

With regard to the probable introduction of *C. rubi* to New Zealand in the 'Cherokee' and 'Comanche' importations, it was the practise to grow the rooted plants under quarantine conditions for a year, and if no pests or diseases were observed, to release the plants to the importer. After 1980, this policy was changed, and imports made as rooted plants were re-propagated by means of softwood cuttings taken from one of the imported plants of each particular cultivar. All of the original imported plant material was then destroyed by autoclaving, as a precaution against the inadvertent release of soil-borne pathogens which may have been present on the roots (Wood 1989). Only the newly propagated material was released to the importer, and only if it had been found to be free from any plant pathogens. With 'Cherokee' and 'Comanche' being imported before 1980, the original rooted plants were sent directly to the LHRC importer where they were established in large half-barrels in a screenhouse. Assuming that *C. rubi* was present in the crowns of these plants, any small rooted plants taken from them could also have been infected, and this may be the method by which the Kumeu Research Orchard plants became infected. However, both cultivars have a self-supporting, upright habit (Jennings 1988), and overseas research has shown that in erect blackberries the fungus is confined to the buds and has not been found in other tissues (Smith & Fox 1991). If the fungus was present it must therefore have been confined to the lower buds of the primocanes and only the tops of the crowns. It is possible that one or more of the other blackberry introductions received from Maryland and grown at Kumeu ('Black Satin', 'Dirksen Thornless', and 'Thornfree') could have been the carrier of the disease though it is thought more likely that they are immune. If any of these cultivars were infected, they may not have shown any noticeable symptoms at Kumeu except for a few affected flowers, spores from these being the source of infection in the nearby commercial Kumeu Boysenberry planting. As LHRC, the Kumeu Research Orchard, and the commercial planting have closed, and the plants in question no longer exist, it is not possible to confirm the trail of infection, but this seems to be the most likely explanation as to how *C. rubi* became established in New Zealand. Had the re-propagation process been in operation in plant quarantine when 'Cherokee', 'Comanche', and the Maryland blackberries were imported, all of the problems which have subsequently occurred with *C. rubi* in New Zealand may not have occurred.

In consideration of the other explanation as to the origin of *C. rubi* in New Zealand, that the disease has been present in wild blackberries since the middle of the 19th century, old archive correspondence on *Rubus* diseases in New Zealand was examined without any such record being found. Loganberries have been found with Boysenberry decline in recent years (Wood & Langford 1996). Prior to this they are known to have been grown in New Zealand since 1901 (Anon. 1901), but there were no reports of Boysenberry decline till the 1990s. Had *C. rubi* been present in New Zealand early this century it would seem likely that other Loganberries or any cultivated blackberries would have become infected and shown symptoms at this time. Wild blackberries with symptoms suspected to be those of Boysenberry decline on their lower shoot growth have been observed since the 1980s at Kumeu, Whakatane, and Motueka (Wood 1998). It has not been determined if *C. rubi* was present in any of them.

In early investigations of Boysenberry decline, before any presence of *C. rubi* was known, the incorporation into new hybrid berry types of resistance to the disease seemed to be the only real solution. Although it was found infected in both Auckland and Whakatane (Table 3), Youngberry offered some hope as a resistance conferring, breeding parent, as it is the most resistant of the Boysenberry types. Symptoms of Boysenberry decline in Youngberry appeared to lack some of the growth and leaf symptoms which occurred on Boysenberry, and it did not become so readily infected. Youngberry growth had not been observed to be proliferated and extended as occurred on Boysenberry. In 1986 on a property at Albany, Auckland, a large block of Youngberry plants was adjacent to a severely affected Boysenberry block, and the grower was confident that the Youngberry block would not be affected. The Boysenberry block was removed some years ago, but flower and some foliage symptoms of Boysenberry decline is now appearing on some of the growth in the Youngberry block. A similar situation occurred on a property at Whakatane. In this instance the Youngberries had been surrounded on three sides by severely affected Boysenberries, but it has only been in recent years that a small amount of infection has appeared. Because of the confusion over Boysenberry and Youngberry types, and the possibility of seedling sports of the two appearing in the field rows and becoming infected, there is a difficulty in determining exactly how resistant Youngberry is. Until recently, the Tayberry was also thought to be resistant, but as some infection has appeared in it (Table 3) this does not now seem likely. In the United States, blackberry growers have been advised to plant and grow cultivars which show resistance to rosette, rather than to rely on chemical means of control (Buckley 1996). This would not solve the present problem of *C. rubi* in Boysenberries in New Zealand, and chemical control would seem to be the only interim measure that can be used. In the long term, if work proceeds in New Zealand to breed a Boysenberry type incorporating resistance to *C. rubi*, blackberries, such as the *C. rubi* immune 'Himalaya Giant' (Jennings 1988) or the cultivar 'Thornfree' may be the best source of a resistant parent.

Red raspberries had, until recently, been thought to be immune to Boysenberry decline, as none had been found naturally infected, and it had not been possible to infect them by graft inoculation (Table 1). As infection has recently been found in a commercial planting of red raspberry at Tauranga, where Boysenberry decline was present in adjacent Boysenberries, this would appear not to be so. *C. rubi* has only rarely been reported in red and black raspberries in the United States (Jennings 1988; Smith & Fox 1991).

Until the 1960s, copper-based sprays had been used for the control of dryberry (*P. Sparsa*) which had appeared affecting some plantings soon after the introduction of Boysenberry to New Zealand. Because of increasing control problems with dryberry, trials of various systemic fungicides in the early 1960s showed that Maneb would give an improved control of dryberry compared with that provided by copper-based sprays (Newhook & Brown 1963; Thiele 1963). Other systemic fungicides gave similar results (Tate 1979) so that Boysenberry growers relied less on the copper-based sprays for dryberry control. Copper-based sprays were also found to be phytotoxic to earthworms, and their use were discouraged for this reason. The systemic fungicides do not appear to have been effective against *C. rubi*. Thus, if spraying with copper-based sprays had not ceased or been restricted, the problem of *C. rubi* and Boysenberry decline may never have arisen or become the major problem it has, as in the United States effective control of *C. rubi* has been achieved with copper sprays timed to coincide with infection periods during the blooming of the rosetted flowers (Smith & Fox 1991).

In summary, both Boysenberry and Youngberry were introduced in the mid to late 1930s, Boysenberry from a nursery in California, an area where *C. rubi* does not occur, and Youngberry

probably from the same source. Distributed throughout New Zealand and grown widely, neither showed any symptoms of Boysenberry decline until 1982, when symptoms were observed on commercial Boysenberries at Kumeu. Although shown to be graft-transmissible, the causal agent was a mystery until recently, when the fungal pathogen *C. rubi* was found in the flowers on some Boysenberry and Youngberry plants, and which appears to be associated with Boysenberry decline. However, flower and foliage symptoms are not identical to those occurring on *Rubus* in the United States, and the possible involvement of *Phormium* yellow leaf phytoplasma, found in some affected plants, has yet to be determined. Of 20 blackberry and hybrid berry cultivars introduced from 1962 and released from plant quarantine by 1982, *C. rubi* seems most likely to have been present in the cultivars 'Cherokee' and 'Comanche' from Arkansas, where the fungus commonly occurs. *C. rubi* may have been present in the upper crowns of these cultivars. Grafting trials showed that a graft-transmissible agent obtained from Boysenberry decline-affected plants, and which could be *C. rubi*, may take several seasons to express symptoms. Thus, rooted plants taken from the imported 'Cherokee' and 'Comanche' crowns, and planted in the dryberry resistance trial at Kumeu could have spread the fungus, once a few sporulating flowers had formed. If no other infectious agent is involved in Boysenberry decline, it should be possible to achieve control with copper-based sprays, but in the long term the production of a Boysenberry type cultivar incorporating resistance to the disease, may be the best solution.

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REFERENCES

Andersen, M. T.; Beever, R. E.; Gilman, A. C.; Liefting, L. W.; Balmori, E.; Beck, D. L.; Sutherland, P. W.; Bryan, G. T.; Gardner, R. C.; Forster, R. L. S. 1998a: Detection of phormium yellow leaf phytoplasma in New Zealand flax (*Phormium tenax*) using nested PCRs. *Plant Pathology 47*: 188–196.

Andersen, M. T.; Longmore, J.; Liefting, L. W.; Wood, G. A.; Sutherland, P. W.; Beck, D. L.; Forster, R. L. S. 1998b: Phormium yellow leaf phytoplasma is associated with strawberry lethal yellows disease in New Zealand. *Plant Disease 82*: 606–609.

Anonymous 1901: Loganberry P.13 *in*: Descriptive catalogue of fruit trees etc. 1901–1902. Warkworth, E. Morrison and Sons, Red Bluff Orchards and Nurseries. 42 p.

Anonymous 1936: The new Boysenberry, the finest vine berry yet developed. Sales brochure, Knott's Berry Place, Buena Park, California, United States. 1p.

Anonymous 1937: Young Berry; Yates small fruits. P. 10 *in*: Nursery catalogue for 1937. Auckland, Arthur Yates & Co. Ltd. 50 p.

Anonymous 1939: The Boysenberry: Yates smallfruits. P. 34 *in*: Nursery catalogue for 1939. Auckland, Arthur Yates & Co. Ltd. 48 p.

Anonymous 1942: Hybrid berries. P. 17 *in*: Miscellaneous fruit. New Zealand Department of Scientific and Industrial Research annual report for 1942. 34 p.

Anonymous 1954 (Undated, but thought to be c. 1954): History of the Boysenberry. Publicity brochure, Bush Berry Advisory Board, Modesto, California, United States. 3 p.

Anonymous 1980: Bramble: cultivars. P. 22 *in*: Todd, J. C. *ed.* Summary of research and experimental work on berryfruits in New Zealand 1979/80. Advisory Services Division, New Zealand Ministry of Agriculture & Fisheries. 65 p.

Anonymous 1997: Berries: acreage, yield, production, price and value by crop, state and United States, 1994–96. *California Fruit and nut Review 17 (March)*: 2.

Bailey, L. H.; Bailey, E. Z. 1978 (revised): Boysenberry. P. 175 *in*: Hortus third. New York and London, MacMillan Publishing Co. Inc. 1290 p.

Baker, R. E. 1949: New blackberry; Boysen variety, shiny type, has flavour resembling a high quality wild berry. *California Agriculture 3 (April)*: 11–14.

Ballantyne, J. A. 1947: The Youngberry and Boysenberry. Introduced berry fruits of value. *The Agricultural Gazette of New South Wales 58 (September)*: 479–482.

Buckley, J. B. 1996: Evaluation of blackberry cultivars and breeding selections for rosette resistance. *Louisiana Agriculture 39 (Summer)*: 25–26.

Butterfield, H. M. 1938: Looking over new berry varieties. *Pacific Rural Press, San Francisco, January 15, 1938*: 64.

Converse, R. H.; Clarke, R. G.; Oman, P. W.; Milbrath, G. M. 1982: Witches' broom disease of black raspberry in Oregon. *Plant Disease 66*: 949–951.

Cook, M. T. 1911: The double blossom of the dewberry. *Delaware College Agricultural Experiment Station Bulletin No. 93.* 12 p.

Cunningham, G. H. 1940: Youngberry; Varietal trials. P. 43 *in*: The Plant Diseases Division. *New Zealand Department of Scientific and Industrial Research Bulletin No. 33.* 56 p.

Darrow, G. M. 1937: Blackberry and raspberry improvement. Pp. 496–533 *in*: Yearbook of agriculture, 1937. United States Department of Agriculture. 1497 p.

Davey, M. 1947: The culture of Boysenberries. *The Orchardist of New Zealand 20 (October)*: 2–5.

Dodge, B. O.; Wilcox, R. B. 1941: Diseases of raspberries and blackberries. *United States Department of Agriculture Farmers Bulletin No. 1488.* 33 p.

Eden, M. A.; Wood, G. A. 1986: Boysenberry disease danger. *Horticulture News 8 (November)*: 25.

Forster, R. L. S.; Andersen, M. T. 1998: Research into phytoplasmas. *In*: The berry report. *New Zealand Commercial Grower 53(August).* Pp. 36–37.

Garner, R. J. 1958: Bottle grafts. The grafters handbook. East Malling Research Station. 260 p.

Gupton, C. L.; Smith, B. J. 1997: Heritability of rosette resistance in blackberry. *HortScience 32*: 940.

Hall, H. K. 1992: Hybrid berry breeding—DSIR research to improve the Boysenberry. *The Orchardist of New Zealand 65 (April)*: 18–20.

Hammett, K. R. W. 1978: Boysenberry: natural resistance to dryberry. P. 25 *in*: Todd, J. C. *ed.* Summary of research and experimental work on berryfruits in New Zealand 1977/78. Advisory Services Division, New Zealand Ministry of Agriculture & Fisheries. 41 p.

Havis, L. 1941: The Boysenberry in Ohio. *Bimonthly Bulletin No. 209, Ohio Agricultural Experiment Station 26*: 36–38.

Hogg, D. J. 1948: Boysenberry culture in Nelson Central. *New Zealand Journal of Agriculture 76*: 363–364.

Jennings, D. L. 1988: Breeding blackberries for eastern and southern North America. Pp. 50–52: Youngberry, Boysenberry and Nectarberry. Pp. 63–64: *Cercosporella rubi* P.106 *in*: Raspberries and blackberries: their breeding, diseases and growth. London, Academic Press. 230 p.

Knott, W. 1935: A new money-making berry. *Better Fruit 30 (December)*: 6.

Langford, G. I.; Jones, A. T.; Wood, G. A. 1995: Control of Boysenberry decline. HortResearch client report to New Zealand Boysenberry Council Ltd. 5 p.

Langford G. I.; Mavromatis, G. 1981: Boysenberry production: New Zealand areas. Pp. 33–34. Boysenberry culture: strains. Pp. 34–36. *In*: A review of the Boysenberry industry. New Zealand Ministry of Agriculture and Fisheries. 61 p.

Meer, F. A. van der 1987: *Rubus* stunt. Pp. 197–203 *in*: Virus diseases of small fruits. *United States Department of Agriculture Handbook Number 631.* 277 p.

Morris, H. F. 1938: Experimental data: disease resistance. Pp. 11–12. Discussion of varieties; Boysen. Pp. 15–16. *In*: Blackberry and dewberry varieties in East Texas. *Texas Agricultural Experiment Station Bulletin No. 558.* 30 p.

Murant, A. F.; Roberts, I. M. 1971: Mycoplasma-like bodies associated with *Rubus* stunt disease. *Annals of Applied Biology 67*: 389–393.

Newhook, F. J.; Brown, I. L. 1963: Control of dryberry disease: results of Boysenberry spray trials. *The Orchardist of New Zealand 36*: 334–337.

Pennycook, S. R. 1998: Blackberry in New Zealand. *Plant Protection Quarterly 13*: 163–174.

Plakidas, A. G. 1934: The rosette disease of blackberries and dewberries. *Louisiana State University College Bulletin No. 250, June 1934.* 8 p.

Plakidas, A. G. 1937: The rosette disease of blackberries and dewberries. *Journal of Agricultural Research 54*: 275–303.

Porter, L. A.; Thompson, A. F.; Broadbent, N. D. 1979: Bramble: cultivars. Pp. 42–43 *in*: Todd, J. C. *ed.* Summary of research and experimental work on berryfruits in New Zealand 1978/79. Advisory Services Division, New Zealand Ministry of Agriculture and Fisheries. 66 p.

Scott, D. N.; Langford, G. I.; Pringle, G. J.; Hall, H. K.; Patel, N. P.; Desborough P. 1993: Industry background: Boysenberries/blackberries/hybrid berries. P. 21 *in*: Review of berryfruit research. *The Horticulture and Food Research Institute of New Zealand Ltd Internal Report No. 93/25.* 35 p.

Sharp, J. E. 1939: Boysenberries in Hawke's Bay. *New Zealand Journal of Agriculture 58*: 355.

Shoemaker, J. S. 1950: Bramble-fruit culture: leading blackberry, dewberry, and related varieties: Boysen. P. 237; Young. P. 238 *in*: Small-fruit culture. Philadelphia, Toronto, The Blackiston Company. 433 p.

Smith, B. J.; Fox, J. A. 1991: Rosette (Double Blossom). Pp. 13–15 *in*: Compendium of raspberry and blackberry diseases and insects. APS Press, The American Phytopathogical Society. 100 p.

Steller, O. A. 1937: The giant Boysenberry goes national—the brambleberry page. *Better Fruit 32 (February)*: 20.

Tate, K. G. 1979: Brambles: dryberry. Pp. 44–45 *in*: Todd, J. C. *ed.* Summary of research and experimental work on berryfruits in New Zealand 1978/79. Advisory Services Division, New Zealand Ministry of Agriculture and Fisheries. 66 p.

Thiele, G. F. 1963: Control of dryberry disease opens way to increased production of Boysenberries. *New Zealand Journal of Agriculture 107 (July)*: 23–25.

Thompson, M. M. 1961: Cytogenetics of Rubus. II. Cytological studies of the varieties 'Young', 'Boysen', and related forms. *American Journal of Botany 48*: 667–673.

Wagnon, H. K.; Williams, H. E. 1970: Blackberry dwarf. Pp. 126–128 *in*: Virus diseases of small fruits and grapevines. University of California, Division of Agricultural Sciences Handbook. 290 p.

Waldo, G. F. 1950: Additional forms possible. Pp.4–5 *in*: Breeding blackberries. *Oregon State College, Corvallis, Agricultural Experiment Station Bulletin 475.* 39 p.

Waldo, G. F. 1968: Blackberry breeding involving native Pacific Coast parentage. *Fruit Varieties and Horticultural Digest 22*: 3–7.

Wilhelm, S. 1951: Diseases of trailing blackberries. *Western Fruit Grower 5 (May)*: 29–31.

Wood, G. A. 1989: Propagation, virus-screening and heat therapy of northern hemisphere imports of *Ribes, Rubus, and Vaccinium. New Zealand Journal of Crop and Horticultural Science 17*: 271–274.

Wood, G. A. 1991: Three graft-transmissible diseases and a variegation disorder of small fruit in New Zealand. *New Zealand Journal of Crop and Horticultural Science 19*: 313–323.

Wood, G. A. 1998: Boysenberry decline. Pp. 31–33 *in*: Virus and virus-like diseases and non-infectious disorders of small fruits in New Zealand. *The Royal Society of New Zealand Bulletin 33.* 86 p.

Wood, G. A.; Charles, J. G. 1989: Boysenberry decline disease investigated. *New Zealand Commercial Grower 44 (January/February)*: 8–9.

Wood, G. A.; Eden, M. A.; Langford, G. I. 1992: Boysenberry decline disease: *New Zealand Commercial Grower 47 (September/October)*: 40–41.

Wood, G. A.; Langford, G. I. 1996: Boysenberry decline: *New Zealand Commercial Grower 51 (November/December)*: 38–39.

Wood, G. A.; Mossop, D. W. 1984: Boysenberry decline. P. 27 *in*: Langford, G. I. *ed.* A summary of berryfruit research in New Zealand 1983/84. Advisory Services Division, New Zealand Ministry of Agriculture and Fisheries. 51 p.

Woodhead, C. E.; Chamberlain, E. E. 1940a: Area under small fruits 1939. P.117 *in*: Report on a survey of small-fruit culture in New Zealand, November–December, 1939. *The Orchardist of New Zealand 13 (July)*: 110–117.

Woodhead, C. E.; Chamberlain, E. E. 1940b: Diseases and pests; Loganberries, Boysenberries, etc. P. 140 *in*: Report on a survey of small-fruit culture in New Zealand, November–December, 1939. *The Orchardist of New Zealand 13 (August)*: 139–141.

Zeller, S. M. 1927: Dwarf of blackberries. *Phytopathology 17*: 629–648.

Zeller, S. M.; Milbrath, J. A. 1940: Blackberry dwarf in Boysenberries and Youngberries. *Plant Disease Reporter 24*: 430.