CAMELLIA FLOWER BLIGHT IN NEW ZEALAND

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ABSTRACT

Camellia flower blight is caused by Ciborinia camelliae. Originating in Japan, the disease spread to the USA in 1938 and was first found in New Zealand in Wellington in 1993. In 1996, the disease was found in Waikanae and Wanganui. A survey carried out from September to November 1997 showed that the disease had spread up the west coast of the North Island as far as New Plymouth. It had also spread over the Rimutakas to the Wairarapa as far as Pahiatua. The disease had spread north from Wellington almost to Taihape and there were two isolated outbreaks, one in Turangi and one at Hatepe near Lake Taupo. The distribution suggests that dispersal of the disease has been primarily by windborne spores, but there is potential for future dispersal from nurseries.

Keywords: ascospores, camellia flower blight, Ciborinia camelliae, distribution.

INTRODUCTION

The camellia is a popular ornamental plant belonging to the Family Theaceae. There are more than 200 species in the genus Camellia (Hagiya 1997) but only four of these are important commercially. Tea is made from the leaves of C. sinensis var. sinensis, and C. japonica, C. reticulata and C. sasanqua are important landscape plants in New Zealand and overseas. There is a wide range of flower shape, size and colour and because the plants are hardy and relatively pest-free, they are common and widespread in New Zealand gardens and public parks.

The fungal pathogen Ciborinia camelliae causes camellia flower blight disease. It is host specific to Camellia species and infects only the blooms. The aesthetic appeal and hence value of camellias is reduced by unsightly blooms. The presence of the disease in New Zealand could threaten an export industry thought to be worth about $2-3 million annually (Bieleski pers. comm.).

Sclerotia of C. camelliae survive over summer and winter in plant debris and produce apothecia in spring during the main camellia flowering season. The apothecia release ascospores into the air and those which land on camellia flowers can cause new infections. Each infection results in a small, brown spot which enlarges and may coalesce with other infections until the entire flower becomes brown and falls from the plant. A characteristic sign of the disease is the grey or white felt-like mycelium around the base of the petals when the sepals are removed. There are no asexual spores or secondary spread. Sclerotia form in the infected flowers and lie dormant in the soil until the next flowering season. Sclerotia of Ciborinia spp. incorporate host tissue and thus differ from those of the related genera Botrytis and Sclerotinia (Kohn and Nagasawa 1984).

Camellia flower blight was first reported and described in Japan in 1919 (Hara 1919). In 1938 it was discovered in San Fransico, USA (Hansen and Thomas 1940) where it was found on camellia plants imported from Japan by the horticultural industry. The disease spread up the west coast to Washington State and to the east coast, where it is now found throughout the southern and southeastern states (Holcomb 1983).

In 1993, camellia flower blight was identified in Wellington, New Zealand (Stewart and Neilson 1993) and a survey of the Wellington region found it to be already widespread in the capital city (C. Hill cited by Frampton, 1994). The disease was found in Waikanae and Wanganui in 1996 but was thought to be limited to these three locations.

In September of 1997, we identified the disease from five different locations in Palmerston North. The discovery of camellia flower blight in Palmerston North prompted us to undertake a survey, the objectives of which were to determine the distribution of the disease in the lower North Island, and its likely method of spread.

METHODS
The survey was carried out over nine days in the period September to November 1997 by driving along the main highways and some back roads, from Wellington north as far as Hamilton. We examined those camellias in public parks and gardens, church yards, roadsides and private gardens which were either accessible from the footpath or where permission was granted to access the property.

Field identification was by flower symptoms and signs: the pale to mid-brown rot of the petals and the ring of grey or white mycelium around the base of the petals. In the laboratory, samples of infected flowers from each location were held under suitable conditions for sclerotial formation to confirm the field identification. Flower blights caused by *Botrytis*, *Monochaetia*, *Penicillium* and *Pestalotia* could be distinguished by microscopic examination.

RESULTS
The disease was found at 62 of the 178 sites surveyed. The locations at which the disease was found in the North Island are shown in Figure 1. From Wellington, the disease was found at frequent intervals up the west coast to Wanganui and through to New Plymouth. The disease was not found on the western side of Mt Taranaki, from Manaia in the south to New Plymouth in the North.

From Wellington eastwards, the disease was found in Upper Hutt and Kaitoke, over the Rimutaka Ranges into the Wairarapa, and as far north as Pahiatua. However, we did not find the disease in Woodville, nor further north in the Hawkes Bay. The disease is less well established in the Wairarapa than on the west coast, with no disease found in some towns.

North of Palmerston North the disease was found at several locations up to Kimbolton. We did not find it at Bulls, but it was present in Marton and Hunterville. The record from Mangaweka was from a single bush that appeared to be a recent planting.

The northern most record of the disease was from Hatepe, on the east shore of Lake Taupo. There was no sign of the disease in the Taumarunui, Te Kuiti, Hamilton, Cambridge, Tokororo, Rotorua or Taupo areas.

DISCUSSION
This survey shows that camellia flower blight is now widespread and well established in the central and southern North Island. The spread of the disease in New Zealand mirrors that of California and the eastern states, where there was a single introduction event from which the disease rapidly became widespread (Alford *et al.* 1961).

We did not find the disease north of Taupo-Te Kuiti nor east in the Hawkes Bay and we consider these negative findings show the limit of the disease distribution thus far. The detection of single diseased blooms at Urenui (northeast of New Plymouth), Turangi, Taihape and Pahiatua (east of Palmerston North) also suggest that these areas represent the disease front.

Before this survey, camellia flower blight was known to be present in Wellington, Waikanae and Wanganui. This distribution suggested that spread of the disease was by human activity moving infected plants or flowers around the country. Our results, however, show that the disease is widespread up the west coast of the North Island, a distribution which suggests that spread by windborne ascospores was the predominant method of dispersal.
Wind dispersed ascospores would explain the widespread distribution of the disease which we found in this survey. Since we could find no camellia plants between Upokorongo and a farm at Kakatahi, which is 45 km to the north, ascospores may be able to travel about 50 km by wind. Camellia flower blight was found on three mature camellia plants at this farm and the owner had not purchased any camellias so that spread on planting material was unlikely to have occurred. These trees were well back from the edge of the garden and could not have been infected by enthusiasts leaning over the fence and touching them, if indeed it is possible to transmit ascospores on skin or clothing. Windborne ascospores would appear to be the only logical method by which the pathogen could have spread to this location.

The disease was first found in Wellington in 1993 but had probably been present for at least two years before then (Stewart and Neilson 1993). If we assume that it started spreading northwards in 1992, then it has taken six years to cover the 355 km from Wellington to New Plymouth, an average of 55–60 km per year.
The disease is likely to continue spreading north and east up the North Island and it is also likely that it has been (or will be) blown (or transported) across Cook Strait. In future, spread of camellia flower blight on planting material could become an important issue as more foci of disease develop on properties near nurseries and garden centres. We found one retail outlet with some plants bearing infected flowers, but there was only one instance in the survey where a diseased site appeared to be linked to a new planting (Mangaweka) and one where there is probably such a link (Hatepe).

REFERENCES