

Rate of spread of myrtle wilt disease in undisturbed Tasmanian rainforests

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Summary

The long-term rate of spread of myrtle wilt disease was monitored in plots within undisturbed forests at six localities in Tasmania representing a range of rainforest types containing myrtle, *Nothofagus cunninghamii*. The incidence of myrtle wilt, indicated by trees showing evidence of attack by the mountain pinhole borer *Platypus subgranosus*, at the start of the monitoring period was 15.8–45.4% of standing trees across the range of sites. After 12–18 y, the incidence was 16.8–50.9%, representing an increase in myrtle wilt of 0.08–0.67% per year.

There was a significant effect of rainforest type on both disease incidence and the level of physical damage to myrtle trees (broken limbs and stem wounds), callidendrous rainforests having higher disease and damage levels than thamnic and implicate stands. The effect of altitude on disease incidence was also significant, with myrtle wilt levels being lower at higher altitudes.

The factors affecting the levels of myrtle wilt and the current significance of the disease in Tasmanian rainforests are discussed; disease levels recorded over the sampling period indicate a currently stable situation.

Keywords: wilts; rain forests; disease surveys; disease prevalence; spread; myrtle wilt disease; *Nothofagus*; Tasmania

Introduction

Myrtle wilt is the main cause of death of myrtle, *Nothofagus cunninghamii* (Hook. Oerst.) in cool temperate rainforest in Tasmania (Elliott *et al.* 1987) and Victoria (Packham and Kile 1992). Tree death results from infection from the pathogenic hyphomycete fungus, *Chalara australis* (Kile and Walker 1987), which enters attacked trees through wounds or via root grafts with nearby diseased trees (Packham 1991, 1994).

The occurrence of dead and dying myrtle trees in cool temperate rainforest was first reported by Howard (1973), who noted that affected myrtles had extensive attack by the mountain pinhole borer, *Platypus subgranosus* Schedl., and for some years this insect was assumed to be a vector of a pathogenic fungus.

Although *P. subgranosus* attack is usually associated with dead and dying trees, Kile and Hall (1988) showed that *Chalara* was not dependent on *P. subgranosus* for entry into trees and that infection occurred prior to attack by the beetles. *Platypus subgranosus* attacks wounded and burnt trees, but only trees infected with *Chalara* suffer sustained attack (Kile *et al.* 1990).

Myrtle wilt is widespread in Tasmanian rainforests. Surveys along transects through a range of undisturbed rainforest types and localities across the state showed that an average of 24.6% (range 9.4–53.4%) of standing myrtles were dead or dying from the disease. At the time of the surveys (mid-1980s), dying trees were occurring at an average rate of 1.6% of live trees per year (Elliott *et al.* 1987). After further investigation of the time taken by attacked trees to die, the mortality rate was revised down to 0.61% (range 0–1.73%) per year (Packham 1994).

The high level of myrtle wilt observed in rainforest areas in Tasmania in the 1970s and 1980s was a great concern to forest managers. The mechanism of spread of this disease was not fully understood at the time, and the longer-term potential for increased incidence was unknown. Disturbance exacerbates the effects of myrtle wilt (Howard 1973) and there were numerous observations by the present authors through the 1970s and 1980s of increased incidence of dying myrtles adjacent to roading and logging disturbance. Monitoring of disease spread in rainforests disturbed by a range of logging and regeneration treatments showed that cumulative mortality of myrtle 10–26 y after treatment ranged from 25.4 to 44.8%. Annual mortality rates from myrtle wilt were initially high at all sites after treatment, but after 3–9 y they had declined significantly to stabilise at background levels previously reported for undisturbed myrtle forests (Elliott *et al.* 2005).

Myrtle wilt was also commonly observed in undisturbed, often remote rainforest areas, many of which were in conservation reserves. Thus, from a nature conservation view as well as a timber production view, knowledge of the potential impact of the disease was required. We established plots to assess the rate of spread of myrtle wilt across a range of rainforest types and localities in order to monitor changes in the incidence of the disease over the medium to long term. This paper reports the changes in the incidence of myrtle wilt in these undisturbed forests over a 12–18 y period.

Methods

Plot establishment

Permanent plots were established at six sites across Tasmania representing major rainforest types as defined by Jarman *et al.* (1984). The forest at all sites had either never been disturbed or had remained undisturbed for several decades with minimal signs of previous disturbance present. Site descriptors, including vegetation types, are listed in Table 1 and site locations are shown in Figure 1.

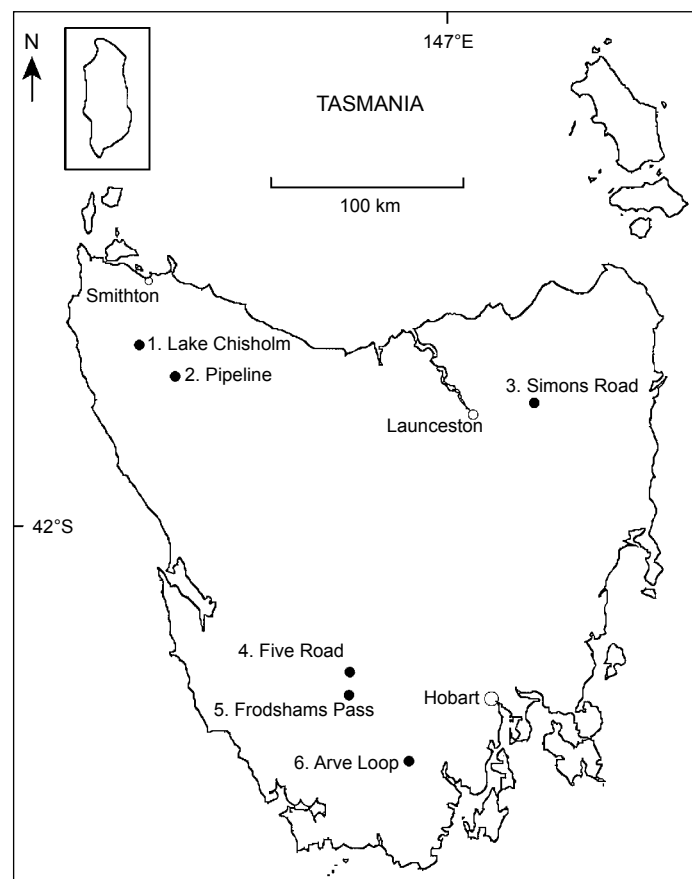


Figure 1. Plot locations

The Arve Loop site was set up in 1982 as a 3.5 ha rectangular plot, and the location and diameter at breast height over bark (dbhob) of 327 myrtle trees > 5 cm diameter were recorded. In 1990, diameters of the plot trees were remeasured and trees that had grown into the 10 cm class in the intervening 8 y at this site were included in the survey, but trees which had grown into the 5–10 cm class were not included.

The other five sites were established in 1988–1989 in homogeneous forest areas of the desired rainforest type. At each site, sufficient circular plots of 50 m radius (0.79 ha) were established to obtain a minimum of about 200 standing myrtle trees with a dbhob ≥ 15 cm (including standing dead trees identifiable as myrtles). At each site, the first plot was located when the first myrtle recently attacked by *P. subgranosus* or dying from myrtle wilt > 100 m from the road edge was found. The centre of the plot was then obtained by measuring 50 m from this tree on the same bearing. Any subsequent plots at the site were established when a currently attacked tree was found, at least 150 m from the edge of the previous plot. Orientation from the first plot depended on the size and shape of the rainforest area. The centre of each plot was marked with a steel picket and all standing myrtle trees over the threshold diameter were numbered with yellow paint on the stem, and distances and compass bearings from the centre peg were recorded. All numbered trees were assessed at each sampling occasion. Trees in these circular rate-of-spread plots which grew into the threshold diameter class (≥ 15 cm dbhob) after the first assessment were included in subsequent assessments, except the most recent one.

Health and damage assessments

At plot establishment and at each subsequent assessment, all numbered standing myrtle trees (alive and dead) were assessed for health status using eight health categories (Table 2). The Arve Loop plot was assessed on seven occasions over 18 y; plots at all other sites were assessed on four occasions over an 12-y period. Attack by *P. subgranosus* was used as an indicator of infection by myrtle wilt disease. Dead and dying trees with evidence of *P. subgranosus* attack and live trees with recent attack were assumed to have myrtle wilt disease. Trees assessed as health status class 2 (healthy but with old *P. subgranosus* attack on the lower stem) were not included in the total of diseased trees

Table 1. Site descriptors for myrtle wilt rate-of-spread plots

Site	Grid reference (1:100 000 series)	Geology	Altitude (m)	Forest type*
Arve Loop	796242	Jurassic dolerite	440–460	<i>Eucalyptus regnans</i> , <i>E. obliqua</i> over thamnian rainforest T1a/T3a
Five Road	526694	Ordovician limestone	440	Scattered <i>E. delegatensis</i> over callidendrous rainforest C1a
Frodshams Pass	496573	Cambrian dolomite	540	Implicate/thamnian rainforest I4a/T1b
Simons Road	442207	Devonian granite	820	Callidendrous rainforest C2a
Lake Chisholm	370444	Precambrian mudstone	120–180	<i>E. ovata</i> , <i>E. nitida</i> over callidendrous/thamnian rainforest CT1a/T3a
Pipeline 18 Mile Peg	583273	Tertiary basalt	500	Thamnian rainforest T1a

*Rainforest type classification after Jarman *et al.* (1984): callidendrous = tall trees, park-like with open understorey; thamnian = trees of moderate height with a shrubby, relatively open understorey; implicate = shorter trees with an open canopy and a tangled understorey

Table 2. Health status classes (HSC)*

1. Healthy
2. Healthy, but with old <i>Platypus subgranosus</i> attack (entry holes) on lower stem
3. Healthy, but with current <i>P. subgranosus</i> attack (frass accumulation on lower stem)
4. Dying (orange/brown foliage retained on the tree) with current <i>P. subgranosus</i> attack
5. Dead <3 y (fine twigs remaining on the tree); <i>P. subgranosus</i> attack (old or current) present
6. Dead >3 y (main branches only present); <i>P. subgranosus</i> entry holes present on lower stem
7. Dead, but with no evidence of <i>P. subgranosus</i> attack
8. Tree alive but with major crown dieback, cause unknown

*From Elliott *et al.* (1987)

as they were considered to have resisted infection by myrtle wilt disease.

The cumulative incidence of myrtle wilt at each site at each assessment was obtained by dividing the number of diseased trees (health status classes 3–6) by the total number of standing myrtle trees (health status classes 1–8), expressed as a percentage.

Trees in the circular plots (i.e. at all sites except Arve Loop) were assessed for recent damage at the first measurement using four damage categories (Table 3).

Analyses

The effects of rainforest type on the initial (1988–1989) incidence of myrtle wilt and of recent damage at the sites where circular plots were established were tested by analysis of variance (ANOVA), adjusted for altitude by using it as a covariate. The data were first transformed using the arcsin of the square root of the percentage of trees diseased or damaged. The Arve Loop plot was omitted in the test on recent damage as this variable had not been assessed throughout the plot at establishment (1982). For the analyses, the Lake Chisholm plot, which was a callidendrous/thamnic rainforest type was considered to be thamnic.

Table 3. Tree damage classes (DC)*

1. No damage
2. Broken limbs in the crown
3. Stem damage
4. Broken limbs in crown and stem damage

*From Elliott *et al.* (1987)

Results

At plot establishment, the cumulative incidence of myrtle wilt ranged from 15.8% at Frodshams Pass to 45.4% at Five Road. After 12–18 y the cumulative incidence across all sites ranged from 16.8% to 50.9% with Frodshams Pass and Five Road still at the low and high points of the range respectively. This represents a slow rate of increase in myrtle wilt incidence over the monitoring period, ranging from 0.08% per annum at Frodshams Pass to 0.67% at Lake Chisholm (Table 4). At some sites there was a slight initial increase in percentage of diseased trees but the rate of increase slowed considerably to become static at many sites, particularly between the penultimate and final measurements (Fig. 2).

The number of trees assessed at each site remained essentially constant through the monitoring period (Table 4). The minor fluctuations in standing tree numbers resulted from addition of trees growing into the diameter range between assessments, and losses from dead trees falling to the forest floor.

There was a significant effect of rainforest type on myrtle wilt incidence at the first measurement (Table 5). The callidendrous sites (Five Road, Simons Road) and the callidendrous/thamnic site (Lake Chisholm) had higher disease incidence than the thamnic and implicate sites (Arve Loop, Frodshams Pass and Pipeline 18 Mile Peg). The effect of altitude on disease incidence was also significant, the occurrence being less at higher altitudes.

The incidence of recent damage in the rate-of-spread plots at the first measurement ranged from 6.8% at Pipeline 18 Mile Peg to 23.9% at Five Road. Callidendrous sites had a higher incidence of recently damaged trees than thamnic and implicate sites (Table 6). The significant effect of rainforest type on the incidence of recent damage is shown in Table 7.

Table 4. Incidence of myrtle wilt in standing trees

Site	No. trees assessed*	Diseased myrtle at plot establishment (%)	Diseased myrtle at final assessment (%)	Increase (% y ⁻¹)
Arve Loop	327 (327)	25.1	32.9	0.43
Lake Chisholm	347 (339)	36.9	44.9	0.67
Five Road	392 (379)	45.4	50.9	0.46
Simons Road	318 (311)	28.0	31.8	0.32
Pipeline 18 Mile Peg	243 (244)	19.8	22.7	0.24
Frodshams Pass	190 (188)	15.8	16.8	0.08

*Number of trees at first assessment; number of trees at final assessment in parentheses

Table 5. ANOVA of percentage of diseased trees by rainforest type at first measurement, adjusted for altitude (data transformed to arcsin square root of percentage diseased)

Source of variation	DF	F ratio	Significance level
Covariate: altitude	1	29.290	0.0325
Main effect: rainforest type	2	96.076	0.0103
Residual	2		

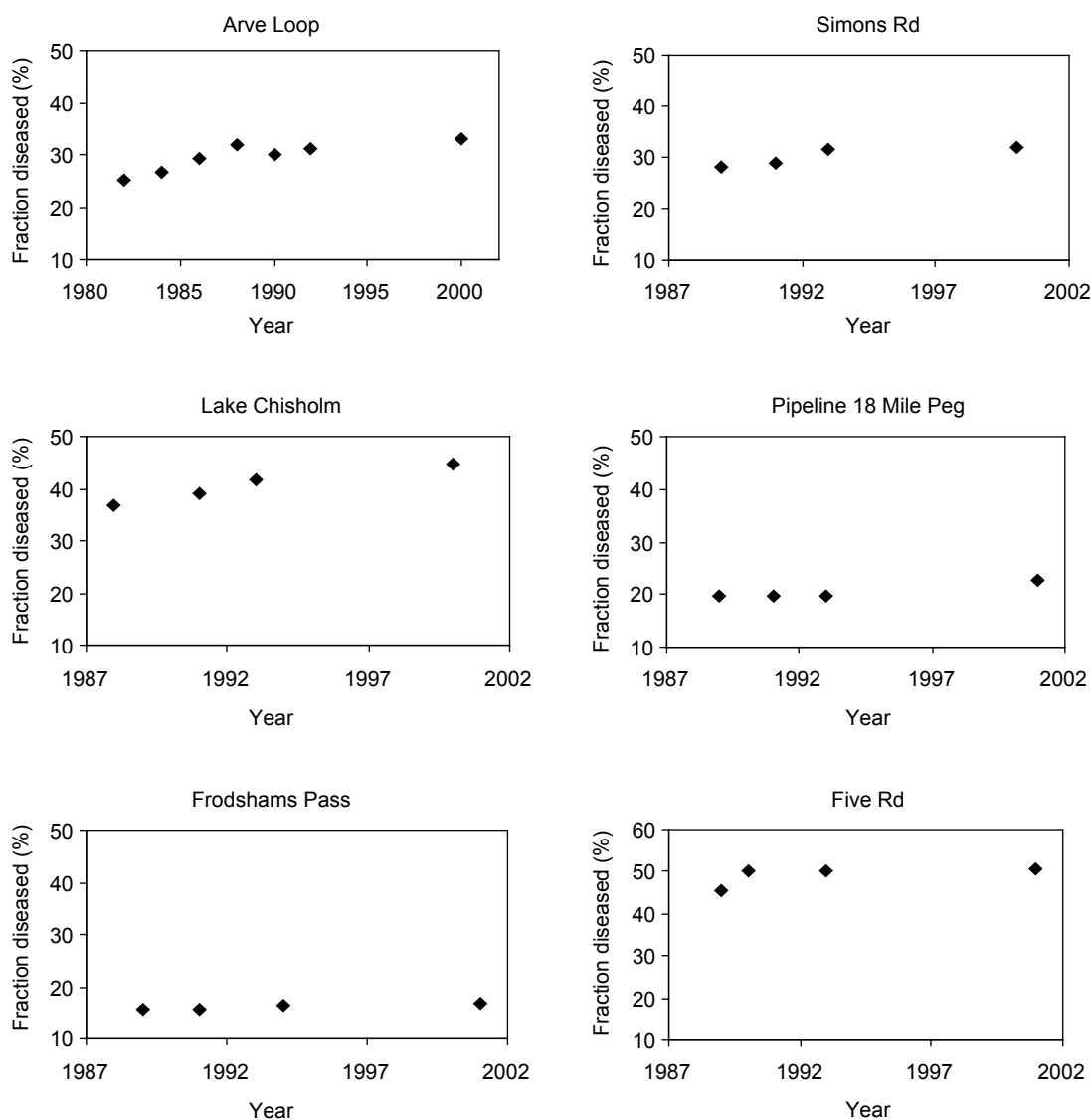


Figure 2. Cumulative incidence of myrtle wilt

Discussion

Packham (1994) concluded that myrtle wilt was probably an endemic disease although levels may have increased in the recent past. Read and Hill (1985) found that Tasmanian rainforest was normally self-replacing, with no major changes in species composition or dominance occurring, and with myrtle maintaining its dominance by seedling regeneration in canopy gaps caused by the death of old myrtles. Myrtle wilt is the prime cause of death

and subsequent gaps in myrtle-dominated rainforest, and is thus an effective agent for the gap regeneration process.

Extensive studies of myrtle wilt in the 1980s and early 1990s established that the average annual mortality from myrtle wilt at the time was 0.61% per annum and that this level of mortality was unlikely to lead to any permanent changes in forest structure (Packham 1994). Although myrtle wilt disease is a continuing phenomenon in Tasmanian rainforests, the low rate of increase

Table 6. Incidence of recent damage in circular rate of spread plots at first measurement (DC = damage class)

Site	Rainforest type	No. of myrtles in damage class				Fraction damaged (DC 2–4/1–4) (%)
		1	2	3	4	
Simons Road	Callidendrous	209	25	12	3	16.1
Five Road	Callidendrous (+ eucalypts)	204	56	6	2	23.9
Lake Chisholm	Callidendrous/thamnic (+ eucalypts)	208	11	10	4	10.7
Pipeline 18 Mile Peg	Thamnic	192	6	8	0	6.8
Frodshams Pass	Implicate/thamnic	152	11	2	2	9.0

Table 7. ANOVA of percentage of damaged trees by rainforest type adjusted for altitude

Source of variation	DF	F ratio	Significance level
Covariate: altitude	1	13.635	0.1661
Main effect: rainforest type	2	260.059	0.0432
Residual	1		

recorded in the present study over 12–18 y of monitoring indicates a currently stable situation. This result supports the prediction of Packham (1994).

Analysis of a sequence of colour aerial photos of an area of undisturbed rainforest taken in 1978–1979, 1984–1985 and 1996–1997 identified 109, 97 and 102 dead or dying tree crowns respectively (Mesibov 2002). Exact rates of spread cannot be calculated from this photo sequence because myrtle wilt may not have been the only cause of death and the tree crowns identified on the photos may not always represent individual trees. However, the study clearly showed that myrtle wilt in this area of Tasmanian rainforest was widely spread in space and time (Mesibov 2002).

The incidence of myrtle wilt disease was generally higher at plot establishment in the sites supporting a callidendrous forest type (Jarman *et al.* 1984). A similar relationship of disease incidence to rainforest type was found in the extensive surveys of undisturbed rainforest by Elliott *et al.* (1987). In callidendrous rainforest in Tasmania, the dominance of myrtle is greater than in thamnic and implicate rainforest and it may be that the favourable growing conditions for myrtle found in callidendrous forest also favour development of the species-specific myrtle wilt disease. Re-analysis of the data set of Elliott *et al.* (1987) by Packham (1994) indicated that the absolute and relative myrtle densities were greater in callidendrous than thamnic or implicate rainforest, and higher in pure rainforest than in mixed forest. The higher density of myrtle stems in the more fertile callidendrous forests may promote more root contacts, thus facilitating disease spread through root grafting. Analysis of the distribution of diseased trees by Packham (1994) showed a distinct clumping effect, supporting a theory that clumping is at least partially caused by the transfer of the disease via root grafts between neighbouring trees.

The incidence of recent damage followed the same trend as disease incidence in relation to rainforest type, although whether

damage is the cause or effect of disease in this case is not clear. Tree wounds provide infection sites for *C. australis* (Kile and Walker 1987) but it is difficult to separate cause and effect because falling stems and branches from dead and dying trees may damage adjacent healthy trees, thereby increasing their susceptibility to infection. However, in mixed forests where overtopping eucalypts frequently shed branches onto the rainforest understorey, groups of damaged myrtles are often found dying from myrtle wilt.

The myrtle wilt rate-of-spread plots are important long-term sites which should continue to be used to monitor the health of myrtle rainforest in Tasmania. It is intended that future assessments will be conducted at intervals of 10 y unless sudden increases in general disease levels warrant shorter intervals.

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