MYRTLE WILT AND ITS POSSIBLE MANAGEMENT IN ASSOCIATION WITH HUMAN DISTURBANCE OF RAINFOREST IN TASMANIA

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ABSTRACT

Chalara australis Walker & Kile is a lethal primary pathogen of *Nothofagus cunninghamii* (Hook.) Oerst. (myrtle) in Tasmanian rainforests. It is a major cause of gaps in myrtle-dominated stands but mortality is often most severe in rainforest subject to human disturbance. Possible strategies could be developed to minimise disease in areas of disturbed rainforest.

Keywords: rainforest; disturbance; *Nothofagus cunninghamii; Chalara australis.*

INTRODUCTION

Nothofagus cunninghamii (myrtle), the most common of the dominant tree species of cool temperate rainforest in south-eastern Australia, is suffering widespread and locally severe mortality (Elliott *et al.* 1987; Kile & Walker 1987). Tree death results from infection and necrosis of root and outer stem wood by the hyphomycete *Chalara australis.* The pin-hole borer *Platypus subgranosus* Schedl attacks the stems of dying trees.

The wilt symptoms which develop in the crowns of diseased trees have led to the adoption of the term "myrtle wilt" to describe the disease and distinguish it from less specific syndromes of morbidity affecting other *Nothofagus* species (Arentz 1983; Skipworth 1983). Myrtle wilt, however, is considered to be a vascular stain disease as the pathogen is not confined to the conducting elements of the xylem in the initial phases of infection and it does not become systemic in the host plant, features associated with vascular wilt pathogens (Green 1981).

Myrtle wilt is a major cause of gaps in Tasmanian rainforests. Initial research on disease impact, etiology, and the relationship of the pathogen to the potential vector *P.*

subgranosus is now being expanded to assess the significance of the disease in the ecology and conservation of *N. cunninghamii*. This paper reviews current information on myrtle wilt and the possibilities for limiting its impact in cool temperate rainforest disturbed by human activity.

DISEASE SYMPTOMS IN INDIVIDUAL TREES

Crown symptoms include chlorosis, necrosis, and abscission of the oldest foliage prior to the sudden general wilting and necrosis of residual foliage (Kile & Walker 1987). Initial symptom expression may vary between trees; individual branches of the upper crown sometimes brown off leaving green branches surrounding or below, while in other trees extensive foliage necrosis and abscission can occur leaving a few distal tufts of green foliage prior to tree death. Hosts may take from 1 to 3 years to die from the time of initial *C. australis* infection.

Root and stem wood infected by *C. australis* is discoloured dark brown. In cross-section this is visible as radial streaks of variable width and length or patches in the sapwood or outer heartwood which may be interspersed with clear wood. Discoloration attenuates upwards in the stem and rarely reaches crown break. Irregular, black, sporulating mycelial felts of *C. australis* may develop on the bark of the lower stem of infected trees and these are a probable source of air- or water-borne inoculum.

Platypus subgranosus attack is initiated on trees with healthy crowns and is a reliable guide to *C. australis* infection in the underlying wood. Infestation is initially often concentrated in one sector of the stem before becoming more widespread on the lower stem and buttress roots in subsequent flight seasons. Tunnelling by *P. subgranosus* could accelerate disease expression by assisting the internal spread of the pathogen within host vascular tissues.

INCIDENCE AND SPATIAL PATTERN OF DISEASE IN UNDISTURBED RAINFOREST

Transect surveys of 20 rainforest sites undisturbed by human activity or recent fire showed cumulative mortality from 9.4% to 53.4% with an average disease incidence of 24.6% (Elliott *et al.* 1987). Incidence decreased with increasing altitude and, when adjusted for this parameter, the callidendrous rainforest subtype (*see* rainforest classifications by Jarman *et al.* 1984) had a higher incidence than the floristically more diverse thamnic-implicate subtypes, respectively. In mixed forest (rainforest with eucalypt overstorey) incidence increased as both relative and absolute measures of myrtle density increased but this trend was not evident in the other forest types. Larger-diameter trees had the highest disease incidence as did trees with stem or crown damage. However, significant variation remained unexplained by the site and stand variables which were measured.

Diseased trees were clumped with the degree of association reasonably consistent across sites but dependent on nearest neighbour distances within sites. Dying trees occurred at an average rate of 2.4 trees/ha or 1.6% of live trees across the sites.

INCIDENCE IN DISTURBED RAINFOREST

Although high levels of the disease occur in undisturbed stands, roading and logging activities have been observed to lead to raised incidence levels.

Results from a series of logging, regeneration, and thinning trials, confirm these observations (J. Hickey pers. comm.). All logging methods which were tested on old rainforest increased disease incidence and mortality levels of the remaining myrtles, compared with undisturbed forest, i.e., selective logging, strip logging, logging leaving shelterwood and seed trees, and also pre-logging soil scarification to improve regeneration. Thinning even-aged myrtle stands at 40 and 65 years significantly ($p < 0.05$) increased mortality.

In some of these logged and thinned areas there was evidence that elevated mortality levels due to myrtle wilt eventually dropped to levels more "normal" in undisturbed forest. Where this happened it took an average of 9 years (range 4–13.5 years).

Transect data from a severely affected roadside area in north-east Tasmania showed that the proportion of diseased trees decreased with distance from the road, the fitted model explaining 53% of the deviance (Fig. 1).

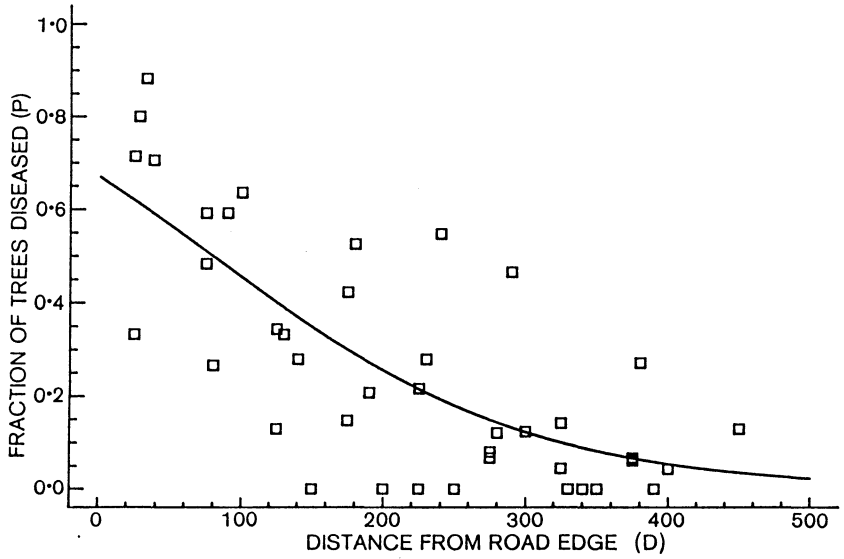

FIG. 1 — Scatter plot of data from multiple transects showing proportion (P) of diseased (dead or symptomatic) trees of *Nothofagus cunninghamii* in relation to metres from a road edge (D) in north-eastern Tasmania. Log it (P) = 0.713 − 0.008896D, percentage deviance explained = 53%.

THE PATHOGEN

Chalara australis grows readily and sporulates profusely in culture on a variety of media. The optimum temperature for growth is approximately 20 °C. The single-celled conidia are produced in chains from the distinctive phialides. Sclerotia (suspected perithecial initials) develop on agar but no teleomorph has been identified for the species (Kile & Walker 1987).

While the fungus shows similarities to other *Chalara* species such as *C. quercina* Henry the anamorph of *Ceratocystis fagacearum* (Bretz) Hunt (cause of oak wilt) and the *Chalara* anamorphs of *Ceratocystis virescens* (Davidson) C. Moreau (cause of sap-streak of sugar maple), it most closely resembles *Chalara neocaledoniae* Kiffer & Delon, a species pathogenic on coffee (*Coffea robusta* Linden) and guava (*Psidium guajava* L.) in New Caledonia (Dadant 1950; Kiffer & Delon 1983). *Chalara neocaledoniae* has smaller phialides and a higher optimum temperature than *C. australis*.

Koch's postulates were established for *C. australis* on seedlings, saplings, and large trees (Kile & Walker 1987). Wood and foliage symptoms were reproduced in hosts of various size and age even though natural infection is not observed in seedlings. These inoculation studies showed evidence of variation in host resistance and in pathogenicity, both of which require further study.

HOST RANGE

Inoculation studies indicate that *C. australis* is relatively host specific. Of 18 Tasmanian native and four exotic species inoculated with *C. australis* (Kile 1989), only the rainforest understorey species *Trochocarpa gunnii* (Hook. f.) Benth. (Epacridaceae) was killed while *Nothofagus gunnii* (Hook. f.) Oerst. was extensively infected and may have died with a longer inoculation period. Other species showed only minor vascular discoloration or were essentially resistant. Natural infection has not been observed in either *T. gunnii* or *N. gunnii*. The marked susceptibility of *T. gunnii* is puzzling as there is no close relationship between the Fagaceae and Epacridaceae.

Inoculation of a limited number of seedlings of exotic *Nothofagus* species showed *N. glauca* (Phil.) Krasser, *N. leoni* Espinosa, and, to a lesser extent, *N. obliqua* (Mirbel) Blume, *N. alpina* Poepp. & Endle., and *N. alessandrii* Espinosa to be susceptible but no deaths occurred (Kile 1989). These species and the Tasmanian endemic *N. gunnii* are all deciduous species grouped in the section *Nothofagus* of the genus. The results indicate some susceptibility to *C. australis* in other members of the genus, particularly the deciduous species, but a more satisfactory assessment must await inoculation studies in mature trees.

DISEASE DISPERSAL

Superficially there is a close association between attack of the stems of myrtle trees by *P. subgranosus* and the development in their crowns of the characteristic wilt symptoms. Howard (1973) hypothesised that the death of myrtle was caused by a pathogenic fungus transmitted by this borer. Kile & Walker (1987) established the veracity of this first element of Howard's hypothesis.

The clumping of diseased trees shown by the survey of Elliott *et al.* (1987), together with the initial observations of Kile & Walker (1987) on the apparent relationship between *P. subgranosus* attack and *C. australis* infection, suggested three hypotheses which alone, or in combination, could explain dispersal and infection by *C. australis*: (a) direct vectoring by *P. subgranosus* (or unknown insect/s), (b) air- and/or water-borne infection of wounds with below-ground spread by root graft or root contact (or

unknown below-ground vectors), or (c) random attacks by *P. subgranosus* on healthy trees or on trees rendered susceptible by stress, followed by infection of the wood via the borer tunnels with air-, water-, or insect-borne inoculum of *C. australis.*

Kile & Hall (1988) provided a detailed assessment of the possible role of *P. subgranosus* in pathogen dispersal from which they concluded that the beetle rarely, if ever, was a direct vector but rather a secondary agent attacking trees already infected by the fungus. This conclusion was based on (a) the very infrequent isolation of *C. australis* from the beetles, (b) lack of infection in disease-free myrtle billets exposed to beetle attack, (c) frequent beetle attack of artificially stressed or wounded trees without infection, (d) evidence of infection of trees in the forest prior to beetle attack, (e) lack of convergence in the life-cycle of the fungus and the beetle in the infected host which would allow emergent beetles to become contaminated with the fungus.

Examples of wound infection, apparently without insect involvement, have been documented (Kile & Walker 1987; Kile & Hall 1988) and this is probably the main means for the establishment of new infection foci. Two potential sources of air-, water-, or insect-borne inoculum occur in rainforest—(a) conidia from mycelial felts on the bark of infected trees or other wood surfaces (Kile 1989), (b) wind-borne frass contaminated with conidia and phialides from beetle tunnelling in infected tissue of living trees.

Inoculum-trapping experiments using fresh billets of stem wood (5–6 cm diam. × 30 cm long) of myrtle and rainwater collection have confirmed the presence within rainforest of air- or water-borne inoculum but quantifying its seasonal availability has proved difficult (Kile unpubl. data). Trapping on wood illustrates a strong summer peak in inoculum levels but it has not been possible to determine whether this indicates greater availability of inoculum propagules or secondary infection between billets during the 1-month exposure periods within the forest (Fig. 2).

Sporulating felts are produced most abundantly during the autumn-winter period (Kile unpubl. data), and a peak in summer could indicate the presence of frass-borne inoculum during the beetle flight season. It is evident, though, that there is the potential for inoculum to be available during most of the year. There is no evidence that insects are involved in transfer of inoculum to wounds.

Clumping of diseased trees and a relationship between incidence and stand density has been considered indicative of below-ground spread (Elliott *et al.* 1987). Although no investigations of the frequency or mechanism of underground transmission have yet been undertaken, it is likely to be a major means of local dispersal.

The third hypothesis is more difficult to establish. Treatments to induce stress in myrtle indicate that *C. australis* renders trees more susceptible to continued *P. subgranosus* attack than sap-ringing or scorching (Table 1). Artificial wounds which expose sapwood result in localised and transient attractiveness of trees to *P. subgranosus* (Kile unpubl. data). These results explain why *C. australis*-infected trees are favoured for beetle attack in the forest.

Thus, while *P. subgranosus* is not a direct vector of *C. australis* it has the potential to be of significance in disease spread through liberation of contaminated frass, creation of wounds (pin holes) in stressed trees, and promotion of spread within trees already infected.

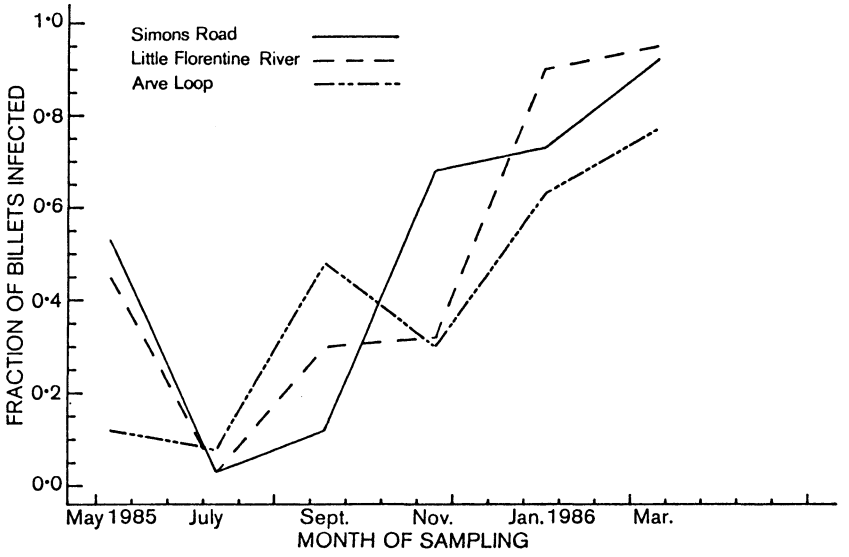

FIG. 2 — Percentage of billets of *Nothofagus cunninghamii* stem wood infected by *Chalara australis* during 1-month exposure periods at three rainforest sites in Tasmania during 1985–86. Presence of infection in individual billets was determined by isolation of the fungus from the wood on 3% malt extract agar.

TABLE 1—Number of male and female *Platypus subgranosus* trapped on sticky traps compared with the number of tunnels initiated on the same trees in five treatments on *Nothofagus cunninghamii* during the 1985–86 beetle flight season (December 1985 – May 1986)*

Treatment of trees	*P. subgranosus*		Total trapped	Total tunnels
	Male	Female		
Chalara australis inoculated	1684	999	2683	3891
Sap ringed	361	333	694	709
Scorched	158	126	284	301
Sterile water injected	53	28	81	61
Control	16	0	16	0
Totals	1972	1486	3757	4962

* Figures are totals for the seven trees in each treatment

OTHER DISEASES AFFECTING STAND STRUCTURE OF *NOTHOFAGUS* COMMUNITIES

Myrtle wilt appears to be unique as an often severe and sustained stand-level disease caused by a primary pathogen in *Nothofagus* forests. Other diseases in *Nothofagus* spp. affecting stand structure have been recorded from New Zealand and Papua New Guinea but mortality has usually been related to environmental stress, particularly drought, followed by infections of secondary pathogens or infestations of pests

(Cartledge *et al.* 1975; Arentz 1983; Skipworth 1983; Hosking & Kershaw 1985; Jane & Green 1986; Hosking & Hutcheson 1986).

Faulds (1977) showed that a fungus, tentatively identified as a *Sporothrix* species, was pathogenic when inoculated into *N. fusca* (Hook.f.) Oerst. This fungus caused somewhat similar effects in the wood and the crowns of this species to those caused by *C. australis* in *N. cunninghamii*. Although infection was associated with attack by *Platypus* spp., it was not established whether the beetles were direct vectors. Tree killing by the fungus, however, appears quite restricted but may be aggravated by stress or forest disturbance (Faulds 1977).

The role, if any, of *C. neocaledoniae* as a pathogen in forests of *Nothofagus* spp. in New Caledonia is unknown. As it attacks exotic species in the Myrtaceae and Rubiaceae (Dadant 1950) and is probably indigenous to the island, infection of native hosts is possible. No *Chalara* species have been recorded on *Nothofagus* spp. in Papua New Guinea (Shaw 1984).

MANAGEMENT OF MYRTLE WILT IN ASSOCIATION WITH DISTURBANCE

A moratorium on rainforest logging in Tasmania is in force until 1990, although mixed forests with more than 5% eucalypt crown cover are logged for regeneration to eucalypt species. It seems likely that significant rainforest areas will eventually be reserved, and that any silviculture of *N. cunninghamii* will be restricted to areas of limited extent.

Myrtle wilt may be a natural cause of gaps which allow stand regeneration in *N. cunninghamii* rainforest. If the host-pathogen relationship is a stable one, then there is little cause for concern about the level of disease expression in large areas of otherwise-undisturbed rainforest. There are, however, other hypotheses which could explain the current levels of disease such as the development of new, more aggressive strains of the pathogen or promotion of disease development through greater human access to and disturbance of rainforest. These are being investigated as part of an assessment of the need for disease management. For the foreseeable future, though, any attempts at management or amelioration of disease will focus on isolated rainforest remnants, and reserves such as national parks and conservation areas disturbed by man for access, or coupes managed for wood production. In such areas aggravated disease may reduce aesthetic values and timber yield and create hazardous trees. As some natural infections will always occur, management will in the main be directed to minimising disease levels.

Prevention of wounds on *N. cunninghamii* in any operations in rainforest is an essential element in any strategy to minimise disease development, whether this be in silvicultural treatments or in creation of access. Assessment of the seasonal availability of inoculum and the period for which wounds are susceptible to infection is necessary to determine if there are periods when rainforest could be disturbed with minimal risk of infection.

The significance of myrtle wilt in *N. cunninghamii* silviculture will depend on the future scale of such activities. In logging trials in rainforest to obtain *N. cunninghamii* regeneration via retained seed trees, the seed trees often die soon after stand treatment

without the opportunity for seed shed (J. Hickey pers. comm.). The majority of trees probably die from *C. australis* infection; during harvesting operations, careful selection of unwounded trees or trees separated from existing infection foci could promote seed-tree longevity.

Thinning may be an option for the intensive management of densely stocked *N. cunninghamii* stands. Retained trees may be wounded during thinning operations, and infection of stumps was observed in a recently thinned pole stand (Kile unpubl. data). The implications of the latter source of infection for survival of crop trees are unknown but management to limit entry of *C. australis* to thinned stands appears necessary. Options could include the timing of operations and the treatment of wounds and stump surfaces, but chemical and biological treatment of wounds to prevent *C. australis* infection has not been investigated.

An important approach to disease management may be delineation of buffer zones to protect core *N. cunninghamii* stands. Disturbance for access increases mortality, probably initially by creating new disease foci through infection of wounds on border trees and later on a continuing basis by below-ground spread between adjacent trees. Severe mortality also has the potential to raise inoculum levels and thereby increase the chances of establishing new infections in adjacent healthy stands. Observations at several locations in Tasmania suggest continuing, albeit slow spread into contiguous "healthy" myrtle more than 15 years after roading (Fig. 1).

Buffer zone size will depend on the rate of disease spread. Plots to measure this rate have only recently been established in Tasmanian rainforest. Within buffers, however, loss of existing trees could be compensated for by regeneration and an initial approach to defining a buffer could be to consider it as a zone in which all existing mature myrtle are not killed within, for example, a period of 50 years of its establishment. By this time myrtle regeneration could form an identifiable forest. Using an estimate for annual below-ground spread of 1–5 m, based on the linear growth rate of the fungus in *N. cunninghamii* stem tissue of up to 1 cm/day (Kile 1989), such a buffer may need to be 50–250 m wide.

Disease development within the buffer zone and the width of the zone might be considerably reduced if the boundary could be carefully formed to minimise infection of the border trees of such a zone. The integrity of a buffer zone may also be affected by air-borne spread which may or may not be related to disturbance in adjacent areas, but there is no basis on which to predict such infections. Sanitation treatment in buffer zones by removal of dead or infected trees could also be considered in especially valuable rainforest areas if the operations could be done without damage to adjacent trees.

As *P. subgranosus* is a secondary agent it seems unlikely that attempts to manage beetle populations would have a significant effect on disease development, except possibly if frass contaminated with *C. australis* is important as inoculum.

CONCLUSIONS

Myrtle wilt does not threaten the survival of *N. cunninghamii* but it is an important disease in Tasmanian rainforests. *Chalara australis* is a potential hazard for *Nothofagus* species growing elsewhere in the world. Infection develops through root

or stem wounds via air- or water-borne inoculum and through local below-ground spread. *Platypus subgranosus* is a secondary agent attacking trees already infected by the fungus but it may liberate inoculum from infected trees. The disease may radically alter stand structure and in public access areas create trees which are hazardous for people and property. There is the potential to develop strategies to minimise disease development in remnant and disturbed rainforest areas.

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