INTERDISCIPLINARY APPROACH TO THE STUDY AND MANAGEMENT OF STEM DEFECT IN EUCALYPTS*

T. WARDLAW,

Forestry Tasmania, GPO Box 207, Hobart, Tasmania, 7001, Australia

C. MOHAMMED†,

CSIRO Forestry and Forest Products,
GPO Box 252-12, Hobart, Tasmania, 7001, Australia

K. BARRY†, A. EYLES†, D. WISEMAN†,

School of Agricultural Science, University of Tasmania,
GPO Box 252-54, Hobart, Tasmania, 7001, Australia

C. BEADLE†, M. BATTAGLIA†,

CSIRO Forestry and Forest Products,
GPO Box 252-12, Hobart, Tasmania, 7001, Australia

E. PINKARD†,

School of Agricultural Science, University of Tasmania,
GPO Box 252-54, Hobart, Tasmania, 7001, Australia

and P. KUBE

Carson Associates Ltd., 34 Parawai Rd., Rotorua 3202, New Zealand

(Received for publication 27 February 2004)

ABSTRACT

In Australia large areas of forest have been closed to industrial forestry and it is necessary to compensate for lost production. Future wood products will come from intensively managed silvicultural regimes — eucalypt regrowth forest and plantations. Solid-wood regimes involving high-cost operations such as pruning and thinning will be economically sensitive to downgrade due to various types of stem defect — fungal and insect damage, staining, kino veins. In Tasmania, research over the last decade has focused on developing management strategies to minimise stem defect caused by decay fungi in both regrowth and plantation eucalypts. Under plantation conditions

---

* Paper presented at the 8th International Congress of Plant Pathology, 2–7 February 2003, Christchurch, New Zealand.

† Also: Co-operative Research Centre for Sustainable Production Forestry

neither *Eucalyptus nitens* (Deane & Maiden) Maiden nor *E. globulus* Labill. shed branches efficiently. Retention of dead branches leads to defects in wood such as a large knotty core or loose knots, making the stem unsuitable for either veneer or sawlog. Pruning of green branches at canopy closure and in subsequent lifts shortly afterwards solves this problem. However, on the more productive warm and wet sites there is a high level of decay infections in *E. nitens* and *E. globulus* via pruning wounds. We are investigating the complex pathological, physiological, genetic, and silvicultural components dictating eucalypt susceptibility to decay infection and its long-term spread into clearwood including: crown characteristics prior to pruning; growth responses to pruning; growth responses to fertiliser; host resistance and antimicrobial defences; and the identity and pathogenicity of decay fungi. An interdisciplinary approach is fundamental to understanding such questions and also to the successful development of site productivity models that include predictions of risk and impact of biotic and abiotic damage.

Keywords: stem decay; wound rot; heart rot; wood-decay fungi; *Eucalyptus nitens*; *Eucalyptus globulus*.

INTRODUCTION

The last two decades have seen a shift in sentiment by society, demanding that larger areas of forest be preserved to protect their natural values. During this period, for example, there have been three major inquiries into forest use in Tasmania (Department of the Arts, Sport, the Environment, Tourism and Territories 1988; Forest and Forest Industries Council 1990; Commonwealth of Australia and State of Tasmania 1997). The net result of these inquiries was that the area of forest in reserves increased dramatically from 13% in 1982 to 40% in 2000. At the same time, however, there is a legislated requirement to maintain a supply of sawlogs from this reduced area of crown forest (Parliament of Tasmania 1990). Intensification of forest management through thinning of native forests and establishment of eucalypt plantations has been adopted as a strategy to increase the yield of sawlogs from this reduced area of crown forest (Forest and Forest Industries Council of Tasmania 1990).

Markets have also been changing. Traditionally eucalypts have been sawn for uses in primarily structural applications such as house framing. However, over the last two to three decades softwoods, particularly *Pinus radiata* D.Don, have displaced eucalypts in structural applications (Ferguson & Dargavel 1978). Increasingly, the future of eucalypt timber is predicted to be in appearance applications such as furniture, panelling, flooring, and internal fittings (Beall 2000; Ye 2000). Sawmill studies (Wardlaw unpubl. data) have shown that the recovery of high-quality boards that meet specifications for appearance uses is more sensitive to log quality than if logs were being sawn for structural products (Fig. 1). Tighter log specifications targeting appearance products would decrease the volume of in-specification logs available for sawing (Fig. 2).

The need for greater productivity, combined with the targeting of products that are more sensitive to log quality, has sharpened our focus on log defects. Stem decay caused by wood-rotting fungi can be a significant defect in eucalypts sawn for timber production (Yang & Waugh 1996). Up until the mid 1980s research concentrated on reporting defects in unmanaged eucalypt forests (e.g., Greaves *et al*. 1965; Elliott & Bashford 1984; Wilkes 1985). Since then, however, research has shifted towards studying the development of defect, particularly fungal stem decay, in eucalypts that have been cultivated under more

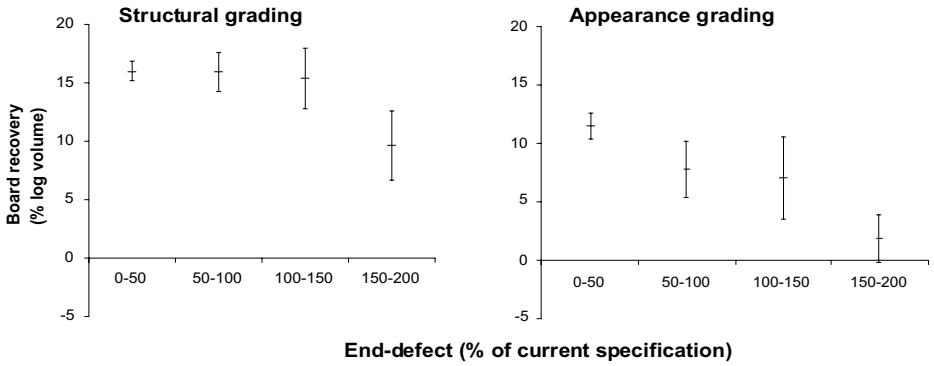

FIG. 1–Recovery (green) of sawn boards that meet either (a) Structural Grade 1 or (b) Select Grade (appearance grading) from regrowth eucalypt sawlogs grouped according to the amount of log end-defect (from Wardlaw unpubl. data). Error bars indicate 95% LSD limits.

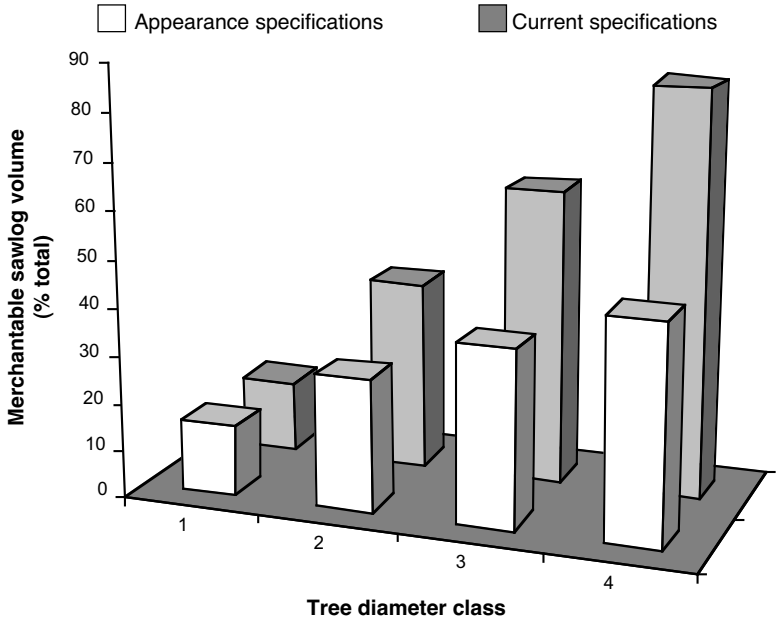

FIG. 2–Percentage of potentially merchantable sawlog volume in each of four tree diameter quartile classes that meets either current specifications for allowable log end-defect or, more stringent, appearance specifications for allowable log end-defect (from Wardlaw 2003).

intensive management regimes. White & Kile (1991) examined the development of decay originating from stem wounds inflicted during thinning operations in natural forests and made predictions of rapid spread of the decay coinciding with the breaching of barrier zones (White & Kile 1994). Wardlaw (1996) reported a high incidence of stem decay originating from natural sources in stands identified for mid-rotation commercial thinning. In eucalypt

plantations managed for solid-wood products, pruning is required to produce logs of sufficient quality for sawing or veneer production (Gerrand *et al.*1997). In the more humid sites of Tasmania, where growth rates are sufficiently high for plantations to be managed economically for solid-wood products, there is a high risk of decay entering through pruned branches (Mohammed *et al.* 1998; Wardlaw & Nielsen 1999).

These studies have formed the basis of prescriptions designed to minimise future losses from stem decay (Pinkard *et al*. 2001; Wardlaw 2003). However, research has continued, with the focus on better understanding the interactions between the host and the decay fungus. It is hoped that through a better understanding of these interactions we will be able to more confidently predict the outcomes of silvicultural intervention and incorporate mitigation measures that minimise the risk of future losses from stem decay. In this paper, we document the interdisciplinary research programme being carried out in Tasmania by the Co-operative Research Centre for Sustainable Production Forestry (CRC-SPF) and Forestry Tasmania into the management of stem decay in eucalypts.

DECAY IN INTENSIVELY MANAGED NATURAL FORESTS

The initial focus of decay research in native forests was on the development of decay originating from stem wounds inflicted during thinning operations. As part of the Young Eucalypt Program, studies by Don White and Glen Kile (White & Kile 1991) demonstrated that *E. obliqua* L'Herit and *E. regnans* F.Muell. carry a high risk of degrade from decay as a result of wounding. A key finding from their research was that the spread of decay accelerated rapidly about 14 years after wounding (White & Kile 1994). This coincided with a breaching of the barrier zone that was established after wounding. Old *et al.* (1993) extended wound-decay research into *E. sieberi* L.A.S.Johnson forests and, in particular, documented the establishment of defect arising from different types of stem wound (Dudzinski *et al.* 1992). These studies highlighted the importance of quality control in thinning programmes and particularly the imperative to minimise the incidence of stem wounds inflicted during thinning. The introduction of quality control and an emphasis on operator training have resulted in the incidence of stem wounding being consistently kept to low levels during thinning operations in Tasmania (Cunningham 1997).

Stem decay can also develop from natural origins. Tim Wardlaw quantified the extent of stem decay arising from natural sources in high-quality ash forests being targeted for intensive management in Tasmania (Wardlaw 1996; Wardlaw *et al.* 1997). He showed the importance of branch shedding as an origin of stem decay; on some sites branch shedding led to the development of extensive volumes of decay in a high proportion of the trees that would otherwise be suitable for retention after thinning (Wardlaw 1996, 2003). The economic impact of such decay was quantified and found to be significant on many sites, particularly in lowland stands of pure *E. obliqua* (Fig. 3). Therefore it was necessary to develop prescriptions enabling tree selection during thinning that reduced the proportion of trees with severe decay retained after thinning. Apart from providing much information about decay processes in young regrowth trees, this research demonstrated the gains that could be made from adhering to tighter thinning prescriptions. Wardlaw (2003) measured trees with severe decay of 27% in a sample of eight regrowth stands scheduled for commercial thinning. Tree selection using tightened prescriptions at thinning was predicted to reduce the incidence of severe decay in the final stand to 14%.

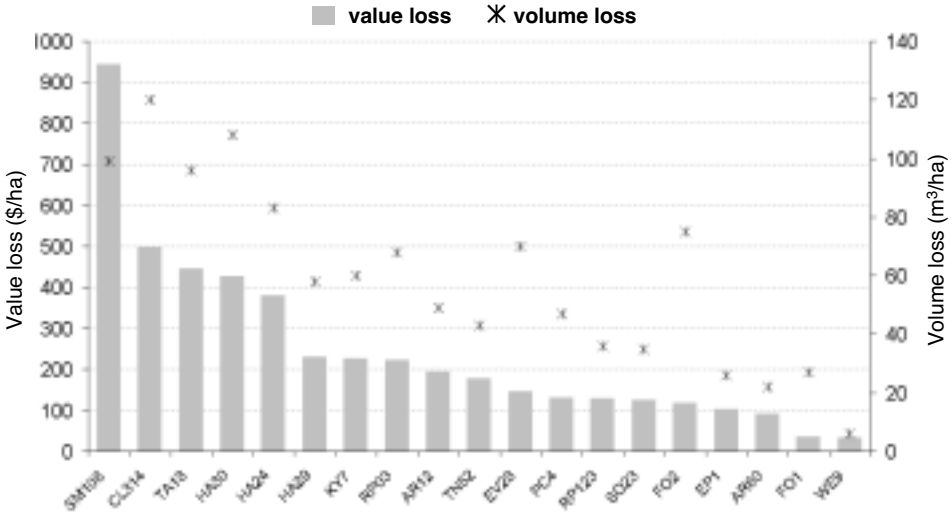

FIG. 3–Predicted losses in sawlog volume and value due to downgrading because of excessive stem decay in 19 regrowth eucalypt coupes in Tasmania. Predictions are based on growth projections to harvest age, 20–30 years after commercial thinning (from Wardlaw 2003).

DECAY IN PLANTATIONS

In the push for sustainable forestry there is a rapidly increasing estate of eucalypt plantations in Australia; most of these plantations are being grown for pulpwood but some are destined for veneer and sawlog production. Of approximately 130 000 ha of eucalypt plantations in Tasmania, 15 000 ha have been pruned for solid wood.

Eucalyptus globulus and *E. nitens,* the main commercial species in Tasmania, do not shed branches in a plantation situation and require pruning to produce wood of sufficient quality for sawn timber and veneer production (Gerrand *et al.* 1997). As the first few experimental plantations reached 6–11 years of age, surveys by Wardlaw & Nielsen (1999) found decay in a substantial proportion of pruned trees. These surveys also showed that unacceptable defects develop from pruning dead branches.

To examine more closely the influence of pruning treatments on the establishment of decay, Mohammed *et al.* (1998) established a trial in *E. nitens* plantations on five contrasting sites in 1996. Destructive sampling done 1 year after pruning showed that decay-risk was associated with pruning live branches. Branch size was confirmed to be a key risk factor: As the diameter of the branch increased above 2 cm, so did the predicted risk of decay (Fig. 4). The study also showed that poor-quality pruning techniques greatly increased the risk of decay. Significantly, highly productive sites, that are most favoured for pruning, carry the greatest risk of decay developing after pruning.

This research produced an immediate response by industry to change prescriptions so that trees selected for pruning had no or few dead branches, or large-diameter (> 3–4 cm) live branches. Since higher quality sites have more vigorous trees and therefore larger branches, pruning prescriptions can only reduce, not eliminate, the number of decay infections that become established after pruning.

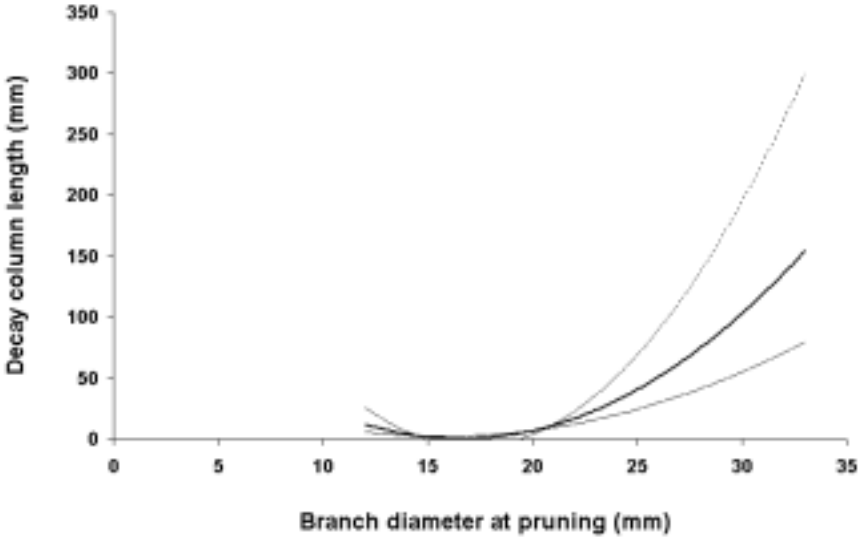

FIG. 4–Predicted length of a decay column (with 95% confidence intervals — dotted line) as a function of the diameter of the pruned branch in *E. nitens* (from Mohammed *et al.* 1998)

DEVELOPMENT OF DECAY OVER TIME

Barry *et al.* (2003) resampled the trial of Mohammed *et al.* (1998) nearly 6 years after pruning. Over that time they found that the incidence of decay in both the pruned and unpruned trees had increased by between two- and five-fold (Table 1). In the pruned trees the decay outbreaks were confined to the knotty core (they had arisen or escaped from branch traces infected at the time of pruning) and the amount of clearwood had increased over time. However, 6 years is only a short time in forestry: plantations managed for solid wood will not be harvested until age 20–35 years. Will the containment of decay in the knotty core observed in the first 6 years after pruning persist over the length of the rotation?

TABLE 1–Number of decay columns per tree (±SE) in *E. nitens* 1 and 5.5 years after pruning at two sites (from Barry *et al.* 2003).

Time since pruning (years)	Flowerdale		Evercreech	
	Pruned	Control	Pruned	Control
1	2.1 (±0.6)	0.8 (±0.3)	1.1 (±0.7)	0.4 (±0.3)
5.5	4.5 (±0.9)	4.1 (±0.8)	3.4 (±0.9)	1.4 (±0.5)

Wardlaw (2003) measured the amount of decay in mid-rotation regrowth trees by destructive harvesting. He then "grew on" the trees to harvest age and, based on the amount of decay he had measured, predicted volume loss and the value of this loss (Fig. 3). In doing so he assumed that incipient decay at mid-rotation would develop into rot by harvest age. However, he really had no information to grow on the decay infections with time. So, without knowledge of the development of decay over time are we making sound predictions? The factors driving both establishment and spread of the decay have to be understood.

HOST RESPONSE TO INFECTION AND THE SPREAD OF DECAY

Tree pathologists often refer to the CODIT model (*sensu* Shigo & Marx 1977) to explain the processes by which the spread of decay is limited within the tree. The difficulty with any model is to validate whether it applies for any particular tree species. Will the containing walls described by the CODIT model effectively restrict decay in eucalypts?

Reaction zones (comprising Walls 1, 2, and 3) are the defence response in trees associated with tissue present at the time of wounding and infection. In *E. nitens* there is sometimes a distinct purple/pink zone between the infected and healthy sapwood (Fig. 5). This zone has features characteristic of active host response: tyloses, a pH that is unfavourable for fungal growth, and antimicrobial compounds (Barry *et al.* 2000). Barry *et al.* (2002) found that increases in the production of antimicrobial compounds in *E. nitens* corresponded with increased aggressiveness of the fungus colonising the wound. This indicates a dynamic response, which appears from our observations to be effective in restricting the spread of decay, at least in the short term (Barry *et al.* 2003; Barry, Pearce, Evans, Hall & Mohammed 2001).

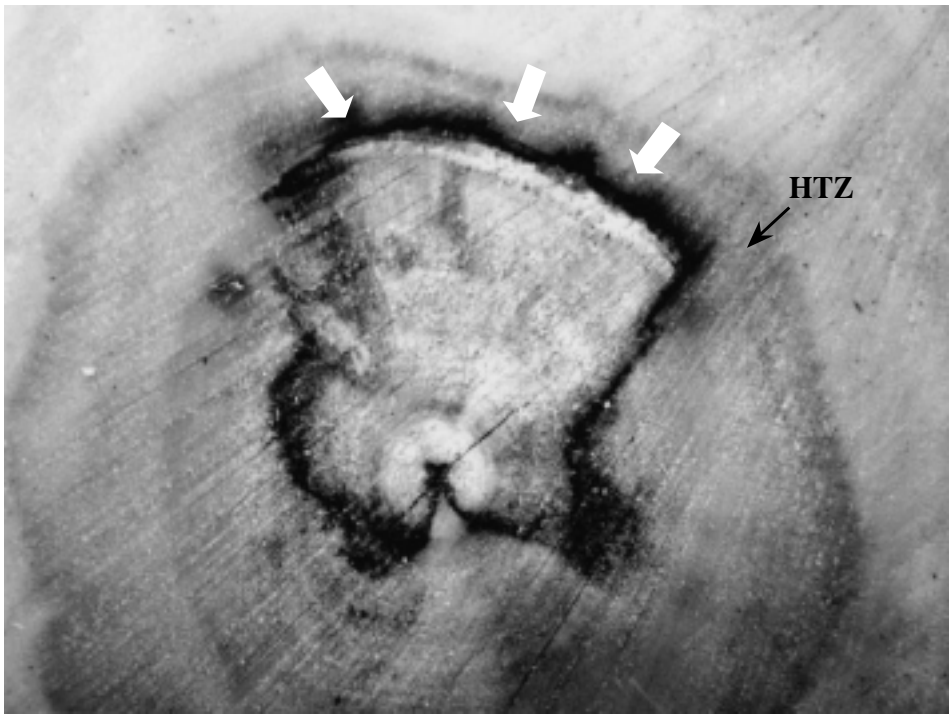

FIG. 5–Transverse surface of an *E. nitens* stem 2 years after pruning, showing a purple reaction zone (arrows) within the heartwood (HTZ — heartwood transition zone is indicated) (from Barry *et al.* 2000).

In eucalypts, kino veins are often seen as a traumatic response to damage to the vascular cambium. This kind of response in new tissue formed after the stress event is often called a barrier zone. *Eucalyptus nitens* does not actually form kino veins, which sets it apart from

quite a few other eucalypt species (Eyles & Mohammed 2002). While not detracting from the importance of kino veins or the barrier zone in hardwoods, Eyles (Eyles, Davies & Mohammed 2003; Eyles, Davies, Yuan & Mohammed 2003; Eyles *et al*. in press (a), (b)) has been investigating wound tissue and considers that its importance in defence has been overlooked. The detection of traumatic oil glands in the wound-associated phloem (Fig. 6) is a new finding for eucalypts (Eyles *et al*. in press (a)). A cocktail of chemicals have been extracted and identified from this region. These chemicals may have a multi-functional role as anti-oxidants and anti-microbials (Eyles, Davies, Yuan & Mohammed 2003; Eyles *et al*. in press (b)) and may make the wound tissue inhibitory to both fungi and pests (mammalian and insect). O'Reilly-Wapstra *et al.* (in prep.) found sideroxylons, a class of formylated phloroglucinol compounds ("fpc"s), reduced the palatability of *E. globulus* leaves to brushtail possums. The same class of compounds has been found in wound tissue formed in response to infection by decay and canker fungi (Eyles, Davies & Mohammed 2003).

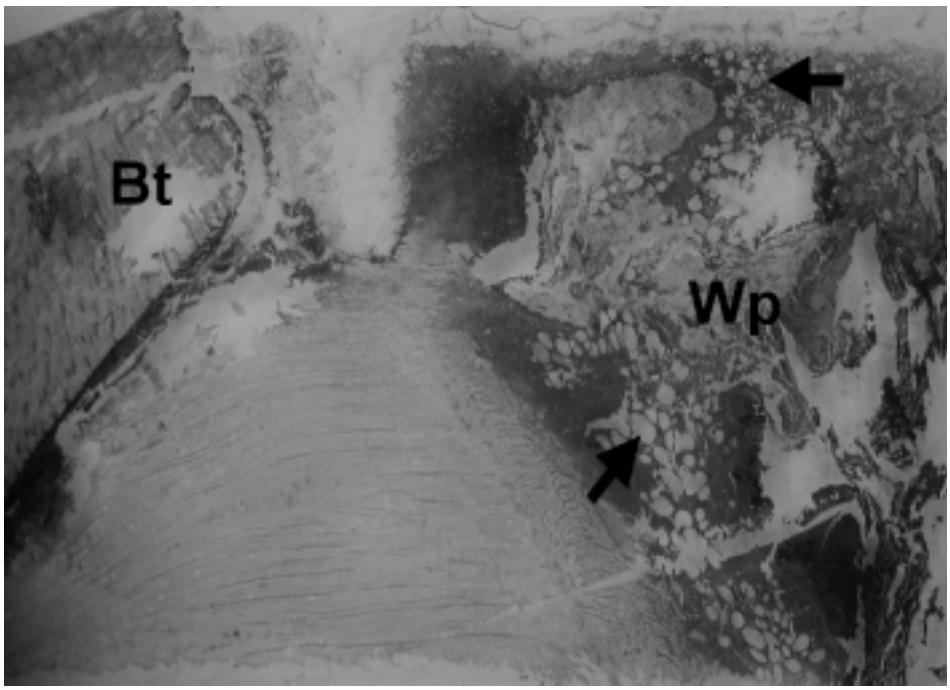

FIG. 6–Radial longitudinal section through a 2-year-old pruned branch wound (Bt) in *E. globulus,* showing wound-associated phloem (Wp) containing abundant oil glands (arrows) (from Eyles *et al.* in press (a)).

EFFECT OF SILVICULTURAL TREATMENTS

Fertiliser Treatment and Decay

Post-establishment fertiliser application is required on many plantation sites in Tasmania. Wiseman (2003) found that the application of nitrogen (900 kg N/ha) and phosphorus (150 kg P/ha) prior to pruning caused a three- to eight-fold increase in the number of decay infections in *E. nitens* (Fig. 7). This increase in decay infection was associated with an

increased longevity of branches resulting in the branches growing to a greater diameter (Fig. 8). Both of these factors are known to increase the risk of decay infections. Similar studies are currently being done in *E. globulus*.

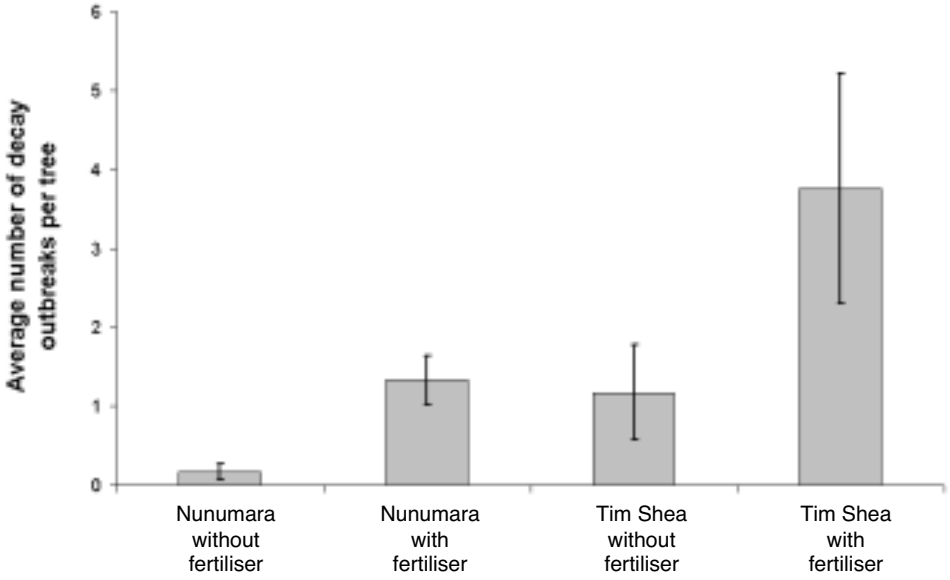

FIG. 7–Average number of decay outbreaks per tree from pruned branches of untreated and fertiliser-treated *E. nitens* at two sites in Tasmania (from Wiseman 2003).

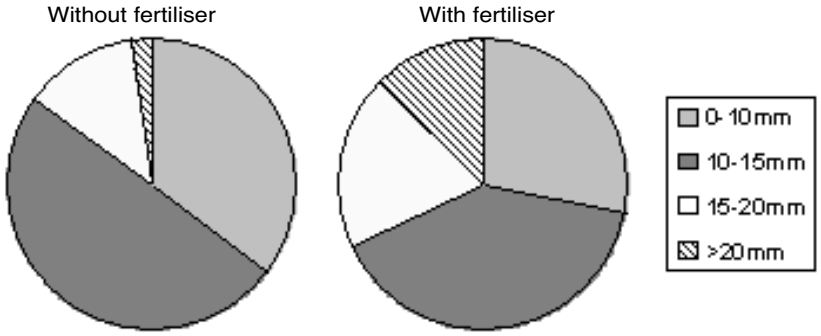

FIG. 8–Proportion of pruned branches in each of four diameter classes from untreated and fertiliser-treated *E. nitens* at Tim Shea (from Wiseman 2003).

Fertiliser treatment of *E. nitens* with nitrogen and phosphorus did not affect chemical properties (lignin, extractives, hydrolysable tannins) of the heartwood or sapwood (Wiseman 2003). The establishment and spread of decay appears not to be correlated with any of these wood properties (Wiseman 2003; P.D.Kube, T.J.Wardlaw & C.A.Raymond unpubl. data). However, we still do not know whether increases in photosynthesis, changes in tissue moisture, and improved carbon uptake after fertiliser treatment mean better resources to fight decay.

Breeding to Select for Greater Decay Resistance

White *et al.* (1999) found no differences in the spread of decay from stem wounds among provenances of *E. regnans*. In the same study, the spread of decay from stem wounds among open-pollinated families of *E. nitens* was much less than was found in *E. regnans* and showed only a modest heritability. However, Kube, Wardlaw & Raymond (unpubl. data) assessed the incidence of wound-rot and heart-rot in 13-year-old *E. nitens* progeny trials and found that they were under moderately strong genetic control with heritabilities of 0.60 and 0.40 respectively. They found no correlation between wound-rot and heart-rot, which suggested that they are separate traits. In addition, neither trait showed strong genetic correlations with diameter, basic density, cellulose content, methanol-soluble extractive content, or branching. These results suggest that deployment using a multi-trait selection strategy among *E. nitens* progeny is unlikely to yield marked increases in decay resistance. However, by selecting only for either wound-rot or heart-rot, significant increases in decay resistance may be achieved. Such a selection strategy may be appropriate for deploying on high decay-risk sites.

Pruning Intensity

The intensity of pruning in eucalypts has a marked effect on growth and the physiological processes contributing to those growth responses. Pinkard & Beadle (1998) found that the removal of up to 50% of the green crown had little effect on growth in *E. nitens,* but heavier pruning caused growth reductions. Similar studies have found that growth of *E. globulus* is more sensitive to the intensity of pruning (Pinkard *et al.* in prep.). Significant growth reductions were measured in *E. globulus* after 50% or more of the green crown was removed. The intensity of pruning also affects establishment of decay. Severe pruning (70% green crown removal) significantly increased the number of decay outbreaks in *E. globulus* (Pinkard *et al.* in prep.). However, more work is needed to determine whether this is simply the result of pruning the larger branches that occur further up the stem, or is the result of a reduced capacity of the tree to contain decay within the stubs of the pruned branches.

IDENTIFICATION OF FUNGI RESPONSIBLE FOR DECAY IN EUCALYPTS

We know that a great diversity of wood-decay fungi colonise eucalypts. However, the identity, abundance, and significance of individual species are poorly known. The need to understand the fungal-host relationship in order to predict the outcome of any decay infection has been shown in European tree-pathogen systems (Schwartze *et al.* 1999). A key issue for decay infections associated with pruning wounds in plantation eucalypts is whether the decay will spread in the tree over time. The outcome of this interaction between the tree and fungus will depend in part on the capacity of the fungus to infect and then cause decay within the tree. We have undertaken artificial inoculation trials with wood-decay fungi to assess their "attack" capability (Barry *et al.* 2002). As we identify a particular fungus that is more aggressive than others then new questions emerge. How prevalent is this fungus? What type of plantation will be at risk of infection by this fungus? In order to answer these questions we must be able to rapidly and systematically identify our fungus.

We now have a collection in excess of 1500 wood-decay isolates from different sources in Tasmania — regrowth eucalypts, plantation trees, rotting logs, and fruit-bodies (identified if possible). We are attempting molecular grouping of these isolates: Even if we cannot ascribe a name to a group we can characterise it. This work is on-going and will probably take several years' research by various people. We are seeing certain groups of isolates associated with a particular type of decay clustering together according to their DNA RFLP profiles (Mohammed *et al*. 2003).

CONCLUSIONS: AN INTERDISCIPLINARY APPROACH TO DECAY RESEARCH

The study of stem decay is time-consuming, expensive, and difficult to conduct. The main imperative driving the decay studies being done in Tasmania is to identify the risks of economic damage and, if necessary, develop strategies to manage those risks. Although this does not preclude opportunities to conduct fundamental research, it does provide a focus for such research. Furthermore, it is neither practical nor sensible to manage decay in isolation from other risks or essential silvicultural treatments required in managing the crop. It is this environment that has shaped the interdisciplinary research programme, combining a mixture of fundamental and applied studies, that we report.

We have used decay surveys, involving the dissection of sample trees, to define the extent of the problem, identify risk factors, and develop interim prescriptions to manage the problem (Wardlaw 1996, 2003; Mohammed *et al*. 1998). More fundamental studies into the nature of the host response to infection have enabled us to make more general predictions of the spread of decay over time (Barry, Davies & Mohammed 2001; Barry, Pearce, Evans, Hall & Mohammed 2001). We are beginning to validate these predictions by assessing the development of decay along a time series (Barry *et al.* 2003).

These fundamental studies have also provided evidence of a more general response by the host to wounding (Eyles, Davies & Mohammed 2003; Eyles, Davies, Yuan & Mohammed 2003; Eyles *et al*. in press (a), (b)), which may also operate against a wider range of damage agents such as mammalian herbivores (O'Reilly-Wapstra *et al.* in prep.). This may offer opportunities to refine selection strategies in breeding programmes to target traits that confer greater resistance to a wider range of damage agents.

Collaboration with tree physiologists has provided the opportunity to examine how the establishment and development of decay are affected by key silvicultural treatments such as pruning (Pinkard *et al.* in prep.) and fertiliser treatment (Wiseman 2003). Ongoing work in this area may us help to understand the physiological basis of the host response to silvicultural intervention and how that influences the establishment and development of decay.

Our research to date has uncovered many pieces of a jigsaw puzzle. There are still many pieces of the puzzle to find and fit, but as we complete more of the puzzle we improve our ability to work out how the pieces link together. The prospect is that as more of the puzzle is completed we will be more confidently able to model and predict the outcomes (in terms of decay risk) under a range of different circumstances. By doing so we will be in a much stronger position to develop optimum prescriptions for achieving overall stand health.

REFERENCES

BARRY, K.M.; DAVIES, N.W.; MOHAMMED, C. 2001: Identification of hydrolyzable tannins in the reaction zone of *Eucalyptus nitens* wood, by high performance liquid chromatography / electron ionization mass spectrometry. *Phytochemical Analysis 12*: 120–127.

———2002: Effect of season and different fungi on phenolics in response to xylem wounding and inoculation in *Eucalyptus nitens*. *Forest Pathology 32*: 163–178.

BARRY, K.M.; HALL, M.F.; MOHAMMED, C.L. 2003: We learn as they grow: Monitoring decay incidence and spread from pruning wounds of *Eucalyptus nitens*. Poster presentation at 8th International Congress of Plant Pathology. 3–7 February, Christchurch, New Zealand.

BARRY, K.M.; PEARCE, R.B.; MOHAMMED, C. 2000: Properties of reaction zones associated with decay from pruning wounds in plantation-grown *Eucalyptus nitens*. *Forest Pathology 30*: 233–245.

BARRY, K.M.; PEARCE, R.B.; EVANS, S.D.; HALL, L.D.; MOHAMMED, C. 2001: Initial defence responses in *Eucalyptus nitens* sapwood following wounding and inoculation, including NMR imaging. *Physiological and Molecular Plant Pathology 58*: 63–72.

BEALL, F.C. 2000: The market potential for eucalypts in North America. Pp. 6–11 *in* "The Future of Eucalypts for Wood Products", Proceedings of the IUFRO Conference, 19–24 March, Launceston, Tasmania.

COMMONWEALTH OF AUSTRALIA AND STATE OF TASMANIA 1997: Tasmanian Regional Forest Agreement. 133 p.

CUNNINGHAM, J. 1997: Development of operational techniques for thinning eucalypt regeneration. *Tasforests 9*: 85–104.

DEPARTMENT OF THE ARTS, SPORT, THE ENVIRONMENT, TOURISM AND TERRITORIES 1988: "Report of the Commission of Inquiry into the Lemonthyme and Southern Forest", Volume 1. AGPS, Canberra. 450 p.

DUDZINSKI, M.J.; OLD, K.M.; GIBBS, R.J. 1992: Minor damage to silvertop ash from thinning operations: Classification of wounds and early tree responses to damage. Report to Department of Conservation and Environment, Victoria. 33 p.

ELLIOTT, H.J.; BASHFORD, R. 1984: Incidence and effects of the dampwood termite, *Porotermes adamsoni*, in two Tasmanian east coast eucalypt forests. *Australian Forestry 47*: 11–15.

EYLES, A.; MOHAMMED, C. 2002: Comparison of CEPA (2-chloroethyl phosphonic acid) induced responses in juvenile *Eucalyptus nitens*, *E. globulus* and *E. obliqua*: a histochemical and anatomical study. *International Association of Wood Anatomists Journal 23(4)*: 419–430.

EYLES, A.; DAVIES, N.W.; MOHAMMED, C. 2003: Novel detection of formylated phloroglucinol compounds (FPCs) in the wound-associated wood of *Eucalyptus globulus* and *E. nitens*. *Journal of Chemical Ecology 29*: 881–898.

———: Traumatic oil glands induced in the wound-associated phloem of *Eucalyptus globulus*: chemistry and histology. *Trees: Structure and Function* (in press (a))

———: Tannins, flavonoids and terpenes detected in the wound wood of *Eucalyptus globulus* and *E. nitens*. *Canadian Journal of Forest Research* (in press (b))

EYLES, A.; DAVIES, N.W.; YUAN, Z.Q.; MOHAMMED, C. 2003: Host responses to natural infection by a stem canker (*Cytonaema* sp.) in the aerial bark of *Eucalyptus globulus*. *Forest Pathology 33*: 1–16.

FERGUSON, I.S.; DARGAVEL, J.B. 1978: Economic considerations. *In* Hillis, W.E.; Brown, A.G. (Ed.) "Eucalypts for Wood Production". CSIRO, Australia.

FOREST AND FOREST INDUSTRIES COUNCIL OF TASMANIA 1990: "Secure Future for Forests and People". Forest and Forest Industries Council of Tasmania, Hobart. 127 p.

GERRAND, A.M.; NEILSEN, W.A.; MEDHURST, J.L. 1997: Thinning and pruning eucalypt plantations for sawlog production in Tasmania. *Tasforests 9*: 15–34.

GREAVES, T.; McINNES, R.S.; DOWSE, J.E. 1965: Timber losses caused by termites, decay and fires in an alpine forest in New South Wales. *Australian Forestry 29*: 161–174.

MOHAMMED, C.; HALL, M.; SAVVA, M. 1998: Reduction of loss from pruning-associated-decay in *Eucalyptus nitens* plantations grown for sawlog and veneer. Report to the Forest and Forest Industries Council of Tasmania. 39 p. + appendices.

MOHAMMED, C.; BARRY, K.; HARRISON, K.; WISEMAN, D.; ZI-QING, Y.; YEE, M.; HOPKINS, A.; WARDLAW, T.; BOUGHER, N.; TOMMERUP, I. 2003: Decay fungi in eucalypts slowly reveal their true identity! *Co-operative Research Centre for Sustainable Production Forestry, Resource Protection Program, Pest off 18*.

OLD, K.M.; DUDZINSKI, M.J.; GIBBS, R.J.; KUBONO, T. 1993: Stem degrade following harvesting damage. Second progress report on collaborative research in regrowth forests between CSIRO and the Victorian Department of Conservation and Natural Resources. CSIRO, Canberra. 48 p.

O'REILLY-WAPSTRA, J.M.; McARTHUR, C.; POTTS, B.M.: Linking plant genotype, plant defensive chemistry and mammal browsing (in prep.).

PARLIAMENT OF TASMANIA 1999: "Forestry Act (1920)". Parliament of Tasmania, Hobart, Tasmania.

PINKARD, E.A.; BEADLE, C.L. 1998: Effects of green pruning on growth and stem shape of *Eucalyptus nitens* (Deane and Maiden) Maiden. *New Forests 15*: 107–126.

PINKARD, E.A.; MOHAMMED, C.; BEADLE, C.L.; HALL, M.F.; WORLEDGE, D.; MOLLON, A.: Growth responses, physiology and decay associated with pruning plantation-grown blue gums (in prep.).

PINKARD, L.; CUNNINGHAM, J.; WILSON, L.; WARDLAW, T.; NEILSEN, B. 2001: Pruning and thinning cool temperate eucalypt plantations for clearwood production. Forestry Tasmania, Hobart, Internal Report. 69 p.

SCHWARZE, F.W.M.R.; ENGELS, J.; MATTHECK, C. 1999: "Fungal Strategies of Wood Decay in Trees". Springer-Verlag, Berlin. 185 p.

SHIGO, A.L.; MARX, H.G. 1977: Compartmentalization of decay in trees. *USDA Forest Service Agricultural Information Bulletin 405*. 73 p.

WARDLAW, T.J. 1996: The origin and extent of discolouration and decay in stems of young regrowth eucalypts in southern Tasmania. *Canadian Journal of Forest Research 26*: 1–8.

———2003: The extent, impact and management of stem decay in young regrowth eucalypt forests scheduled for thinning in Tasmania. Ph.D. thesis, University of Tasmania. 244 p.

WARDLAW, T.J.; NEILSEN, W.A. 1999: Decay and other defects associated with pruned branches of *Eucalyptus nitens*. *Tasforests 11*: 49–57.

WARDLAW, T.J.; SAVVA, M.H.; WALSH, A.M. 1997: Stem decay in final-crop eucalypts from regrowth forests identified for intensive management. *Tasforests 9*: 123–138.

WHITE, D.A.; KILE, G.A. 1991: Thinning damage and defect in eucalypts. Pp. 152–177 *in* Kerruish, C.M.; Rawlins, W.H.M. (Ed.) "The Young Eucalypt Report". CSIRO, Melbourne.

———1994: Breakdown of barrier zones and prediction of the spread of discolouration and decay resulting from stem wounds in *Eucalyptus regnans* and *E. obliqua*. *European Journal of Forest Pathology 24*: 71–78.

WHITE, D.A.; RAYMOND, C.A.; KILE, G.A.; HALL, M.F. 1999: Are there genetic differences in susceptibility of *Eucalyptus nitens* and *E. regnans* stems to defect and decay. *Australian Forestry 62(4)*: 368–374.

WILKES, J. 1985: Host attributes affecting patterns of decay in a regrowth eucalypt forest. I. Patterns of natural decay. *Holzforschung 39(1)*: 17–22.

WISEMAN, D. 2003: Fertiliser produces larger branches and higher incidence of wood decay in *E. nitens. Co-operative Research Centre for Sustainable Production Forestry, Resource Protection Program, Pest off 24*.

YANG, J.L.; WAUGH, G. 1996: Potential of plantation-grown eucalypts for structural sawn products. II. *Eucalyptus nitens* (Dean & Maiden) Maiden and *E. regnans* F. Muell. *Australian Forestry 59(2)*: 99–107.

YE, K. 2000: Market opportunities for eucalypts in China. Pp. 12–18 *in* "The Future of Eucalypts for Wood Products". Proceedings of the IUFRO Conference, 19–24 March, Launceston, Tasmania.