LETTER TO THE EDITOR

Shoot dieback epidemics in Pinus radiata *in New Zealand:*

is there an endophyte story?

Summary

Shoot dieback, specifically leader dieback, has caused considerable stem malformation in *Pinus radiata* in New Zealand. While seldom totally absent, it has also occurred in some major episodes. Immediate causes of the dieback have included: pathogens, climatic damage, and nutrient deficiencies, acting individually or in combinations. Pathogens can be identified by isolating the organism(s) from affected material, backed up by confirming pathogenicity. Climatic injuries can be addressed by planting on appropriate sites, and good establishment and tending. Nutrient deficiencies, if identified, can be corrected by applying appropriate fertilisers.

I focus here on two spectacular episodes of dieback on the North Island volcanic plateau, one in the early 1930s, and the other with an initial peak around 1967. Of special scientific interest is that these major outbreaks affected first-crop stands, but not second crops. While the evidence is largely anecdotal, this difference has been stark and calls for explanation. To account for the difference fully seems to require some additional biotic effect or effects. Recent appreciation of the prevalence, diversity and potential influence of fungal endophytes in pine needles suggests that changes over time in endophyte populations may have played a role in the observed difference. Various lines of study are readily possible for investigating this postulate, and some are listed. Also, possible wider implications are outlined.

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Editor's Note:

The aim of an opinion piece, such as this, is to stimulate scientific discussion on a particular topic.

Alternative viewpoints are welcomed and should also be submitted as Letters to the Editor at the following address: nzjfs@scionresearch.com

Introduction

Shoot dieback has often been recorded in plantations of *Pinus radiata* D.Don in New Zealand. The condition is seldom totally absent, but it has also occurred in some spectacular episodes. Dieback involving leaders can readily cause lasting and serious stem malformation. This occurs in the form of forks or multileaders, which are often associated with kinks in stems. In extreme occurrences of dieback, leaders and almost all main laterals are repeatedly affected. This can result in severe and repeated malformation, and also seriously curtail stand growth.

Fortunately, the incidence of dieback usually trails off after the crop is around 5 – 7 years old. However, this means that the effects of dieback are manifested mainly in the butt log which is potentially the most valuable part of the tree.

Height growth, log quality and recoverable wood production can all be reduced substantially if leader dieback is at all prevalent. This has obvious economic implications, forcing the forest grower to accept increased growing costs and/ or reduced net value of the log harvest. Diameter growth and total stem volume production are, however, are much less readily affected. The exception is at the level of individual trees that lose competitive crown status through loss of height growth.

Even if no obvious permanent malformation results, timber quality tends to be adversely affected. Timber degrade can result in two different ways. The first is through grain distortion and associated formation of reaction wood. The second is through increased branch sizes which reflect periods when several laterals became competing leaders.

Dieback occurs in *Pinus radiata* in all parts of New Zealand, and I review causes in general. Nevertheless, my focus is on the patterns of occurrence in the volcanic plateau of the North Island (the "pumicelands"), which is very roughly a triangle of land extending from north and northeast of the middle of the island with the third side being the coastline. This is characterised by distinctive soils, terrain, and climate. Soils are almost all recently derived from rhyolitic tephra (i.e. pumice), with distinctive geochemistry. The tephra over most of the area is of the air-fall kind. However, there are areas of both flow tephra (from *nuées ardentes*) and water-borne pumice, these areas being hollows or flats with distinctive mesoclimates. The macroclimate is strongly oceanic, and characterised by high rainfall (generally 1100 – 2000 mm/annum) which has an even seasonal distribution and is associated mainly with mild to warm temperatures.

The volcanic plateau contains extremely important forest plantation resources. The plantations occupy over 500,000 ha (cf Ministry of Agriculture and Forestry, 2010), of which over 90% would be in *Pinus radiata* and mostly very productive. This represents nearly one-third of the country's commercial forest resource. However, the region has been associated with spectacular past episodes of dieback which. Yet those episodes have not recurred in subsequent rotations on the same sites. To explain this is a challenging puzzle.

During recent years, major advances have come in the awareness of the prevalence and possible roles of fungal endophytes living in tissues of conifers. In the light of past occurrences of the dieback, and of past understanding of the causes, a role of endophytes is postulated as a clue to the puzzling historical picture. Avenues for follow-up research are proposed.

Known and presumed causes

Various causes of dieback have been postulated, but fall into three main categories: pathogens, climate and nutrient deficiencies, which can operate individually or in combinations.

Pathogens, notably *Allantophomopsis pseudotsugae* (M. Wilson) Nag Raj (syn.*Phomopsis strobi*)[1,2] (Birch, 1935) and *Diplodia pinea* Kickx (Chou, 1976a), have been implicated in major outbreaks affecting young crops, although *D. pinea* has been widely implicated in scattered occurrences as well.

Among climatic factors, frost damage (Burdon, 2001) can cause shoot dieback, often affecting almost all upper shoots. However, this tends to be confined to very young trees. Dieback can also occur after drought periods. Such dieback can be associated with attack by *D. pinea* (Wright & Marks, 1970), in what is termed "autumn brown top" in Australia. This condition is often evident in *P. radiata* after droughts in Canterbury, in the South Island of New

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[1] *Birch, T. C. (1932). [Unpublished report].* Report on diseases of pines in the pumice country. *Wellington, New Zealand: New Zealand State Forest Service.*

[2] *Birch, T. T. C. (1933). [Unpublished report].* Supplement to Birch (1932).

Zealand, where it can also be severe in *P. ponderosa* Lawson. Attack by *D. pinea* attack can also occur on shoots injured by hail (Scott, 1960), but that is uncommon in New Zealand.

Will (1978, 1985) found that boron deficiency, like frost, can cause dieback of upper shoots, often occurring repeatedly and/or affecting multiple shoots. Again, however, this tends to be confined to young stands. Dieback caused by boron deficiency tends to be exacerbated by drought. Poor mobility of boron within the plant, combined with fluctuations in boron availability, accounts for the pattern of such dieback.

Apart from boron deficiency, other nutrient deficiencies may be involved. Calcium deficiency has been implicated in dieback, sharing with boron the same immobility within the plant. For instance, dieback was prevalent in many stands on 'gumland clay' soils which occur north of about Lat. 37½° S. in New Zealand. Such soils were diagnosed and successfully treated as being acutely phosphorus-deficient. Without superphosphate applications, the trees were very slow-growing, spindly and prone to dieback (Will, 1978, 1985). There is reason to believe that calcium deficiency caused the actual dieback which is not necessarily associated with acute phosphorus deficiency (Will, 1978, 1985). On such gumland clay sites, foliar levels of calcium are typically low, and the superphosphate used to correct phosphorus deficiency has a high calcium content which would account for correcting the dieback. On the volcanic plateau, however, there has never been reason to suspect calcium deficiency as a significant cause of dieback

Acute magnesium deficiency, in the form of severe Upper Mid-Crown Yellowing (Beets & Oliver, 1998) can lead to dieback of first-order laterals, but this tends to affect somewhat older trees. Extreme cases, however, can involve the leader as well (P. N. Beets pers. comm., 2008). In any event, magnesium deficiency is widespread in the volcanic plateau (Will, 1978. 1985), and is evidently induced by high levels of potassium (Beets et al., 2003).

Birch[1] (1936) noted a form of dieback in the volcanic plateau, which he termed "stag headedness". He could not attribute it to any pathogen, and the pattern of occurrence made very unlikely any effects of frost injury or boron deficiency.

Identifying causes

Identifying causes of particular cases of dieback can be difficult, especially if multiple causes are involved. Moreover, practical considerations often dictate that causes be identified definitively in only small samples of individual occurrences. One must then make inferences concerning the remaining occurrences in a particular episode or occurrences in a consistent pattern.

If a pathogen is involved it is usually possible to identify it from affected material. Demonstrating, then, that the isolated organism can cause the disease establishes its role as a pathogen, by satisfying Koch's postulate.

Nutrient deficiencies can be diagnosed with confidence from appropriate foliar sampling and chemical analysis. This can be confirmed if the problem is corrected by applying fertiliser containing the nutrient in question.

Cases of multiple causes of dieback have been demonstrated. As mentioned earlier, drought can be a predisposing factor for not only attack by *Diplodia pinea* but also boron deficiency. High temperatures and humidity (which may occur after drought) will favour attack by *D. pinea* (Eldridge, 1961), while exposure to frost that caused no visible injury has been shown to allow attack by *Allantophomopsis* (Birch, 1935). Indeed, even more complex co-occurrences of conditions may be involved. For instance, certain hollows and river flats on the volcanic plateau of central North Island, New Zealand are characterised by a combination of: 'ponding' of cold air; periods of high humidity; and the occurrence of either coarse flow tephra or coarse, water-borne pumice wash. This combination, the 'unholy trinity', may be conducive to frost damage, pathogen attack, and boron deficiency, making diagnosis of the causes of shoot dieback very complicated.

Another problem arises in postulating or revising putative causes of dieback reported in the past. Regarding the "stag headedness" reported by Birch[1] (Birch, 1936) but not attributed to any detectable pathogen; it can only be speculated that this was attributable to magnesium deficiency akin to very severe cases of Upper Mid-Crown Yellowing described by Beets and Oliver (1998).

Historical picture

Dramatic episodes of shoot dieback in *Pinus radiata* growing on the volcanic plateau have been reported during two periods, one in the early 1930s and the other in the late 1960s. The first episode involved quite large areas of first-rotation crop[1,2], mostly around Lat. 39° S. Very high rates of shoot dieback were noted on sites that were subject to ponding of cold air. Many of the sites were at high altitudes (>500 m) on river flats of the upper Rangitaiki River. Other sites affected, judging from levels of persistent severe malformation, included river terraces of the Waikato River, which are at lower altitudes. The sites concerned mostly had soils that are now recognised as having been formed on flow tephra or by coarse, water-borne pumice wash. In addition, leaders of scattered trees showed dieback on the remaining sites in the region. The intensive occurrences of dieback were attributed by Birch mainly to the pathogen "*Phomopsis strobi*" attacking shoots that had been subjected to severe, unseasonable frost. In fact, Birch (1935) demonstrated that inoculation with the pathogen could cause dieback on shoots that had been exposed to frost without causing visible frost injury. Anyway, the stands where dieback was very intensive showed severe and permanent malformation of the butt logs. Records of the extent of recurrence of dieback during the following few years have not been found. However, the lack of reports, and the recovery of trees above the butt logs (evidenced by very little fresh malformation in upper logs) leave no doubt that the dieback abated almost completely.

Second crops of *Pinus radiata* on the same sites have shown very little dieback and consequent stem malformation, to the extent that specific documentation was deemed to be unwarranted. So, despite the lack of formal documentation, there can be no doubt that the amount of shoot dieback was vastly less than in the first crops. No unequivocal causes have been found for the contrast in either the extent or effects of dieback between the first and second crops on the same sites. Certainly, it is very difficult to invoke changes in mesoclimates as the cause of differences between the prevalence of dieback in first- and second- rotation crops.

The second major episode of dieback was first noted in 1967, in first-rotation crops at two sites in Tarawera Forest (Lat. 38° S). Almost every tree was affected, often repeatedly.

One site was Fenton's Mill Flat, very near Kawerau, and the other was called "Death Valley", at the foot of Mount Edgecumbe. Both sites had similar pumice soils with a surface layer of basaltic scoria, and a particular combination of other distinctive features. Both were enclosed hollows, at low altitude and were often isolated from sea breezes. As such, they were often subject to high temperatures, high humidities, and wide diurnal temperature fluctuations. Dieback appeared extreme at Fenton's Mill Flat, creating much doubt as to whether a satisfactory crop could ever be harvested. Yet, only three years later, after a thinning, the stand showed little outward sign of the outbreak. At Death Valley the dieback was markedly more extreme. The dieback occurred so repeatedly as to check growth very severely and leave much persistent malformation.

The good and speedy recovery of trees at Fenton's Mill Flat was presumably helped by a very high site index (reflecting warm temperatures, abundant rainfall, good rooting depth and generally good soil fertility for a forest site) that would allow extremely rapid stand development. Comparing severity of dieback in different episodes cannot be done reliably. There is no doubt that, in 1967 and following years, the dieback in Tarawera Forest was worse in Death Valley than in Fenton's Mill Flat. The degree and speed of recovery of tree form in Fenton's Mill Flat, compared with the persistence and severity of malformation on sites affected severely by the 1930s episode, suggests that the dieback was more severe in the 1930s. However, the degree of eventual recovery in Death Valley (Figure 1) suggests that factors other than initial severity of dieback may influence degree of recovery. Possibilities are: higher stocking reflecting better post-planting survival, much warmer site conditions being conducive to good recovery, and superior genetic stock resulting from the practice of select-tree seed collections.

The episodes of dieback in Tarawera Forest prompted vigorous research into possible causes. Subsequent results confirmed that *Diplodia pinea* was a common factor and also established that *D. pinea* could function as a primary pathogen of uninjured, unripened shoots (Chou, 1976a, b).

While the intensive episodes of dieback in 1967 were highly localised, a lower level of dieback also occurred in first-rotation plantings of *Pinus radiata* at surrounding high-index sites. There was enough dieback at these sites to cause worrying levels of stem malformation. Accordingly, a pilot programme of selection for resistance to *Diplodea pinea*-associated dieback was begun in 1970 (Burdon et al., 1987) at both Fenton's Mill Flat and Death Valley. Trees were selected at Fenton's Mill Flat on the basis of almost no sign of dieback, in addition to good form and vigour. At

FIGURE 1: Trees in 'Death Valley', Tarawera Forest in 1977 recovering from very severe and repeated early dieback that greatly checked height growth for some years. Tree in middle has had branches cut off to show resulting ramicorn branches and stem kinks.

Death Valley, mainly trees with the fewest signs of dieback were selected. In Death Valley, an area of first-crop trees was felled to make way for some experiments to test selections for putative resistance. However, the incidence of dieback in this second rotation was so low that the experiments were, in effect, abandoned. Similarly, the second crops of *P. radiata* in surrounding sites have shown strikingly less leader dieback than the first crops. This lower level of dieback in second-rotation crops mirrors closely the episode of intensive dieback in the 1930s. As with the dieback occurring in the 1930s, it seems very unlikely that post-harvest modification of mesoclimates could account for the drop in dieback in the second crops. The evidence is admittedly anecdotal, yet the gaps in the records are themselves testimony to the stark difference in dieback between the first and second crops. This difference is of much scientific interest, and satisfactory explanation may eventually have major practical implications.

From the late 1960s till 1980, New Zealand Forest Biology Survey Annual Reports were produced by D.J. Kershaw (unpubl.). Generally, they mentioned observations of *Diplodia pinea*-associated shoot dieback in *Pinus radiata*, but without repetition of the intensive outbreaks. Later, however, a severe outbreak of dieback in coastal Hawke's Bay was recorded by Stoodley[3]. Dieback occurred "within a month" (reportedly within two to three weeks) after an exceptionally hot and humid day during a 1981-2 summer drought.

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[3] *Stoodley, M. A. (1985). [Unpublished report].* A study of Diplodia wilt in drought stressed *Pinus radiata* in coastal Hawkes Bay. *(Forest Health and Improvement Project Record No. 734). Rotorua, New Zealand: Forest Research Institute.*

Interpretation of historical picture

The extent and severity of the early 1930s episodes of shoot dieback seems largely attributable to a combination of factors: a large area of stands all within a vulnerable age band and located on high-hazard sites, and at least one extreme climatic event (according to Birch[1]) in the form of a very late frost. Admittedly, more of a mix of age classes in the second rotation on such site types would make outbreaks less striking. Also, a genetic shift towards a short-internode branch habit, resulting from both natural selection (Burdon et al., 1996) and artificial selection (Burdon et al., 2008), may have made individual cases of leader dieback less noticeable, because trees of this branching habit can replace damaged leaders with less visible malformation. Such effects, however, seem grossly inadequate to explain the difference in shoot dieback between rotations.

It is uncertain whether the first rotations affected in the 1930s were subjected to decisively more severe climatic events than second crops on such sites. That possibility, however, seems most unlikely for the outbreaks during the late 1960s in Tarawera Forest. The same holds for the evident non-recurrence of the nuisance level of dieback that occurred extensively in first-rotation stands planted in much of the region after about 1950. While one cannot rule out decisive differences in climatic events between rotations, in the case of the 1930s outbreaks, or in mesoclimate in the case of Death Valley, climatic effects provide a very unconvincing explanation for the lack of significant second-rotation outbreaks.

A really decisive effect of a genetic shift, in either the host or the pathogen, also seems very unlikely. Despite promising results from a glasshouse test of selections for resistance to *Diplodia pinea*[4] (Burdon et al., 1982), a field progeny trial[5] indicated a very limited selection response under field conditions. Surprisingly, these field-trial results occurred despite quite good resolution of progeny differences[5]. For a convincing explanation we must consider the possibility of biotic factors involving both host and pathogen, which I will now address.

Biotic interactions?

The behaviour of plant pathogens and pests can be much influenced by the presence of other microorganisms (e.g. Blakeman & Fokkema, 1982; Eyles et al., 2010; Fravel, 1988; Rishbeth et al., 1988). Microorganisms associated with plants fall into several categories which include: phylloplane species, living on leaf (or possibly stem) surfaces; rhizosphere species profiting from root exudates; mycorrhizal symbionts; and full endophytes that inhabit various plant tissues beneficially, or at least harmlessly, to the hosts (Carroll, 1988). Demarcation between true endophytes and latent pathogens, however, can often be unclear.

In a second rotation after afforestation there could well be a markedly different assemblage of microorganisms associated with the trees compared with in a first rotation. While of no direct application to dieback, it is noteworthy that within the life of a pine crop, there can be a marked succession of mycorrhizal symbionts (Chu-Chou, 1979). A second crop would likely start with a different assemblage of such symbionts compared with the first, a question being how persistent such a difference would be in the life of a second crop. The accumulation of duff would seem highly conducive to a different assemblage of other microorganisms at the start of a second rotation. Also, there may well be quite a different phyllosphere, the assemblage of phylloplane microorganisms on the surfaces of leaves and shoots, in a second crop compared with the first.

In another context, it has been observed that some phylloplane fungi (*Cladosporium* spp.) can behave antagonistically towards the poplar leaf rust pathogen *Melampsora larici-populina* Kleb. (Sharma & Heather, 1981); which led these authors to postulate that a build-up of *Cladosporium* had contributed to reduced severity of leaf rust within a few years after *Melampsora* arrived near Canberra, Australia. Variations in phyllosphere organisms during the life of a tree, and between rotations, could be studied, as could possible antagonisms between them and known dieback-causing pathogens.

A specific case of apparent cross-protection against a pathogen of *Pinus sylvestris* L. has been reported by Minter (1981). Only in the absence of *Lophodermium conigeum* (Brunaud) Hilitz., did *Lophodermium seditiosum* Minter et al. behave as an aggressive foliage pathogen, although it was not established whether *L. conigeum* was behaving as a true endophyte or as a weak pathogen or as a saprophyte.

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[4] *Burdon, R. D., Currie, D., & Chou, C. K. S. (1976). [Unpublished report].* Response to inoculating with *Diplodia pinea* in progenies of apparently resistant trees of *Pinus radiata*. *(Genetics and Tree Improvement Internal Report No. 122). Rotorua, New Zealand: Forest Research Institute.*

[5] *Burdon, R. D. & Low, C. B. (1980). [Unpublished report].* First assessment of field progeny test of *Pinus radiata* for resistance to Diplodia infection. *(Genetics and Tree Improvement Internal Report No. 187). Rotorua, New Zealand: Forest Research Institute.*

Endophytic fungi may well have played a role in the great reduction of shoot dieback in *P. radiata* after the first rotation. In fact, there are indications that they could be important. It has recently become clear that endophytes are remarkably abundant in conifers, at least in the seeds and foliage (Ganley et al., 2004; Ganley & Newcombe, 2006 and references therein). Indeed, we now know enough—but not yet too much—to postulate a huge range of possible effects, which I will now outline.

Endophytes can be antagonistic to both pathogens and animal pests. Of outstanding importance is the known role of an endophyte in ryegrass (*Lolium* spp.) conferring resistance to an important insect pest (Easton, 2007). In respect of pathogens, there can be a direct antagonistic effect of an endophyte to some pathogen(s), or an endophyte can make its host resistant to one or more pathogens. Such resistance may range from being broadly systemic (termed Systemic Acquired Resistance) to much more localised (Eyles et al., 2010). Conversely, some endophytes might increase susceptibility of the host to certain pathogens, and/or influence the role of environment in favouring the pathogen in the classical host-pathogen-environment 'triangle'. Depending on the mode of transmission of endophytes, there are many possible ways in which the assemblage of endophytes may change during the life of a stand, and from the first crop to the second.

An intriguing conundrum exists concerning *Diplodia pinea*. It has reportedly occurred in an "endophytic" state (Eyles et al., 2010) which would be widely viewed as latent pathogenicity. Such a state, however, seems hard to reconcile with the involvement of *D. pinea* in autumn brown top, which occurs only after some wet or damp weather following the actual drought (Wright & Marks, 1970). Damp conditions may not provide sufficient moisture for trees to recover from drought stress or injury, but there may be enough for spore germination. Spores of sapstain fungi, which include *D. pinea*, can germinate very rapidly, so this scenario seems a more likely possibility than the moisture activating latent pathogen infection. These fungi can evidently spread rapidly in wood (Eden et al., 1997), although just how rapidly *D. pinea* can spread in standing trees has not been quantified (D. O'Callahan [Eden] pers. comm., 2011). In any event, drought stress as a predisposing factor for fungal attack is well established (Desprez-Loustau et al., 2006 and references therein).

Possible studies

Numerous lines of study come to mind for investigating the role of endophytes. They include:

- Study of populations of seed-borne endophytes and latent pathogens. This can now be accomplished with modern DNA analysis much more easily than in the past. (The presence of various fungi as loose spores in seeds with unsealed seed coats needs to be taken into consideration).

- Study of conditions for survival or elimination of seed-borne endophytes—and latent pathogens.

- Study of endophyte assemblages at different stages of the crop life cycle. This is likely to be a major undertaking as it would involve investigations in the nursery and at different ages in field.

- Compare endophyte assemblages between first and subsequent rotations. Existing sites with good 'back-to-back' comparisons may be scarce so new trials may need to be established.

- Study of relationships between endophyte assemblages and observed disease in the field.

- Laboratory studies of possible antagonisms between known endophytes and known pathogens.

- Inoculation studies in material of known endophyte status, with environmental conditions as a controlled variable.

Isolating, identifying and characterising the roles of such microorganisms will be very challenging even with the benefit of current DNA technologies. In addition to the specific importance of the studies in finding the possible keys to the difference in dieback between first and second crops, all the above investigations are justifiable as areas of major scientific interest in their own right. Above all, these studies could be crucial to developing new tools for biological control of serious pathogens and even insect pests.

While endophytes represent the area of exciting science, and seem likely to exercise greater protective effects, the

possible role of shoot-surface organisms would still warrant study. Comparisons of the microorganisms between age classes and rotations seem indicated, as would study of the impacts of cultures of these organisms on spore germination and hyphal development of pathogens. Study of competitive interactions among shoot-surface organisms appears to be less difficult than study of the assemblage and roles of endophytes, although various interactions may be laboratory-specific.

Concluding comments

The dieback story has raised the possibility of the role of endophytes in at least modulating the incidence and severity of disease. But the story probably has a much wider significance. Given that *Pinus radiata* is widely grown in New Zealand on sites of fungal disease hazard, the appearance of a new and severe disease may require the availability and understanding of as many mechanisms as possible for managing the disease. New diseases affecting this species in the last 20 years or so include pitch canker in California (Storer et al., 1997), and needle death in Chile associated with *Phytophthora pinifolia* (Durán et al., 2008). As early as the 1960s, an often-severe needle blight caused by *Dothistroma pini* Hulbary had started to affect *Pinus radiata* in anumber of countries. Importantly, all these diseases have struck without prior record of being significant in *P. radiata*. This unpredictability makes it doubly important to acquire fundamental knowledge of factors affecting pathogen attack, rather than just addressing proven countermeasures against pathogens of known significance.

Acknowledgements

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