ARMILLARIA ROOT DISEASE OF *PINUS RADIATA* IN NEW ZEALAND. 2: INVASION AND HOST REACTION

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ABSTRACT

The root systems of thirteen 13-year-old *Pinus radiata* D. Don trees with basal resinosis caused by *Armillaria novae-zelandiae* (Stevenson) Herink in Kaingaroa Forest were excavated and the boles were sectioned. The extent of infection in bark and wood of boles and roots was determined. *Armillaria novae-zelandiae* appeared to be able to survive and spread in bark, leaving the vascular cambium and the inner living phloem largely intact. Infected bark exhibited special characteristics including wide, light-coloured, resin-soaked layers of dead phloem separated by thick periderms often containing stone phellem, small mycelial fans, and layers of old resin. Penetrations to and death of sections of the cambium occurred on all but one of the trees, but the areas involved were small (mostly <40 mm in tangential and 100 to 150 mm in longitudinal extent). Most were located between major lateral roots, and in none did the dead area extend higher than 300 mm above the root collar. Penetrations generally occurred during the dormant season and a strong host reaction the following growing season resulted in well-developed calluses, which healed xylem penetrations within a few years. Invasion of the cambium by *A. novae-zelandiae* occurred at various dates from 1990 to the current growing season, and could not be simply related to stress caused by silvicultural operations or climatic conditions. *Armillaria novae-zelandiae* is likely to survive on infected trees to harvest, causing minor scarring and, in a few trees, resin soaking of xylem, but only at the very base of the tree. A small increment loss is also likely. After harvest and planting, the pathogen may again cause mortality in young plantations.

Keywords: infection; invasion; host reaction; roots; root collar; callus; resin; *Armillaria novae-zelandiae*; *Pinus radiata*.

INTRODUCTION

Armillaria root disease caused by either of the two New Zealand species of *Armillaria* (*A. novae-zelandiae* and *A. limonea* (Stevenson) Boesewinkel) has been observed on *Pinus radiata* for many years. In young (<5-year-old) plantations the disease causes scattered

mortality, which is most serious on sites that have a large amount of inoculum at planting, such as those previously occupied by native forests (Hood & Sandberg 1993a). In older stands, mortality is rare, but infection by *Armillaria* species does persist and can be found at the root collar of many trees where it can be identified by the combination of resinosis and rhizomorphs. Small mycelial fans can also be found in the outer bark. In such stands Armillaria root disease does not cause significant tree mortality or obvious crown symptoms, and infection of this type has been called "chronic" (Hood & Sandberg 1993b). MacKenzie (1987) and Hood *et al.* (2002) showed that the extent of chronic infection of individual trees can change over time, the direction and magnitude of such change probably depending on stand and site conditions, weather, and management practices. Such chronic infections also appear to result in small but significant increment losses (Shaw & Calderon 1977; MacKenzie 1987; Kimberley *et al.* 2002).

Smaller trees tend to be more severely infected (MacKenzie 1987), but cause and effect are confounded. Survival of infection until harvest appears to be assured, and there is little doubt that without remedies such as stump removal (Self & MacKenzie 1995), there will again be mortality caused by the disease during the early stages of the next rotation (Self *et al.* 1998).

Several studies in New Zealand have been designed to follow the disease over time (MacKenzie 1987; Hood *et al.* 2002). In such studies, it is important to keep disturbance of infected trees to a minimum because disturbance might well affect subsequent disease development. Hence, in these studies, as well as in descriptions of Armillaria root disease severity (Shaw *et al.* 1976; Shaw & Toes 1977), the extent of infection by *Armillaria* species has been described as the degree of "girdling" — by which is meant the lateral extent of resinosis visible on the outside of the bark at the root collar. Information on root infection and on necrosis of phloem and the vascular cambium caused by *Armillaria* species, as well as on host reactions to invasion by the parasite, is not available from such studies. The purpose of the study reported here was to elucidate the aetiology of the disease by describing the location of the parasite in bark, wood, and roots of affected trees, the extent of necrosis of such tissues, and host reactions to invasion.

METHODS

The investigation was conducted in Kaingaroa Forest in 1998, in a stand of *P. radiata* in its thirteenth growing season since planting. The site chosen was Cpt 365 in the surrounds of a New Zealand Forest Research Institute study dealing with the effects of various thinning regimes on Armillaria root disease (Hood & Sandberg 1993b). Stand stocking was 612 stems/ha following a non-commercial thinning undertaken 6 years after planting at a stocking of 983 stems/ha. *Armillaria novae-zelandiae* was by far the most common species of *Armillaria* found in this area (Hood *et al.* 2002), and 13 trees with various degrees of infection at the root collar, as shown by the extent of basal resinosis, were selected to represent the range of diameters, pruning regime, crown form, and infection severity found in the stand. All trees were located within 100 m of each other. The following tree parameters were recorded: height; diameter at breast height (dbh); live crown ratio (as a percentage of tree height, the base of the live crown being defined as the point halfway between the lowest living branch and the lowest fully live whorl); degree of pruning (in three classes — high (>3m), low (<3m), and none); branching habit (in three classes — fine, medium, and coarse);

and the presence or absence of a sunken face in the area of resinosis, possibly indicating reduced increment or death of a section of cambium.

The root system of each tree was then excavated to a depth of at least 0.7 m or to a root-restricting layer, and for a 1-m radius around each tree. All roots were examined for infection by *Armillaria* species, and for each root infection the following measurements were recorded: root diameter at the proximal end of the infection, length (as determined by the area exhibiting copious resinosis), and direction of root from the tree. The bark was then peeled from each infection to determine whether the root xylem had been penetrated by the pathogen. In all bole or root infections, infection by *Armillaria* species was verified by the presence of rhizomorphs or mycelial fans in the bark or both. Small roots (<20 mm diameter) were severed and removed after inspection; large roots were left in place. A set of photographs was taken of each excavated root system. The extent of the basal stem infection was recorded as the percentage of the tree circumference occupied by each separate patch of resinosis.

Finally, the upper roots were severed at the edge of the excavation, and the trees were pulled over using a hand winch to lift the root system out of the ground. All soil was cleared from each root system and further infections were noted. Disks were then cut at the centre of the main basal infection, at dbh, and at various heights between dbh and the main basal infection. The basal section was planed, and all penetrations by *Armillaria* species were recorded by year and tangential extent. Healthy and infected bark structure was described.

RESULTS

Soils

The soil type at the study site was Kaingaroa sand (Vucetich *et al.* 1960; Vucetich & Pullar 1964; Will & Knight 1968; Rijkse 1988), a welded impeded pumice soil (Hewitt 1993). Soils had been much disturbed by site preparation (V-blading). The forest floor was 20–40 mm thick with a very thin humus layer and rapid incorporation of organic matter into a well-developed, 100-to 200-mm-thick A-horizon. The mineral soil consisted of layers of fine ash and pumice, some highly compacted and others unconsolidated. The order, composition, and thickness of the layers varied considerably between trees and that variation extended over the whole depth of excavation.

Soils contained old well-rotted roots and stumps (occasionally containing pieces of sound wood) of the previous rotation stand of *Pinus nigra* ssp. *laricio* (Poir.) Maire. Some of these had been disturbed by site preparation for the current stand. In several cases rhizomorphs of *Armillaria* species were found associated with this material.

Root Systems

The upper root systems of the 13-year-old trees examined consisted of a set of three to six major lateral roots and a similar number of fine (<20 mm diameter) primary roots. The major laterals generally extended out near the soil surface to a distance of at least 1 m from the tree base before turning down. The smaller upper laterals formed a network of fine roots lying within a few centimetres of the mineral soil surface. Lateral roots of the same tree were often grafted together when in contact with each other. A rapidly tapering and somewhat fluted

taproot with a diameter directly below the upper laterals of slightly less than tree dbh generally extended down for at least 500 mm. Additional lateral roots developed from the taproot, often well below the upper laterals. These lower laterals, typically 20–50 mm in diameter, were generally short, angled downwards steeply, and ended in horizontal plates of highly divided small roots lying on top of consolidated layers of ash. On several trees the root system was distinctly two-level with a lower set of laterals developing below a well-consolidated layer of ash or pumice. Upper lateral roots commonly extended several metres from the tree base; lower laterals tended to be much shorter, and seldom reached more than 1 m from the tree.

Bark Structure

In tree sections unaffected by Armillaria root disease, bark on the lower bole consisted of a well-developed (10- to 30-mm-thick) rhytidome and a 2- to 4-mm layer of living phloem. The rhytidome consisted of layers of old dead phloem and cortex separated by up to eight successive necrophylactic periderms (Mullick & Jensen 1973) (sometimes described as secondary or wound periderms — Srivastava 1964) (Fig. 1a). In contrast, root bark consisted of a 2- to 4-mm-thick layer of phloem and cortex protected by the original exophylactic periderm (primary periderm), and no rhytidome. The division between stem and root bark was generally sharp, and the thick bole rhytidome sometimes appeared as a skirt around the base of the tree buried within the top layers of the forest floor. Periderm formation in *P. radiata* is similar to that in most other conifers. The first necrophylactic periderm on the bole forms when the bole reaches about 100 mm diameter, and after that new necrophylactic periderms form about once a year. On the major lateral roots the original exophylactic periderm is not replaced until much later, unless there are root injuries.

Description of Sample Trees

The 13 sample trees are described in Table 1. Individual trees ranged in dbh from 170 to 350 mm, and from 15 to 19 m in height. Live crown ratio ranged from 40% to 80%. Pruning occurred over the period 1990 to 1993, and the two high-pruned trees were pruned at least twice. Most trees exhibited a single patch of basal resinosis extending over 15% to 70% of the circumference. In some trees, however, there were two or more separate patches of resinosis separated by vigorous lateral roots and the stem bark above them. All these trees had a low live-crown ratio, and all were of below average height (Table 1). All but No. 9 were classified as having a fine branch habit.

Bole Infection by *Armillaria* species

Infection by *Armillaria* species was evident in basal cross-sections in both bark and wood. Infected bark exhibited irregular periderm formation (Fig. 1b). Individual sections of phloem isolated by periderms were often much wider than normal. Periderms in infected tissue were wider and darker, and often had layers of stone phellem (cork consisting of large, loosely packed, thick-walled cells) to the outside of regular phellem. Old dead phloem layers in infected bark retained a light pink colour and were resin soaked and hard compared to the medium brown and softer dead phloem of the normal rhytidome (Fig. 1a). When infected dead bark was split tangentially, small mycelial fans or impressions left by such fans in resin-

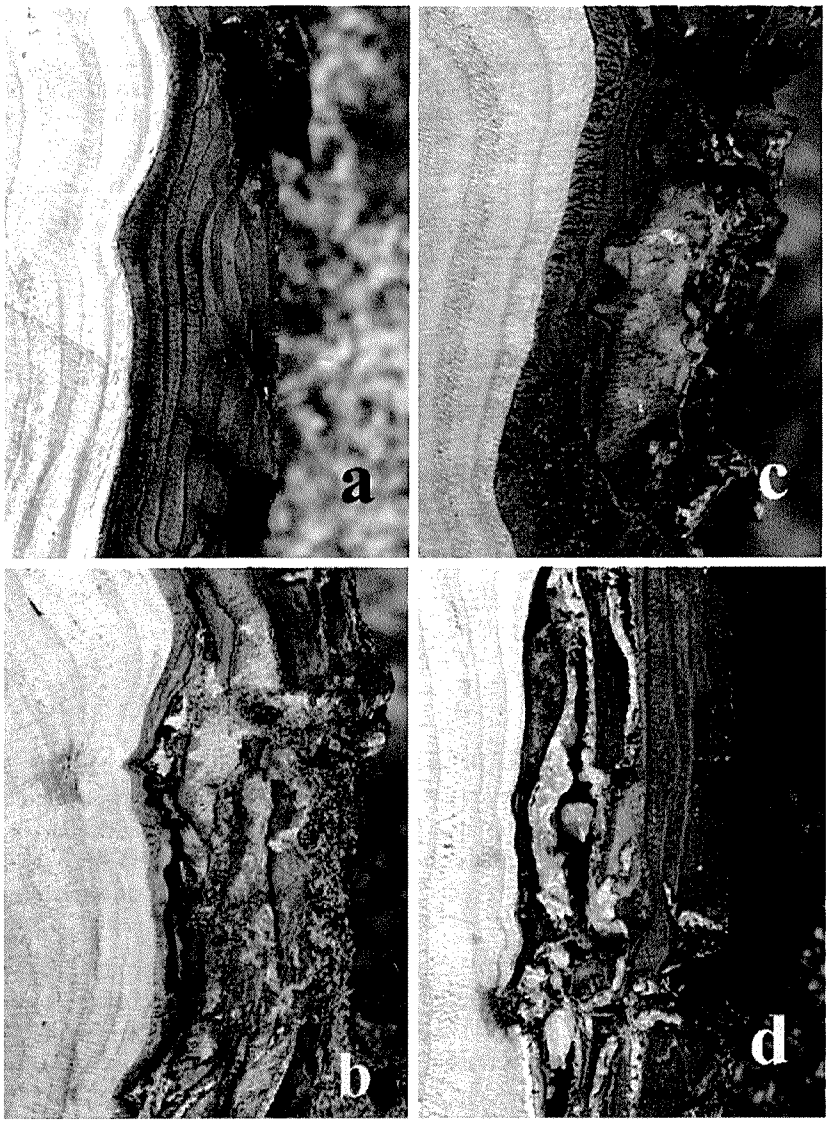

FIG. 1–Symptoms of infection by *Armillaria* species in the basal bark of boles of 13-year-old *P. radiata*.

(a) Normal basal bark. Only the inner layer of phloem is alive.
(b) Bark formed in the presence of *Armillaria* species. Note the pronounced inner periderms and the wide, resin-soaked layers of old phloem. The cambium and inner phloem are all alive in this section.
(c) The outer rhytidome exhibits characteristics of diseased bark while, at the upper end of the photograph, the inner bark appears normal.
(d) The outer rhytidome consists of normal bark while the inner bark is diseased.

soaked old phloem tissue were often evident (Fig. 2a). Finally, layers of old resin were found in the infected rhytidome of all trees.

TABLE 1–Sample tree characteristics and extent of resinosis caused by *Armillaria* species at the root collar.

Tree No.	Dbh (mm)	Height (m)	Live crown ratio	Resinosis (percentage of circumference per patch)*			Pruning†	Branch habit‡	Depression§
1	240	17.5	65	40	–	–	L	F	Y
2	350	18	65	25	10	–	L	M	Y
3	310	19	55	45	–	–	N	M	Y
4	180	15	50	20	–	–	N	F	Y
5	250	17.5	75	25	10	5	L	M	N
6	310	18.5	80	55	20	–	N	C	Y
7	280	16	65	30	10	–	L	M	Y
8	190	16.5	45	55	–	–	H	F	N
9	170	16	35	70	–	–	N	M	N
10	210	17	45	70	–	–	L	F	N
11	230	16	50	65	–	–	L	F	N
12	210	16.5	40	35	5	–	L	F	N
13	250	19	60	15	–	–	H	F	Y

* Up to three patches per tree
† Pruning: N = none; L = low, to <3m; H = high, to >3m.
‡ Branching habit: F = fine; M = medium; C = coarse.
§ Depression: Y = present; N = absent.

At the lateral edges of infected bark patches there was often evidence of tangential spread of the pathogen. At such locations the outer rhytidome consisted of normal bark whereas the inner rhytidome exhibited mycelial fans and characteristics of infected bark (Fig. 1d). In a few areas the reverse was observed, namely normal rhytidome adjacent to the living phloem and typical infected rhytidome on the outside (Fig. 1c). Presumably this indicated retreat of the pathogen.

The vascular cambium and the innermost layer of phloem lying underneath infected bark were usually alive. However, all but one tree (Tree No.3) showed evidence of penetration to the cambium. A typical example is shown in Fig. 2b. Here a longitudinal strip of cambium was killed during the 1992 dormant season. Some resin-soaking of the xylem directly beneath the dead cambial surface is evident. During the following growing season, a callus formed on both sides of the dead surface, leaving a strip of resin-soaked bark embedded in the wood. Two years later the healing was complete, and a continuous layer of living phloem covered the old wound. However, the bark to the outside of this phloem remained infected.

The infections, observed at the root collar of the 13 trees, that penetrated to and killed a section of vascular cambium are listed in Table 2 by date of invasion of the cambium and tangential extent. Of the 42 penetrations recorded, five exceeded 25 mm in tangential extent, and only one was >100 mm. Thirty-four of the penetrations occurred during the dormant season, as shown by the fact that the dead xylem surface was continuous with the end of an annual ring. The eight instances of penetration to the cambium during the growing season occurred on four trees, and in all of these the dead xylem surface was parallel to the annual ring, indicating very rapid penetration followed by arrest of further spread. Only a single

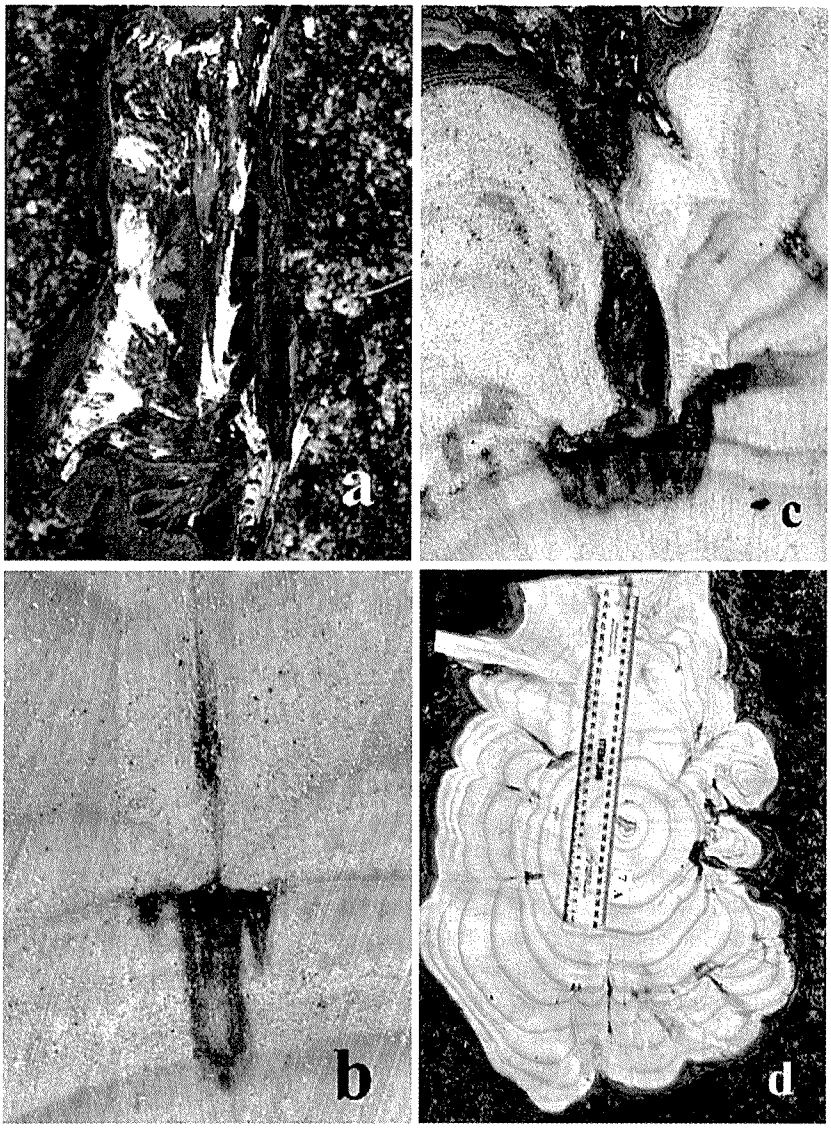

FIG. 2–Symptoms of infection by *Armillaria* species in bark and wood of the lower bole in 13-year-old *P. radiata*.

(a) Mycelial fan between bark scales. Note the impression left in the resin-soaked bark which remains long after the mycelium has died.

(b) A typical small, now healed-over penetration. Note the resin-soaking directly below the affected area, the coincidence of the dead surface with the end of the annual ring, and the form of subsequent annual rings showing how the wound was healed by callus formation.

(c) The single instance observed in which the original penetration by *Armillaria* species (the section parallel to the annual ring) was followed by callus formation, and during the next dormant season, the callus on the right was invaded and partially killed by the pathogen.

(d) Basal cross-section of Tree No. 7 showing several penetrations by *Armillaria* species.

TABLE 2–Tangential extent (mm) of cambial sections killed by *Armillaria* species, by date of penetration. Numbers in parentheses represent penetrations during the growing season starting in the year indicated. All others describe penetrations during the dormant season.

Tree No.	Year of penetration							
	1990	1991	1992	1993	1994	1995	1996	1997
1	–	–	–	–	–	–	–	7
2	–	–	–	–	–	(14) (5)	—	–
3	–	–	–	–	–	–	–	–
4	–	–	–	–	–	–	–	2, 3
5	4	–	2	–	4	3	3	–
6	–	–	20, 22, 5, 145	11, 10	–	–	–	–
7	(17)	15	27, 11	(22) (4) (4)	–	–	–	–
8	–	–	–	–	3, 5	6	–	–
9	–	–	–	–	–	–	12, 11, 17	2
10	–	–	–	–	40, 9, 6	–	3	–
11	–	–	–	–	–	(7)	3, 6	–
12	52 (7)	–	–	–	–	2	–	–
13	–	–	–	–	40, 37	–	–	–

extension of the killed area in the year after the initial invasion was observed (Fig. 2c). The basal disk of Tree No.7 is illustrated in Fig. 2d.

None of the penetrations extended >250 mm above or >300 mm below the base of the bole (as defined by the lower edge of the stem rhytidome), and most were located between major laterals and had a longitudinal extent of 100 to 150 mm. None of the penetrations extended into the upper surfaces of major roots, but they did occasionally extend a small distance (<100 mm) along the lateral or lower surface of major roots. The zone of resinosis on bark always extended beyond the area of xylem penetration, both tangentially and longitudinally. Many penetrations had healed completely, and were visible only in cross-section.

The xylem directly below old penetrations was resin-soaked to a depth of 10 to 30 mm. Resin-soaking extended longitudinally a few centimetres above and below the killed xylem surfaces. In a few trees (Trees No. 2, 7, and 12) small flecks of resin-soaking occurred throughout the xylem. Tree No.6 had a large pie-shaped area of resin-soaking that extended from the surface killed by *Armillaria* species to the pith, and was visible as a small area of resin-soaking around the pith 300 mm above the base of the tree.

The date of penetration varied within and between trees. The oldest occurred in 1990, and the most recent during the current growing season. The number of penetrations per tree varied from none (Tree No.3) to seven (Tree No.7). On any one tree, penetrations tended to occur over a 1- to 3-year period, but Tree No.5, with penetrations dating from 1990 to 1996, was a clear exception. Half of the eight growing season penetrations occurred on a single tree (Tree No.7). The number of trees examined in this study was insufficient to draw conclusions

about the relationship between tree characteristics and the number, date, and size of penetrations by *Armillaria* species.

Root Infection by *Armillaria* species

Twenty-four root infections were found, and the number of root infections per tree varied from zero (five trees) to six (Tree No.13) (Table 3). In about half the infections, the pathogen had penetrated to and killed part of the cambial surface. Penetrations to the xylem on roots occasionally extended over >50% of the root circumference. All penetrations occurred during the dormant season, and instances of further extensions in subsequent years were not observed. Healing by callus formation of such necrotic areas was slower than on stems: in several roots, calluses >5 years of age had not yet closed the wound. Only a single instance of complete girdling, resulting in death of the distal portion of the root, was recorded (the smallest root on Tree No.13). It may be that additional small roots killed by the pathogen were missed because they had deteriorated too far.

TABLE 3–Location and size of root infections.

Tree No.	Root diameter (mm)	Distance from bole (mm)	Length (mm)	Penetration to xylem
1	–	–	–	–
2	120	250	150	Y
	150	550	200	N
3	–	–	–	–
4	80	300	80	Y
	40	500	100	N
5	–	–	–	–
6	30	400	100	Y
	110	300	120	N
	80	250	100	N
7	100	400	200	Y
	50	450	100	N
	100	100	150	Y
	40	150	100	N
8	60	300	300	N
9	–	–	–	–
10	–	–	–	–
11	80	400	120	Y
	90	0	300	Y
	70	450	200	N
	40	300	100	N
	120	350	150	Y
12	60	250	200	Y
13	150	400	150	N
	10	120	200	Y
	40	0	250	N
	30	500	100	Y
	20	350	100	Y
	60	750	100	Y

DISCUSSION

Apparently *A. novae-zelandiae* can survive and spread in the inner rhytidome of 13-year-old *P. radiata* without entering the xylem. Occasional penetrations to the vascular cambium and xylem do occur, but these are quickly healed by the host tree. Most of the penetrations recorded in Table 2 had healed completely, although the bark lying to the outside of these old penetrations was still infected. Moreover, the area of infected bark (as determined by resinosis, abnormal bark formation, and the presence of mycelial fans) was always greater than the area of dead cambial surface. One consequence of this behaviour is that some infections by *Armillaria* species will probably survive to the next rotation. After harvest, the xylem of stumps and roots will likely be invaded rapidly and form the inoculum base in the next stand and the origin of mortality during the first few years of such stands

Host reactions involving callus formation are a common response to invasion by *Armillaria* species (Morrison, Merler & Norris 1991). However, the reaction observed in these trees was unusually strong and effective, particular in boles. The strongly pathogenic species *A. ostoyae* (Romagn.) Herink on conifers in western North America can be contained by host reactions involving callus formation, but many infections continue to spread from year to year until the host is girdled and killed (Morrison, Williams & Whitney 1991). Such continued development was not seen on the 13-year-old trees in this study. Only a single instance of continued spread for a second year was observed in boles, and that infection was effectively contained by a callus formed in the following year. On roots all penetrations that were observed occurred in a single year, and continued spread was not observed. However, it is possible that on small roots continued spread over time may occur, with older parts of these roots deteriorating rapidly. The methods employed in this study could have missed such roots.

Armillaria species appear to stimulate the vascular cambium to produce extra phloem, and induces host reactions of resinosis and the production of well-developed necrophylactic periderms. These periderms apparently serve to prevent invasion of the inner living phloem and cambium most of the time. Occasionally, however, the pathogen is able to breach these defense structures and invade and kill a section of cambium. Full characterisation of these periderms and of the substantial layers of phloem will require further work. The pathogen appears to be able to invade previously healthy bark by advancing in the inner rhytidome. In Fig. 1d the outer rhytidome consists of normal layers of old phloem and periderms, while the inner, more recently formed rhytidome exhibits the characteristics of infected bark. The pathogen may also retreat; for instance, in Fig. 1c the outer rhytidome consists of infected bark and the inner of normal bark. These interpretations support earlier findings that the area of infected bark can either increase or decrease over time (MacKenzie 1987).

Uninfected roots on trees this age do not have a rhytidome. However, root infections by *Armillaria* species always exhibited necrophylactic periderms and resin-impregnated old phloem with small mycelial fans, similar to that found on boles. Presumably the formation of such periderms is stimulated when the pathogen first contacts the root surface, or perhaps penetrates the exophylactic periderm.

There may be some question whether all the cases of "penetrations to the cambium" were in fact caused by *Armillaria* species as wounding of all kinds could produce a similar host reaction. However, the bark to the outside of the wound was clearly infected, and there was

no evidence of bark removal, which would be necessary in order for a scarring wound to reach the xylem. No attempts were made to isolate the pathogen from wood.

The earliest penetrations recorded occurred in 1990. It is likely that penetrations earlier than 1990 (when trees were <5 years old) resulted in death of the host. Inspection of trees killed by *Armillaria* species in three young plantations in Kaingaroa Forest revealed that the pathogen invaded and girdled the trees near the root collar, killing the vascular cambium and living phloem in a single dormant season (evidence: the last xylem ring was complete). Thus it appears that invasion by *Armillaria* species is largely restricted to the dormant season, and that during the subsequent growing season further spread is stopped and healing of the wound by callus formation is initiated. On small trees *Armillaria* species have the ability to girdle the tree in a single season, and that may be the reason why mortality attributable to Armillaria root disease is common in young plantations but rare in trees >8 years of age (Hood 1989).

During excavation special attention was paid to possible entry points for *Armillaria* species into tree boles. On young, recently killed trees it is usually possible to identify the root along which the pathogen advanced to the tree base, but in this study no such roots were found, except possibly in Tree No.13. At least two explanations come to mind. It may be that bole infections were caused by direct infection via rhizomorphs arising from inoculum from the previous stand. In about half the infected trees, old dead roots and stumps were found in close proximity to the tree base, but in the other half such inoculum was not evident. It may also be that the pathogen reached the tree base via a small root several years ago, and that these small roots had deteriorated to the point that they could no longer be recognized at the time of inspection. Most likely both types of pathway occurred.

Root infections described in this study occurred almost exclusively in the A horizon and within 250 mm of the mineral soil surface. Contacts between trees and between the current crop and old inoculum are much more probable here than lower down as the upper lateral roots extended several metres out from the tree base and were in contact with roots of neighbouring trees. Lower roots seldom grew more than a metre from the tree base, and there was no root contact among trees at this level.

The impact of Armillaria root disease on volume increment and wood quality is always a central practical concern (Kimberley *et al.* 2002). The descriptions and interpretation of *Armillaria* species on young *P. radiata* presented above may shed some light on this issue. Undoubtedly the extra tissues formed in infected bark, as well as resinosis, represent a drain on host carbohydrate reserves. However, the total dry weight of extra materials so produced is likely to be less than 1% of the dry weight of bole xylem formed by the tree each year. Also, the degree of infection found in this study is judged to have at worst a small effect on the functioning of the tree translocation systems. No large roots were girdled and killed, and losses of functional xylem area due to invasion of xylem by *Armillaria* species were quickly restored by compensatory growth during the following seasons. The cross-sectional area of xylem killed by the pathogen was seldom more than 10% of the functional xylem. Excessive production of phloem under infected rhytidome tissue implies that some stimulus arising from the parasite reaches the vascular cambium. It may well be that the living phloem underneath infected bark produces anti-fungal agents such as various phenolics, which typically require substantial energy expenditures by the host. Garraway *et al.* (1991) discussed such reactions and suggested that evidence for their occurrence was uncertain.

Loss of carbohydrate attributable to excessive rates of metabolism in tissues around the infected areas may well represent the largest energy drain on the host.

In summary, it appears that *Armillaria* species on young *P. radiata* (in this study almost certainly *A. novae-zelandiae*) lives primarily as a bark saprophyte. Presumably it derives the necessary nutrients and energy from dead phloem recently isolated by host periderms. It appears that the vascular cambium responds to the presence of the pathogen in the inner rhytidome by excessive phloem production, and necrophylactic periderms produced in such phloem are particularly well developed. These host reactions produce distinct symptoms in the rhytidome, which may be useful for recognition of infection by *Armillaria* species. On occasion the parasite will penetrate to the cambium and kill it. This happens mostly during the dormant season, and such penetrations induce a strong host response involving callus formation that quickly heals the wound. Of course, the same species of *Armillaria* on different hosts, and possibly on older *P. radiata*, does live primarily in wood where it survives in domains bordered by zone lines. It is not immediately clear why bark infection is restricted to the very base of the tree. In 13-year-old trees a well-developed rhytidome extends several metres up the tree. Perhaps this tissue is too dry for *Armillaria* species except where it is in contact with the forest floor.

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REFERENCES

GARRAWAY, M.O.; HÜTTERMAN, A; WARGO, P.M. 1991: Ontogeny and physiology. Chapter 3, Pp. 21–47 *in* Shaw, C.G. III; Kile, G.A. (Ed.) " Armillaria Root Disease". *United States Department of Agriculture Forest Service, Agriculture Handbook 691.*

HEWITT, A.E. 1993: New Zealand Soil Classification. *Landcare Research New Zealand Limited, Landcare Research Science Series No.1.* 133 p.

HOOD, I.A. 1989: Armillaria root disease in New Zealand forests. *New Zealand Journal of Forestry Science 19*: 180–197.

HOOD, I.A.; SANDBERG, C.J. 1993a: *Armillaria* populations in a *Pinus radiata* plantation on a former indigenous rainforest site. *New Zealand Journal of Forestry Science 23*: 62–77.

———1993b: *Armillaria* – a hidden disease of *Pinus radiata*. *New Zealand Forestry 38*: 29–32.

HOOD, I.A.; KIMBERLEY, M.O.; GARDNER, J.F.; SANDBERG, C.J. 2002: Armillaria root disease of *Pinus radiata* in New Zealand. 3: Influence of thinning and pruning. *New Zealand Journal of Forestry Science 32(1)*:

KIMBERLEY, M.O.; HOOD, I.A.; GARDNER, J.F. 2002: Armillaria root disease of *Pinus radiata* in New Zealand. 6: Growth loss. *New Zealand Journal of Forestry Science 32(1)*:

MacKENZIE, M. 1987: Infection changes and volume loss in a 19-year-old *Pinus radiata* stand affected by Armillaria root rot. *New Zealand Journal of Forestry Science 17*: 100–108.

MORRISON, D.J.; MERLER, H.; NORRIS, D. 1991: Detection, recognition and management of Armillaria and Phellinus root diseases in the Southern Interior of British Columbia. Canada – British Columbia Partnership Agreement on Forest Resource Development: FRDA II. *British Columbia Ministry of Forests, Victoria, B.C., FRDA Rep. 179.* 24 p.

MORRISON, D.J.; WILLIAMS, R.E.; WHITNEY, R.D. 1991: Infection, disease development, diagnosis and detection. Chapter 5, Pp. 62–75 *in* Shaw, C.G. III; Kile, G.A. (Ed.) " Armillaria Root Disease". *United States Department of Agriculture Forest Service, Agriculture Handbook 691.*

MULLICK, D.B.; JENSEN, G.D. 1973: New concepts and terminology of coniferous periderms: Necrophylactic and exophylactic periderms. *Canadian Journal of Botany 51*: 1459–1470.

RIJKSE, W.C. 1988: Soils of the Kaingaroa Plateau, North Island, New Zealand. *New Zealand Soil Bureau District Office Report RO14.*

SELF, N.M.; MacKENZIE, M. 1995: Intensive site preparation to control Armillaria root disease in second-rotation *Pinus radiata*. *New Zealand Journal of Forestry Science 25*: 111–116.

SELF, N.M.; HOOD, I.A.; KIMBERLEY, M.O.; SHU, Q.L.; GARDNER, J.F. 1998: Distribution and incidence of Armillaria root disease in *Pinus radiata* plantations throughout New Zealand. Pp. 137–147 *in* Delatour, C.; Marçais, B.; Guillaumin, J.-J.; Lung-Escarmant, B. (Ed.) Proceedings of the Ninth International IUFRO (Working Party S2.06.01) Conference on Root and Butt Rots, 31 August – 7 September 1997, Carcans-Maubuisson, France. *Institut National de la Recherche Agronomique (INRA) Editions Les Colloques No. 89.*

SHAW, C.G. III; CALDERON, S. 1977: Impact of *Armillaria* root rot in plantations of *Pinus radiata* established on sites converted from indigenous forest. *New Zealand Journal of Forestry Science 7*: 359–373.

SHAW, C.G. III; TOES, E.H. 1977: Impact of Dothistroma needle blight and Armillaria root rot on diameter growth of *Pinus radiata*. *Phytopathology 67*: 1319–1323.

SHAW, C.G. III; SIJNJA, D.; MacKENZIE, M. 1976: Toetoe (*Cortaderia fulvida*) – A new graminaceous host for *Armillaria* root rot. *New Zealand Journal of Forestry 21*: 265–268.

SRIVASTAVA, L.M. 1964: Anatomy, chemistry and physiology of bark. *International Review of Forestry Research 1*: 202–277.

VUCETICH, C.G.; PULLAR, W.A. 1964: Stratigraphy and chronology of Late Quaternary volcanic ash in Taupo, Rotorua, and Gisborne districts. Part 2. *New Zealand Geological Survey Bulletin 73*: 43–88.

VUCETICH, C.G.; LEAMY, M.L.; POPPLEWELL, M.A.; URE, J.; TAYLOR, C.R.; WILL, G.M.; SUTTON, J.A.; BLAKEMORE, L.C. 1960: Soils, forestry and agriculture of the northern part, Kaingaroa State Forest and the Galatea Basin. *New Zealand Department of Scientific and Industrial Research, New Zealand Soil Bureau Bulletin 18.*

WILL, G.M.; KNIGHT, P.J. 1968: Pumice soils as a medium for tree growth 2. Pot trial evaluation of nutrient supply. *New Zealand Journal of Forestry 13*: 50–65.