



How do *Phytophthora* spp. de Bary kill trees? †

Elaine M. Davison

Department of Environment and Agriculture, Curtin University, GPO Box U1987, Perth, WA 6845, Australia

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e.davison@curtin.edu.au

Abstract

Phytophthora spp. de Bary are being increasingly recognised as pathogens that cause tree death, without necessarily having any clear understanding of how this happens. Suggested mechanisms include:

- extensive fine-root necrosis especially on wet or drought prone sites, leading to reduced water uptake, crown decline and death, e.g. *Phytophthora quercina* T. Jung infection of European oaks;
- root and stem cankers resulting from phloem invasion and cambial death, leading to death of basal buds and carbon starvation of the root system, e.g. *Phytophthora alni* Brasier & S.A. Kirk infection of alders;
- xylem invasion, leading to reduced conduction, hydraulic failure and death, e.g. *Phytophthora ramorum* Werres, De Cock & Man in 't Veld infection of tanoaks; and
- hormonal imbalance and/or damage from toxins, e.g. *Phytophthora cinnamomi* Rands infection of eucalypts.

These possible mechanisms are reviewed, together with different hypotheses of why trees die, and the predisposing environmental stresses that contribute to tree death. Extensive xylem invasion provides a mechanistic explanation of how death occurs, but is the least frequently reported symptom of *Phytophthora* infection.

Keywords: cankers; carbon starvation; fine-root necrosis; hydraulic failure; xylem invasion.

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Introduction

Phytophthora spp. de Bary are being increasingly recognised as pathogens that cause the death of trees and other woody plants in natural ecosystems. Some of these pathogens such as *Phytophthora cinnamomi* Rands and *Phytophthora multivora* P.M. Scott & T. Jung are soil borne and infect roots; others such as *Phytophthora ramorum* Werres, De Cock & Man in 't Veld are aerial pathogens infecting trunks, branches, twigs and leaves. Although some hosts die rapidly

there is no clear understanding of how this happens, and why other hosts are apparently little affected. Pathogenicity experiments demonstrate the ability to infect seedlings, saplings, roots and branches, but the ability to infect a relatively small plant is not necessarily the ability to kill a large tree.

Trees are great survivors. They have pre-formed physical and chemical barriers that minimise invasion

by pests and pathogens, and contain large water and carbon reserves that enable them to respond to damage and survive periods of stress (Manion, 1981), although these reserves may be insufficient to enable them to survive extreme climatic events (McDowell et al., 2008). So, how much damage can trees sustain before they die, and where are they most vulnerable?

Plant pathologists need good explanations of how *Phytophthora* spp. kill trees. Explanations proposed in the past are that death results from fine root necrosis, stem and root cankers, xylem invasion leading to hydraulic failure, or disruption of host physiology caused by toxins, elicitors and hormone imbalance. These explanations are not mutually exclusive. What is the evidence for these different explanations? What testable predictions can be developed that will refine our understanding of how and why death occurs?

This paper first considers the different types of infection that have been suggested (fine root necrosis, cankers, xylem invasion and disruption of host physiology). It then briefly outlines different hypotheses of why trees die, and then considers the many stress factors that are commonly associated with infection and death. Finally it makes some suggestions about additional work that would aid our interpretation of how *Phytophthora* spp. kill trees.

Suggested ways in which *Phytophthora* spp. can cause tree death

Fine root necrosis

The peripheral root system of trees is essential for the uptake of water and nutrients. Death of fine roots, caused by soil borne *Phytophthora* spp., has been repeatedly demonstrated in pathogenicity experiments (e.g. Jönsson, 2004; Jung et al. 1996; Newhook, 1959; Robin & Desprez-Loustau, 1998; Vettraino et al., 2003). Decline, dieback and reduced growth are the types of symptoms that would result from such infections

in mature trees, if they are sufficiently extensive and persist long enough for the reduction of stored water and carbon reserves.

Death resulting from fine-root necrosis caused by *Phytophthora cinnamomi* has been used to account for death or symptoms of decline in a number of tree species, notably root rot of avocados (*Persea americana* Mill.) in California (Wager, 1942; Zentmyer & Thorn, 1967), little-leaf disease of *Pinus echinata* Mill. in south eastern USA (Campbell, 1949), and death of *Pinus radiata* D. Don shelterbelts in New Zealand (Newhook, 1959). Fine-root necrosis has also been suggested as the mechanism by which *Phytophthora cinnamomi* killed *Eucalyptus marginata* Sm. (jarrah) trees in Western Australia. This latter problem was known as "jarrah dieback", and referred to groups of *E. marginata* trees that died suddenly for no apparent cause. Podger et al. (1965) found that these *E. marginata* deaths, and deaths of many mid and understorey forest species such as *Banksia grandis* Willd., were associated with site infestation by *Phytophthora cinnamomi*. However, there was no evidence of cankers or rotting of structural roots in affected *E. marginata* trees. Also, *Phytophthora cinnamomi* was isolated from soil and other forest species more frequently than from *E. marginata* (Table 1) so it was concluded that the *E. marginata* deaths resulted from fine-root necrosis (Zentmyer, G. A. University of California, Riverside, unpublished report¹; Batini & Hopkins, 1972).

Based on the early circumstantial evidence described above, plus field surveys (Pratt & Heather, 1973) and field inoculations (Malajczuk et al., 1977), it was generally accepted, during the 1970s, that eucalypt decline and deaths in Australia resulted from a fine root necrosis caused by *Phytophthora cinnamomi* (Zentmyer, 1980). Site infestation, as demonstrated by soil baiting, was associated with major changes in the health of many species of eucalypts (Marks et al., 1972; Marks et al., 1975; Weste & Taylor, 1971). There were, however, difficulties in demonstrating extensive fine-root necrosis on infested sites, although recovery

TABLE 1: Frequency of the isolation of *Phytophthora cinnamomi* from various hosts in the jarrah forest of Western Australia. Isolation methods: direct plating onto 3P or P₁₀VP agar or by lupin baiting (Erwin & Ribeiro 1996). Data from the CSIRO isolation books, May 1965 to December 1968, viewed with permission February 1980.

Host	Sample size	Isolation frequency (%)
<i>Eucalyptus marginata</i> (jarrah)	100	5
<i>Banksia grandis</i>	121	29
Other plants	546	19
Soil samples	1163	23

¹ Unpublished report to the Forests Department Western Australia, May 1968. Included as Appendix I in Wallace, W. R. (1969). [Unpublished report]. Progress report on jarrah dieback research in Western Australia. Perth, Australia: Forest Department of Western Australia.

from fine roots could be increased by summer irrigation (Shea et al., 1980; Shea & Dell, 1981). In fact, the only consistent symptom that has been found is tylosed sapwood in the roots, a symptom now known to be caused by waterlogging (Davison & Tay, 1985).

The association between site infestation with *Phytophthora* spp. and declining stands of European oaks has been demonstrated by many workers (Balci & Halmschlager, 2003; Brasier et al., 1993; Gallego et al., 1999; Jung et al., 1996; Jung et al. 2000). Many oak species are affected, including *Quercus cerris* L., *Quercus ilex* L., *Quercus petraea* (Matt.) Liebl., *Quercus robur* L., and *Quercus suber* L.. Jung and Blaschke (1995) showed that there were far fewer fine roots on root segments from declining *Q. robur* trees than from healthy trees; fine root necrosis caused by *Phytophthora quercina* T. Jung and other *Phytophthora* spp., is believed to be a major cause of this decline (Jönsson, 2004; Jung et al. 2000; Vettraino et al., 2002). However, stem and root cankers caused by *Phytophthora* spp. also occur in trees on some sites and contribute to symptoms of branch dieback, production of epicormic shoots and crown transparency.

Cankers of stems and roots

A canker is “a sunken necrotic lesion of main root, stem or branch arising from disintegration of tissues outside the xylem cylinder ...” (Federation of British Plant Pathologists, 1973). Infection will lead to disruption of the movement of photosynthate and other substances through the phloem resulting in the death of dormant buds. If the dead bark is shed and the sapwood exposed, the canker may be an entry point for wood-rotting fungi. Symptoms that will result from large or numerous cankers include: crown transparency; branch dieback; reduced growth rate; and slow decline.

Phytophthora spp. are important causes of cankers on branches, trunks, root collars and roots of woody plants. Tree death results from extensive cankers, especially at the root collar. Many of these infections result in bleeding cankers, so called because they produce a tarry exudate. Examples of canker diseases include stripe canker of cinnamon trees caused by *P. cinnamomi* (Rands, 1922), trunk cankers and collar rot of apple caused by *Phytophthora cactorum* (Lebert & Cohn) J. Schröt. (Baines, 1939), and collar rots of *Castanea sativa* Mill. and *Fagus sylvatica* L. caused by *Phytophthora cambivora* (Petri) Buisman, *P. cinnamomi* and *Phytophthora syringae* (Kleb.) Kleb. (Day, 1938). More recent examples include: phytophthora disease of *Alnus glutinosa* (L.) Gaertn. and other *Alnus* spp. Mill. caused by *Phytophthora alni* Brasier & S.A. Kirk (Gibbs et al., 1999; Jung & Blaschke, 2004); ink disease of *Quercus robur* caused by *P. cinnamomi* (Robin, 1992), scion cankers in almond caused by *P. citricola* Sawada (Browne & Viveros, 1999); and trunk canker

of *Quercus* spp. and *Lithocarpus densiflorus* (Oerst.) Rehder caused by *P. ramorum* (Rizzo et al., 2002). Infection is mainly in the bark and cambium, although there may be a very narrow band of discoloration in the underlying sapwood (Baines, 1939; Day, 1938; Rands, 1922).

Pathogenicity tests using wound inoculation of large stems and roots result in large and conspicuous lesions in the inner bark and cambium that are limited by host responses (Tippett et al., 1983; Tippett et al., 1985). Lesion lengths resulting from such experiments are often used to compare the pathogenicity of different *Phytophthora* spp. or isolates, or to compare the susceptibility of different hosts (e.g. Broadbent et al., 1996; Gabor & Coffey, 1991; Moralejo et al., 2009; Robin & Desprez-Loustau, 1998; Utkehede & Quamme, 1988).

Xylem invasion and hydraulic failure

Although some infected trees decline over many years, some appear to die almost “overnight”, with no prior symptoms that the tree is under stress (Rizzo et al., 2002; Tainter et al., 2000). Sudden tree death is a spectacular and frightening occurrence. Wilting and death of the crown implies hydraulic failure resulting from blocked xylem vessels which could be the result of extensive invasion of the sapwood.

Although the sapwood underlying phytophthora cankers is sometimes discoloured (Baines, 1939; Day, 1938; Rands, 1922; Rizzo et al., 2002) there have been very few records of isolations specifically from the xylem of living trees. Brown and Brasier (2007) isolated several *Phytophthora* spp., from discoloured xylem underlying phloem cankers, with invasion by *P. ramorum* occurring up to 25 mm into the wood of *Q. cerris* and *Acer pseudoplatanus* L.. Greslebin and Hansen (2010) were able to isolate *Phytophthora austrocedri* Gresl. & E.M. Hansen from discoloured sapwood internal to phloem lesions in *Austrocedrus chilensis* (D. Don) Florin & Boutleje. Further evidence of xylem invasion is given by Parke et al. (2007) and Collins et al. (2009), who showed that *P. ramorum* is able to invade the sapwood of *Lithocarpus densiflorus*, resulting in tylosed xylem vessels and reduced hydraulic conductivity. *Phytophthora ramorum* was isolated from, or detected in, both symptomatic and symptomless sapwood.

The extent of xylem invasion in healthy hosts varies between species (Davison et al., 1994). In *Eucalyptus marginata*, *Phytophthora cinnamomi* invades a narrow band of sapwood, forming an inapparent infection (“any infection that gives no overt signs of its presence”, Federation of British Plant Pathologists, 1973), and persisting for at least 3 months as a latent infection (“an inapparent infection that is chronic and in which a certain host-parasite relationship is established”,

Federation of British Plant Pathologists, 1973). In *Pinus radiata*, however, *Phytophthora cinnamomi* invaded radially into the sapwood, and then was rapidly contained within an area of non-conducting wood and died out (Davison et al., 1994). A much more extensive inapparent infection occurs in the sapwood of *Banksia grandis* when inoculated in summer (Figure 1, Davison et al., 1991). Such extensive, inapparent infections may be common in *Banksia* spp. because Smith et al. (1997) also recorded xylem invasion by *P. cinnamomi* in inoculated stems of *Banksia brownii* Baxter ex R.Br..

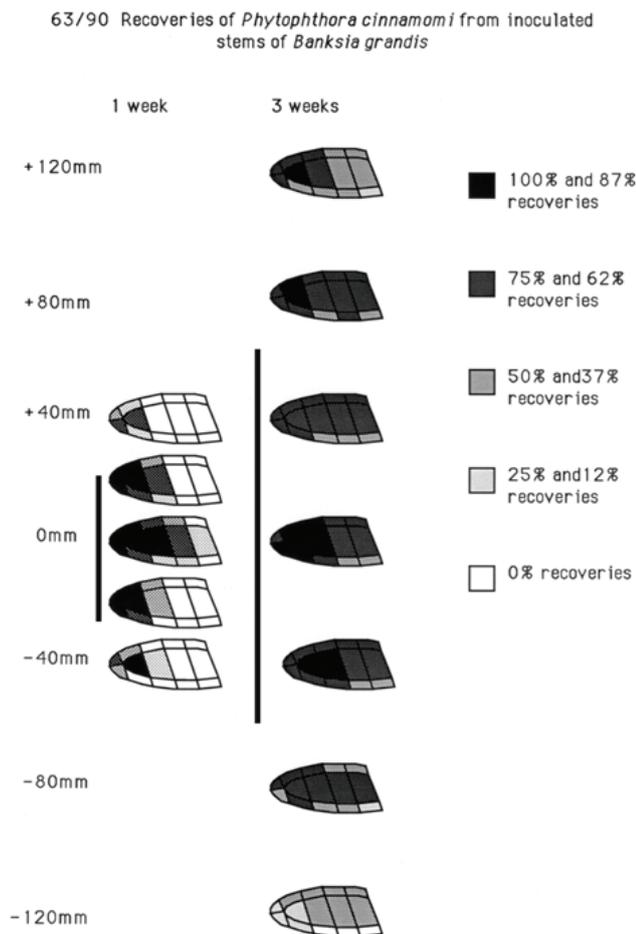


FIGURE 1: Inapparent infections in the bark and wood of *Banksia grandis* stems in summer, 1 and 3 weeks after wound inoculation by *Phytophthora cinnamomi*. The stem was cut into discs at different distances from the inoculation point (40 mm at week 1 and 120 mm at week 3) and each disc split into 5 mm wide strips and the bark and wood plated separately; there were eight replicate stems at each harvest time. The mean extent of the apparent infection in the bark is shown by the vertical bar. There were no symptoms of discolouration in the wood. From Davison et al. (1991).

These observations show that xylem invasion occurs in some pathosystems but such infections may not be immediately apparent because there is either no or very limited discolouration of the sapwood. Xylem invasion in live trees may, therefore, be more widespread than is generally recognised and may be a precursor to xylem dysfunction leading to hydraulic failure and tree death.

Effects on host physiology – hormonal imbalance

During the 1980s there were several investigations to try to understand why susceptible eucalypts growing on sites infested with *Phytophthora cinnamomi* developed water deficits and died (Dawson & Weste, 1982; Weste, 1980). Dawson and Weste (1984) showed that when root tips of seedlings of *Eucalyptus sieberi* L.A.S.Johnson (susceptible) and *Eucalyptus maculata* Hook. (resistant) were inoculated with *P. cinnamomi*, hydraulic conductivity in the root system of the susceptible plants was reduced significantly. This resulted in symptoms of water stress, even though less than one sixth of the root system was infected. As histological examination failed to show xylem damage (Cahill et al., 1986), Dawson and Weste (1984) suggested that this reduction in hydraulic conductivity might be the result of a pathogen-mediated hormonal imbalance.

Cytokinins are synthesised in root tips, so diseased plants with fewer healthy root tips than uninfected plants are likely to have reduced cytokinin levels in the xylem. Cahill et al. (1985; 1986) showed that infection of *Eucalyptus marginata* roots by *Phytophthora cinnamomi* resulted in a significant decrease of zeatin-type cytokinins in xylem exudate within 3 days, whilst there was no comparable decrease in the field tolerant *Corymbia calophylla* (Lindl.) K.D.Hill & L.A.S.Johnson. They suggested that this decrease, which preceded the reduction in hydraulic conductivity, would trigger an increase in abscisic acid (ABA) concentration in buds and leaves, resulting in the typical droughting response of stomatal closure. The ultimate effect would be wilting and death. Further experiments were conducted by Maurel et al. (2004) who investigated the role of chemical signals produced in roots on stomatal conductance and transpiration of *Castanea sativa* saplings that were either infected with *P. cinnamomi* or droughted. They showed that water uptake, stomatal conductance and predawn leaf water potential were lower in infected and droughted plants, and there was a trend of decreasing conductance with increasing concentration of ABA in xylem sap collected from the upper part of the main stem. They did not attempt to determine however, whether there was an increase in the delivery rate of ABA from roots to shoots, or whether their observed increase resulted from reduced dilution of ABA as a consequence of slower transpiration (Jackson, 2002).

Effects on host physiology – damage from toxins

Weste and Cahill (1982) found that there was increased ion leakage from seedling roots of a number of Australian native plants following infection by *Phytophthora cinnamomi*. They suggested that this could be caused by a toxin. Some *Phytophthora* spp. produce mobile toxins that cause physiological injury resulting in symptoms of leaf necrosis and wilting (Devergne et al., 1992; Grant et al., 1996; Kamoun, 2006; Mezzetti et al., 1994). Woodward et al. (1980) suggested that if fungal toxins are involved in pathogenesis, this would explain how a relatively small amount of infected tissue might account for tree death. They showed that water soluble β -glucans produced in culture filtrates of *P. cinnamomi*, *Phytophthora cryptogea* Pethybr. & Laff. and *Phytophthora nicotianae* Breda de Haan are toxic to seedlings of the field-susceptible *E. sieberi*, inducing wilting, however, they also induced similar symptoms in the field resistant *Eucalyptus cypellocarpa* L.A.S.Johnson.

Another group of potentially toxic molecules that are released into culture filtrates are elicitors. These are peptides that are xylem mobile and can cause a hypersensitive reaction in tobacco leaves (Devergne et al., 1992; Kamoun et al., 1993; Ricci et al., 1992). Maurel et al. (2004) investigated the effect of the elicitors parasiticein and cryptogein, which are similar to cinnamomin produced by *Phytophthora cinnamomi*, on leaf physiology of *Castanea sativa*. They found that while both ABA and root infection induced a rapid decrease in stomatal conductance, the elicitors did not. Somewhat similar results were obtained by Fleischmann et al. (2005), using the *Phytophthora citricola* – *Fagus sylvatica* pathosystem, who found that although addition of citricolin induced a hypersensitive reaction in tobacco cell suspensions there was no comparable response in suspensions of beech cells.

Phytophthora ramorum produces at least two physiologically active elicitors (Manter et al., 2007). When infiltrated into leaves of both woody hosts (*Lithocarpus densiflorus*, *Umbellularia californica* (Hook. & Arn.) Nutt., *Rhododendron macrophyllum* D. Don ex G. Don) and a herbaceous non-host (tobacco) these elicitors cause a decrease in chlorophyll fluorescence and an increase in hydrogen ion (H^+) uptake and ethylene production, which are interpreted as a hypersensitive-like response. Manter et al. (2007) suggest that differences between species results from the timing and degree of this hypersensitive response.

Results from these physiological studies on seedlings have been extremely valuable in dissecting out the events that follow root infection and that lead to wilting and death. However, it remains unclear as to whether these changes are sufficiently large and long lasting to result in the death of mature trees.

Events that precede rapid tree death

Tree death is considered “sudden” if no symptoms are visible prior to mortality. Observations in *Eucalyptus marginata* trees, however, indicate that severe water deficits precede sudden death (Shea et al., 1982). This has been confirmed by observations of *E. marginata* trees that were part of growth and peneology trials (Crombie & Tippett, 1990; Davison, 1994; Davison & Tay, 1995a). An example is shown in Figure 2. In this example, and others given in Davison and Tay (1995a) the death of the foliage occurred within a four-week period during summer and autumn, the time between successive observations. The death of the foliage was preceded by sustained shrinkage for several weeks or months at 1.3 m, as shown by dendrometer band measurements, indicating that the trunk was drying out. Figure 2 shows that maximum girth occurred 18 months before the crown died. In several trees new leaves were present in the crown immediately before foliar death. It is likely that hydration of foliage immediately prior to death resulted from the removal of stored water in the wood, rather than from conduction of water from the roots. It is assumed that xylem dysfunction in the roots and trunk must have preceded foliar death, with the tree ultimately dying from hydraulic failure.

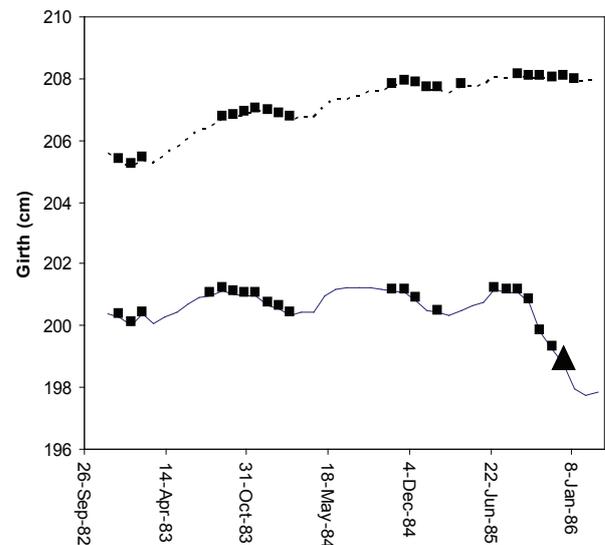


FIGURE 2: Changes in girth (measured with a dendrometer band at 1.3 m height) and leaf production of two *Eucalyptus marginata* trees located at a perched-water-table site at Ross Block, measured between November 1982 and April 1986. Site details are given in Davison and Tay (1989). Tree R10 (solid line) was on a site infested by *Phytophthora cinnamomi*, tree R22 (dashed line) was on an adjacent, uninfested site. Squares indicate when new leaves were present in the crown, the triangle indicates when the foliage died. (Modified from Davison (1994). Reproduced with permission from the Royal Society of Western Australia).

Hypotheses relating to tree death

Stress factors

Stress factors, which can be both abiotic and biotic, are believed to be important predisposing factors in the decline and death of many trees. They are frequently cited as being important in phytophthora diseases (Brasier et al. 1993; Jung, 2009; Newhook, 1959; Portela et al., 1999).

Houston (1987) proposed that diebacks and declines are initiated by these predisposing factors, and facultative pathogens are able to attack and invade hosts that would normally be able to contain such infections. Although environmental stresses alone can kill trees, those that survive usually recover once the stress has ceased. Direct and indirect effects of these environmental stresses are on the uptake and movement of water and minerals, photosynthesis, and the storage and movement of carbohydrates. These effects in turn reduce the host's ability to compartmentalise damaged tissues (Shigo, 1985) resulting in increased damage by pests and pathogens.

Manion's decline-disease theory is similar (Manion, 1981). He proposed that three different types of stresses (predisposing, inciting and contributing) result in dieback and death. These abiotic and biotic stresses are somewhat interchangeable, depending on when they occur in the chronology of the decline. Predisposing stresses are long term factors such as climate, moisture, nutrition and host genotype while inciting stresses are short term factors such as frost, drought, insect defoliation and mechanical injury. Contributing factors are pests and pathogens that are ubiquitous and usually present at low levels on healthy trees, but which multiply on stressed trees that have impaired ability to respond to damage.

Population dynamics

A very different approach was suggested by Mueller-Dombois et al. (1983). They proposed that stand level dieback of *Metrosideros polymorpha* Gaudich. forests in Hawaii, and group deaths of other tree species in North America, are a result of cohort succession, and regard synchronised tree declines as a problem of tree population dynamics, not pathology. Gerrish (1990) suggested that a credible physiological mechanism for cohort senescence is carbon starvation resulting from a decline in the ratio of photosynthesis to respiration as a tree ages. Thus a mature tree, in comparison with a young tree, would have less available resources for mobilisation during periods of stress. However the explanation that *Metrosideros* decline results from cohort senescence is not universally accepted. Hodges et al. (1986) suggest that it can be interpreted as a typical decline disease *sensu* Houston, in which predisposing factors include poor soil drainage,

drought and nutrition, resulting in trees which are less able to respond to attack by *P. cinnamomi*, *Armillaria mellea* (Vahl) P. Kumm. and the borer *Plagithmysus bilineatus* Sharp, 1896.

Drought and carbon starvation

The different responses to drought, that affect the ultimate survival of trees, have been analysed by McDowell et al. (2008). They suggest that isohydric trees that maintain large margins of hydraulic safety by regulating water loss through stomatal closure, are vulnerable to hydraulically mediated carbon starvation, and, therefore, have reduced ability to respond to biotic agents. Anisohydric trees that continue to transpire during drought, are less likely to die from carbon starvation, but more likely to die from hydraulic failure. In pinyon-juniper woodlands, pinyon (*Pinus edulis* Engelm.) is isohydric while juniper (*Juniperus monosperma* Sarg.) is anisohydric. During a prolonged, severe drought a greater proportion of pinyon trees died compared to juniper trees. They hypothesise that death from carbon starvation will occur when the drought is long enough to reduce photosynthesis sufficiently so that stored carbon is depleted by just maintaining the trees' metabolism. Death from hydraulic failure occurs when a plant exceeds its threshold for irreversible desiccation before carbon starvation sets in. Pests and pathogens can exacerbate both carbon starvation and hydraulic failure, and can also multiply on a host that has reduced ability to contain damage.

These different hypotheses reflect the different approaches by pathologists, physiologists and ecologists. Many of the stresses that they consider are similar; it is the timing and emphasis that differ.

Confounding factors

Poor drainage and reduced soil aeration

While stress caused by too little water is a possible factor in tree death many reports of root infection and tree deaths mention the role of too much water, i.e. poor drainage. Rands (1922) for example, stated that cinnamon canker caused by *Phytophthora cinnamomi* occurred most frequently on trees growing on poorly drained soils; avocado root rot was specifically described as a problem on soils with poor internal drainage (Crandall, 1948; Stolzy et al., 1966; Wager, 1942); another example is the death of *Pinus radiata* shelterbelts in New Zealand reported by Newhook (1959) which followed exceptionally heavy rainfall. This is unsurprising because most *Phytophthora* spp. require free water for sporangial production and zoospore discharge (Duniway, 1975; Ioannou & Grogan, 1984). Zoospores migrate both actively and passively through the soil solution. They are attracted to roots by both molecular signals and electric fields,

binding to specific sites on the root surface prior to penetration (Deacon & Donaldson, 1993). Repeated cycles of infection are setup by a number of flooding and draining cycles.

Soil saturation produces major physical and chemical changes in soil. The most rapid effect is the depletion of oxygen through the respiration of soil microorganisms, so that the soil solution becomes hypoxic and anoxic (Drew, 1992). Soil becomes anoxic within 24 hours in climates with high soil temperatures, but this occurs more slowly in temperate regions. Hypoxia and anoxia reduce both the rate and number of sporangia produced by *Phytophthora* spp. (Duniway, 1975 ; Mitchell & Zentmyer, 1971). They do not reduce the proportion of encysted zoospores that germinate, but do reduce their subsequent growth (Davison & Tay, 1986).

Soil saturation provides a conducive environment for root infection, and is a standard procedure in determining the pathogenicity of *Phytophthora* spp. in infested soil (e.g. Dawson & Weste, 1982; Fleischmann et al. 2002; Greslebin & Hansen, 2010; Jung et al. 1996) by increasing the number of root lesions, but not necessarily increasing lesion length (Burgess et al., 1998; Davison & Tay 1987). Flooding periods are usually of one- or two days duration and set up cycling infections: sporangia are formed when there is adequate aeration, while zoospore discharge and root infection occur each time the soil is saturated.

Hypoxia and anoxia also have an immediate effect on the metabolism of root tips which switch from aerobic to anaerobic respiration (Drew, 1997). Anaerobiosis produces less adenosine-5'-triphosphate (ATP) than aerobic respiration so that cell division and root elongation cease, water uptake is reduced, root tip cells die, and ethanol and other metabolites leak from the root tips. These effects on the root system affect the growth of the aerial parts of the plant resulting in stomatal closure, epinasty, reduced leaf expansion and increased leaf senescence (Jackson, 2002; Koslowski, 1986). Signals from roots to shoots are xylem mediated and include reductions in the flux of zeatin riboside cytokinins and ABA, which occur rapidly after the onset of flooding (Smit et al., 1989). In tomato, for example, one or more factors that reduce stomatal aperture were detected in xylem sap within 2 to 4 hours of the plants being flooded at 25 °C (Else et al., 2006).

Many of the effects of soil saturation on roots are similar to those that occur when root tips are infected by *Phytophthora* spp., including reduction in cytokinins in xylem extrudate, leakage from roots, and reduced stomatal conductance (Cahill et al., 1985; 1986; Maurel et al., 2004; Weste & Cahill, 1982). This is unsurprising as similar tissues are damaged. As noted above, the immediate effects of hypoxia and anoxia

on root metabolism also reduce the roots' ability to respond to infection (Burgess et al., 1998).

Soil compaction, whether by stock or agricultural machinery, also results in hypoxic soil conditions. Compaction may be on the surface, or at depth creating a plough pan. Such conditions are unfavourable to root growth, and are associated with more severe symptoms of chestnut ink disease caused by *Phytophthora cinnamomi* (Portela et al., 1999).

On poorly drained field sites, soil saturation may persist for days, rather than for hours. This results in the death of submerged roots, leading to a shallow root system and a reduced root to shoot ratio (Koslowski, 1986). The rate at which these changes occur depends on species susceptibility to these site conditions. Trees on such sites are more susceptible to drought, compared to similar trees on better drained sites (which will have a larger rooting volume).

Host anatomy

One interesting question is whether there is an effect of the position of damage, such as a canker, on water movement through the sapwood. This could be mediated by the occlusion of xylem vessels internal to the canker, and by the formation of narrow, traumatic xylem vessels internal to a regenerating cambium (Blaschke, 1994; Tippet & Hill, 1984). This will be most damaging to species with narrow sapwood.

In Proteaceae from the south west of Western Australia, and also in *Eucalyptus marginata*, conductance through lateral and horizontal roots decreased towards the root collar, and this decrease was the result of narrow xylem vessels at the distal end of the root (Davison & Tay, 1995a; Pate et al., 1995). A canker at the root collar is therefore likely to have a greater effect on conduction than one further from the base of the tree, not just because all of the roots converge at this point, but because vessel diameters are small and potentially restrictive to flow. An example of this is given by Hardy et al. (1996) who reported that cankers caused by *Phytophthora cinnamomi* in recently dead *E. marginata* and *Corymbia calophylla* saplings were consistently at the root collar and lignotuber.

Discussion

Trees contain large water and carbon reserves that enable them to respond to damage and survive periods of stress (Manion, 1981). If these reserves are reduced by extreme climatic conditions and/or by pathogens, a tree may reach a tipping point when it is so damaged that it dies. Infection by *Phytophthora* spp. has the potential to cause, or contribute to such damage.

Reduced conduction of water through the sapwood can lead to hydraulic failure, wilting and death. *Phytophthora* spp. that are known to invade the sapwood and thus have the potential to kill trees in this way are *P. ramorum* in *L. densiflorus*, (Collins et al., 2009; Parke et al., 2007) and *Q. cerris* (Brown & Brasier, 2007), and *P. cinnamomi* in *Banksia* spp. (Smith et al., 1997; Figure 1). It is likely that elicitors and toxins contribute to xylem damage. Xylem vessels provide a route for rapid axial spread throughout the tree with bark cankers developing adjacent to infected sapwood (Brown & Brasier, 2007). This type of infection is potentially the most damaging, but is the least frequently reported.

Extensive sapwood invasion may be common, but unrecognised, in other pathosystems if an inapparent infection is formed internal to conspicuous bark cankers. Chestnut blight, caused by *Cryphonectria parasitica* (Murrill) M.E. Barr, provides a relevant example (Ewers et al., 1989; McManus et al., 1989; McManus & Ewers, 1990). This disease is characterised by wilting, yet the most conspicuous symptoms are bark cankers. *C. parasitica* invades the sapwood in addition to infecting the phloem. Xylem vessels associated with the colonised sapwood are blocked by tyloses, resulting in hydraulic failure.

The isolation of *Phytophthora* spp. from the wood of a recently dead tree, however, does not necessarily indicate that sapwood invasion was the cause of death. Figure 2 shows that even though symptoms appear suddenly, the tree may have been declining for many months before the foliage dies, so that *Phytophthora* spp. and other pathogens will have been able to colonise tissues that would normally compartmentalise invasion. In recently dead *E. marginata* trees for example, *P. cinnamomi* was isolated from the wood in the roots and at the root collar (Dell & Wallace, 1981; Hardy et al., 1996; Shea et al., 1982; Shearer et al., 1981), an observation that is inconsistent with the results of pathogenicity tests which show that lesions are limited by host responses to the inner bark, cambium (Tippett et al., 1983) and a narrow band of sapwood adjacent to the canker (Davison et al., 1994). These differences can be reconciled if it is assumed that one or more stress factors reduce the host's ability to contain infection.

Fine root necrosis is another type of damage that will reduce water uptake and the amount of water stored within a tree. This type of infection is often assumed to occur as a result of infection by *Phytophthora* spp. when these pathogens are isolated from the rhizosphere soil at the base of declining trees (Balci & Halmschlager, 2003; Pratt & Heather, 1973; Jung & Blaschke, 1995; Jung et al., 1996; Jung et al., 2000). However, this evidence is circumstantial and it is frequently noted that affected trees are on poorly drained sites. Therefore, root damage caused by

hypoxic and anoxic soil conditions will also result in reduced water uptake and root death (Drew, 1997).

Many phytophthora diseases of trees fall into the category of declines and diebacks (Houston, 1987; Manion, 1981). Suggested predisposing site and weather factors include: heavy rainfall and poor soil drainage (Hodges et al., 1986; Jung, 2009; Newhook, 1959; Stolzy et al., 1966); and compaction (Portela et al., 1999), which will affect water uptake. Reduced water availability during drought is another predisposing factor. (Balci & Halmschlager, 2003; Brasier et al., 1993; Jung, 2009)

Reduced carbon availability will result from prolonged stomatal closure in anisohydric trees during drought (McDowell et al., 2008), and when the soil is hypoxic or anoxic (Koslowski, 1986; Jackson, 2002). Consequently, carbon starvation is likely to be an important physiological factor for trees growing on either seasonally droughty or wet sites. Any reduction in the amount of stored carbon will affect both the rate of root regeneration, and the trees' ability to respond to, and compartmentalise damage (Burgess et al., 1998).

Symptoms of decline and dieback imply that a large proportion of the root system must have been damaged. Although measurements of height and diameter increment are not strictly comparable with observations on crown transparency, work with other root and collar rot pathogens of forest and plantation trees has shown that growth is only reduced when there is either at least half of the large roots or root collar infected (Bloomberg & Hall, 1986; Froelich et al., 1977; Shaw & Toes, 1977). Most work on the amount of damage to roots systems by *Phytophthora* spp. diseases has been done with seedlings and saplings and these have shown high levels of infection and a large proportion of deaths (Gallego et al., 1999; Jönsson, 2004; Vettraino et al., 2003). However, excavations of live *Eucalyptus marginata* trees showed that less than 5% of the roots had *P. cinnamomi* cankers (Davison & Tay, 1995b; Shearer & Tippett, 1989). It is difficult to see how this low proportion of infected roots will cause tree death.

Trying to determine how and why trees die is not easy. A tree's size, for example, makes it difficult and expensive to excavate, so that many investigations are based on small sample sizes and a 'best bet' of where damage is most likely to have occurred. Less obvious symptoms, such as occluded xylem vessels and low levels of starch may be missed. The timing of tree death also presents a problem for researchers because the 'point of no return' may have occurred many months or years before symptoms become apparent. The physiological tolerance of each tree species needs to be considered, together with its genetic diversity. Each habitat is complex, representing a snapshot of past seed availability, environmental conditions

and successional changes (Jensen, 1986). As each pathosystem is unique, there is far less opportunity to review results and refine ideas than is possible with agricultural crops.

There are still many unresolved questions relating to how *Phytophthora* spp. kill trees. The most likely way is by xylem invasion leading to hydraulic failure. Attempted isolations from symptomless sapwood close to bark cankers would show whether xylem invasion is common. Wound inoculation tests using large stems or roots would show how extensively the sapwood can be colonised, and whether such infections result in reduced conduction of water. Another line of investigation would be to determine how reduced carbon availability affected the ability of the host to compartmentalise infections of the fine roots, phloem and wood.

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