25 Oomycete Diseases

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25.1 Pathogens, Significance and Distribution

The most important oomycete forest pathogens comprise two genera: *Pythium* and the formidable genus *Phytophthora*, whose name appropriately means 'plant destroyer'. *Pythium* spp. cause seed and root rots and damping-off diseases that thwart seedling establishment, and have been implicated in helping to drive forest diversity patterns through increased disease pressures on seedlings closest to their mother tree (Janzen, 1970; Connell, 1971). In contrast, *Phytophthora* spp. can cause disease at every life stage of forest trees, from root to crown, and from trunk cankers to foliar blights (Erwin and Ribeiro, 1996). They are remarkably flexible and effective pathogens with an unusual genetic architecture that may favour the rapid evolution of pathogenicity (Jiang *et al*., 2008; Raffaele *et al*., 2010; Seidl *et al*., 2011). Outbreaks of disease caused by *Phytophthora* spp. (especially when they have been introduced to new systems) have been documented with dramatic, and sometimes disastrous, effects since the mid 1800s. European and North American chestnuts (*Castanea* spp.) began dramatic declines from chestnut ink disease, a root rot caused in part by the extreme generalist *Phytophthora cinnamomi* Rands (Crandall *et al*., 1945; Anagnostakis, 1995). *P. cinnamomi* is notorious for the massive mortality it has caused in jarrah (*Eucalyptus marginata* Donn ex Sm.) forests in Western Australia, where it was first observed in the 1920s (Podger, 1972). *P. cinnamomi* causes root disease in agricultural and forest systems worldwide with varying degrees of virulence, but as Phytophthora dieback it has been seen to kill 50–75% of the species in sites in Western Australia, in some cases leaving every tree and much of the understorey dead (Weste, 2003). Shearer *et al*. (2004) estimate that of the 5710 described plant species in the South West Botanical Province of Western Australia, approximately 2300 species are susceptible, of which 800 of these are highly susceptible.

More recently, the trunk canker caused by *Phytophthora ramorum* Werres, De Cock & Man in't Veld has caused a devastating die-off of oaks (*Quercus* spp.) and tanoak (*Lithocarpus densiflorus* (Hook. & Arn.) Rehder) in western North America (Rizzo and Garbelotto, 2003), spreading from a relatively minor foliar blight of ornamentals in nurseries to a fatal scourge in US wildlands and UK gardens. In contrast to *P. cinnamomi*, which is a root pathogen

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transmitted by soil and water, infection by and transmission of *P. ramorum* occur primarily above ground. While the two species have much in common, e.g. extremely broad host ranges and disastrous consequences of introduction on native ecosystems, their contrasting modes of transmission span the range for oomycete pathogens. Here, we treat these two species and the extensive literature surrounding them as case studies for oomycete diseases and their management.

A great many more species of *Phytophthora* have an impact on forest systems, and new species are discovered almost yearly. Most of these are likely to have been established for some time (even if they are relatively recent introductions), but have only just noticed because of increased sampling and new molecular tools (Jung *et al*., 2002, 2011; Hansen *et al*., 2003; Balci *et al*., 2008; Burgess *et al*., 2009; Scott *et al*., 2009). Others are entirely new, however, notably a novel *Phytophthora* species complex that has begun to decimate alders (*Alnus* spp.) in Europe (Brasier *et al*., 1995, 1999). In addition to the more detailed descriptions of *P. cinnamomi* and *P. ramorum*, we briefly describe a selection of those species currently known to have the greatest impacts on forest systems. For a more complete description of the biology and disease control of older *Phytophthora* spp., refer to Erwin and Ribeiro (1996).

P. cinnamomi has a worldwide distribution, and is the cause of Phytophthora root and collar rot (synonyms: ink disease of hardwoods, Phytophthora dieback, littleleaf disease of pines, stripe canker of cinnamon). *P. cinnamomi* was confirmed as a cause of ink disease, along with *P. cambivora* (Petri) Buisman in Europe, in the early 1900s (Day, 1938), and as the cause of 'jarrah dieback' in 1965 (Podger *et al*., 1965; Podger, 1972). The pathogen causes root rots on a huge number of species (Hardham, 2005). Disease severity ranges from asymptomatic to fatal, depending on the host and environmental conditions, with among the worst observed in the jarrah 'graveyards' of Western Australia. It has been documented to live saprophytically, and to persist in moist soil for as long as 6 years (Zentmyer and Mircetich, 1966). However, the work of McCarren *et al*. (2005) provides strong evidence that *P. cinnamomi* is unable to survive saprophytically in the absence of host plants. In dry conditions, it persists in the hardpan layer, where deep lateral roots may be in contact with free water even when the surface soil is dry (Shea *et al*., 1983; Kinal *et al*., 1993; Shearer *et al*., 2010). It favours warmer temperatures than most *Phytophthora* spp., but has a wide range of conducive temperatures, facilitating its worldwide distribution.

P. ramorum is the cause of sudden oak death trunk canker, ramorum blight and dieback, and is distributed in nurseries in the western USA, western Canada and throughout Europe. It is present in gardens in the UK and wildlands in the USA. Diseases caused by *P. ramorum* emerged in the mid 1990s as foliar and twig blight of nursery ornamentals in Europe (Werres *et al*., 2001), and as fatal canker disease of oaks and tanoaks in a 300 km stretch of the California coast (Rizzo *et al*., 2002). The twig and foliar blight infects hosts in nearly every plant family, and is the primary source of inoculum even in the US epidemic (Garbelotto *et al*., 2002). The canker form in true oaks has not been documented to contribute to pathogen spread, but tanoaks are distinctly susceptible to both the sporulating foliar and twig disease and the fatal canker (Davidson *et al*., 2008). The disease has been responsible for the deaths of hundreds of thousands, if not millions of oaks and tanoaks (Plate 30), the near extirpation in tanoak in some parts of its range, and millions of US dollars incurred in costs due to quarantine and the monitoring of nurseries.

Pythium spp. (e.g. *P. debaryanum* R. Hesse, *P. irregulare* Buisman, *P. ultimum* Trow) are distributed worldwide, are root pathogens and a major cause of seed rots and damping-off diseases. These diseases are a major cause of seedling mortality, and have long been thought to help drive diversity in tropical forests through density-dependent mortality (Augspurger, 1984; Bell *et al*., 2006). They have also been documented in temperate systems (Packer and Clay, 2000, 2003). See Gilbert (2002) for a review of the role and limitations of pathogens as natural enemies in driving forest diversity, as proposed by Janzen (1970) and Connell (1971). Put simply, the Janzen–Connell hypothesis posits density-dependent

feedback, wherein the highest density of seeds and their pathogens or other natural enemies will both occur close to a parent tree. Seeds may have to travel some distance to escape their enemies, thus helping to drive forest diversity.

For forest managers, seed rots and damping-off diseases are a hindrance to reseeding efforts, and may inspire the use of nursery-grown seedlings rather than starting new trees from seed, bringing with them the potential to spread nursery diseases into wildlands. *Pythium* spp. have been implicated in the decline of spruce (*Picea* spp.) and beech (*Fagus* spp.) in Bavaria (Nechwatal and Osswald, 2001), the decline of Spanish oaks (Romero *et al*., 2007), and as contributing to growth reductions of Scots pine (*Pinus sylvestis* L.) in Scotland, especially when mixed with *P. cinnamomi* or *Fusarium* spp. (Chavarriaga *et al*., 2007). Features of the *P. ultimum* genome sequence are consistent with its status as a necrotroph and generalist pathogen of seedlings; it lacks cutinases to break down plant cuticles, lacks xylanase to digest complex polysaccharides, and does not have the RXLR effectors (effector proteins that can be delivered to the inside of host cells to manipulate innate immunity) that characterize *Phytophthora* pathogenicity (Lévesque *et al*., 2010).

A number of other well-known *Phytophthora* spp. (that is, other than *P. cinnamomi* and *P. ramorum*) are described below. *Phytophthora alni* Brasier & S.A. Kirk is distributed in Europe (Brasier *et al*., 2004b) and Alaska (Adams *et al*., 2008). This oomycete is a self-fertile complex of hybrids causing fatal root and collar rots in alders. First isolated in 1993 and 1994 in the UK (Brasier *et al*., 1995), the new species was eventually found to be a recent hybrid of *P. cambivora* and a species similar to *P. fragariae* Hickman (Brasier *et al*., 1999), probably with several instances of hybridization and backcrossing (Ioos *et al*., 2006). Diseases caused by the *P. alni* hybrid complex are characterized by root rot, dark and sometimes bleeding cankers on the trunk and/or crown decline, although asymptomatic infections are common, and are an important source of inoculum in infested watersheds (Lonsdale, 2003; Elegbede *et al*., 2010).

Phytophthora cactorum (Lebert & Cohn) J. Schröt. is globally distributed and causes disease on a wide range of species, from forest trees to ornamentals. It has been associated with canker diseases on maple (*Acer* spp.) (Caroselli and Howard, 1939; Erwin and Ribeiro, 1996), beech (Jung *et al*., 2006), horse chestnut (*Aesculus hippocastanum* L.) (Brasier and Strouts, 1976) and white poplar (*Populus alba* L.) (Cerny *et al*., 2008), and with declining oaks in Europe (Jung *et al*., 1996); it has long been associated with root rots of conifers.

Phytophthora cambivora has a worldwide distribution and, as already noted, is co-implicated with *P. cinnamomi* as the cause of ink disease of chestnut in Europe, both in its historic form, dating to before 1800 (Day, 1938; Crandall *et al*., 1945), and in its recent resurgence (Vettraino *et al*., 2001, 2005). The infection spreads from roots into the collar of the tree, and frequently causes death within 2 years. The disease may also move more slowly, and cause the slow decline of the crown, followed by death within several years (Day, 1938). Ink disease is characterized by a black exudate from a lesion that spreads upwards from the soil. The pathogen is soil borne and heterothallic, and is favoured by wet soil and by cooler temperatures than is *P. cinnamomi* (Erwin and Ribeiro, 1996).

With *P. cambivora* and *P. cinnamomi*, the more weakly pathogenic *P. citricola* Sawada is among the species most frequently isolated on symptomatic chestnuts in Europe (Vettraino *et al*., 2001, 2005). It is homothallic, and produces oospores that may contribute to its relatively stronger ability to persist in soil during the dry season (Vettraino *et al*., 2001). It is an important pathogen worldwide, and can infect a wide range of hosts. However, with the advent of molecular tools, isolates previously described as *P. citricola* based on morphological characteristics are now shown to be part of a species complex (Burgess *et al*., 2009; Jung and Burgess, 2009) to include *P. plurivora* T. Jung & T.I. Burgess and *P. multivora* P.M. Scott & T. Jung (Scott *et al*., 2009). For example, in Western Australia, the sequencing of the internal transcribed spacer (ITS) region of 73 isolates previously thought to be *P. citricola* revealed that no single isolate corresponded to *P. citricola s.*

(Burgess *et al*., 2009), and it appears that *P. citricola* is not present in wildlands in Western Australia. Therefore, it is likely that many diseases previously linked to *P. citricola* may well not be, and more work is required on the *P. citricola* species complex to fully understand the diseases they cause.

Phytophthora kernoviae Brasier, Beales & S. A. Kirk is the cause of a trunk canker of beech trees, and of leaf and twig blight and dieback in the UK and New Zealand. Although homothallic, *P. kernoviae* shares many characteristics with *P. ramorum*, and was in fact first discovered in 2003 during a survey of symptomatic trees at sites in Cornwall that were first found to be infested with *P. ramorum* (Brasier *et al*., 2004a). It is an aggressive pathogen of beech trees, causing large and fatal cankers. Like *P. ramorum*, however, its transmission is from foliar lesions on ornamentals, including *Rhododendron* spp. (Brasier *et al*., 2005). In addition to foliar symptoms, root infections have been observed on common rhododendron (*R. ponticum* L.), the primary agent of spread to canker hosts in woodlands, and these potentially complicate removal and eradication efforts (Fichtner *et al*., 2011).

Phytophthora lateralis Tucker & Milbrath causes a root rot of Port Orford cedar (*Chamaecyparis lawsoniana* (A. Murray bis) Parl.) in the Pacific Northwest of the USA and in British Columbia (Canada). It has recently been discovered in forest soils in Taiwan (Brasier *et al*., 2010) and in a new outbreak in Brittany (France) (Robin *et al*., 2011); otherwise, it is a nursery pathogen in Europe. *P. lateralis* is highly specific, infecting primarily *Chamaecyparis* spp., although Pacific yew (*Taxus brevifolia* Nutt.) is a minor host in North America (DeNitto and Kliejunas, 1991). Port Orford cedar is highly susceptible, and in Oregon forests it has caused major losses. There has been only one report of the aerial transmission of *P. lateralis* in the USA (Trione and Roth, 1957). Notably, a recent outbreak of disease caused by *P. lateralis* in France has been primarily foliar, and shows signs of aerial transmission (Robin *et al*., 2011).

Phytophthora palmivora (E.J. Butler) E.J. Butler is the cause of rots, blights and cankers, from root to fruits. It infects plant hosts worldwide, but is most important as a forest pathogen in the tropics, where it is a major pathogen of rubber (*Hevea brasiliensis* (Willd. ex A. Juss.) Müll. Arg.) and cacao (*Theobroma cacao* L.) trees. It is heterothallic, and produces caducous (shedding) sporangia and chlamydospores. Importantly, Arnold *et al*. (2003) and Herre *et al*. (2007) demonstrated the role of endophytes in host resistance by showing that their presence protected cacao trees from disease caused by *P. palmivora*.

Phytophthora pinifolia Alv. Durán, Gryzenh. & M.J. Wingf. is remarkable because it is the first known oomycete foliar pathogen of pines (*Pinus* spp.). It was discovered in Chile in 2003 in plantations of Monterey pine (*P. radiata* D. Don), where it is beginning to have a major impact on forestry. All size classes of trees are susceptible, and disease is characterized by the rapid death and drop of needles in mature trees and the death of seedlings (Durán *et al*., 2008).

25.2 Diagnosis

Infections caused by oomycetes are not always readily diagnosed, especially in novel outbreaks. The intrinsic difficulty lies in the different types of disease that may emerge when different pathogen species are involved, when outbreaks occur in sites that may be less or more conducive to disease because of soil and/or environmental parameters, when different hosts are involved, and even when different lineages or genotypes of the pathogen and/or the host are involved. The combination of two or more of the factors above may lead to symptoms and to epidemiological patterns that are different from those expected, and may be hard to identify.

The detection and identification of *Phytophthora* spp. have been recently reviewed by Martin *et al*. (2012). Soil-borne and water-borne species such as *P. cinnamomi*, *P. cambivora* and *P. lateralis* in general cause at first an underground infection of fine roots that may or may not, depending on host species and on site, proceed to an infection of the main roots, the root crown, and even of the lower part of the bole. In the absence of above-ground signs

of infection, diagnosis must be based on retrieval of the pathogen directly from the soil, and on symptoms such as reduction in growth rate, early foliar senescence and, eventually, drying of the canopy as indicators of infection (Campbell and Copeland, 1954; Zentmyer, 1980; Brasier *et al*., 1993). Some host species may be relatively tolerant of root infection by soil-borne *Phytophthora* spp., making diagnosis extremely difficult; in these cases symptoms often accelerate and lead to tree mortality only when other debilitating factors such as drought are superimposed on the root infection. Often, the only way to confirm root infections without extensive excavation is to bait the pathogen from the soil around trees. Baiting techniques to isolate *Phytophthora* spp. from soil are varied, and are described in detail by Tsao (1983) and Erwin and Ribeiro (1996), while modifications are described by Jung *et al*. (2000) and Scott *et al*. (2009).

In other pathosystems, including white oaks (*Q. alba* L.) in Mexico (Tainter *et al*., 2000), chestnuts in North America (Crandall *et al*., 1945), and manzanitas (*Arctostaphylos* spp.) in California (Swiecki *et al*., 2003) infected by *P. cinnamomi*, Port Orford cedar infected by *P. lateralis* in western North America (Tucker and Milbrath, 1942; Zobel *et al*., 1982; Hansen *et al*., 2000; Oh *et al*., 2006), alders infected by *P. alni* in Europe (Brasier *et al*., 1995; Streito, 2003) and chestnuts infected by *P. cambivora* (Day, 1938; Robin *et al*., 2006), host tolerance, while sometimes present and epidemiologically important, is quite low and infection of the roots underground leads to obvious symptoms and signs above ground in a broader range of environmental conditions. Outbreaks can be identified by thinning canopies and mortality of trees in groups, often associated with girdling lesions advancing from the root collar into the lower parts of the bole. Lesions are normally brown to cinnamon and black in colour, extensive in the phloem, while limited in the bark and the xylem, and are characterized by irregular margins at times marked by a dark line. Lesions can sometimes be identified as areas of sunken bark but, in general, lesion identification requires an excision of the outer layers of bark to expose the phloem. To confirm infection, it is often necessary to isolate the pathogen by plating a small chip from the edges of a lesion. Plating techniques and media selective for *Phytophthora* spp. are described in detail by Erwin and Ribeiro (1996).

One of the best known symptoms associated with above-ground stem colonization by *Phytophthora* spp. is the so-called 'bleeding' or 'seeping', a symptom known as 'gummosis' in some tree species, which can be described as the production of a dark and viscous substance on the outside of the bark. This 'bleeding' corresponds to the lesion in the phloem but in the absence of any obvious mechanical damage or insect-generated wound. 'Bleeding' symptoms on the trunk, if correctly diagnosed, can help to determine the presence of an infection by a *Phytophthora* species; however, not all hosts will develop such symptoms, and there is considerable variation even within some species.

Similar girdling lesions and associated bleeding are also characteristically associated with infections by aerial Phytophthoras, or with aerial infection caused by soil-borne *Phytophthora* spp. In these cases, lesions can occur on the stems, branches and even twigs without any clear association with root crowns (collars). In fact, in the case of *P. ramorum*, large aerial trunk lesions disappear just below the soil level (Davidson *et al*., 2003). Lesions caused by these aerial species can lead to the development of different symptoms, depending on the tolerance of the host and, especially, on the location of the lesion. As a result, highly susceptible hosts infected in the lower part of the main trunk may be rapidly girdled with a simultaneous death of the entire canopy. Conversely, a lesion on a branch may lead to death of the branch alone, without immediate consequences for the whole tree. In most cases, aerial *Phytophthora* spp. girdle and kill the infected portion or the whole individual; thus the slow decline associated with root infection by some soil-borne *Phytophthora* spp. seems to be less frequently observed. In many cases, epicormic adventitious branches are produced right below the lesion; although these sprouts rarely survive long, they may help to identify a *Phytophthora* lesion. Drought-adapted species such as California coast live oak (*Q. agrifolia* Née) may be effectively girdled, but maintain a canopy that remains green and even breaks bud and

grows for up to several years after the stem is girdled, thanks to the presence of stored carbohydrates. These trees undergo a progressive drying of the foliage, are often affected by accelerated decay processes, and become attractive to insect infestations even if the canopy has not obviously turned brown. The presence of entry holes by bark beetles and of fruiting bodies of wood decaying fungi (both basidiomycetes and ascomycetes) in several portions of the main stem is a clear indication that the tree is functionally dead.

Another clear distinction between soil-borne or waterborne and aerial *Phytophthora* spp. is the pattern of spread of the disease. Infection by soil-borne or waterborne species appears to be clearly associated with bodies of water, areas where water accumulates – such as poorly drained draws (shallow gullies) and flood areas, underground water tables, and finally unpaved roads or even dirt tracks. This association is lacking for aerial species and is replaced by the spatial aggregation of infections in areas adjacent to sporulating hosts.

In complex forest ecosystems such as the coastal forests of California, or the jarrah forest and banksia (*Banksia* spp.) woodlands of Western Australia, an infestation by a *Phytophthora* spp. can also be diagnosed via susceptible indicator plants that develop symptoms more rapidly than the tree hosts of interest. Leaf lesions on California laurels (*Umbellularia californica* (Hook. & Arn.) Nutt.) and twig infections of tanoaks, Pacific madrones (*Arbutus menziesii* Pursh), and huckleberries (*Gaylussacia* spp.), just to name a few species, often precede lethal girdling stem lesions of oaks and tanoaks and can strengthen the diagnosis of an infestation by *P. ramorum*, even in the absence of lethal infections on tree hosts. Similarly, the mortality of shrubs and herbaceous hosts such as bull banksia (*B. grandis* Willd.) and balga (*Xanthorrhoea preissii* Endl.) in the jarrah forest of Western Australia can be rapid and precede any symptoms on trees, thus providing a valuable indicator of an expanding infestation (Shearer and Tippett, 1989).

Whether from soil, from an indicator plant, or from a tree, the identification of the *Phytophthora* species involved can be of paramount importance in predicting risk and in assessing the need for management. Many *Phytophthora* spp., both native and introduced, can coexist in forest ecosystems. Morphological identification of the pathogen in culture remains the best approach, but often the pathogen is extremely hard to isolate from some host species or at certain times of year (e.g. during dry spells, or at unfavourable temperatures). Additionally, new species are continually being described, while traditional taxonomic expertise is becoming more limited. For these reasons, diagnosis can be significantly enhanced by DNA-based techniques and immunoassays. Sequencing one or two diagnostic loci and comparing the sequences with those in public databases may suffice to identify a species that is not familiar to the researcher. When no cultures can be obtained, immunoassays and polymerase chain reaction (PCR)-based protocols can confirm the presence of a *Phytophthora* species directly from infected plant tissue or from soil and water (Table 25.1). It should be highlighted that the pitfalls of diagnosis based solely on molecular techniques are many, and include both false negative and false positives diagnoses. Currently, the advantages and disadvantages of molecular identification approaches have been somewhat adequately investigated only for *P. ramorum* (Hayden *et al*., 2006; Kox *et al*., 2007; Lane *et al*., 2007; Vettraino *et al*., 2010). Hüberli *et al*. (2000) discuss the problems of misdiagnosis based on false negative isolation by culture for *P. cinnamomi*.

25.3 Infection Biology

Oomycetes are not true fungi; they are instead classified with the brown algae, or water moulds. Their vegetative hyphae resemble fungi, but unlike true fungi, *Pythium* and *Phytophthora* cell walls do not contain chitin; they require an outside source for sterols, and infection occurs through either direct germination of asexually produced sporangia, or through encystment and germination of the biflagellate, swimming zoospores that emerge from sporangia (Alexopoulos *et al*., 1996; Erwin and Ribeiro, 1996).

Phytophthora spp. are generally hemibiotrophic, first infecting living tissue, and then

Table 25.1. Taxon-specific PCR-based methods for the detection of *Phytophthora* spp.

Target	References
Multiple taxa:	
P. cambivora, *P. cinnamomi*, *P. citricola*, *P. citrophthora*, *P. infestans*, *P. kernoviae*, *P. nemorosa*, *P. nicotianae*, *P. pseudosyringae*, *P. quercina*, *P. ramorum*	Érsek *et al.*, 1994; Böhm *et al.*, 1999; Schubert *et al.*, 1999; Nechwatal *et al.*, 2001; Ippolito *et al.*, 2004; Martin *et al.*, 2004; Schena *et al.*, 2006, 2008; Langrell *et al.*, 2011
P. alni complex	Ioos *et al.*, 2005; Bakonyi *et al.*, 2006
P. cinnamomi	Schubert *et al.*, 1999; Kong *et al.*, 2003; O'Brien, 2008; Williams *et al.*, 2009; Langrell *et al.*, 2011
P. lateralis	Winton and Hansen, 2001
P. ramorum	Kroon *et al.*, 2004; Martin *et al.*, 2004; Tomlinson *et al.*, 2005, 2007; Hayden *et al.*, 2006; Hughes *et al.*, 2006; Schena *et al.*, 2006; Belbahri *et al.*, 2007; Bilodeau *et al.*, 2007a,b

growing into and between plant cells while leaving a necrotic lesion behind, whereas *Pythium* spp. may be either hemibiotrophs or necrotrophs (Latijnhouwers *et al.*, 2003). Hyphae originating from direct germination of sporangia enter the plant through stomata, lenticels or other openings. Zoospores germinate by first encysting on the plant surface (roots, stems or leaves), after which either an appressorium forms to break the host cell wall, or a germ tube may enter through stomata. Rapid cycling of infection followed by asexual sporulation in good conditions for disease development allow for huge increases in inoculum loads over short time periods.

Zoospore encystment is triggered by pressure, electrochemical charge and chemical signalling. Zoospore movements have been documented to respond to chemical exudates from plants, to light, to gravity and to charge (Hardham *et al.*, 1994; Hardham, 2001, 2007). Zoospores can travel short distances (millimetres to centimetres) under their own power, or spores can passively travel long distances in flowing water. Propagules of *Phytophthora cinnamomi* have been documented in flowing groundwater up to 5 m below the soil surface, where free water may persist even when the surface dries (Shea *et al.*, 1983; Kinal *et al.*, 1993; Shearer *et al.*, 2010). Some species are also able to shed sporangia, which can then disperse by wind or water. On landing, new infections may occur either through direct germination or indirectly by the release of zoospores. Even after the wind dispersal of sporangia, neither direct germination nor zoospore release may occur in the absence of free water (Judelson and Blanco, 2005), and zoospores and sporangia are relatively fragile and short lived.

Oomycetes are persistent in soil, especially if it remains damp. *Pythium* spp. can persist as a saprobe or as resting oospores (Martin and Loper, 1999), and *Phytophthora* spp. may persist in soil or gravel for years as oospores, chlamydospores or, sometimes, saprophytically (Zentmyer and Mircetich, 1966; Hwang and Ko, 1978; Weste and Vithanage, 1979; Linderman and Davis, 2006; Shishkoff, 2007). Chlamydospores are asexual spores with a surprising degree of intraspecific variation in structure, wall thickness and tolerance of extreme environments, while oospores are produced by sexual recombination, and tend to be thick walled and quite persistent (McCarren *et al.*, 2005).

The frequency of chlamydospore and oospore production is governed by environmental conditions as well as by the biology of the particular species in question. The production of both structures tends to be favoured at temperatures somewhat below optimum growth temperatures, requires adequate moisture and nutrients, and is influenced by the chemical environment (Mircetich *et al.*, 1968; Tsao, 1969; Cother and Griffin, 1974; Ribeiro, 1983; Erwin and Ribeiro, 1996). Homothallic species can produce oospores by self-fertilization, but heterothallic species require contact between opposite mating types.

Both *Phytophthora ramorum* and *P. cinnamomi* are heterothallic, albeit that in their introduced ranges they appear to reproduce mostly asexually, even when both mating types are present (Dobrowolski *et al*., 2003; Ivors *et al*., 2006; Prospero *et al*., 2007; Mascheretti *et al*., 2008; Goss *et al*., 2009). None the less, Brasier (1971, 1975) showed that A2 mating types of several heterothallic species, including *P. cinnamomi*, could be stimulated to produce self-fertilized oospores by antibiotic compounds from *Trichoderma* spp. Some plant root exudates have a similar effect; A2 *P. cinnamomi* cease the production of sporangia and generate self-fertilized oospores in response to exudates from avocado (*Persea americana* Mill.) (Zentmyer, 1979) and acacia roots (Jayasekera *et al*., 2007).

25.4 Epidemiology

25.4.1 Mode of dispersal

A primary distinction in the epidemiology of oomycete pathogens is whether they are dispersed aerially, or solely by water and soil. Species with caducous sporangia, including most of the foliar oomycete pathogens (Erwin and Ribeiro, 1996), may travel by wind as well as by water splash, flowing water and in soil. For example, Davidson *et al*. (2005) documented *Phytophthora ramorum* inoculum in rainwater, streams and even in soil on hikers' shoes. A more recent genetic analysis of gene flow in *P. ramorum* revealed a bimodal peak, whereby most genotypic similarity was detected at 15 m, the range for water splash, with a second peak at 1000 m, the likely range for wind dispersal (Mascheretti *et al*., 2008). Analysis of individual populations could not definitively distinguish between new infestations from wind- or human-mediated dispersal, although at least some wind transmission was probable in the study populations. New infestations of *P. ramorum* have been detected at 4000 m from the nearest known population in Oregon (Hansen *et al*., 2008), where there is an active programme to eradicate the pathogen wherever it is detected (Goheen *et al*., 2002; Kanaskie *et al*., 2006, 2008). Hansen *et al*. (2008) observed that new Oregon infestations often appeared as the infection of several trees with the same pathogen genotype at once, suggesting that wind dispersal might occur as relatively rare, mass movement of spores by turbulence events. At distances greater than 1–4 km, human-mediated movement through nursery plants or soil is the most important mechanism for spread.

Soil-borne pathogens are more reliant on humans for both medium and long-range dispersal, although waterways may carry propagules for longer distances, and spread from soil moisture within an infested site can be extremely efficient, reaching up to 100% infection within a short time (Colquhoun and Hardy, 2000; Hansen *et al*., 2000). Roadways are especially important. An analysis of the spread of *Phytophthora lateralis* on Port Orford cedar in the USA determined that 26 of 36 new infestations were due to vehicle traffic on roads, and the rest were from foot traffic (Jules *et al*., 2002). Roads have been similarly implicated in the spread of *Phytophthora cinnamomi* into the jarrah forest in Western Australia, through vehicle traffic and through the earth movement required for their construction (Shearer and Tippett, 1989). Bauxite mining in Western Australia is a particular problem for the spread of *P. cinnamomi* in the jarrah forest. The mining process moves huge quantities of soil, redirects drainage patterns, and necessitates the construction and heavy use of new roads, all of which have the potential to spread the pathogen and have been the subject of a containment effort (Colquhoun and Hardy, 2000; Colquhoun and Kerp, 2007).

Human activities may lead to unexpected or unusual disease forms. In nurseries with artificial irrigation and abundant water splash, needle infections of firs (*Abies* spp.) by *P. cinnamomi* have been reported (McCain and Scharph, 1986). Recently, Robin *et al*. (2011) reported foliar disease caused *P. lateralis* on Port Orford cedar in landscapes in France, where it previously had been seen only in nurseries. Trione and Roth (1957) reported an infrequent aerial habit of *P. lateralis* in USA forests, but aerial disease has not since been reported, nor has it been reported to play any role in disease dynamics.

P. lateralis was previously confined to nurseries in Europe, so the new report is of both a new range and a new form. Evidently, some component of the new environment has triggered a shift in the pathogen's mode of transmission, with potentially important implications for epidemiology in the new range (Robin *et al*., 2011).

The movement of nursery plants as a source of spread of *Phytophthora* spp. cannot be underemphasized. Nursery plants have played a major role in the evolution and spread of, among others, the *Phytophthora alni* complex (Brasier *et al*., 1999; Brasier, 2000; Jung and Blaschke, 2004), have had a demonstrable role in the spread of *P. cinnamomi* (Hardy and Sivasithamparam, 1988) and are responsible for the rapid spread of *P. ramorum* throughout much of the USA and Europe (Ivors *et al*., 2006; Mascheretti *et al*., 2008, 2009). Brasier (2008) documents the many introductions of destructive pathogens from trade in plant products, and the threat that they pose to ecosystems and economies.

25.4.2 Host range

Pathogens with large host ranges will have different ecological and epidemiological dynamics from specialists. It is no coincidence that the extreme generalists *P. cinnamomi* and *P. ramorum* have both led to spreading epidemics of forest disease. *P. cinnamomi* is estimated to have more than 3000 plant hosts (Hardham, 2005), while the much more recently emerged *P. ramorum* has a smaller, but ever-growing list of known hosts that encompasses nearly every family of plants (Garbelotto *et al*., 2002; Grünwald *et al*., 2008). A wide host range opens the range of niches available to the pathogen. The probability of an individual spore encountering a susceptible host is increased, thereby increasing the probability of transmission. Generalists are more likely than specialists to become established in new environments because of the greater likelihood of finding a susceptible host. They may even be more likely to jump to new hosts, through inoculum rain from a nearby susceptible species that could eventually include a variant able to overcome the previously resistant host's defences (Parker and Gilbert, 2004).

None the less, all hosts may not be equal. Of special concern are tolerant species, which support pathogen infection with minimal or no disease symptoms or fitness effects, but nevertheless support pathogen sporulation and transmission. Roy and Kirchner (2000) modelled the potentially insidious nature of host tolerance on host–pathogen co-evolution; in contrast to host resistance, in which some intermediate level of virulence is theoretically optimal, tolerance interactions can be expected to lead to ever-increasing pathogen virulence and aggressiveness. Real interactions are most likely to involve some mixture of resistance and tolerance.

California laurel trees are a case in point: they restrict infection by *P. ramorum* to their leaves, where they experience only minor physiological damage from ramorum blight (DiLeo *et al*., 2009). The pathogen sporulates prolifically from California laurel, however, and the trees have played a major role in the epidemiology of sudden oak death in California. As always, the interactions of environment, hosts and pathogens all play a role in disease dynamics. Davidson *et al*. (2011) attribute the quicker disease progress of sudden oak death in California redwood (*Sequoia sempervirens* (Lamb. ex D. Don) Endl.) forests than occurs in similar mixed evergreen forests to the greater propensity of California laurel leaves in evergreen forests to drop their leaves during the dry summer months. Because California laurel trees in redwood forests are more likely to keep their diseased leaves, there is a greater supply of inoculum at the ready when moist conditions in the spring once again favour disease. The sporulation of *P. ramorum* and *P. kernoviae* has been documented from entirely asymptomatic fruits and rhododendron leaves used as baits (Denman *et al*., 2008); the role of asymptomatic infection in disease dynamics hence merits further study. A genetic analysis of a newly observed infestation of *P. ramorum* in northern California revealed that the pathogen population had in reality been in residence and reproducing for some time (S. Mascheretti

and M. Garbelotto, 2011, unpublished results); it is entirely possible that this was in latent, asymptomatic infections.

25.5 Management Strategies and Tactics

25.5.1 Avoidance

A seemingly obvious technique for avoiding oomycete diseases is to choose forestry sites outside the pathogen's environmental limits (Table 25.2). Temperature effects depend on the pathogen and host of interest. Every pathogen has its own temperature requirements for growth and sporulation; temperature and phenology can have just as important but often overlooked effect on host susceptibility. California coast live oaks in the western USA are the most susceptible to sudden oak death in the springtime during a flush of active cambial growth (Dodd *et al.*, 2008). This period of increased susceptibility has an unfortunate synchronism with both the time for maximum susceptibility of the vector, California laurel, and the time of maximum pathogen sporulation (Hüberli *et al.*, 2012). Locating planting sites in regions that are unfavourable or marginal for pathogen growth and transmission may avoid or minimize disease. Pythium diseases of seedlings may be avoided to some extent by simply sowing seed when the weather shifts to slow pathogen growth and speed plant growth, depending on the particular host–pathogen combination (Tainter and Baker, 1996). In Australia, avoidance of *Phytophthora cinnamomi* involves extensive mapping to determine where the pathogen is present and where it is not present in wildlands. Strict hygiene and quarantine protocols are then implemented in those plant communities that are disease free, and considered to be susceptible or at risk to the pathogen (Dell *et al.*, 2005). In Oregon, consideration of the proximity to known infestations is recommended for avoidance of *P. lateralis*, as well as the placement of sites upslope from roads and on convex slopes to minimize exposure to new inoculum (Hansen *et al.*, 2000).

Table 25.2. Avoidance strategies available to control oomycete diseases.

Avoidance strategy	References
Canopy architecture	
Depending on their transmission mode, open canopies help to avoid soil-borne species, but aid their aerial transmission	Tainter and Baker, 1996; Kelly and Meentemeyer, 2002; Swiecki and Bernhardt, 2002, 2006; Rizzo *et al.*, 2005
Inoculum sources	
Monitoring/mapping pathogen locations; proximity to infectious hosts; thinning or removal of infectious hosts	Shearer and Tippett, 1989; Kelly and Meentemeyer, 2002; Goheen *et al.*, 2004; Meentemeyer *et al.*, 2004; Davidson *et al.*, 2005, 2008; Maloney *et al.*, 2005; Rizzo *et al.*, 2005; Valachovic *et al.*, 2008, 2010; Cobb *et al.*, 2010; Davis *et al.*, 2010
Site selection	
Climate unfavourable to pathogen; water dynamics to carry potentially infested water away	Tainter and Baker, 1996; Hansen *et al.*, 2000; Tooley and Browning, 2008; Fichtner *et al.*, 2009; Hüberli *et al.*, 2012
Soil characteristics	
Permeable, well drained, loamy, organic content	Campbell and Copeland, 1954; Hoitink *et al.*, 1977, 1997; Weste and Vithanage, 1979; Shea *et al.*, 1983; Hardy and Sivasithamparam, 1991; Kinal *et al.*, 1993; Tainter and Baker, 1996; Fichtner *et al.*, 2009; Shearer *et al.*, 2010; Shearer and Crane, 2011

Avoidance of aerially dispersed oomycetes

One of the most important tools for the avoidance of aerially dispersed Phytophthora diseases is the minimization of spore sources. Proximity to the infectious host California laurel is a major risk factor for sudden oak death in California (Kelly and Meentemeyer, 2002; Meentemeyer *et al*., 2004; Davidson *et al*., 2005, 2008; Maloney *et al*., 2005; Cobb *et al*., 2010; Davis *et al*., 2010). Selectively thinning California laurel has been suggested as an inoculum reduction strategy (Rizzo *et al*., 2005; Valachovic *et al*., 2010). Thinning treatments can range from pruning the lower branches of California laurels, as recommended by Rizzo *et al*. (2005), to the complete removal of tanoak and California laurel hosts (Goheen *et al*., 2004; Valachovic *et al*., 2008, 2010). Thinning treatments that span this continuum have been initiated in California (Valachovic *et al*., 2010; EFSA, 2011). Preliminary results have shown that removal of California laurel from a 10–15 m boundary surrounding oak trees reduces spore loads to levels that usually avoid infection of the trees (EFSA, 2011).

Aerially dispersed oomycetes make use of the same resting soil structures as their soil-borne kin. Shishkoff (2007) found that chlamydospores of *Phytophthora ramorum* persisted for over a year in potting mix and sand, and germinated near plant roots. Chlamydospore survival has been shown to be decreased by soil drying (Fichtner *et al*., 2007), and chlamydospores have been inactivated *in vitro* at temperatures at and below –10°C and at 40°C (Tooley and Browning, 2008). A study of the persistence of chlamydospores under forest conditions found that survival of *P. ramorum* chlamydospores was minimized by redwood soil (steaming the soil did not change suppression), and that the infection of plant material by splash from the soil was decreased but not eliminated by the presence of leaf litter (Fichtner *et al*., 2009). None the less, spread of *P. ramorum* was found to be greater in redwood forests than in mixed evergreen forest, perhaps in part owing to the greater leaf abscission by foliar hosts in those forest types (Davidson *et al*., 2011).

Closed canopies and dense stands promote Phytophthora and Pythium diseases; sunlight and open stands can help avoid root diseases, particularly damping-off (Tainter and Baker, 1996). However, increased airflow in open stands may in fact aid transmission of aerially-dispersed *Phythophthora* spp. (Kelly and Meentemeyer, 2002; Swiecki and Bernhardt, 2002, 2006; Rizzo *et al*., 2005).

Avoidance of soil-and water-dispersed oomycetes

Soil characteristics play a large role in the development of soil-borne Phytophthora and Pythium diseases, and much can be accomplished by choosing appropriate sites for plantations or reforestation. Well-drained, loamy soils with a high organic content are widely recommended for avoiding root diseases. Good drainage minimizes the time in contact with free water that is required for oomycete infection; high organic content reduces oospore and chlamydospore survival times (Weste and Vithanage, 1979), and it also harbours microbes that may outcompete poorly saprophytic *Pythium* spp. Organic soils in the form of composts have long been found to suppress a number of *Phytophthora* and *Pythium* spp. (Hoitink *et al*., 1977, 1997; Hardy and Sivasithamparam, 1991). The microbial community no doubt plays a large role in the protective effect; although in some cases the composts offer disease protection even after sterilization (Fichtner *et al*., 2009).

A study of different disease progress rates for *Banksia* spp. inoculated with *Phytophthora cinnamomi* in different soil types from across a threatened park system supported the overall rating of soil types, with the greatest mortality in sands and the least mortality in loams (Shearer and Crane, 2011). An impermeable concretized duricrust has been linked to Phytophthora root and collar rot in eucalyptus and banksia forests (Shea *et al*., 1983; Kinal *et al*., 1993; Shearer *et al*., 2010); free-flowing water collects above this layer, remaining in contact with deep taproots even as the surface soil dries, thereby facilitating disease. Likewise, the relationship between soil quality and risk for littleleaf disease caused by *P. cinnamomi* on pines in the south-east USA is

well established, and soils are rated before planting on a scale of 1–100. Impermeable, highly eroded soils with strongly mottled, firm subsoil are most at risk (Campbell and Copeland, 1954; Tainter and Baker, 1996).

The reduction of spore loads by host removal has also been tried for soil-borne species. In Western Australia, attempts were made to remove the highly susceptible bull banksia using silvicultural techniques (stump poisoning or by fire) to reduce inoculum loads of *P. cinnamomi*, especially where jarrah grew on soils considered to be highly conducive to the pathogen (Shearer and Tippett, 1989).

25.5.2 Exclusion

Exclusion of nursery pathogens from forests

The exclusion of nursery pathogens from forested areas is a critical issue for forest health, as exemplified by the epidemics of both *Phytophthora cinnamomi* and *P. ramorum*. Long-distance movement of plants and soil should be avoided; planting projects using seedlings should use locally grown sources and scrupulously disease-free plants (Table 25.3). Seedlings should be sourced from nurseries that have a strong emphasis on hygiene; measures include the use of new seedling containers and of container media that have been adequately composted or steam pasteurized, irrigating with water known to be *Phytophthora* free and keeping containers on benches off the ground (Hardy and Sivasithamparam, 2002). Even local sourcing for plants does not guarantee against the emergence of new pathogens, as nurseries can select for the evolution of new hybrids such as *P. alni*.

Exclusion of aerial Phytophthoras

The current policies for exclusion of *P. ramorum* have relied on inspection protocols for nurseries and quarantines at the level of political boundaries. Within the USA, there has been a concerted eradication effort in Oregon, and the regulation of movement of host plants among infested counties within states or across state borders. A coordinated effort to slow the spread of *P. ramorum* to uninfested regions has been lacking in the California, the largest part of the pathogen's naturalized range, although a number of small projects have been initiated at the local level (Rizzo *et al*., 2005; Alexander and Lee, 2010; Valachovic *et al*., 2010).

Exclusion of soil-borne oomycetes

Western Australia has been the site of a more successful coordinated effort to exclude soil-borne species from new locations. Bauxite mining in jarrah forest clears about 550 ha of

Table 25.3. Exclusion strategies available to control oomycete diseases.

Exclusion strategy	References
Forest sanitation	
Monitoring/mapping pathogen locations; restricting vehicle movement from infested to uninfested areas; cleaning vehicles before entering uninfested areas; preventing infested and uninfested soils from mixing; preventing water draining from infested to uninfested areas; education of public and forestry workers	Colquhoun and Hardy, 2000; Hansen *et al*., 2000; Colquhoun and Kerp, 2007
Nursery sanitation	
Use of new seedling containers, container media pasteurized; irrigation water *Phytophthora* free; water splash kept off leaves and wetness time minimized; containers kept off the ground; suppressive composts or fungicides avoided; sustained heat treatment to kill resting structures in plant or soil material via composting, solarization, oven treatment or autoclaving	Le Bihan *et al*., 1997; Hardy and Sivasithamparam, 2002; Harnik *et al*., 2004; Swain *et al*., 2006; Tooley *et al*., 2008

forest and moves approximately 6 million m^3 of soil annually, so the potential for spreading *Phytophthora cinnamomi* is high (Colquhoun and Hardy, 2000). *P. cinnamomi* occurs in a mosaic throughout the jarrah forest and a range of procedures have been developed for every stage of the mining and restoration process to ensure that the pathogen is not spread. These procedures include: (i) knowing where the pathogen is through detailed mapping for the presence of *P. cinnamomi* using susceptible indicator species and soil baiting; (ii) restricting vehicle movement from infested to uninfested areas; (iii) cleaning vehicles before entering uninfested areas; (iv) preventing infested and uninfested soils from mixing; (v) preventing water draining from infested to uninfested areas; (vi) training all field staff and planners; and (vii) monitoring the spread of the disease attributable to mining and investigating the causes (Colquhoun and Kerp, 2007). As a result of these procedures, and despite the logistics of moving huge volumes of soil, it is estimated that the rates of spread of *P. cinnamomi* attributable to mining activities equate to 6×10^{-4} ha for every hectare mined (Colquhoun and Kerp, 2007). Therefore, it is possible to manage the soil-borne species of *Phytophthora* given adequate resources and infrastructure.

Port Orford cedar disease from *P. lateralis* has been the target of a similar effort to contain spread from new areas. The disease spreads primarily from mud on vehicles along roads and then through waterways (Jules *et al*., 2002). Techniques in place for controlling spread have been outlined in Hansen *et al*. (2000), and include: (i) road closures – permanent and wet season; (ii) re-engineering roads, elevating and paving surfaces, and redirecting runoff away from Port Orford cedar stands; (iii) controlling the use of water for dust control and firefighting to prevent infested water sources from being sprayed on to stands; (iv) public education about risk of spread; and (v) restricting harvest operations.

Sanitation

For all oomycete disease, the sanitation of infested soil, water, and plant materials is critical to prevent pathogen spread. Heat to 40°C effectively kills even resting chlamydospores of *Phytophthora ramorum* (Harnik *et al*., 2004; Swain *et al*., 2006; Tooley *et al*., 2008). This temperature can be achieved via direct exposure to the sun, by a solarisation process or by composting. Simply piling dead wood in the sun rather than in the shade is a wise choice for helping to minimize pathogen survival, or a more formal composting protocol can be used to assure the destruction of propagules in plant material (Swain *et al*., 2006). Solarization has been shown to be as effective as chemical fumigants in eliminating *Pythium* spp. from nursery soil (Le Bihan *et al*., 1997) and eradicating *Phytophthora cryptogea* Pethybr. & Laff. from infested plots of a loamy soil up to 30 cm depth (Kaewruang *et al*., 1989).

In addition to the sanitation of water used for dust control and firefighting that was previously mentioned, landscape and park settings should avoid irrigating with stream or pond water during times of maximum sporulation, although there is some evidence that passing through sprinklers reduces inoculum levels of *P. ramorum*, and lowers disease incidence in nursery plants (Tjosvold *et al*., 2008). Standard irrigation practices are recommended, including avoiding water splash on leaves, avoiding watering drought-adapted species during the dry season, or avoiding watering in the evenings, when free water and leaf wetness times will be longest and so allow the maximum opportunity for oomycete infection. Tjosvold *et al*. (2008) found that direct application of infested irrigation water to leaves of nursery stock was required for ramorum blight to occur. However, infection of the aerial parts of plants by *Phytophthora* via the roots should not be completely discounted, as *P. ramorum* infection from infested soil has been documented on various ornamental species (Parke and Lewis, 2007; Shishkoff, 2007). In production nurseries, it is critical to avoid the use of fungicides including phosphonates, which can suppress disease symptoms, and thus allow the sale and movement of apparently disease-free nursery stock into the wider environment.

Eradication

In contrast to its wide establishment at the time of its discovery in California, sudden

oak death was first detected as nine infestations in a small area in Oregon (Goheen *et al*., 2002; Hansen *et al*., 2008). Consequently, an eradication programme for *P. ramorum* was initiated in Oregon, involving cutting and burning all host plants within 15–30 m (2001 and 2002) or 100 m (2003 onwards) of the detection site, followed by aerial detection surveys and stream leaf baiting to detect new infestations (Table 25.4). This programme has successfully eradicated the pathogen within microsites, and has doubtlessly slowed the spread of the epidemic in Oregon. Yet eradication has not been successful on a landscape scale: new infestations have been detected each year following the initiation of the eradication programme, at up to 4 km from the nearest known infestation (Kanaskie *et al*., 2006, 2008; Hansen *et al*., 2008).

A recent eradication programme for *P. cinnamomi* has also had local success. Working on the idea that denying the pathogen living roots for nutrition would ruin its competitive ability in soil, the programme involved: (i) physical and herbicidal removal of all plants from the site, to a distance of 4–10 m beyond the disease front; (ii) root barriers to prevent the intrusion of roots from adjacent sites; (iii) treatments of the soil surface with triadiazole and metalaxyl; (iv) fumigation of the deep soil and soil surface with metham-sodium. The pathogen was completely eradicated from one site, and largely controlled at the other (Dunstan *et al*., 2010).

25.5.4 Protection

Systemic chemical protection: phosphonates

Systemically translocated chemicals containing phosphonates (including fosetyl-aluminium and potassium salts of phosphorous acid) are among the most important agents for controlling oomycete diseases (Cohen and Coffey, 1986; Smillie *et al*., 1989; Guest and Grant, 1991) (Table 25.5). Phosphonates have two modes of action, depending on dose (Smillie *et al*., 1989). At high concentrations, they decrease pathogen growth and sporulation directly (Wilkinson *et al*., 2001a,b; Garbelotto *et al*., 2009). At low concentrations, they act indirectly by stimulating host defences. There is some evidence that host defences may be stimulated by changes in pathogen metabolism, rather than by an effect of phosphonate on the host itself. Low concentrations of phosphonates have been found to alter the metabolism of *Phytophthora* spp. without slowing hyphal growth (Grant *et al*., 1990).

Phosphonates have long been observed to have varying actions among individual hosts; one hypothesis is that the chemicals act primarily to stimulate extant host defences (Grant *et al*., 1990; Guest and Bompeix, 1990; Daniel and Guest, 2005). A study by Pilbeam *et al*. (2011) compared the histological responses to phosphonates of resistant and susceptible lines of infected eucalyptus and revealed that while phosphonate application

Table 25.4. Eradication strategies available to control oomycete diseases caused by *Phytophthora* spp.

Eradication strategy	References
P. cinnamomi	
Physical and herbicidal removal of all plants to 4–10 m beyond disease front; root barriers; triadiazole and metalaxyl treatments of soil surface; fumigation of deep soil	Dunstan *et al*., 2010
Locally successful; landscape effects unknown	
P. ramorum	
Physical, herbicide, and burn removal of hosts within 100 m of infestation; aerial monitoring for new sites	Goheen *et al*., 2002; Kanaskie *et al*., 2006, 2008; Hansen *et al*., 2008
Locally successful, but spread continues at landscape level	

Table 25.5. Strategies for protection against and therapy for Phytophthora and Pythium diseases.

Protective or therapeutic strategy	References
Biological control	
Composts and organic soils suppress Phytophthora and Pythium diseases, as does the planting of plant hosts which act to reduce environmental spore loads	Hoitink *et al.*, 1977, 1997; Shea *et al.*, 1978; Hardy and Sivasithamparam, 1991; D'Souza *et al.*, 2004; Fichtner *et al.*, 2009
Copper compounds	
Give surface protection and have a long use history; no therapeutic benefit; ecotoxic	McCallan, 1949; Hirst *et al.*, 1961; Frank *et al.*, 1976; Heitefuss, 1989; Erwin and Ribeiro, 1996; Howard *et al.*, 1998; Pietrzak and McPhail, 2004; Bünemann *et al.*, 2006; Garbelotto *et al.*, 2009
Phenylamide compounds (e.g. metalaxyl)	
Active only on oomycetes, by inhibitin g RNA synthesis; resistance in pathogens is common; protective and therapeutic; inhibit spore production and chlamydospore germination	Cohen and Coffey, 1986; Erwin and Ribeiro, 1996
Phosphonate compounds	
Preventive and therapeutic effects; act directly to suppress pathogen and indirectly to stimulate host defences	Cohen and Coffey, 1986; Smillie *et al.*, 1989; Grant *et al.*, 1990; Guest and Grant, 1991; Tainter and Baker 1996; Jackson *et al.*, 2000; Wilkinson *et al.*, 2001a,b; Dobrowolski *et al.*, 2008; Suddaby *et al.*, 2008; Garbelotto and Schmidt, 2009; Garbelotto *et al.*, 2009; King *et al.*, 2010; Pilbeam *et al.*, 2011

equalized responses in resistant and susceptible lines after several days, their early histological responses were quite different. Responses were stronger in the susceptible line, and included increases in lignin and suberin production. The resistant line increased suberin alone, and the stimulation of mitosis and callus formation was observed in both lines.

In the field, 75% of tanoak trees in 32 plots that were treated once annually with a bark application of phosphonate (plus a surfactant) as described by Garbelotto and Schmidt (2009) were healthy after 5 years, compared with only 55% of untreated trees (M. Garbelotto and D.J. Schmidt, 2011, unpublished results).

Pathogen resistance to phosphonates is believed to be unlikely or slow to develop because of its complex mode of action, yet reduced sensitivity to the chemical has been documented. In particular, Dobrowolski *et al*. (2008) were able to experimentally select for phosphonate resistance in *P. cinnamomi* isolates, and documented reduced sensitivity to phosphonate in isolates originating in sites with long-term histories of phosphonate usage.

Systemic chemical protection: phenylamides

Phenylamides, of which metalaxyl is the most commonly used, are water soluble and are rapidly translocated into plant tissues from roots, shoots and leaves. They act by inhibiting ribosomal RNA synthesis in oomycetes, and *only* in oomycetes (Erwin and Ribeiro, 1996). They can be very effective at inhibiting sporangial production and chlamydospore germination at concentrations as low as 1 μg ml^{-1} (Cohen and Coffey, 1986). Resistance to these compounds has become a major problem, however, and necessitates the combination with or alternation of metalaxyl with other fungicides. Metalaxyl was found to be effective in controlling *P. ramorum in vitro*, but drenches were not effective for controlling sudden oak death on California coast live oak *in planta* (Garbelotto *et al*., 2009).

Surface chemical protection: copper compounds

Copper sulfate, as the famous Bordeaux mixture, has been used to control plant disease since the very beginning of plant pathology. Copper compounds are used extensively as foliar sprays and trunk paints to control aerial *Phythophthora* spp., but they offer a strictly protective function, having no action once infection has occurred (McCallan, 1949; Heitefuss, 1989; Erwin and Ribeiro, 1996). Copper hydroxide is effective in controlling ramorum blight on California laurel leaves, resulting in almost complete reduction of lesions on treated leaves for at least 4 weeks after applying a foliar spray at 460 μg ml^{-1} (Garbelotto *et al*., 2009). Soil treatments of copper sulfate have been shown to have some protective effect against *P. cinnamomi*: the pathogen was not fully killed by copper sulfate treatments *in vitro* or in gravel and topsoil, but reductions in plant infection rates were observed in treated soil (Howard *et al*., 1998).

Copper is an environmental pollutant, and copper compounds have negative long-term impacts through their accumulation in soil and accompanying heavy metal toxicity (Hirst *et al*., 1961; Frank *et al*., 1976; Bünemann *et al*., 2006). In soil layers which are slow to turn over, copper may be available to leaching for very long periods of time (Pietrzak and McPhail, 2004).

Biological protection

The role of biological compounds in protection from oomycete diseases is highlighted by the suppressive action of composts on *Phytophthora* spp. and *Pythium* spp. Unsterilized composts may suppress a number of *Phytophthora* and *Pythium* species (Hoitink *et al*., 1977, 1997; Hardy and Sivasithamparam, 1991); documentation of suppression by sterilized soils is more rare (Fichtner *et al*., 2009). Consequently, non-sterilized composts should not be incorporated into container media, as *Phytophthora* spp. could be spread in stock that is infested but made symptomless by suppression (Hardy and Sivasithamparam, 1991).

Plant species may also be used for biological control. *Acacia* spp. have been documented to suppress *P. cinnamomi* sporulation (Shea *et al*., 1978) and to protect bull banksia from infection by *P. cinnamomi* when they were planted together in a field experiment (D'Souza *et al*., 2004). Before being deployed to either reduce the number of susceptible hosts or to actively suppress the pathogen, a plant species must be determined to be truly resistant. Hosts with asymptomatic infections will not have the desired effect; these species will rather allow inoculum to continue to build (D'Souza *et al*., 2005).

25.5.5 Resistance

A breeding programme for host resistance to manage *P. lateralis* disease in Port Orford cedars that relies on results from multiple inoculation techniques has experienced recent success, with resistant genotypes now commercially available (Sniezko *et al*., 2012; Table 25.6). The success is a result from a concerted effort to identify resistant trees from survivors in diseased areas that begun in 1996, followed by propagation, crosses and multiple inoculation assays, and importantly, by a combination of greenhouse testing with field validation (Oh *et al*., 2006; Sniezko, 2006). The programme has focused on quantitative (partial) resistance, which in this and other systems has been less likely to be overcome by the evolution of virulence in the pathogen (Carson and Carson, 1989; Kinloch *et al*., 2008).

Heritable variation in resistance to oomycete pathogens has been observed in other systems. Resistance in Monterey pine to littleleaf disease has been a consideration for breeding since its documentation in 1984 (Butcher *et al*., 1984). Selection and propagation efforts are likewise underway for chestnut trees resistant to ink disease caused by *P. cinnamomi* and *P. cambivora* (Robin *et al*., 2006; Miranda-Fontaina *et al*., 2007), while a genomics screening effort is in progress to find molecular markers for resistance to *P. cinnamomi* in chestnut that may be used for early screening (Olukolu *et al*., 2012). A project

Table 25.6. Use of resistance to manage oomycete diseases caused by *Phytophthora* spp.

Resistance programme	References
Chestnut (*Castanea* spp.) resistance to *P. cinnamomi* and *P. cambivora* ink disease Jarrah (*Eucalyptus marginata* Donn ex Sm.) lines resistant to *P. cinnamomi* Monterey pine (*Pinus radiata* Donn ex Sm.) resistance to *P. cinnamomi* Port Orford cedar (*Chamaecyparis lawsoniana* (A. Murray bis) Parl.) resistant to *P. lateralis* based on multiple inoculation techniques; lines commercially available Tanoak (*Lithocarpus densiflorus* (Hook. & Arn.) Rehder) resistance to *P. ramorum*	Butcher *et al.*, 1984; Stukely and Crane, 1994; Oh *et al.*, 2006; Robin *et al.*, 2006; Sniezko, 2006; Miranda-Fontaina *et al.*, 2007; Stukely *et al.*, 2007; Hayden *et al.*, 2010; Olukolu *et al.*, 2012; Sniezko *et al.*, 2012

to survey variation in quantitative resistance to sudden oak death in tanoaks throughout their range had identified families that perform better than average in laboratory and field trials; these families have potential to form the basis of a resistance breeding programme (Hayden *et al.*, 2010, 2011).

25.5.6 Therapy

Phosphonate and phenylamide compounds may be used for therapy as well as for protection (Table 25.5). In New Zealand, stands of Monterey pine were rehabilitated by the aerial application of phosphonates applied at 568 kg ha^{-1} (Tainter and Baker, 1996). The application resulted in a suite of improvements that together limited the pathogen: crown improvement, root rejuvenation and mycorrhizal gains; and improved soil aeration and reduced waterlogging because of improved crown condition and increased transpiration. Phosphonate injections of oaks and tanoaks (Plate 31) can provide therapy if applied in the early stages of the disease (Garbelotto and Schmidt, 2009). Resistant and susceptible lines of eucalyptus seedlings show histological changes in response to phosphonate treatment after infection by *P. cinnamomi*; these promote callus formation by stimulating mitosis and the production of lignin (resistant lines), and lignin and suberin (susceptible lines) (Pilbeam *et al.*, 2011). In grass-tree (*Xanthorrhoea australis* R. Br.) seedlings, potassium phosphonate limited the growth of *P. cinnamomi* to the root cortex and the cell membrane retracted from the cell wall. Additionally, phenolic compounds and electron dense substances were deposited around the wall of infected and neighbouring cells. In contrast, in seedlings not treated with potassium phosphonate, hyphal growth became intracellular and progressed into the vascular tissue (Daniel *et al.*, 2005). At a broader scale, phosphonate applied as a foliar spray has been shown to control *P. cinnamomi* in a range of Australian native plant species in two natural plant communities (Tynan *et al.*, 2001).

Metalaxyl is especially useful for therapy because of its inhibitory effects on spore production and chlamydospore germination (Cohen and Coffey, 1986), and its rapid translocation to new tissues (Erwin and Ribeiro, 1996). Widespread problems with resistance do limit its usefulness though.

The amendment of soils may have some therapeutic effect. Tainter and Baker (1996) recommend 5–10–5 commercial (NPK) fertilizer at 2273 kg ha^{-1} applied with 454 kg ammonium sulfate and repeated every 4 years for the therapy of pine trees in the early stages of development of littleleaf disease. Increased nitrogen offsets the declining crown, and may be sufficient to maintain the trees for some time. An especially conducive year for disease may offset any therapeutic effect, however.

25.5.7 Integrated disease management

Oomycete diseases are so widespread and so versatile that no one management tactic can be completely successful. Any site-level management plan must take into account the environment, the host, the pathogen and human activities, and work to reduce inoculum load, likelihood of infection and severity of the disease all together.

From a broad perspective, nurseries are the most important venues for disease management because of their role in incubating and spreading pathogens worldwide (Hardy and Sivasithamparam, 2002; Brasier, 2008; Frankel, 2008). Hardy and Sivasithamparam (2002) provide recommendations for nursery sanitation that should be followed for all commercial operations, whether for forest trees or ornamentals. While composts and chemical treatments may be useful to suppress disease in natural settings, they should be avoided in nursery settings because of their potential to disguise infected but asymptomatic plants that are harbouring resting spores (Hardy and Sivasithamparam, 1991). If they are to be effective, enacting limitations on the movement of pathogens from nurseries should not rest solely on foresters, but must include the landscape industry and components of public education and public policy.

Management programmes for outbreaks of soil-borne Phytophthora disease in wildlands have depended on regulatory and local support, as well as on extensive monitoring, sanitation and quarantine programmes (Hansen *et al*., 2000; Colquhoun and Kerp, 2007; Frankel, 2008; Alexander and Lee, 2010). Large-scale management of aerial Phytophthoras provides a special problem because of the potential for long-distance dispersal and complex host relationships. Mbah and Gilligan (2010) and Ndeffo Mbah and Gilligan (2010), using the *P. ramorum* epidemic in the USA as a model system, provide decision-making models for the allocation of detection, eradication and treatment resources, recommending equal effort towards detection and eradication, with treatment directed at infectious foliar hosts.

Successes in Western Australia demonstrate that the management of Phytophthora disease can be accomplished on a landscape scale, given enough scientific knowledge and political will. While the number of known *Phytophthora* spp. is increasing steadily, an understanding of the ecology of soil-borne and aerial Phytophthoras is also growing, allowing further avenues for management (Rizzo *et al*., 2005). Future management successes will depend on the combination of an increasing body of research with the political will to mount a coordinated response.

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