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FOREST PATHOLOGY

A0005

Root and Butt Rot Diseases

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Introduction

P0005 Diseases caused by root rots figure prominently amongst the most-studied pathologies of forest trees. Indeed, root and butt rots cause more economic damage to commercial forestry in the temperate world than any other known type of disease. While this notoriety underlines the negative impact of this type of disease on timber production, a much more dynamic and positive role can be assigned to root rots in natural ecosystems. Root rots are one of the driving forces ensuring spatial and temporal diversification of forests. While root rots as a whole encompass both generalistic and host-specific pathogens, aggressive primary microbes, and secondary opportunistic ones, their overall effect at the stand level is to accelerate, and sometimes cause, a patchiness in which some tree species are preferentially affected and weaker individuals culled. Because of their selectivity at the species and at the individual tree level, root diseases play a significant role in determining the structure and composition of a forest. Individual trees and/or clusters of individuals are taken out, and the gaps created allow for tree regeneration. Often, more resistant seral species will substitute the more susceptible pioneering species leading to forest succession. Ecotones between gaps and closed canopy offer rich and diverse habitats, home to a substantial amount of the local biodiversity. Finally, a further outcome of root rots is nutrient recycling: this is achieved by breaking down the chemically complex woody substrate in a synergistic activity with other wood decay fungi, bacteria, and wood-boring insects.

P0010 As stated above, concerns about root rots become serious when timber production is involved. Unfortunately, most habitat modifications, including but not limited to those related to logging, appear to increase the damage caused by root rots. In many cases, human activities allow for the establishment of or increase in root rots. Once disease is established, an irreversible process starts in which root rots will play a significant role in shaping the future of that forest. When root diseases affect ecosystems characterized by poor soils, limited host variability, or limiting climatic conditions, their impact may be significant even in the absence of further human activities.

Causal Agents of Root Rots: Establishment Strategies and Population Genetics

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P0015 Root diseases can be caused by a wide range of organisms including oomycetes, ascomycetes, and basidiomycetes. Root and butt rots, instead, are exclusively caused by fungi belonging to the homobasidiomycetes. The three genera *Armillaria*, *Heterobasidion*, and *Phellinus* have broad worldwide distribution and probably are responsible for the majority of root diseases in temperate forests. Other less frequently encountered genera include *Inonotus* and *Phaeolus*.

P0020 All of the known root rotting organisms reproduce sexually. A fertile cell layer (called the hymenium) borne by the sexual fruit bodies of these fungi produces haploid meiospores. Hymenia can be porous or gilled (**Table 1**). Fruit bodies produced by root rot fungi can be an excellent diagnostic clue and include true mushrooms (*Armillaria*), bracket or shelflike conks (*Heterobasidion*), and relative inconspicuous resupinate fruit bodies entirely supported by the surface on which they are growing (*Phellinus weirii*) (**Table 1**). It should be noted that by the time the fruit bodies are produced, root rots are already in an advanced stage. Most root rot agents can be cultured, and in culture some produce asexual mitospores called conidia that may have diagnostic value.

P0025 In general, airborne basidiospores are the main means for primary infection by a root rot pathogen, and these allow for the infestation of new areas. Vegetative or somatic spread of the fungal mycelium is the main means for secondary spread, allowing for the expansion of those individuals originally established through the primary infection process. This expansion may occur either by tree-to-tree contagion through root contacts and grafts, or by free growth of the pathogen in the soil through specialized structures such as mycelial cords or rhizomorphs.

T0005 **Table 1** A summary of characteristics of some important root rot diseases

Pathogen	*Disease*	*Hosts*	*Range*	*Decay*	*Fruit body*
Armillaria spp. complex	Oak root fungus; honey mushrooms	Very broad range, both angiosperms and conifers	Worldwide	White stringy	Fleshy gilled mushroom
Heterobasidion spp. complex	Annosus root disease	Primarily conifers, some angiosperms	Worldwide	White laminar	Perennial and annual porous brackets
Phellinus weirii spp. complex	Laminated root rot	Conifers	Western North America, Eastern Asia	White laminated decay, pitted	Resupinate porous
Inonotus tomentosus	Tomentosus root disease	Spruce, pines	Pacific Northwest, USA–Canada	White	
Phaeolus schweinitzii	Velvet top fungus	Conifers	Worldwide	Brown cubicle	Fleshy, velvety corrugated, porous, short or no stipe

Root rotting basidiomycetes colonize a substrate and utilize it as they are growing. In this light, they can be considered territorial organisms, which physically occupy an area and do not easily allow other conspecific organisms to coexist in the same niche. While this phenomenon can be explained partially by the depletion of available nutrients by the pioneering individual, it has been shown that this exclusion of other individuals starts significantly earlier than nutrient depletion. Like most other wood-rotting basidiomycetes, species causing root rots have developed a multilocus system called vegetative (or somatic) incompatibility (VI). When two individuals belonging to the same species meet, a chemically mediated reaction results in the inhibition of growth of both individuals. The area between the two colonies is characterized by sparse or no fungal growth and represents one of the territorial borders of fungal clones or genets. Self-protection from viruses or pathogens present in other individuals is another function attributed to VI systems.

P0030 The VI system is active only in diploid ($2n$) and dikaryophytic ($n+n$) isolates. In dikaryons, the two parental nuclei pair up but do not undergo karyogamy. The lack of an active VI system in haploids allows mating to occur. Mating does not involve any specialized sexual structures but is attained by the simple fusion of vegetative haploid hyphae. The resulting $2n$ or $n+n$ thallus is long lived and leads to the production of fruit bodies and of meiospores. The haploid phase in nature is regarded as relatively short-lived and unfit, but some species, especially those relying heavily on primary infection (e.g., *Heterobasidion* spp.), are known to have long lived and virulent haploid phases.

P0035 Genetic studies on the clonal distribution of most root disease organisms have often shown complex patterns of colonization characterized by areas colonized by several individuals. In some cases both haploids and dikaryons coexist and there is evidence of genetic exchanges among individuals. VI barriers have thus to be interpreted not as absolute boundaries but as dynamic areas with potential interspecific interchanges.

P0040 Some root disease organisms, especially within the genera *Armillaria* and *Phellinus*, show a remarkable genetic homogeneity over large areas. In some cases, tens of acres have been shown to be colonized by a single individual, secondarily spreading among root systems of an entire forest for thousands of years. The case of a 33-acre (13.4 ha) genet of *Armillaria bulbosa* in northern Michigan has been reported as the largest living organism on earth. This case exemplifies a root disease organism for which secondary infection plays a very significant role and appears to be a much more frequent event than primary infection.

P0045 The relative importance of primary vs. secondary infection is of paramount importance not only for understanding the biology and epidemiology of root diseases but also for management purposes. Pathogens like *Armillaria bulbosa* or *A. mellea* have the ability to spread secondarily over significant distances. When these organisms colonize woodlands extensively, there is often a carry-over of the pathogen into future generations; in these cases, new mortality may not necessarily be caused by new primary infection, but by the remnant of infections established at the site in previous rotations. The clear-cutting of infested stands may not resolve the problem, as most root disease organisms will survive for decades in larger woody debris and in stumps. A study of *Armillaria* infections in the south of France showed that the disease spread radially from a source point in all directions, causing a classic root disease center. Natural regeneration of the stand would

always occur in the middle of the infection center, after the root disease had expanded and the pathogen in the oldest infection area was no longer viable. Interestingly, as the nucleus of regeneration expanded, its outer margins would eventually overlap with the outer margins of the disease center, where the pathogen was still viable. These regeneration patches would then become infected in the areas of overlap and start dying back inwards towards the center of the infection center.

P0050 Plantations of forest trees or woody crops in areas previously infested by a root disease like *A. mellea* may result in regeneration failures. In these cases, it may be important to identify and isolate sources of secondary infection, while ensuring trees are vigorous and less prone to become infected. When dealing with root diseases characterized by abundant primary infection events such as *Heterobasidion* spp., forest management should focus on minimizing those activities likely to create good primary infection courts, e.g., wounds on roots, stems and branches, and stumps. In general, heavy use of the land and logging will exacerbate these types of root diseases, with losses becoming apparent only after several years.

F0005 **Figure 1** A section of the cut stump of a white fir (*Abies concolor*) in a California site infested by the root pathogen *Heterobasidion annosum.* A white laminated decay caused the rot pockets visible in the sapwood. Trees affected by sapwood rot will have reduced vigor and may decline rapidly at the onset of further problems.

S0015 Symptoms Caused by Root Diseases

P0055 The lag between infection and the visible development of symptoms is due to the obvious fact that most symptoms caused by root diseases are in the roots. It has been shown that up to 50% of the root system needs to be affected before any symptoms of tomentosus root disease may be visible above ground. Colonization of the root system may thus go undetected for long periods of time. Although roots of all sizes can be infected by root pathogens, the most reliable underground symptom is the decay of woody roots. Root diseases are equipped with many enzymatic systems and can cause a brown (lignin not being utilized by the pathogen) or a white (lignin utilized by the pathogen) rot. Root rots can also be further characterized by being cubicle, pitted, laminar, and so forth. **Table 1** summarizes the types of rots caused by some important root diseases. Roots can be decayed in two ways: either by starting from the cambium (root girdling) and then proceeding inwards (sapwood rot) (**Figure 1**), or by decaying the central portion of the root (heart rot). The first pattern will result in the rapid death of infected roots and of the root collar, and in a relatively rapid death of the host by girdling. The second type may kill the smaller woody roots by physically breaking them down, but the larger ones may be hollowed and remain physiologically functional for long periods of time. Infection will proceed into the tree sapwood (or in some cases the heartwood), resulting in a physiological loss of vigor due to the loss of functional outer sapwood. Sapwood infection might make trees more vulnerable to other pests, diseases, and unfavorable climatic conditions. This type of decay, when advanced and extensive, may result in the significant weakening of the roots and lower bole of infected trees. Infected trees will be mechanically compromised and more likely to crash due to the effects of wind, rainstorms, or snowfall.

P0060 These two major patterns of root rotting are often determined not only by the pathogen species, but also by the host being infected. It is not uncommon to have the same pathogen species colonizing roots in two different ways when infecting two different hosts. Patterns of butt decay will also depend both on species of the pathogen and of the plant host. Not all root infections proceed into the tree butt. When surveying for incidence of root diseases, results have been different depending on whether surveys were based on root sampling or basal wood coring of tree buttresses. At any rate, root diseases figure as one of the most significant causes of cull in timber production, and the problem intensifies with each new rotation.

P0065 The time lag between root infection and the onset of symptoms above ground may vary depending on climate, host and pathogen species, and tree age.

Smaller trees with smaller root systems may die rapidly after infection, even within the same season. In contrast adult trees may survive for decades after infection. When infection levels start affecting tree physiology and vigor, visible symptoms can be seen in the crown. Infected individuals will display slower growth. Tree crowns will appear thin as a result of both the slower growth and the shorter retention time of leaves. For instance, pines infected by a root disease may only keep needles produced in the current year while 2- and 3-year-old needles may be prematurely dropped. A single cluster of 1-year-old needles at the end of a barren branch gives these branches the so-called "lion's tail" appearance. Root infection will also result in a shift in the color of the foliage, which generally appears less vibrant than the foliage of healthy individuals.

P0070 All of the above symptoms indicate trees with significantly altered physiology. The obvious question is whether such symptoms may always be considered as the effect and not the cause of infection by a root disease. The issue of causality is an important one and it has not been fully investigated. There are several studies indicating a strong correlation between physiological stress and infection by root diseases. These studies have shown that infected trees grow slower, that trees exposed to air pollution are more likely to be found infected, and that short-term draughts increase the frequency of trees with root diseases. Finally, trees affected by root diseases will be more attractive to bark and ambrosia beetles, and may provide an initial substrate for the growth of beetle populations that may then overflow and affect healthy trees. While there is enough evidence to suggest that these symptoms are the effect of infection by root diseases, there is still the possibility that these pre-existing symptoms may facilitate infection by root pathogens, and this awaits further clarification.

P0075 An interesting perspective, when correlating root diseases and tree stress, may be to differentiate between primary aggressive pathogens and secondary less virulent ones. While primary pathogens can cause significant disease and alter the tree physiology with or without pre-existing tree stress, secondary ones require a pre-existing or concomitant stress factor (including other pathogens) in order for disease to develop. In the Sierra Nevada of California, for instance, rhizomorphs of the nonaggressive *A. gallica* can be found on the surface of the roots of large numbers of true firs (*Abies* spp.). Although the rhizomorphs are growing ectotrophically on the roots, there is no or little associated decay. *Armillaria* decay, though, may start soon after the same roots are infected by the more aggressive pathogen *Heterobasidion annosum*.

P0080 Those rhizomorphs could almost be interpreted as exploratory outposts ready to capitalize on the availability of weakened trees. This exploratory function of a secondary pathogen, although apparently extremely costly, allows for the rapid utilization of a substrate that may otherwise be fully colonized by the aggressive primary pathogen or other competitors. The above example highlights another important feature of root pathogens capable of secondary (vegetative) spread: i.e., their ability to maintain a functional networks covering a sizeable area and several plants. Most of the network may be maintained at comparable levels until resources become available in one spot. At that point, most of the resources of the network are dynamically allocated to increase the utilization of that substrate. The apparent new attack on a host (e.g., a weakened tree), may be the result of energy reallocation through an already established network, rather than being the result of the advancement of an infection front.

P0085 At later stages, symptoms of root disease include progressive dieback of the crown, starting from the top downwards, and the presence of signs of decay. Fungal fruit bodies are produced either on the colonized woody substrates (normally at the root collar or on the roots) or on the duff layer thanks to the presence of masses of finer roots. Rhizomorphs are specialized structures produced by many *Armillaria* species; they consist of a strand of hyphae encased by a highly hydrophobic melanin layer. Rhizomorphs, commonly referred to as "shoestrings" because of their appearance, can grow freely in the soil and allow the pathogen to move from tree to tree without the need to follow root contacts. The consistency, thickness, and cross-sectional structure of rhizomorphs can be a useful diagnostic tool to differentiate among *Armillaria* species. Rhizomorphs are commonly found on the roots and root collars of host trees, but they can also be found on several meters of the main bole, growing under the bark. Another sign of decay associated with root diseases is the presence of mycelial mats. *Armillaria* mycelial mats appear as fans radiating under the bark from the root collar. Wood in an advanced state of decay will present cavities often colonized by the white mycelium of root pathogens like *Heterobasidion* spp. or *Phellinus weirii*. The mycelium of the latter species is characterized by the presence of hair like structures called setae. Advanced decay also results in obvious punks and defects in the wood, often associated with resinosis, sap bleeding, or wetwood.

P0090 Root disease centers can be differentiated from clusters of trees killed by pest outbreaks or abiotic factors because tree mortality is not synchronous but occurs over a period of time. In the middle of the root disease center, there are those trees initially killed by the disease; some of them may have already been windthrown. More recent mortality may be present in a ring encircling the older mortality. Recently killed trees will still bear most of the dead foliage, while relatively older mortality will be characterized by trees made barren by winter storms. Symptomatic live trees may represent the visible edge of the root disease center (**Figure 2**). Studies on control options aimed at halting the secondary spread of the root pathogen *Heterobasidion annosum* in ponderosa pine (*Pinus ponderosa*) stands have highlighted the fact that the pathogen had already infected several tiers of apparently asymptomatic trees outward and beyond the apparently symptomatic ones. Root disease centers may thus be larger than judged by the presence of visible symptoms. It has been noted that enlargement of root disease centers does not proceed indefinitely but eventually stops. The factors regulating the spread of root disease centers are only marginally known.

Figure 2 A typical root disease center caused by *Heterobasidion annosum* in the mixed conifer forest of the Sierra Nevada, California. While true firs appear symptomatic or dead, pines (distinguishable by the longer needles) are not affected. This species of the pathogen in fact is specialized on true firs, Douglas-fir, and sequoias, and is very different from the species found on pines. Stumps and trees dead for several years are visible in the center, the location where the pathogen had originally established itself. Secondary growth leads to an expansion of the mortality center. Symptomatic trees and trees recently killed by the pathogen (note the brown–orange foliage still borne by the branches) mark the expanding edge of the pathogen. F0010

S0020 The Ecology of Root Diseases

P0095 While root pathogens vary in their ability to freely grow in the soil (**Table 1**), their biology is intimately linked to its properties. In general, loose, well-drained soils, poor in organic matter, are extremely conducive to the development of root diseases. The ecological and trophic requirements of these pathogens are rather species-specific, and root pathogens have evolved to minimize spatial overlap amongst individuals from the same species or from species having identical requirements. When requirements differ, spatial overlap may occur, as each species may be utilizing a different niche. In general, primary infection of root pathogens is successful only during the very initial stages of decay. In the case of conifer stumps infected by *Heterobasidion annosum*, infection rates drop dramatically each day after logging and become fairly minimal after 5 or 6 days. *Heterobasidion* spp. are not good competitors and need to arrive on substrates that are relatively available. Thus other wood-inhabiting fungi have the ability to exclude this pathogen from a substrate otherwise available. This basic observation has resulted in a biological control approach prescribing the application of strong competitors on the wood surface at the time of logging. Because with time there is a sharp decrease in infection success by spores of this pathogen, competitors only need be active for a few days in order to be effective.

P0100 Precipitation requirements may differ among root pathogens but in general mild temperatures and good precipitation favor sporulation. Dry weather, or temperatures under 10°C or above 25°C, are in general unfavorable to sporulation and infection alike. Excessive rain may also be unfavorable to root pathogens relying mostly on airborne inoculum (e.g., *Heterobasidion*). In the case of pathogens relying more heavily on secondary spread (e.g., *Armillaria*), heavy rains can at times cause a temporary anoxia in

the root systems and trigger a more aggressive root colonization by the pathogen. In areas with cold and dry winters, sporulation by *Heterobasidion* spp. is extremely low. Sporulation by the same species in areas with mild winters continues throughout the year; winter sporulation is relatively abundant in areas where precipitation, even if in the form of snow, occurs mostly in the winter season.

P0105 Although fruit bodies of most root pathogens produce unimaginably large numbers of basidiospores, their role is not always clear. This is particularly true for root pathogens relying heavily on secondary spread. While basidiospores can travel a long distance, sometimes on the order of hundreds of kilometers, spore densities undergo a huge dilution after the first few meters. The likelihood of primary infection is thus directly correlated with distance from a source point. While the migration of even a few spores may be significant for areas still not colonized by a root pathogen, it is unlikely the few spores from a distant source would have a huge impact on the large number of locally produced spores.

P0110 The presence of marked host specificity among morphologically indistinguishable populations of the same pathogen is prominent in root diseases. In fact the discovery of host specificity within the broad host range of pathogens *Armillaria* and *Heterobasidion* was a milestone towards the better understanding of the concept of biological species for the fungi. This host specificity was backed up by genetic studies indicating that populations specializing on different groups of hosts were genetically isolated from one another. The term intersterility group (ISG) was coined in the late 1970s to describe these host-specialized and reproductively isolated root pathogen populations. In the case of *Heterobasidion annosum*, a series of crosses between the two North American ISGs revealed for the first time the genetic system regulating intersterility and consequently speciation among these host specialized groups. Nowadays, thanks to the advancement of our genetic techniques, most of these biological species have been elevated to the rank of species (**Tables 2–4**).

Host specificity has several relevant consequences: P0115

1. In a mixed forest not all trees species will be equally susceptible.
2. Disease severity in a mixed forest should be less than in a forest comprising a single susceptible species.

T0010 **Table 2** Details on some common taxa within the *Armillaria* species complex

Pathogen	*Main hosts*	*Range*	*Disease*	*Primary pathogenicity*[a]
Armillaria ostoyae	Conifers	Worldwide, circumboreal	Cambium and sapwood rot	+ + +
Armillaria borealis	Conifers	Northern Europe, Asia	Butt rot	+
Armillaria calvescens	Hardwoods	North America	Butt rot	+
Armillaria cepistipes	Hardwoods	Europe, North America, Japan	Butt rot	+ +
Armillaria mellea	Hardwoods, conifers	Worldwide, circumboreal	Cambium and sapwood rot	+ + +
Armillaria gallica	Hardwoods	Worldwide, circumboreal	Heartrot and sapwood rot	+ +
Armillaria sinapina	Hardwoods, conifers	North America	Heartrot and sapwood rot	+
Armillaria luteobubalina	Hardwoods	Australia	Heartrot and sapwood rot	+ +

[a] Refers to ability of killing hosts: + + +, more aggressive (primary pathogen); + +, less aggressive; +, secondary pathogen.

T0015 **Table 3** Details on taxa within the *Heterobasidion annosum* species complex

Pathogen	*Main hosts*	*Range*	*Disease*	*Primary pathogenicity*[a]
H. annosum	Pines, spruce	Europe, Asia	Root and root collar girdling	+ + +
H. parviporum	Spruce	Europe, Asia	Heartrot	+
H. abietinum	True fir	Central and southern Europe	Sapwood rot and heartrot	+ +
H. annosum P ISG	Pines, incense cedar, juniper	North America	Root and root collar girdling	+ + +
H. annosum S ISG	True firs, sequoia, Douglas-firs	Western North America	Sapwood rot and heartrot	+ +

[a] Refers to ability of killing hosts: + + +, more aggressive (primary pathogen); + +, less aggressive; +, secondary pathogen.

T0020 **Table 4** Details on taxa within the *Phellinus weirii* species complex

Pathogen	*Main hosts*	*Range*	*Disease*	*Primary pathogenicity*[a]
Phellinus weirii	Western redcedar	Western North America	Sapwood rot and heartrot	+ +
Phellinus henrichii, North American biological species	Douglas-fir, mountain hemlock, and other conifers	Western North America	Root girdling and sapwood rot	+ + +
Phellinus henrichii, Asian biological species	Conifers	Japan, Siberia	Root girdling and sapwood rot	+ + +

[a] Refers to ability of killing hosts: + + + more aggressive (primary pathogen), + +, less aggressive.

3. Susceptible species may be replaced by more resistant ones (succession).

P0120 It should be noted that host specificity might be relaxed when infection occurs on stumps rather than on live trees. This phenomenon has been reported for *Heterobasidion* species both in Europe and North America. In the case of this pathosystem, stumps may have a triple effect: (1) they greatly enhance primary infection, (2) allow for the establishment of a pathogen species once rare, and (3) allow for two or more species to grow in the same habitat. This close proximity, rarely present in nature, where different species are segregated on their specific plant hosts, has resulted in interspecific hybridization and gene introgression with unpredictable outcomes for California forests. Furthermore, as stated above, once stumps are infected, they can be the source of inoculum for several decades. Fruit bodies may in fact proliferate on stumps, and at the same time the infection of the stumps' root system represents a source of secondary infection.

S0025 ## Root Rot Pathogens Shape our Forests

P0125 Forest management and the resulting forest structure and composition have a significant impact on root disease epidemiology, and conversely root disease will affect forest structure and composition. Disease severity tends to be higher in forests frequently logged and thinned, in forests with higher tree density, and in older forests. Logging and thinning provide either an abundance of primary infection courts (e.g., for *Heterobasidion annosum*) or may facilitate the secondary root colonization of the remaining stumps by *Armillaria* spp. Denser stands will facilitate secondary spread directly by making root contacts more abundant, and indirectly because of the overall reduced vigor of trees growing in overcrowded stands. Older stands are subject to greater amounts of root diseases; this is due to several factors including the greater availability of a substrate (i.e., roots) in larger trees, the tendency of older trees to be more prone to decay processes, and the loss of structural integrity allowing for some secondary root rots to colonize trees previously unavailable. Finally, larger trees will favor extensive secondary spread because of the presence of larger and far-reaching root systems. Furthermore it has been shown that some root rots grow faster in larger roots. This relationship appears to be true especially if the pathogen is not capable of growing ectotrophically on the root surface (*Heterobasidion*, *Phaeolus*): if ectotrophic growth is possible (*Armillaria*, *Phellinus*), the pathogen may not be limited by the presence of thin roots or the absence of root grafts.

P0130 Root diseases will in turn affect forest structure and composition. Root diseases create gaps where regeneration, often by different, light-loving, and more resistant plant species, will take place. Trees on the edge of the gaps will be released and their growth rates increased. In general, root diseases will create ecotones with increased biodiversity. It is clear that trees infected by root diseases become more attractive to bark and ambrosia beetles. Beetle attacks will generally significantly accelerate tree mortality. Diseased trees will also allow for the local growth of beetle populations. When beetle populations surpass certain thresholds, attacks may include neighboring healthy trees and expand tree mortality beyond that predictable solely by the effects of the root disease. Broad generalizations should be avoided when looking at the complex interactions between two natural disturbance agents such as root diseases and insects. In the case of *Inonotus tomentosus*, for instance, diseased trees were shown to be more attractive to beetles only where endemic beetle populations were present.

P0135 Droughts, pollution, and wind have been shown to interact with root diseases in increasing tree mortality. Root diseases will also affect nutrient cycling at the landscape level. In the case of ecosystems in harsh environment these effects may be long term. It has been determined that the openings in mountain hemlock (*Tsuga*) stands caused by the pathogen

Phellinus weirii in Oregon result in negative levels of carbon production for about 100 years. Increased nitrogen mineralization processes follow the edge of the expanding root disease center, with important consequences for growth rates of the regeneration and resistance of the remaining standing trees. In this case, therefore, the effects of root diseases may be long lasting.

P0140 In some instances, root disease centers are characterized by a complexity in the pathogen population that does not fit the picture of individual infections spreading radially in all directions as shown for *Armillaria* spp., *Inonotus tomentosus*, and *Phellinus weirii*. An alternative hypothesis has been formulated suggesting that initial infection processes may trigger a chain effect in neighboring trees (for instance by allowing more oxygen into roots of neighboring trees grafted to decayed roots) where resident infections may be latently waiting to be activated. Genetic data from true fir infection centers caused by *H. annosum* in California partially support this hypothesis.

S0030 Management Options for Root Diseases

P0145 Root diseases are virtually impossible to eradicate once they are established, and their introduction may have a long-lasting impact on forest ecosystems. These diseases may also shape the future of forests: once introduced and established they may make a return to the original type of forest an unlikely outcome. For instance, logging and fire suppression have allowed for the establishment of large mortality centers caused by *Armillaria* and *Heterobasidion* spp. in the Yosemite Valley in California. These pathogens will now in turn limit the possibility of regeneration of the forest, and are likely to lead the landscape towards a mosaic of open meadows interspersed by clusters of trees.

P0150 When forests are threatened by root pathogens, approaches can be taken to limit their establishment and spread. These interventions are often complex and long term and may be worth executing only if dealing with fragile ecosystems or when the disease has resulted in hazardous trees. Once the biology of the causal organism is understood, precautions can be taken to minimize levels of primary infection. For instance, in the case of *H. annosum* in pine, precautions include treating stumps (important primary infection courts) with chemicals such as borate or urea or, alternatively, applying the biological control fungus *Peniophora gigantea* at the time of cutting. In the case of *H. annosum* in true fir, thinning wounds may represent the most important source of primary infection courts, and thus a strategy aimed at limiting thinning entries or wounding of standing trees may be helpful. Operations should preferably be done when environmental conditions are unfavorable to the pathogen; this strategy has been adopted with some success by logging *Heterobasidion*-infested pine plantations in the southern USA during the hot summer months.

P0155 Lowering stand density may regulate secondary spread of the pathogen. Early precommercial thinning, when trees may be too small to be favorable infection courts, or the use of prescribed fires may help achieve this goal. When high-value sites are in question (e.g., campsites, public areas, etc.) a physical disruption of the vegetative growth of the fungus can be attempted. In southern California, trenching the soil and removing two tiers of apparently asymptomatic trees beyond the visible outer margin of an infection center successfully halted the spread of *H. annosum* in pine. In the case of pathogens capable of growing freely in the soil via rhizomorphs, this attempt may be futile, as portions of root systems left behind may actually be ideal colonization substrates for *Armillaria* spp.

P0160 If the removal of an individual tree is in question, pathogen identification is essential. For instance, the stump of a felled tree will provide an ideal substrate for *Armillaria* and become a source of inoculum if other trees are in the same vicinity. On the other hand complete removal of the tree, including the root system, may beneficially lower the amount of inoculum.

P0165 If a forest is affected by a pathogen with restricted or defined host range, it may be possible to increase the percentage of trees more resistant to the pathogen. Because resistance is often only partial, it may be a good idea to avoid complete removal of the susceptible species, as the resulting selection pressure on the pathogen may result in the infection of plants normally considered nonhosts.

P0170 When dealing with trees at the urban–wildland interface, avoid any horticultural treatment or practice that may result in physiological stress. Often even single events, e.g., the overwatering of a tree in the summer, may result in the colonization of that tree by a root pathogen that originally was growing ectotrophically on the roots. In some cases, when trees are affected by root disease but appear to decline very slowly, the application of insecticides on the bark before the tree is attacked may significantly prolong its life. The effects of such treatments are short-lived and valid only for conifers with a known beetle infestations. These treatments require the use of chemicals with a relatively broad spectrum of action and should be used sparingly and only in high-value landscape situations.

S0035

List of Technical Nomenclature

See also: **Entomology**: Bark Beetles (00030). **Forest Pathology**: Diseases of Forest Trees (00061); Phytophthora Root Rot of Forest Trees (00064). **Tree Physiology**: Root System Physiology (00102); Stress (00105).

Further Reading

Goheen DJ and Hansen EM (1993) Effects of pathogens and bark beetles on forests. In: Schowalter TD and Filip GM (eds) *Beetle–Pathogen Interactions in Conifer Forests*, pp. 175–196. San Diego, CA: Academic Press.

Hansen EM and Goheen EM (2000) *Phellinus weirii* and other native root pathogens as determinants of forest structure and process in Western North America. *Annual Review of Phytopathology* 38: 515–539.

Otrosina WJ and Scharpf RF (eds) (1989) *Proceedings of the Symposium on Research and Management of Annosus Root Disease* (Heterobasidion annosum) *in Western North America*, 18–21 April 1989, Monterey, CA: US Department of Agriculture Forest Service.

Shaw CG III and Kile GA (eds) (1991) *Armillaria Root Disease*. Agriculture Handbook no. 691. Washington, DC: US Department of Agriculture Forest Service.

Slaughter G and Rizzo D (1999) Past forest management promoted root diseases in Yosemite Valley. *California Agriculture* Xx–xx.

Smith ML, Bruhn JN, and Anderson JB (1992) The fungus *Armillaria bulbosa* is among the largest and oldest living organisms. *Nature* 356: 428–431.

Woodward S, Stenlid J, Karjalainen R, and Hüttermann A. (eds.) (1998) Heterobasidion annosum: *Biology, Ecology, Impact and Control*. Wallingford, UK: CAB International.