CHAPTER 19

DISEASES AND THE ECOLOGY OF INDIGENOUS AND EXOTIC PINES

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19.1 INTRODUCTION

Many of the major diseases of pine were characterized as long ago as Hartig (1874), but their role in the ecology and biogeography of this and other tree genera has gone largely unrecognized. However, with the burgeoning interest in ecologically-based management of forest resources and forest health, there has been increasing attention given to the pivotal role of pathogens in forest ecosystems (Castello, Leopold & Smallidge, 1995; Haack & Byler, 1993; Monnig & Byler, 1992; van der Kamp, 1991; Worrall & Harrington, 1988). Pines, pathogens, insects and fire have evolved complex relationships that determine the dynamics of many Northern Hemisphere ecosystems. With these relationships in mind, sound management practices can minimize losses in commercial forests or otherwise optimize the benefits of natural forests to humans. Pines planted outside of their native range often encounter new relationships and present substantial management problems, while introduced pathogens have proven devastating to other pine ecosystems.

Among the pathogens causing mortality in natural pine ecosystems, dwarf mistletoes and root diseases are perhaps the most significant. These diseases interact strongly with bark beetles and fires in maintaining the heterogeneity of the forest landscape and in driving successional patterns. Fire suppression has increased the management problems caused by dwarf mistletoe, brown spot needle blight, fusiform rust and some root diseases, while the creation of stumps during harvesting and thinning has proven advantageous to Armillaria root rot, annosum root rot and black stain root disease. Pines on degraded, old field sites have proven susceptible to another root disease, littleleaf disease of southern pines. Stem rots of living pines were substantial contributors to cull in harvesting old growth forests, but with the shorter “pathological rotations” today, they now are valued as important wildlife management tools. The roles of diseases in natural pine ecosystems have often clashed with human interests and human manipulations, but with understanding of these often subtle relationships, wiser management practices follow.

Serious losses due to disease are commonly associated with pines, either native or exotic, grown in plantations. Cankers and foliage diseases in natural forests generally have a minor impact, primarily because of the resistance in the host population that has developed through millennia of selection pressure. Monocultures and the selection by humans of fast growing species and genotypes can inadvertently shift the host population to susceptibility, e.g., fusiform rust. Pines planted outside of their native range may encounter stronger disease pressures and be devastated by diseases that are minor in their native habitat. Perhaps the best examples of such enhanced susceptibility are Sphaeropsis canker and red band needle blight. The pathogens that cause these diseases are now established in many important pine growing regions in the Southern Hemisphere and have dramatically altered forestry there.

The importance and potentially devastating impact of tree diseases was recognized early in the 20th century with severe epidemics associated with the introduction of new pathogens to native forest ecosystems. Notable examples are chestnut blight and Dutch elm disease. On pine, the first such disaster was white pine blister rust, which became established twice in North America in the early 1900s. This disease threatens to eliminate some pine ecosystems and removes white pine as a management option in others, and this has, in turn, exacerbated other disease problems. Other pine disease epidemics associated with introduced pathogens include Scleroderris canker and pitch canker, the latter of particular concern in recent years. Perhaps the most disastrous introduction was the appearance of the pinewood nematode in Japan. Unfortunately, the globalization of timber and other industries, particularly the intercontinental movement of raw timber and packaging materials (e.g., dunnage), will likely allow future introductions.

A comprehensive treatment of all diseases of pines is clearly beyond the scope of this chapter. More comprehensive coverage of the diseases of pines, their symptomatology and epidemiology can be found in Boyce (1961), Manion (1981) and Sinclair, Lyon & Johnson (1987). Our aim here is to give an overview of the major disease classes affecting pine, with an emphasis on their ecology and impact on indigenous and exotic pines. Pathogens causing tree mortality are emphasized because they are most important in the ecology of pine. The specific diseases mentioned above will be used as examples and discussed in more detail, generally discussing first those affecting the indigenous forests, then those having greater

impact in exotic plantings. Pathogens introduced to new environments will be covered towards the end of the chapter. Finally, we will conclude with an assessment of the future of diseases in the ecology and biogeography of pines.

19.2 DWARF MISTLETOE

It has long been recognized that *Arceuthobium*, a genus of parasitic higher plants, has a major effect on pine ecosystems (Weir, 1916; Gill, 1935). Heavily infected hosts die, but most losses in commercial forests are due to reduced growth rate of the diseased trees. In the forests of the Northern Region of the Rocky Mountains in the USA, there are in excess of 1.4 million hectares of infested forests, with annual losses of about 600,000 cubic meters (Monnig & Byler, 1992). Throughout the western USA, the annual losses due to dwarf mistletoe have been estimated at 7.5 million cubic meters (Hawksworth & Wiens, 1972). Seed production and wood quality of diseased trees may also be reduced. Heavily infested stands are also prone to fire.

Members of *Arceuthobium* are obligate parasites on the family Pinaceae and a few other coniferous hosts (Hawksworth & Wiens, 1996). *Pinus* is their primary host genus; 33 of the 47 recognized taxa have pines as their principal hosts, and about one-third of the pine species are affected. The genus *Arceuthobium* is widespread throughout most of the Northern Hemisphere and, to a limited extent, the Southern Hemisphere. The genus is best represented in Mexico (22 species) and the western USA. *Arceuthobium* species, like their hosts, are largely temperate plants. No species are found in low-elevation, moist, tropical forests. In the Caribbean, the only known species is *A. bicarinatum*, occurring in Hispaniola on *P. occidentalis*, a relatively high elevation pine species. *Arceuthobium hawksworthii* infects *P. caribbaea* var. *hondurensis* in the uplands of Belize. Curiously, dwarf mistletoes are not found on the pines of eastern North America. Two old world species occur on pines; *A. minutissimum* Hook. parasitizes *P. wallichiana* in the Himalayas, and *A. pini* Hawksworth & Wiens occurs on *P. tabulaeformis* in China.

The dwarf mistletoes have a unique biology (Hawksworth & Wiens, 1996). They obtain their water, minerals and carbohydrates through an elaborate endophytic system in the branchwood and phloem of the host. Usually, there is swelling of infected branches, and prolific branching of the host results in conspicuous witches' brooms up to several meters in diameter. The endophytic system may be limited to 20 cm of the host branch, or, in the case of the systemic dwarf mistletoe species, a single endophytic system may extend throughout the branches of a witches' broom. Years following infection, flowering shoots (male or female) develop each year along the endophytic system, and the fruit that develop each contain a single seed that is forcibly discharged at maturity. The seeds generally travel horizontally less than 10 m. Successful development of an endophytic system depends on the discharged seed adhering to a living needle and then sliding down the needle to the branch after the sticky coating of the seed is wetted by rain. The life cycle of the dwarf mistletoe species is 3 to 7 years, and with the limited dispersal of the seeds, the pathogen moves slowly compared to fungal pathogens on pine. Maximum spread is from an infested overstory to the understory. Lateral spread through a dense, even-aged stand is slow, less than one meter per year, even if the stand is comprised mostly of the principal host.

The dwarf mistletoes show host preferences but have curious ranges of occasional hosts. About two-thirds of the dwarf mistletoe species parasitize tree species other than their primary host, often parasitizing hosts in other genera, while species closely related to the primary host are immune (Scharpf, 1984). It has been difficult to identify resistant individuals of the primary host species. However, Roth (1966) identified a "drooping needle" race of ponderosa pine that was resistant, presumably because discharged dwarf mistletoe seeds that adhere to the needles, slide to the ground after being wetted by rains instead of sliding to the branch at the needle base, which is the normal site of infection.

Where they occur, dwarf mistletoes generally have a dramatic impact on the ecology of their hosts. Fire plays a major role in dwarf mistletoe ecology, but the relationships to fire are complex (Alexander & Hawksworth, 1975; Wicker & Leaphart, 1976). Killed trees and dead witches' brooms on the ground can serve as major fuels for fires, and witches' brooms on living trees may serve as fire ladders on which ground fires can climb to the crowns. Medium

intensity fires may develop to kill non-host species less fire resistant than the primary host and may leave infected trees for later overstory to understory spread of dwarf mistletoe seed in the regenerating stand. Alternatively, an intense fire could kill all the trees on the site and eliminate the pathogen. The invasion of the burned area by regenerating trees from the perimeter generally proceeds much faster than the invasion of the dwarf mistletoe. Thus, fire tends to sanitize the stand. But even this scenario may favor the dwarf mistletoe in the long run if the seral host species is perpetuated on the site and the non-host climax species are avoided. Dwarf mistletoes generally use pioneer or seral species as their principal hosts, and if no fire occurs in a heavily infested seral stand, the stand may be accelerated to the resistant climax vegetation.

Climate, topography, and other site factors may influence the distribution and intensity of dwarf mistletoes, but it is the dynamics of the forest that most affects the dynamics of the pathogen population (Parmeter, 1978). Fire and cutting history are critical factors. Severely infested stands may have substantial accumulation of fuel for fires, and it is believed that pockets of heavy infestation can become foci of major forest fires, which tend to sanitize stands. Large clearcuts, with no residual host trees, can work like fire to eliminate the disease from managed stands, and this is an extremely effective control strategy. This control practice illustrates how understanding of the natural dynamics of the ecosystem can be translated into sound disease management. Still, poor harvesting practices have maintained this easily-controlled disease as a management problem.

19.2.1 *Lodgepole pine dwarf mistletoe*

Arceuthobium americanum Nutt. ex Engelm. has the largest geographic distribution of any of the species (Hawksworth & Wiens, 1972). Its distribution coincides closely with its principal hosts, *Pinus contorta* var. *latifolia* and var. *murrayana*, but it is common on *P. banksiana* in the western portion of that host's range. More than half of the lodgepole timber type is infested in many areas, particularly from British Columbia through Colorado (Hawksworth & Johnson, 1989). It is the most important disease of this species, and infested stands show a dramatic yield loss and substantially increased mortality rate (Hawksworth & Johnson, 1989).

In contrast to some other pine diseases, dwarf mistletoe does not appear to increase the susceptibility of pine to attack by bark beetles (Stevens & Hawksworth, 1984). But dwarf mistletoe and the mountain pine beetle are important biotic components in the fire cycle of lodgepole pine (Brown, 1975), a species with generally serotinous cones and a heavy dependence on fire for regeneration. Dwarf mistletoe contributes to the ground fuel, and the large witches' brooms and the foliage they trap provide vertical fuel continuity and a "fire ladder." The ensuing fire may ensure regeneration of more host material for the dwarf mistletoe, but an intense fire may remove all the host material and the mistletoe shoots, and the pathogen would have to re-invade the stand from the perimeter, which is a very slow process.

The fire cycle of infested lodgepole pine stands has been largely suppressed, or at least postponed, and this suppression has probably dramatically increased the incidence of dwarf mistletoe (Alexander & Hawksworth, 1975) and resulted in an unprecedented buildup of fuels from dwarf mistletoe and mountain pine beetle mortality. This buildup has been likened by Monnig & Byler (1992) to " . . . holding water behind a leaky dam. We can draw the water down gradually or we can wait for the dam to break."

A lodgepole pine overstory that does not burn can become dominated by the more shade-tolerant spruce or fir as the dwarf mistletoe suppresses host growth and kills the pine. Unless sufficient fuel accumulates and an intense fire returns the stand to pine, the climax species will eventually dominate the stand (Hawksworth & Johnson, 1989). A different situation may be occurring in some Oregon stands of *P. contorta* var. *murrayana*, in which mortality of branches and trees caused by *A. americanum* can result in an environment favoring regeneration of the host (Wanner & Tinnin, 1989). But generally, dwarf mistletoe infestations without fire will accelerate succession to the more shade-tolerant climax species or, if the climax vegetation fails to develop, the dwarf mistletoe continues to intensify.

19.3 STEM ROTS

Decay fungi were major contributors to cull in the harvesting of old-growth forests, but significant stem rot is generally restricted to mature trees (Basham & Morawski, 1964). There is a "pathological rotation" age, after which there is a significant economic loss due to decay (Boyce, 1961), but the actual rotation age for pines in most forests would be substantially less. In unmanaged forests, living trees with stem rot usually survive for decades or even centuries, and these hollowed trees, living or dead, serve as important habitat for a variety of wildlife. Because of the importance of stemwood decay to wildlife management, there have been attempts at increasing the incidence of decay in living trees by artificial inoculations (Parks, Bull & Filip, 1995).

Aside from the vital role of sapwood in conducting water and nutrients, the structural integrity of stemwood must be maintained for physical support. Some deterioration of the heartwood can take place with little loss of stem strength; a pipe is nearly as strong as a solid cylinder (Peters, Osenbruggen & Shigo, 1985). Yet, at least the outer core of conducting sapwood and some of the heartwood is needed, and most large and long-lived tree species have evolved effective strategies to protect these tissues from the destructive enzymes of all but those few wood decay fungi specialized to that host genus or family.

Compared to the heartwood of other genera in the Pinaceae, *Pinus* species are moderately resistant to decay (Wenger, 1984). The resinous response of living sapwood to wounding probably contributes substantially to the resistance of pine roots and stems (Gibbs, 1968). Shallow stem wounds to pine trees generally resist infection by airborne spores of the stem-rotting fungi, but once established in the heartwood, they may encroach on the oldest growth rings of the sapwood. Decay may proceed in the heartwood column for meters above and below the point of entry, while decay of the sapwood is relatively limited (Basham, 1975). Still, these fungi kill and decay sapwood, and the moniker of "heartrot fungi" is not completely accurate.

Pine species vary significantly in susceptibility to decay, perhaps in relation to their life history strategies. Loehle (1988) found that longevity of gymnosperm species can be predicted, to some extent, by their resistance to wood decay. Generally, tree species that invest more energy into wood protection live longer but grow more slowly. Decay susceptible pioneer species on good sites may invest in fast growth as a mechanism to avoid decay problems, but these species may deteriorate quickly once they reach their maximum size and growth is slowed (Loehle, 1988). On harsh sites, resistance to decay may not be necessary for longevity. It has been speculated that many pine sites are too dry for substantial decay activity, and others sites are presumably too cool. For instance, wood of the slow growing bristlecone pine (*P. longeava*) can harbor a significant number of wood decay fungi, but the survival of the oldest pines in a sound condition for thousands of years on cold and dry sites may be because the activities of wood decay fungi are limited there (Lindsey & Gilbertson, 1983).

19.3.1 *Red ring rot*

Although there are many species of fungi adapted to the stems of living pines, one stands out. *Phellinus pini* (Thore:Fr.) Pilat (syn: *Fomes pini*), the cause of red ring rot, is widely distributed throughout the natural range of pines in the Northern Hemisphere (Haddow, 1938). It is the most economically important of the stem rotters of the Pinaceae and has been a topic of interest since it was recognized as a decay fungus by Hartig (1874). But the biology and taxonomy of this fungus are still poorly known. Decay columns of 2 meters or more in height in living pines may yield polypored fruiting bodies that produce wind-disseminated basidiospores. Old branch stubs are not important infection courts, though the fungus may move into the stemwood through living branches. It is believed that most infections originate from deep stem wounds or broken tops or through infections by rust fungi (Basham, 1975). Decay is generally noted in pines of 60 years or more in age, but infection may take place decades earlier (Boyce, 1961).

Pines with red ring rot may be cull to wood-cutters in old growth forests, but they are an important resource to wildlife managers (McFarlane, 1992). Red ring rot is the primary stem rot

of southern yellow pines in the southeastern USA, and an endangered species there, the red cockaded woodpecker, nests almost exclusively in living pines with red ring rot (Conner & Locke, 1982; Jackson, 1977). The birds attempt to make nests in large living trees. The copious resin flowing down the stem apparently provides some protection from snakes and other predators. Nests are generally completed only in those trees with decay columns, presumably because they make for easier nest building. The shortened rotation ages of the managed pine forests result in a scarcity of stem rotted pines, and those few infected trees of suitable size for nesting may be set aside for wildlife management.

19.4 ROOT ROTS

Perhaps no other group of diseases has a greater impact on the ecology of indigenous conifer forests than the root rots, and human activities appear to enhance the incidence of many of these diseases. Losses in commercial forests can be substantial in some regions, such as in western USA, where Smith (1984) estimated that 18% of tree mortality was attributable to root diseases, with annual volume losses of 6.7 million cubic meters. Aside from mortality, root rots predispose trees to uprooting and windsnap, which is a common cause of canopy gaps, although such gap initiations are often considered as merely blowdown trees, and the importance of root rotters in the dynamics of forest ecosystems is largely underestimated (Worrall & Harrington, 1988). Root diseases are also extremely important in supporting endemic populations of bark beetles through predisposition (Cobb *et al.*, 1974; Goheen & Hansen, 1993). Substantial reductions in growth may occur, and decay of the butt log is an important source of cull, but the most important root rotters kill trees relatively quickly by attacking the cambium in advance of decay. *Armillaria* species and *Heterobasidion annosum* kill pines in this way, and these pathogens will be discussed specifically.

The root rotters of pine have a common biology (Harrington, 1993). They cause wood decay and produce airborne basidiospores from macroscopic conks or mushrooms. Colonization of fresh stumps or wounds of living trees by these spores are rare but important events. Wounds on living pines are relatively resistant to infection, presumably due to the heavy resin response (Gibbs, 1968), and exposed heartwood or deep sapwood wounds are often necessary for infection. Suitable infection courts may have been relatively rare in natural forests, but some of the most important root rotters today can colonize fresh stump tops (Hodges, 1969; Rishbeth, 1988). Once established, the mycelium of a root rot fungus may persist on a site in decayed roots for decades or centuries and spread underground as asexual thalli or clones, colonizing and decaying the root wood of diseased or killed trees. The roughly circular pockets of tree mortality are known as "infection centers," which may expand radially at a rate of a half meter or more per year.

Compared to other conifer genera, pines are relatively resistant to root rot diseases. Forests with well-established clones of virulent root rotters may consist of mixtures of susceptible and resistant tree species, the latter often predominating within the clones of root rot fungi. van der Kamp (1991) has referred to such forests as "root disease climaxes." In the Pacific Northwest of the USA and British Columbia, ponderosa and lodgepole pine are more resistant to *Phellinus weirii* (Murr.) Gilb. than are the other Pinaceae (Hadfield & Johnson, 1977). In unmanaged mountain hemlock forests, distinctive circular rings of mortality associated with individual clones of *P. weirii* are evident, with seedlings of the relatively resistant lodgepole pine colonizing the center. In time, the disease pushes the forest composition towards pine (Dickman & Cook, 1989). Similarly, Douglas-fir mortality due to various root diseases encourages the domination of some sites by ponderosa pine.

In many regions, indigenous pine species have been favored in harvesting, and this "high-grading" has left disease- and pest-prone forests of fir and other species. Fire exclusion has had a similar effect. Root diseases have become major management problems as forests have shifted in composition from pines (particularly ponderosa pine) to Douglas-fir and true fir in the interior of the Pacific Northwest (Hagle & Byler, 1994; Monnig & Byler, 1992). On wetter sites that are prone to root disease, the resistant *Pinus monticola* does particularly well, but since the introduction of *Cronartium ribicola*, the cause of white pine blister rust, *P. monticola* has been of limited use, firs predominate, and the root disease situation there continues to worsen (Byler, Marsden & Hagle, 1990; Monnig & Byler, 1992).

19.4.1 *Armillaria root rot*

Species of *Armillaria* play an important role in the decomposition of root-wood and stumps, but some of these same species are important killers of pines and other hosts (Kile, McDonald & Byler, 1991). Epiphytic rhizomorphs of *Armillaria* species are common on living roots, and butt rot colonization patterns are known, but pathogenic colonization of living trees typically occurs under the bark as a sheet or fan of mycelium, killing the cambium and later decaying the underlying wood. Young trees and stressed trees are most often killed, but mortality of vigorous trees may occur when the pathogen increases its inoculum potential.

The fungus can survive for decades in decayed wood, which serves as a food base from which fruiting bodies (mushrooms) or vegetative structures (rhizomorphs) may develop. This food base can provide the inoculum potential for infection of living roots (Redfern & Filip, 1991). The capacity of the thallus to infect depends, in part, on the volume of decayed wood and the species of wood colonized, and so the size of the root system colonized and the species composition of the stand are important. Pines in stands mixed with oaks or pines planted on old hardwood stands that had oak as a component can be particularly vulnerable. Mortality of larger trees has been associated with predisposing stresses such as drought, flooding, pollution, defoliation by insects, and other diseases, including root diseases (Wargo & Harrington, 1991). Often, *Armillaria* deals the final blow to a tree that has been subjected to a long series of biotic and abiotic stresses, but *Armillaria* can kill trees on its own if it has a suitable food base from which to work.

The growing list of recognized *Armillaria* species includes pathogens and saprophytes that decay rootwood in most forests of the world (Kile *et al*., 1994). In the Southern Hemisphere, pine mortality has been associated with *Armillaria* species on cut-over indigenous forests (Hood, Redfern & Kile, 1991; Lundquist, 1993; MacKenzie & Shaw, 1977; Wingfield, Swart & von Broembsen, 1989). Mortality may not persist through subsequent rotations, however, and Armillaria root rot may not become a persistent management problem in tropical or southern temperate pine plantations.

Until relatively recently, Northern Hemisphere *Armillaria* species were inappropriately lumped by pathologists into one species, *A. mellea* (Vahl:Fr.) Kummer. There are now at least 13 clearly defined species in the Northern Hemisphere alone, and most can be found on pines, though not all cause tree mortality (Harrington, Worrall & Baker, 1992). *A. mellea* is most common in milder temperate regions, particularly Mediterranean climates, where it is a common cause of mortality of ornamental, fruit and forest trees. *Armillaria tabescens* (Scop.:Fr.) Emel. is known in southeastern USA, eastern Asia and southern Europe as a pathogen of both hardwoods and conifers, including pines. More important on pine is *A. ostoyae* (Romagnesi) Herink, a pathogen of many conifer species and also on hardwoods in the cooler coniferous forests of Europe (Guillaumin *et al*., 1993), Asia (Sung *et al*., 1991) and North America (Harrington, Worrall & Baker, 1992).

In pines, much of the mortality is associated with relatively young plantings on sites where *A. ostoyae* was previously established, but mortality of large, overstory pines is common in some forest situations, particularly in relatively dry forests. Kile *et al.* (1991) discuss such infection centers in the drier pine forests of western North America and the French Pyrenees, where the disease is known as “ring disease” because of the distinctive expanding rings of mortality. In dry forest types, clones of *A. ostoyae* are usually infrequent but quite large, often covering several hectares or more (Anderson *et al*., 1979; Shaw & Roth, 1976). Rizzo & Harrington (1993) speculated that clones in such dry environments are able to expand, unimpeded by competition from other *A. ostoyae* clones, using the killed trees as further inoculum to expand the center. In contrast, clones tend to be more abundant and smaller in moist ecosystems, perhaps due to the abundance of fruiting bodies in moist climates (Anderson *et al.*, 1979). Here, the entire forest floor may be a mosaic of various clones, which do not tend to intermingle because of somatic incompatibility (Rizzo, Blanchette & May, 1995; Rizzo & Harrington, 1993). Where static clones expend much of their energy in maintaining their genetic integrity and space, little inoculum potential may develop, and mortality is restricted to young saplings and stressed overstory trees.

19.4.2 *Annosum root rot*

Heterobasidion annosum (Fr.) Bref., also known as *Fomes annosus* (Fr.) Cke. and *Fomitopsis annosa* (Fr.) Karst., is one of the most important pathogens on pines. Host-specialized and partially intersterile groups of *H. annosum* have been identified (Harrington, Worrall & Rizzo, 1989; Korhonen, 1978; Stenlid & Swedjemark, 1988). Mortality of mature pines has been associated with a pine form of *H. annosum* in Europe, known as the "P type" (Korhonen, 1978). In North America, a similar pine form of the fungus is recognized (Harrington *et al.,* 1989).

Before tree harvesting and the creation of managed monocultures of pines, annosum root disease must have been a relatively rare disease. The infection of freshly-cut pine stumps by *H. annosum* was recognized by Rishbeth (1951), who elucidated the epidemiology of the disease in pine plantations and noted that competing fungi, most notably *Phlebiopsis* (*Peniophora*) *gigantea* (Fr.) Jul., could exclude *H. annosum* from stumps if applied quickly after harvesting. This led to one of the most effective biological controls of a plant disease. Treatment of freshly cut stumps with *P. gigantea*, borate, urea or other materials may effectively exclude *H. annosum* from colonizing the stump (Korhonen *et al*., 1994; Pratt, 1994). Such stump top treatments are routine in many managed pine forests and are mandated on Forestry Commission lands in Great Britain.

Healthy pine trees may become infected with *H. annosum* when their roots are in contact with decayed roots of killed trees or stumps (Hodges, 1969). Once inside a living pine root, the fungus generally progresses in the xylem, near the cambium, and the root wood may become resin-impregnated in response to the pathogen colonization. Decay of the root wood generally follows cambium killing, but the butt rot pattern of colonization does occur (Greig, 1995). Vegetative spread of the pathogen through root systems may be up to 1 m per year, but on average, radial expansion of infection centers is much less, especially if the pine trees are not large (Slaughter & Parmeter, 1995). Compared to some other root rotters, infection centers associated with *H. annosum* have a limited life, generally less than 20-30 years in northeastern California (Slaughter & Parmeter, 1995).

Plantation forestry and poor management practices appear to be worsening the impact of annosum root rot (Smith, 1989; Stambaugh, 1989). The disease can be severe on pines planted on agricultural lands, particularly those on alkaline soils. Lack of competing forest fungi on such sites apparently favors stump colonisation by *H. annosum* (Schönhar, 1988). Scots pine forests of Europe are severely affected, as are North American pine forests, especially southern yellow pine forests of the Southeast, red pine plantations in the Northeast, and ponderosa and Jeffrey pine forests of the Far West. A noteworthy example is in the Yosemite Valley of California, where fire exclusion has increased the basal area and density of ponderosa pine and incense cedar (*Calocedrus decurrens* (Torr.) Florin), both hosts of *H. annosum* (Marosy & Parmeter, 1989; Sherman & Warren, 1988). Heavy mortality of pine by *H. annosum* and bark beetles has recently shifted this now dense forest more to *Calocedrus*, which is more prone than pine to windthrow if infected with annosum root rot. The hazard to humans and property associated with uprooted pine, and especially cedar, infected with *H. annosum* has resulted in a major management problem in one of the most heavily used recreational sites in the USA.

19.5 OTHER ROOT DISEASES

The aforementioned root rots are generally more important to the ecology of pines than are other root diseases. But these other root diseases, which are caused by fungi that do not decay wood, may be of local importance. For instance, *Rhizinia undulata* Fr.:Fr., an ascomycete, has altered management practices in some parts of the world where substantial seedling mortality may occur in plantings on slash-burned sites (Wingfield, Swart & von Broembsen, 1989). Two other root diseases will be discussed in more detail. These two fungi and the diseases they cause have little in common, except that they attack pine roots, they do not cause wood decay, and they both predispose pines to bark beetle attack.

19.5.1 *Black stain root disease*

This disease has a spotty distribution across its known range of western North America, where it is of local importance but may have a dramatic impact on the landscape. The distribution of the disease has probably not changed much with human activity, but the disease may well be more common today than in previous years due to enhanced vector activity, particularly by the creation of stumps and the attraction of vectors to disturbed stands, where infection centers originate. In contrast to the root rot fungi, *Leptographium* (*Verticicladiella*) *wageneri* (Kendr.) Wingf. produces its spores (conidia) in sticky drops on top of stalked structures (conidiophores) within insect galleries. Like root rot diseases, black stain can spread through pine root systems to adjacent trees. The preference for monocultures of susceptible pines in some regions can enhance tree-to-tree spread, and fire exclusion may similarly provide heavily stocked stands of large pine trees.

Three host-specialized varieties with overlapping geographic distributions are recognized (Harrington & Cobb, 1986, 1987; Zambino & Harrington, 1989). The species was originally described from pinyon, and this variety infects *Pinus monophylla* and *P. edulis* throughout the southwestern USA and north to southern Idaho. Variety *ponderosum* occurs on the hard pines in the Pacific Coast states, Idaho, Montana and British Columbia, Canada. Variety *pseudotsugae* is a pathogen on Douglas-fir throughout the western USA and British Columbia. In each of these hosts, hyphae of *L. wageneri* grow through the tracheids only, at least in the early stages of the disease, and it moves systemically throughout the root system and into the roots of adjacent trees through root contacts, and it may grow for short distances through soil (Cobb, 1988; Cobb *et al.*, 1982).

Infection centers are initiated when root-feeding insects, primarily bark beetles, carry the pathogen into the roots of a living tree. Vector biology has been most studied in Douglas-fir, but similar vector relationships appear to occur in pine. Species of the root feeding bark beetle genus *Hylastes* (Coleoptera: Scolytidae) are the primary vectors (Goheen & Cobb, 1978; Harrington, Cobb & Lownsberry, 1985; Witcosky, Schowalter & Hansen, 1986). After emergence from trees killed by black stain, new adults may feed on roots of healthy trees before breeding and egg-laying, and this so-called maturation feeding has been proposed as a crucial stage for introduction of *L. wageneri* into a living host and initiation of a new infection center (Harrington *et al*., 1985). Populations of vectors may be artificially high in some managed forests because the stumps left after harvesting and thinning of stands provide abundant breeding material. The disease is strongly associated with stand disturbances such as roadside construction, tractor logging or stand thinning (Hansen, 1978; Harrington *et al*., 1983), presumably because the vectors are attracted to wounded trees and fresh stumps (Harrington *et al*., 1985). Although initiation of new infection centers may be less frequent in pine than in Douglas-fir, black stain root disease has been associated with disturbances in pine stands (Cobb, 1988). A less efficient vector system or smaller vector population may explain the relatively rare initiation of new infection centers in pines. Aside from the vectors, other bark beetles play an important role in killing pines with black stain root disease, and the disease contributes to the potential brood material for endemic populations of stem feeding bark beetles (Cobb *et al*., 1974; Goheen & Cobb, 1980; Landis & Helburg, 1976; Wagener & Mielke, 1961).

The disease has been a major factor in the management of pinyon at some locations, such as Mesa Verde National Park in southwest Colorado (Landis & Helburg, 1976), where black stain is the chief cause of pinyon mortality, and 12,000 dead and dying trees were removed in 1932 through 1934 (Wagener & Mielke, 1961). Overmature pinyon stands with high stocking densities and little or no juniper can be created through fire suppression, and when these occur on cool sites with deep and moist soils, local epidemics of black stain root disease can be found. The disease is clearly restricted to the coolest of sites, mostly at the upper elevations of the host distributions (Landis & Helburg, 1976; Wagener & Mielke, 1961), perhaps because lower elevation sites are too warm for growth and survival of *L. wageneri* (Harrington & Cobb, 1984). Similarly, the disease is found on pinyon west of the continental divide in Colorado but has not been reported from the east side (Landis & Helburg, 1976).

The hard pine variety, var. *ponderosum*, occurs primarily on ponderosa, Jeffrey and lodgepole pines (Harrington & Cobb, 1986). The disease is found at the higher elevations of

ponderosa pine in northern California, and the disease is not known in southern California. New infection centers arise very rarely, but where they occur, they may cover many hectares (Cobb *et al.*, 1982). Sites where the disease is severe are typically those suited to the Sierra mixed-conifer forest type, but fire, mud slides or stand management practices, particularly logging followed by burning, have created nearly pure stands of ponderosa pine (Cobb, 1988). In one such area, the rate of radial spread of infection centers ranged from 0 to 7 m/yr, with the density of ponderosa pine in the stand explaining much of the variation (Cobb *et al.*, 1982). Black stain root disease reduces the ponderosa pine component in these nearly pure stands and creates openings for the more shade-tolerant conifer species, thus helping to maintain the natural heterogeneity of the conifer forests in this region.

19.5.2 *Littleleaf disease*

Many species of *Phytophthora* are pathogens of plants, including pines in nurseries, but only one has been known to have a major impact in pine stands. *Phytophthora cinnamomi* Rands has a very broad host range, and most species of pine show some degree of susceptibility (Zentmeyer, 1980). The natural range of the fungus is not clear, but it may be native to southeastern Asia. It is now widely distributed throughout mild temperate, subtropical and tropical regions of the world (Zentmeyer, 1980). Fortunately, *P. cinnamomi* does not persist in the soils of many cooler coniferous forests, presumably because freezing soils impede the overwintering survival of the fungus (Benson, 1982), and moist soil conditions are needed during warm periods for the fungus to be active. Exotic plantings of pines in the Southern Hemisphere have been known to be affected by *P. cinnamomi* (Newhook, 1959; Wingfield *et al.* 1989). In indigenous forests, the pathogen has been less of a problem, except in the southeastern USA, where loblolly, and especially shortleaf, pines are vulnerable on some sites. Here, the disease is known as littleleaf disease.

In pines and many other hosts, the fungus primarily kills only the small-diameter feeder roots (Zentmeyer, 1980). Slowed growth, chlorotic crowns with small leaves, and crown dieback may be evident for many years before death. Littleleaf disease is most prevalent on old-field pine forests that previously were agricultural lands in the Piedmont region of the Southeast. The pathogen often works in concert with poor soils and bark beetles to form a complex decline syndrome (Belanger, Hedden & Tainter, 1986). Shortleaf pine on eroded or compacted soils of poor fertility are particularly vulnerable to littleleaf disease. Trees under 20 years of age are rarely diseased, and affected trees are often killed by bark beetles, particularly the southern pine beetle, *Dendroctonus frontalis* Zimmermann. The disease is one of the reasons that loblolly pine has been favored in plantations over shortleaf pine, and this has lead to some other management problems, such as fusiform rust.

19.6 CANKER DISEASES

A great number of canker diseases of pines exist, and these vary considerably in their biology, symptoms and impact (Boyce, 1961; Manion, 1981, Sinclair, Lyon & Johnson, 1987). Cankers are defined as necrosis of the inner bark tissues of stems or branches, but in this treatment of canker diseases, we are excluding cankers associated with dwarf mistletoes, wood decay fungi, and the rusts. Vigorously-growing pines in their natural range generally are highly resistant to canker diseases, but stresses are important predisposing factors for canker diseases, and there is strong evidence that some of the most conspicuous canker fungi have been introduced to new locations.

Most canker diseases, as well as most of the needle diseases, are caused by Ascomycete fungi that generally have two distinct spore stages, often a wind-dispersed sexual spore (ascospore) and a rainsplash-dispersed asexual spore (conidium). They generally require wounds to infect their hosts. These wounds are provided by insect feeding, wind and hail damage, or even natural growth cracks. There are, however, some canker pathogens that infect unwounded foliage or young pine tissue.

Annual cankers occur when the pathogen is active in the branch or stem for only one season and are commonly associated with wounds on stressed trees. These infections are usually excluded by the tree when the stress abates. Perennial cankers are caused by pathogens, such as *Atropellis* species, that are relatively weak but most active when trees are not

growing actively, such as in the late summer and early winter (Sinclair, Lyon & Johnson, 1987). In these cases, tree growth in spring results in the development of a band of callus tissue, which temporarily impedes the pathogen but then is transgressed towards the end of the growing season. This cycle leads to the development of concentric rings of callus tissue, and thus the term “target canker” is often applied. Perennial cankers develop slowly and tree mortality seldom results. The so-called diffuse cankers are caused by virulent pathogens where the host shows little resistance to infection, and layers of callus are not generally evident. In these cases, trees or their branches are rapidly girdled and killed.

We discuss in detail three important canker diseases that illustrate various ecological and biogeographical concepts. With each of the three, there is evidence that the pathogens have been introduced to new pine-growing regions, and this is where their impact has been greatest.

19.6.1 *Scleroderris canker*

Scleroderris canker is caused by the fungus *Gremmeniella abietina* (Lagerb.) Morelet (syn. *Ascocalyx abietina* (Lagerb.) Schlaepfer or *Scleroderris lagerbergii* Gremmen, also known in Europe by the conidial state name *Brunchorstia pinea* (P. Karst.) Hoehn. The disease has been known in Europe since 1888 and in North America since the 1950s. Confusion surrounding the various reported forms of the pathogen (Skilling, 1977) and a severe epidemic of the disease in New York in the 1970s prompted a symposium in 1983, the proceedings of which were published and summarized by Manion & Skilling (1983). Serious discrepancies have remained concerning the various forms of the pathogen in North America and Europe.

An Asian race is apparently restricted to *Abies*, whereas the so-called North American and European races have wider host ranges but occur primarily on pines (Yakota, Uozumi & Matsuzaki, 1974; Dorworth, 1981; Skilling, Kienzler & Haynes, 1984). Yakota (1983) also reported on the rare occurrence of a pine form of the fungus in Japan on *P. strobus*. The confusion over the pine forms in Europe and North America appears to be clarified by recent reports using randomly amplified polymorphic DNA. Lecours *et al.* (1994) grouped European isolates into three amplitypes. A cold-adapted amplitype occurred on *P. cembra*, *P. mugo*, *P. sylvestris* and *Larix lyalli* above 2500 m elevation in the Alps; this amplitype apparently corresponds to *G. abietina* var. *cembrae* Morelet. A northern amplitype was found above 66 degrees latitude on *P. sylvestris* and planted *P. contorta*. The third European amplitype was more widely distributed, from Scandinavia to northern Italy. In a more detailed study, Hellgren & Högberg (1995) also differentiated the northern amplitype from the more generally distributed European amplitype. According to Hamelin, Ouellette & Bernier (1993) and Lecours *et al.* (1994), it is the generally distributed European amplitype that has been referred to as the "European race" in North America, i.e., the pathogen responsible for the epidemic in New York in the 1970s. The "North American race," which has been associated with epidemics on young *P. resinosa* and *P. banksiana* across the Lake States and Ontario since the 1950s, was not found in European samples.

With this delineation of amplitype or races, the ecology of the disease needs to be re-evaluated. Some general trends are evident, however. Symptoms can include browning of needles and needle cast, yellowing at the bases of needles, twig cankers, die-back of branches or death of shoots, with a concomitant yellow-green discoloration of the cambium under the cankers. In some cases the branch cankers can spread to the main stems of trees causing stem cankers and deformation (Manion, 1981; Sinclair, Lyon & Johnson, 1987). Mortality of mature trees is sometimes evident in epidemic situations.

Severe epidemics of the disease have been associated with particular climatic conditions and plantings of exotic germplasm. The disease is favored by cooler temperatures and is most problematic in plantation areas where cold air accumulates, such as in depressions (Aalto-Kallonen & Kurkela, 1985; Dorworth, 1972; Patton, Spear & Blennis, 1984). Some forms of the pathogen are most active when host tissue is dormant in winter months and under snow cover (Dorworth, 1972; Patton, Spear & Blennis, 1984). In Scotland, the disease is most serious in Corsican pine (*P. nigra* var. *maritima*) on north-facing slopes (Read, 1968). As in many other canker diseases of pines, tree species or seed sources planted out of their natural range are more susceptible, and predisposing stresses are important. In the Great

Lakes region of USA and Canada, the disease is most problematic in relatively young plantings of infected nursery stock, which can be avoided by the use of fungicide treatments in nurseries (Skilling & Waddell, 1970).

As mentioned above, the Scleroderris canker epidemic on *P. resinosa* and *P. sylvestris* in New York during the mid 1970s was apparently caused by the amplitype found throughout most of Europe, where it is not dependent on snow cover for infection, and it infects large trees (Hellgren & Högberg, 1995). It is likely that this form of the pathogen has been introduced to several locations between New York and the Maritime Provinces of Canada. As pointed out by Manion & Skilling (1983), the New York epidemic was associated with cooler than normal temperatures, and non-local seed sources had been used in the affected plantations. The epidemic ultimately involved mortality of mature trees over about 14,000 ha, prompting considerable concern and quarantine restrictions. Fortunately, the feared wide-spread devastation did not materialize, though the disease persists.

19.6.2 *Pitch canker*

Pitch canker is caused by the fungus *Gibberella fujikuroi* (Sawada) Ito but is best known by its conidial state, *Fusarium subglutinans* (Wollenweb. & Reinking) P. Nelson *et al.* f. sp. *pini*. Pine species differ in their susceptibility, but most species appear to be susceptible to some degree, and even Douglas-fir has been recorded as a host (Storer *et al*., 1994). The disease was first described from the southeastern United States (Hepting & Roth, 1953), where it is particularly damaging on young *P. elliottii*, *P. echinata* and *P. virginiana* (Dwinell, Kuhlman & Blakeslee, 1981; Dwinell, Barrows-Broaddus & Kuhlman, 1985). Although discovered in the 1940s, it only became serious in the Southeast in the mid 1970s and has particularly been a problem in seed orchards. Since 1987, the pathogen has been recognized in four new geographic areas (Viljoen, 1995). There is sufficient cause to expect that pitch canker threatens the genetic base of many pine species in various parts of the world. There is an urgent need to launch a coordinated effort to study this relatively unknown disease.

The disease has been most studied in the southeastern USA. Symptoms include shoot death and cankers on branches and stems. Cankers are typified by copious resin exudation and pitch soaking of the wood, often through to the pith. The major infection propagules of *F. subglutinans* f. sp. *pini* are the asexual conidia. The sexual state of the fungus has been reported under laboratory conditions (Kuhlman *et al*., 1978) but has not been found in nature. Wounds are required for infection. Wind, hail damage, infections by other pathogens, such as fusiform rust, and insects, including the pine tip moth (*Rhyaciona subtropica*), the deodar weevil (*Pissodes nemorensis*) and bark beetles (Coleoptera: Scolytidae), may provide suitable wounds. In the Southeast, where infection of trees in seed orchards is a management problem, efforts are made to reduce the occurrence of mechanical damage to trees.

Pitch canker was discovered in California for the first time in 1986 on radiata pine near Santa Cruz (McCain, Koehler & Tjosvold, 1987), where it has been largely confined to landscape plantings. Very little genetic variation is found in the pathogen in California compared to Florida, strongly suggesting that the California population is derived from a recent introduction (Correll, Gordon & McCain, 1992). It continues to spread to new areas of California and to additional species of pine, including the three native *P. radiata* stands on the mainland (Dallara *et al*., 1995; Storer *et al*., 1994). Thus, the genetic base of this important pine species is threatened.

In California, the pitch canker pathogen has established an association with various insects, such as the engraver beetles *Ips mexicanus* Hopkins and *I. paraconfusus* Lanier, which are able to transmit the fungus to mature pines (Fox, Wood & Koehler, 1990; Fox *et al*., 1991). These insects may be more efficient vectors of the pathogen than the weevils and moths that have been associated with the fungus in the Southeast (Blakeslee & Foltz, 1981; Runion & Bruck, 1985). Indications are that the spread and importance of pitch canker internationally will depend to some extent on the insects with which it becomes associated. Strategies to manage these insects may be used to reduce disease incidence in the future.

Pitch canker was recorded from Japan for the first time in 1988 on *P. luchuensis* (Muramoto,

Tashiro & Minamihashi, 1988) and has subsequently spread and increased in importance (Kobayashi & Kawabe, 1992; Kobayashi & Muramoto, 1989; Muramoto & Dwinell, 1990). The most recent discovery of the pitch canker pathogen has been in South Africa, where it has been associated with a devastating disease of *P. patula* in a large commercial nursery (Viljoen, Wingfield & Marasas, 1994; Viljoen et al., 1995). Thus far, the disease has not been reported on established trees in plantations in this area. The occurrence of the disease in this nursery suggests that the pathogen might have been introduced on seed. Fraedrich & Miller (1995) demonstrated that *F. subglutinans* can be isolated from seed.

The recent outbreaks of pitch canker favor the hypothesis that the pathogen has been newly introduced into these areas. Although the biogeography of the disease is not well documented, the disease has also recently been recognized on many native species in Mexico, where it is considered to be severe in some areas (Rodriques, 1989; Santos & Tovar, 1991). Apparently, the disease is well established there, and this could be the area of origin of the pathogen. An earlier hypothesis (Berry & Hepting, 1959) was that the pathogen in the southeastern USA had originated in Haiti, where it was well established on *P. occidentalis*.

19.6.3 *Sphaeropsis canker*

The disease, caused by *Sphaeropsis sapinea* (Fr.) Dyko & Sutton but perhaps better known by the earlier name *Diplodia pinea* (Desm.) Kickx *et al.*, occurs throughout the world where pines are grown (Ivory, 1994; Sutton, 1980). The disease is particularly common in Africa (Ivory, 1994). There is some indication that races of *S. sapinea* exist and that these vary in their ability to kill trees (Palmer, Stewart & Wingfield, 1987; Smith & Stanosz, 1995; Swart *et al.*, 1991). Although the fungus has been rarely associated with episodic disease outbreaks on pines in their native range, it is most devastating in exotic plantings, both in landscapes and in intensively managed, exotic plantations.

Sphaeropsis sapinea causes annual cankers and might be considered to be more opportunistic than the aforementioned canker pathogens. Sphaeropsis canker may involve a multiplicity of symptoms, including die-back and death of pines after hail, die-back of young shoots, stem and branch cankers, cone infections, blue stain of freshly felled timber and root disease (Gibson, 1979; Sinclair, Lyon & Johnson, 1987; Swart & Wingfield, 1991). Although the pathogen is able to infect and kill young growing shoots (Peterson, 1977), it is most often associated with wounds on stressed trees. Wounds suitable for infection result not only from hail but from wind damage, pruning and insect feeding. Incidental wounds are common in plantations, but the cankers generally do not extend far beyond the localized sites of infection. Not pruning during warm and wet weather and avoidance of excessive pruning are important means to reduce losses (Swart & Wingfield, 1991). In many landscape planted pines, cankers usually develop after the hosts produce cones, on which fruiting structures of the pathogen are generally abundant. Conidia that are rain-splashed from the cone scales are the major source of inoculum (Peterson, 1977).

The fungus is common throughout the native range of pines, though it appears to cause little damage in natural settings. It can be a problem in nurseries near cone-bearing pines (Palmer & Nicholls, 1985), however, and pines planted out of their natural range are particularly vulnerable. For instance, Sphaeropsis canker has been severe on *P. nigra* in the Midwest and Northeast of the USA (Peterson, 1981). *Sphaeropsis sapinea* has been effectively introduced into every part of the world where pines are grown, perhaps because the fungus is common on pine seeds (Fraedrich & Miller 1995). Preliminary studies (Wingfield, unpublished) indicate that *S. sapinea* is represented by a high degree of genetic diversity in South Africa. Given the fact that the fungus is known only in its asexual form, this would imply that it has been introduced into the area many times and from many parts of the world.

The die-back and death of trees associated with *S. sapinea* has been more important in southern Africa than in any other part of the world (Swart & Wingfield, 1991; Wingfield, 1990). Here, extensive losses occur annually in pine plantations damaged by hail storms (Swart & Wingfield, 1991). This is due to a high incidence of hail damage (wounds) and a high degree of susceptibility in two of the most important pine species (*P. patula* and *P. radiata*) in the region. Cankers after hail develop extremely rapidly, perhaps due to the stress associated

with hail damage rather than to infection of hail wounds. The fungus can reside as an endophyte in living twigs and cones in a latent phase without causing symptoms (Smith *et al.*, 1995). Where hail damage is common, *P. radiata* and *P. patula* should not be planted.

19.7 NEEDLE DISEASES

Trees in their natural range are generally not seriously affected by foliage diseases because of the high level of resistance in the native population, and mortality due to foliage disease is relatively rare. Genotypes adapted to areas of low disease pressure, however, may be seriously affected when planted in areas of high disease pressure. Exotic plantings, off-site plantings and pines planted for Christmas trees are examples where foliage diseases may have significant impact.

Two general classes of needle diseases occur on pine. The needlecasts are associated with fungi that usually infect young needles and remain latent in the needle for months or years before symptoms develop. After the infected needles are cast, fruiting structures develop and sexual spores (ascospores) are discharged, generally as new needles emerge in the spring. Common genera of needlecast fungi on pine include *Lophodermium*, *Lophodermella*, *Ploioderma*, *Cyclaneusma* and *Elytroderma*. The latter genus is unusual in that twigs are also infected and witches' brooms develop. *Elytroderma deformans* (Weir) Darker occurs in western North America and *E. torres-juanii* in southern Europe (Sinclair *et al.*, 1987). The other needlecast fungi can be of major importance in Christmas tree production, and *Lophodermium seditiosum* Minter, Staley & Millar is considered to be a virulent and primary pathogen.

The other class of needle disease is the needle blights, many of which involve both primary (ascospores) and secondary (conidia) inoculum. The sexual fruit bodies develop towards the end of the growing season and are relatively uncommon in many of these fungi but may allow for genetic recombination in the pathogen and commonly provide a means for over wintering. In contrast, the asexual conidia are produced abundantly during wet periods of the growing season and are responsible for an increase in the inoculum load and the rapid development of epidemics, usually in wet weather. Infections occur either directly through the epidermis or via stomata. These lead to spotting and banding symptoms and, eventually, needle death and defoliation. Severely diseased trees may show substantially reduced growth rates or mortality. Species of *Mycosphaerella* infecting pines are the most noteworthy of needle blight fungi (Evans, 1984). *Mycosphaerella gibsonii* is perhaps endemic to the Himalayas and has been recorded from Asia, Africa, Papua New Guinea and Jamaica (Ivory, 1994). Two better known *Mycosphaerella* species will be considered here in more detail.

19.7.1 *Red band needle blight*

Dothistroma or red band needle blight is caused by the fungus *Mycosphaerella pini* E. Rostrup apud Munk (*Schirria pini* Funk & Parker) but is better known by its asexual state name, *Dothistroma septospora* (Dorog.) Morelet (syn. *Dothistroma pini* Hulbary). It is the best known and most studied needle blight of pine and has caused extensive damage and defoliation of pines in many parts of the world (Gibson, 1972). The pathogen is native on pines in the Northern Hemisphere and was first recognized and described from eastern Europe in 1911 (Gibson, 1972), but it is now widely distributed. In most cases where this disease has imparted serious losses, the pathogen has been introduced or the host planted outside of its natural environment. In such situations, it has been necessary to plant alternative species or to resort to complicated and extensive management strategies.

Ivory (1994) speculated that there are two primary origins of the species, a short-spored form endemic to Eurasia and found in Asia, Australasia and South America (*D. pini* var. *pini*) and an intermediate-spored form endemic to Central America and found in Africa (*D. pini* var. *keniensis* Ivory). The long-spored form (*D. pini* var. *lineare* Thyr & Shaw) is found in North America and France and may have a separate origin. Sutton (1980) considered these morphological distinctions debatable, and the definitive genetic work has not been done.

The first symptoms of infection are yellow bands on mature needles at the bases of branches and in the lower half of the crown, and the bands later turn red in color, thus leading to the name red band needle blight. Infected needles are killed and drop, and conidia produced from

them are splashed to other needles. Temperatures of between 15 and 20 C with extended periods of moisture favor severe infection, thus accounting for the severe infection levels in regions such as the East African Highlands and the North Island of New Zealand (Gibson, 1979).

A wide range of pine species have been recorded as hosts (Ivory, 1994; Sutton, 1980), but *P. radiata*, *P. nigra* and *P. ponderosa* are by far the most susceptible species, at least when planted outside of their natural ranges (Gibson, 1979). The disease has caused substantial damage to exotic pine plantings in the USA and Europe (Gibson, 1972; Peterson, 1967). Most plantings of *P. ponderosa* in eastern North America have failed because of this disease. Red band needle blight is absent in the limited natural stands of *P. radiata* in California, but further north along the coast, the disease has been devastating in some *P. radiata* plantations (Cobb & Miller, 1968).

The fungus was recognized as the causal agent of severe defoliation of *P. radiata* in Tanzania in 1957 and, thereafter, spread rapidly throughout central and southern Africa (Gibson, 1972). The disease thus led to the abandonment of *P. radiata* as a plantation species in many parts of Africa and the use of alternative species, such as *P. patula* (Ivory, 1987). The disease then appeared in Chile and New Zealand in the early 1960s, where it has caused substantial damage, also to *P. radiata*. In areas of New Zealand where the disease has been severe, chemical control using copper fungicides was effectively practiced for many years (Gibson, 1975). The fact that *P. radiata* trees develop adult resistance to this disease at about 15 years has reduced the need for chemical treatments throughout the rotation (Ivory, 1972). In recent years, substantial progress has also been made in breeding for resistance to this disease (Carson & Carson, 1989).

19.7. 2 *Brown spot needle blight*

This disease is caused by *Mycosphaerella dearnessii* Barr (syn. *Scirrhia acicola* (Dearn.) Siggers), which is also known by its asexual state name, *Lecanosticta* (*Dothistroma*) *acicola* (Thuem.) Syd. apud Syd. & Petrak (Evans, 1984). It is best known for the damage that it has caused to longleaf pine (*P. palustris*) in plantations in the southeastern USA (Boyce, 1961; Sinclair *et al*., 1987), but it can be important elsewhere. Symptoms and epidemiology are very similar to red band needle blight, though its impact has been generally less.

The symptoms of the disease vary greatly amongst hosts, but chlorotic spots or bands with dead brown centers are common. Disease tends to develop on foliage on older parts of trees, and eventually spreads to younger foliage. Severe infection can result in needle-free branches with tufts of diseased current year needles at their apices. New infections become established in spring and early summer. These usually result from conidia that are produced in sticky masses from conidial stromata and are rainsplash disseminated. Sexual fruiting bodies may develop in these same structures after approximately two months, though the sexual state is not common. Rate of symptom development after infection is extremely variable but is favored by warm and wet weather (Kais, 1975).

Longleaf pine develops through a grass stage, which is particularly conducive to the development of brown spot needle blight. This host has a fire tolerant bud in the grass stage, and as fire burns the needles, it prevents the buildup of inoculum. Fire suppression in plantations has, thus, led to severe disease. Heavy infection can result in serious delay (4 to 10 years or more) in trees emerging from the grass stage. Control of brown spot needle blight with fire or chemical sprays is essential in longleaf pine plantations (Gibson, 1979; Sinclair *et al*., 1987). Brown spot needle blight has also been exacerbated by the fact that seedlings are commonly infected in nurseries, providing an initial inoculum for disease development. Considerable effort has been expended in the development of disease resistant seed sources (Snyder & Derr, 1972). This disease has made longleaf pine less desirable as a plantation species, and this has contributed to the favoring of other southern pines, which are more susceptible to fusiform rust and southern pine beetle.

Although brown spot needle blight has been most serious and is best known on *P. palustris* in the South, it has a wide host range amongst species of pine (Gibson, 1979; Sinclair *et al*., 1987). It also occurs in northern USA and Canada, and it is scattered in South America and

Europe, perhaps through introductions. Northern USA isolates differ from those in the southern USA, and isolates from China are apparently of the southern USA type (Zheng-Yu, Smalley & Guries, 1995). The disease is severe in parts of China, where it became common after large-scale import of slash pine seed from the USA in the 1970s, although Zheng-Yu *et al.* (1995) believe the pathogen was present in China before then. Brown spot needle blight has resulted in serious losses to certain provenances of *P. sylvestris* grown for Christmas trees in the USA (Skilling & Nicholls, 1974). This high value crop is dependent on a dense and high quality foliage. Short needle varieties of Scots pine from Spain and France are highly susceptible to brown spot and are not suitable for Christmas tree production. Shearing of needles in wet weather enhances spore dispersal and should also be avoided (Skilling & Nicholls, 1975).

19.8 RUST DISEASES

Members of the order Uredinales are obligate plant pathogens. These fungi are called rusts due to the fact that, at some point in their life cycle, they produce masses of dry spores that are often rust colored. There are many species of pine rusts and most of these reside in the stem rust genera *Cronartium*, *Endocronartium* and *Peridermium*, or in the foliar rust genera of *Coleosporium* and *Melampsora*. On pine hosts, they occur only in the Northern Hemisphere where they can damage stems, foliage or cones. Infections by stem rust fungi can lead to mortality, particularly when young trees are infected, and these are the most damaging of the pine rusts. Most noteworthy are white pine blister rust and fusiform rust, both caused by species of *Cronartium*.

The rust fungi are all biotrophic (obligate pathogens) and most have complex life cycles, with up to five different spore stages involved in the completion of a single generation. *Cronartium* species infect stems of pine and are heteroecious, that is, they produce some of their spore stages on an alternate, dicotyledonous host. The typical life cycle begins with aecia and aeciospores that develop after the fertilization of two haploid gametes produced on the pine tissue in structures called pycnia. The product of this fertilization gives rise to aecial structures from which masses of dikaryotic (two paired nuclei) aeciospores are produced. Galls or swellings generally develop on branches or stems producing aecia, and these tissues generally survive for many years unless killed by weakly pathogenic canker fungi, the activities of insects or other animal feeding (Byler, Cobb & Parmeter, 1972).

Aeciospores are wind dispersed and infect leaves of the dicotyledonous host, on which uredinia and the dikaryotic urediniospores are produced. The uredinial stage is often referred to as the repeating stage because the urediniospores infect the dicot host, thus building the rust population through several generations in a single growing season. Towards the end of a season and as temperatures drop, uredinial infections give rise to the telial stage, which produces teliospores. Upon germination of teliospores, the dikaryotic nuclei fuse to form a diploid and, after meiosis and mitosis, four haploid basidiospores are formed. The basidiospores can infect only pine and not the dicot host. These infections result in the formation of pycnia and spermatia, thus completing the typical heteroecious rust life cycle. The basidiospores are generally the most delicate spore stage and are most sensitive to environmental conditions. Years of optimal conditions for basidiospore infection, known as "wave years," are associated with fusiform rust and white pine blister rust epidemics. In the former, these occur every 3 to 5 years, but if they occurred every 1 or 2 years, it would be impossible to grow plantation pines in the South (Powers, Schmidt & Snow, 1981).

The impact of stem rusts is certainly increased by the trend to establish even aged stands of fast growing pine for commercial purposes (Peterson & Jewell, 1968; Powers, Schmidt & Snow, 1981). Risks associated with movement of pine rusts among continents are adequately illustrated by the damage that white pine blister rust has caused in North America. The potential of most pine rusts to become established in the Southern Hemisphere is reduced by the fact that most rusts require two specific hosts. In contrast, there is substantial risk associated with the potential introduction of members of the stem rust genera *Endocronartium* or *Peridermium*, which are derived from species of *Cronartium* (Vogler, 1995) but produce aeciospores that infect pine (autoecious). The most important of the pine to pine rusts in terms of quarantine procedures is *Peridermium* (*Endocronartium*) *harknesii* J. P. Moore, which is known to infect the widely planted *P. radiata* (Parmeter & Newhook, 1967) and has

been introduced to new areas of North America on nursery stock (Peterson, 1981).

19.8.1 *Fusiform rust*

This disease, caused by *Cronartium quercuum* (Berk.) Miyabe ex Shirai f. sp. *fusiforme*, is one of the most important diseases of forest trees in the USA (Czabator, 1971). The pathogen is restricted to the southern parts of the country (Maryland to Florida to Texas), with losses most severe from Louisiana to South Carolina. Oaks are the alternate host. Many two and three needle pines are susceptible to the various host-specialized forms of *C. quercuum*, but the pathogen that causes fusiform rust (f. sp. *fusiforme*) most severely damages slash and loblolly pines (Burdsall & Snow, 1977). Infected trees less than 5 years of age generally die, and older trees may be deformed or break at the point of infection. Powers, Schmidt & Snow (1981) estimate annual losses due to degrade of timber alone at $75 million. Another estimate places losses due to fusiform rust at $130 million annually (Anderson & Mistretta, 1982).

Fusiform rust has likely been a major factor in the biogeography of the southern pines. Loblolly pine is most susceptible when planted in the southern part of its natural range and slash pine when planted north of its natural range (Powers, Schmidt & Snow, 1981). In the "rust corridor" between the core distributions of these rust susceptible species, the rust resistant longleaf and shortleaf pines were once more prevalent, but slow growth, regeneration difficulties, and problems with brown spot needle blight and littleleaf disease, respectively, have made these species less desirable for plantation forestry. Dinus (1974) notes that the fusiform rust corridor runs along the northern boundary of the original longleaf pine forest type, where loblolly pine and oaks tended to be interspersed with longleaf pine. Heavy disease pressure here likely gave an advantage to rust-resistant longleaf pine before human intervention.

Dinus (1974) speculates that fusiform rust was rare before 1900, in part because of the prominence of the fire resistant longleaf pine then. Harvesting practices and fire control reduced this species to perhaps 10% of its previous volume. Loblolly and slash pine regenerated well on cutover longleaf pine sites. Factors contributing to the high impact of fusiform rust today include the increased susceptibility of pines due to vigorous growth, a shift to plantations versus natural regeneration, use of the most rust-susceptible species (loblolly and slash pines) for 98% of all plantings, and control of wildfires, which leads to an increase in the incidence of the alternate hosts, oaks, and a decrease in the incidence of the rust resistant longleaf pine (Dinus, 1974; Powers, Schmidt & Snow, 1981).

Considerable effort has been expended on reducing the impact of fusiform rust in recent years. Hazard ratings of sites is useful (Powers, Schmidt & Snow, 1981). Efforts have concentrated on the selection of resistant provenances of loblolly and slash pines and the development of hybrids with shortleaf pine (Carson & Carson, 1989; Peterson & Jewell, 1968; Powers, Schmidt & Snow, 1981; Schmidt, Powers & Snow, 1981). Techniques for rapid and effective inoculation of seedlings and the early detection of susceptible stock have been developed and are used with considerable success (Wells & Dinus, 1978). Selection of resistant stock is complicated by the fact that the pathogen has a variable genetic base and is continuously adapting to rust resistant material (Powers *et al.*, 1981; Snow, Dinus & Walkinshaw, 1976). With the ecology of the disease and the southern pines in mind, Dinus (1974) argues for a greater emphasis on longleaf pine, which is also more resistant to attacks by the southern pine beetle, another major management problem in southern pine forests.

19.8.2 *White pine blister rust*

This rust disease, caused by *Cronartium ribicola* J.C. Fisch. ex Rab, is one of the most important and intensively studied tree diseases. It is notorious on white (five needle) pines in North America. The pathogen is native to Asia (Kaneko *et al.*, 1995) but was introduced to Europe and from there to eastern and western North America at the beginning of this century (Peterson & Jewell, 1968; Ziller, 1974), when planting stock was being produced in Europe and shipped to the United States. Establishment and spread of the disease in North America was effective and rapid due to the susceptibility of native pines relative to Asian and European white pines (Hoff, Bingham & McDonald, 1980), climatic conditions favoring

infection, and the wide scale presence of the alternate hosts (various species of *Ribes*, the currents and gooseberries). As mentioned earlier, the loss of western white pine as a management option on some forest sites has led to other pest problems, notably root rots. This is just one of the irrevocable effects blister rust has had on the white pine forests of North America.

Cronartium ribicola has a life cycle that spans between three and six years. During mid-summer to autumn, basidiospores are produced from teliospores on *Ribes*. These spores are airborne for relatively short distances, and those landing on pine needles may germinate and penetrate stomata during cool, wet conditions. High hazard regions are those where average temperatures in summer (July) are below 21 C with prolonged wet periods (Sinclair, Lyon & Johnson, 1987; Van Arsdel, Riker & Patton, 1956). In some regions, conditions in most years are too dry and warm during these periods. Infections proceed from needles to twigs, from twigs to branches, and from branches to the trunk of trees. Inconspicuous pycnia develop on the surface of infected tissue one to two years after infection. These give rise to conspicuous aecia, which burst through the stem tissue as blisters of fungal tissue (peridium) containing masses of orange aeciospores. Chlorosis of killed branches (flags) or tree tops develop as the infected area dies. Small trees are most vulnerable due to the fact that infected branches are relatively close to the main stem. Pruning branches of young trees on low to moderate hazard sites is an effective control. As the forest canopy begins to close, damage due to white pine blister rust subsides because spores produced on the alternate host below the canopy are less common and susceptible tissue is less accessible to them. Infections of older trees most commonly result from basidiospores that have originated outside that stand and develop at the tops of the trees.

During the early years of blister rust in North America, a vigorous program to eradicate alternate hosts from stands of white pine was undertaken. This was later abandoned in most areas after it was shown to be ineffective (Boyce, 1961; Peterson & Jewell, 1968). However, Ostrofsky *et al.* (1988) showed substantial benefit of a 70 year *Ribes* eradication program in Maine. Hazard ratings for sites based on climatic conditions during basidiospore release have been developed. White pines on steep slopes and near areas where *Ribes* are abundant (e.g., along streams and in openings in the forest canopy) are particularly vulnerable (Hunt, 1983).

Virtually all of the white pines of the world are susceptible to white pine blister rust to some degree (Hoff *et al*., 1980). Considering that the introduced *C. ribicola* is the only stem rust on the white pines in North America, it is somewhat surprising that some level of resistance is found in populations of most of these species. Breeding for disease resistance has been successful in North America, although the presence of many races of the pathogen that differ in virulence complicates these efforts (McDonald *et al*., 1984; Peterson & Jewell, 1968). Surprisingly, a major, dominant gene for resistance in sugar pine has held up well in the field (Kinloch & Dupper, 1987). Kinloch (1992) has speculated that this single gene may have evolved through selection pressure imposed by pinyon rust, which does not occur on sugar pine, but the gene is more frequent in sugar pine populations where pinyon rust is prevalent.

Most attention has been given to the commercially important white pines *P. strobus*, *P. monticola* and *P. lambertiana*, but the disease can be even more severe on other North American species. Limber pine is devastated in Alberta and elsewhere (Ziller, 1974). Whitebark pine is threatened by extinction in some regions, and the whitebark pine ecosystem is being converted to a spruce-fir ecosystem through the actions of blister rust and fire suppression (Keane, Morgan & Menakis, 1994; Krebill & Hoff, 1995). Many wildlife depend on the large seeds of whitebark pine, and these animal populations will continue to be affected.

19.9 DISEASES CAUSED BY NEMATODES

In contrast to the situation with agronomic crops, nematodes are not generally considered to be important pathogens of pines. Various root infesting nematodes are, however, known on pines and some of these can be moderately important in nurseries. The importance of soil-borne nematodes in agriculture has grown with repeated rotations and other intensive farming practices, and it is thus felt that nematodes have the potential to become important in

plantation forestry in the future. In recent years a nematode with an insect vector has come to be recognized as one of the most serious pathogens of pines. This unique nematode is commonly referred to as the pinewood nematode or pine wilt nematode.

19.9.1 *Pine wilt*

The pine wood nematode, *Busaphelenchus xylophilus* (Steiner & Buhrer) Nickle, apparently is native to North America but is currently active and killing large numbers of native pines in Asia, most notably in Japan (Mamiya, 1983; Wingfield, 1987a). The pine wood nematode is a classic example of an introduced pathogen on a population apparently lacking resistance. Where the nematode is native in North America, local pine species are highly resistant and unaffected.

The nematode has a fascinating and complex biology (Mamiya, 1983; Wingfield, 1987a). Its vector, long horn beetles (Coleoptera: Cerambycidae), themselves of secondary importance, emerge from dead trees in spring carrying large numbers of nematodes and proceed to maturation feed on the shoots of healthy pines. Nematodes enter the maturation feeding wounds and multiply rapidly in the resin ducts, resulting in rapid wilting of the host, perhaps due to toxins produced by the nematode (Bolla *et al.*, 1987). In dying and dead trees, the nematodes enter a mycophagus phase, where they feed on blue stain fungi. Dying trees are then attractive to the mature beetles that oviposit in the bark, thus completing the life cycle of the beetles and providing the nematodes in these trees with vectors for the next season.

When the pinewood nematode was first discovered as a pathogen in the USA in 1979 (Dropkin & Foudin, 1979), it was feared that it would have a very devastating impact (Dropkin *et al.*, 1982). The nematode had, however, been recorded in the country in 1929, although this was under another name (*Aphelenchoides xylophilus* Steiner & Buhrer) and in the absence of the knowledge of its pathogenic nature. It was later shown that in the USA, *B. xylophilus* commonly occurs in trees or tree parts dying due to factors other than nematode infestation (Wingfield *et al.*, 1982). It was furthermore shown that the nematode is not pathogenic on native North American pine species (Wingfield, Blanchette & Nicholls, 1984; Wingfield, Bedker & Blanchette, 1986). An ecologically important discovery was also that it is not only transmitted during feeding of beetles on healthy trees but also when the beetles oviposit in dying trees (Wingfield, 1983). Transmission during oviposition implies that *B. xylophilus* can maintain an exclusively mycophagus life cycle, which appears to be the norm in North America (Wingfield, 1987b).

Although the pine wilt disease was recognized as serious in Japan during the early part of this century (Mamiya, 1987), it was not until 1971 that the nematode was found to be the causal agent of this serious malady (Kiyohara & Tokushige, 1971). It seems likely that pine wilt disease will ultimately eliminate highly susceptible pine species such as *P. thunbergii* and *P. luchuensis* from Asian landscapes (Wingfield *et al.*, 1984; Mamiya, 1987; Wingfield, 1987a). Control of pine wilt is virtually impossible in Asia due to its introduced and epidemic nature. Despite this, considerable effort has been made to protect valuable shade and ornamental trees using insecticide sprays and chemical injection. Suppression of vector populations in Japan by removing borer infested trees from forests is vigorously pursued. Various attempts have also been made to implement a variety of forms of biological control, although the impact of these strategies is at this stage not known.

A similar but apparently different nematode occurs in European pines without causing disease. *Pinus sylvestris* is, however, highly susceptible to infection by *B. xylophilus* where it is planted in the USA, particularly in warmer regions. There is considerable concern that, if introduced, the pine wilt nematode could cause an epidemic in Europe. Indeed, this is the basis for very strict control measures pertaining to trade in conifer timber between North America and Europe.

Table 19.1. Discoveries, introductions and epidemics in the pathology of *Pinus*.

1870s	Hartig writes the first book on tree diseases describing Armillaria root rot, annosum root rot, red ring rot, a stem rust and a needle rust of pine.
1900s	White pine blister rust is introduced twice to North America on nursery stock raised in Europe.
1910s	Weir describes the biology and management of dwarf mistletoe.
	Sudden death of pine trees noted in Japan, now believed due to pine wood nematode.
	Sphaeropsis canker reported in Southern Africa.
1930s	Fusiform rust emerges as a management problem for southern yellow pines in the USA.
1940s	Phytophthora cinnamomi associated with littleleaf disease on old field sites in the southeastern USA.
	Pitch canker recognized in the southeastern USA.
1950s	Stump top colonization associated with the epidemiology of annosum root rot, and biological control developed.
	Red band needle blight appears in Africa, eliminating radiata pine as a commercial species in some regions.
1960s	Red band needle blight appears on radiata pine in Chile and New Zealand.
1970s	Cavities in living trees caused by stem rotters recognized as important wildlife habitat.
	Root rotted trees found to support endemic bark beetle populations.
	Pine wood nematode identified as cause of pine wilt in Japan and recognized as a pathogen in USA.
	New York (USA) epidemic of Scleroderris canker caused by European race.
	Brown spot needle blight appears in China and red band needle blight in Australia.
1980s	Characterization of host specialized variants or species of root pathogens in *Armillaria, Heterobasidion annosum*, *Phellinus weirii* and *Leptographium wageneri*.
	Pitch canker discovered in California (USA) and Japan. Also reported from Mexico, but perhaps indigenous there.
1990s	The importance of root rots and other diseases in the dynamics of forest ecosystems increasingly recognized.
	Pitch canker pathogen reported from South Africa.

19.10 CONCLUSIONS

Diseases have had a very substantial impact on pines in natural ecosystems as well as in plantations. We are gradually gaining an understanding of the complex and often subtle roles that pathogens play in natural forest ecosystems. This knowledge will be an important aid in the development of sound management practices, which should align our interests more closely to the natural dynamics of the forest. For instance, tree harvesting should mimic the effects of indigenous mortality agents, and fire needs to be incorporated into sustainable management of many pine ecosystems. Plantation forestry can also incorporate disease resistance and heterogeneity into unnatural pine ecosystems and thus lessen the impact of some diseases.

In spite of this awareness and the advantages of a biorational approach to forest management, indications are that diseases will become increasingly important in the future. We make this prediction based on the following scenarios:

a. Most of our so-called natural forest ecosystems are not following the natural dynamics of forest succession and rejuvenation. Fire has been a major component of pine ecosystems, and fire suppression is changing the composition of our forests, often to more disease prone species and abnormally high stocking levels. Loss of otherwise disease resistant species due to introduced pathogens will also make management of natural ecosystems more difficult.

b. The growing pressure to conserve natural forest environments is likely to increase the importance of plantation forestry. Plantations of native pine species have given rise to serious disease problems that were previously of minor consequence. These diseases will be favored by genetic uniformity of the host trees, often a low level of disease resistance, and in some cases, by the creation of fresh stumps.

c. The most devastating epidemics have arisen through the introduction of pathogens into new environments (Table 19.1). Globalization of our economies and the movement of people, packaging materials, seed and raw timber continue to increase, and the introduction of new pathogens into new environments thus seems inevitable.

d. Although evidence to this effect is still rudimentary, there is reason to believe that plant pathogens are rapidly evolving. Minor pathogens are presented with new niches through introductions to new ecosystems and alteration of the composition or dynamics of others. It would appear that agricultural practices have opened the door of opportunity for many new pathogens of crops, and, likewise, forestry practices may be putting considerable selection pressure on new pathogens to fill new niches.

The above may be perceived as a rather pessimistic view of the future. At this point, it is germane to note that considerable progress has been made during the course of the last century in our understanding of tree diseases (Table 19.1), and disease problems have been very effectively managed in many situations. The study of tree pathogens should be considerably enhanced in order to equip us with the wherewithal to deal with future disease problems. Efforts to exclude pathogens from new environments through more effective quarantine strategies should also be vigorously supported.

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