have not been developed as tools to control rust diseases of forest trees.

Control of Rust Fungi of Forest Trees

Forest pathology has been greatly influenced by attempts to control rust diseases. Avoidance of areas of high inoculum is generally recommended for new plantings of all forest trees. In the famous case of the introduced *Cronartium ribicola*, or white pine blister rust, extensive and expensive efforts were made to eradicate the alternate host (i.e., species of Ribes), albeit without much success. Pruning, avoidance of areas in which the environment favors infection by *C. ribicola*, nursery applications of fungicide, and programs to breed for resistance are all components of the arsenal deployed against white pine blister rust.

Breeding for resistance to the native, fusiform rust of loblolly and slash pines in the southern United States has also been combined successfully with silvicultural techniques of intensive management. Breeding for resistance to poplar rust is an ongoing battle both in Europe and North America, but again this subject is presented in more detail in this volume and elsewhere.

See also: **Pathology**: Diseases of Forest Trees. **Tree Breeding, Practices**: Breeding for Disease and Insect Resistance.

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Stem Canker Diseases

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Introduction

Canker diseases are caused by a diverse array of pathogenic fungi, and are grouped together based on similarities in the symptoms they produce on their host plants. Our treatment will be restricted to diseases caused by fungi in the group known as Ascomycetes. This includes most of the biotic pathogenic agents responsible for cankers, but does exclude at least one noteworthy group: the obligate parasites known as rust fungi, some of which cause diseases referred to as cankers (*see* **Pathology**: Rust Diseases). Even with this limitation, canker diseases represent a heterogeneous grouping, which is unified more by the nature and location of the damage on the tree than the appearance of the diseased tissue.

Pathology

A canker would typically be defined as a more or less sunken area on a stem or branch where pathogen growth has killed the underlying cambium, and which is often bordered by host-produced callus tissue. Thus, it is the combination of no growth where host tissue is killed and a somewhat elevated border of callus produced in response to infection that defines a depressed area known as a canker. However, many so-called canker diseases do not have symptoms at all similar to the forgoing description, and among the pathogens that produce typical cankers, some will do so only on certain plant parts, such as large-diameter branches, because younger branches die too quickly after infection for a canker to develop.

To account for variation in the appearance of cankers induced by different pathogens, three subgroupings have been recognized: perennial, annual, and diffuse cankers. Perennial cankers would most closely match the description given above. As the

name implies, perennial cankers persist for many years. Their appearance may change over time, reflecting the dynamic nature of the interaction between host and pathogen. For example, the sunken appearance becomes increasingly apparent as surrounding healthy tissue expands while growth has ceased in the cankered area. Concentric rings may develop around the canker due to annual production of callus tissue in response to infection (these have been termed 'target cankers'). In some cases the callus may expand into the interior of the canker and even cover it completely. The appearance of the canker may help to identify the causal agent, and many publications and internet resources provide photographs that are useful for diagnostic purposes.

Annual cankers are those that do not persist past a single year, so there is insufficient time for differential growth of infected and healthy tissue, and/or production of callus to produce the distinctive appearance of a perennial canker. Annual cankers are often associated with an effective host response that contains the infection, and the affected area may be overgrown with callus, which effectively 'heals' the wound. On the other hand, those in the third category, diffuse cankers, expand rapidly and outpace radial growth of the tree. The inability of the host to contain the pathogen effectively often results in girdling of the affected branch or stem. This type of canker would commonly be found in young tissue on a highly susceptible host.

In summary, canker diseases are those that affect branches and stems of various age and size classes, where growth of the pathogen leads to death of the colonized tissue, which, combined with structural alterations to the bordering healthy tissue, may or may not lead to a sunken appearance. Where cankers occur on young branches near the growing tip, the end result will likely be girdling of the affected branch and the disease might well be placed in the category of 'shoot blight' or 'tip dieback.' The same pathogen growing on larger diameter branches may produce a more discernible canker; *Diplodia pinea*, described below, is one example of a pathogen that produces both types of symptoms.

Economic Impact of Cankers

Where damage is restricted to tip dieback, the principal economic impact will be reduced growth due to loss of photosynthetic tissue and redirection of resources to defense and away from growth. This effect will be greatly magnified when cankers form on larger branches, increasing the proportion of crown affected. Cankers on the main stem can affect the entire tree by compromising transport of water to the canopy and photosynthate to the roots. Cankers on the main stem can also lead to differential rates of radial expansion, enhancing the risk of structural failure and providing points of entry for other wound-infecting pathogens, such as wood-decay organisms. Wood quality is affected as well. A higher proportion of non-woody cells reduces the strength of the wood, whereas discoloration and resin accumulation (two commonly co-occurring symptoms) impair the appearance of the wood and reduce its permeability to preservatives.

In general terms, one might ask how canker diseases developed into problems for forest trees, and if there are common features that can explain these syndromes and thereby suggest ways in which their impacts can be minimized. A first step toward answering such questions is to consider the diversity of pathogens that are associated with canker diseases and the nature of their relationships with host trees. Not surprisingly this exercise quickly exposes our lack of knowledge of pathogen biology and the host–pathogen interactions that give rise to disease syndromes. In particular, we are largely ignorant of how contemporary associations came to be; i.e., what was the relationship between host and pathogen prior to human intervention and how has the latter altered the situation? Although the information available on these subjects is incomplete, some well-studied examples provide a basis for speculation on hypothetical pathways to pathogenicity that may account for much of the present inventory of canker diseases.

Ecology of Canker-Causing Pathogens

We can begin by considering the ecological role of canker-causing pathogens in a native forest. Trees are primary producers in forested ecosystems and as such represent a source of fixed carbon to be exploited by other organisms, including fungi. In principle, fungi could access the nutrients available in standing trees through overt pathogenesis, leading to death of the host and assimilation of its constituent parts. No doubt fungi have pursued this strategy at various times, but it has not led to associations of long duration, for obvious reasons. Host trees susceptible to such an onslaught would be eliminated from the forest and leave relatively few propagules to seed later generations. Populations would thus be extirpated or, through natural selection, enriched with individuals more resistant to the aggressive pathogen. In the latter case, presumably either the pathogen would be eliminated for lack of a suitable host or a more stable relationship would ensue. Such a relationship could involve limited colonization of

living trees, whereby the pathogen obtains nutrients without severely impacting the host.

Weakly parasitic associations could develop as described above, or more directly through selection from strictly saprobic fungi for strains that are sufficiently invasive to gain limited access to host tissue prior to its demise. Regardless of how it came about, fungi employing this strategy can persist, albeit with limited growth in most cases, at the expense of their host, and may further benefit from their proximity to the underlying tissue, which can support more extensive growth when the tree, or a part of it, is no longer living. Such a relationship is obviously beneficial to the fungus, with a more variable and less predictable impact on the host. The literature on forest pathology clearly shows that many fungi exploit trees in this way and they are regarded as benign colonizers of nonliving tissue or minor pathogens of little consequence.

Relationships between organisms are inherently dynamic as they are subject to alteration due to physiological or genetic changes in either participant, and to changes in the ambient environment. These factors provide a framework within which pathways to pathogenesis can be recognized. In the context of a native forest, trees growing on sites to which they are well adapted could be expected to tolerate the presence of weakly parasitic fungi in their bark and to a lesser degree in more internal tissues. In fact, healthy trees commonly support the growth of many fungi within their living tissues and may never suffer ill effects from them (such fungi are often referred to as endophytes). On the other hand, trees growing under less suitable circumstances may be unable to devote the resources necessary to limit growth of parasitic fungi, and one or more of them may thus be allowed to grow aggressively and cause visible damage to the tree. Such parasites are generally referred to as opportunistic pathogens, and it seems likely they serve as one means by which the geographic range of a tree species is enforced. It follows from the nature of this relationship that its pathogenic manifestation is seldom observed, but its historical role in shaping biogeographical patterns in the forest may be profound.

Incidence and Severity of Canker Diseases

Evidence for an effect of growing conditions on the incidence and severity of canker diseases may be found in observations of numerous host–pathogen combinations. For example, in a US Forest Service publication on diseases affecting native conifers on the Pacific Coast, 12 of the 14 canker diseases included are described as being more severe on trees growing on poor sites and/or subjected to drought stress; the absence of this qualification in two cases may be due only to incomplete information on the diseases. A particularly dramatic example of site effects on disease severity is provided by cypress canker, caused by *Seiridium cardinale*. This disease is unknown in native stands of Monterey cypress, which are found on the California coast where temperatures are moderate and fog is frequent, but is common and often fatal to planted Monterey cypress in warm, dry environments farther inland. Thus, for a forestry enterprise and for landscape plantings, proper site selection is an important component of managing canker diseases.

Physical location will not be the only determinant of the growing environment to which trees are subjected. For example, stand density can impose limits on access to light and soil moisture, which may lead to stress that, in turn, may promote the activity of otherwise minor pathogens. This density-dependent effect of plant pathogens provides one mechanism for natural thinning, which will tend to promote stocking levels that are more sustainable. Just as dense stands can render light-deprived trees more prone to damage from canker diseases, shade-suppressed lower branches on otherwise vigorously growing trees are often preferred sites for development of these pathogens. As a result, weakly parasitic fungi participate in the early stages of nutrient cycling by initiating the breakdown of plant parts no longer making a positive contribution to the carbon economy of a growing tree.

Many factors that influence tree health and vigor can render the impact of canker diseases more severe. One commonly cited predisposing factor is water stress brought about by an abundance of neighboring trees and/or insufficient precipitation. *Diplodia pinea*, for example, is known to be more damaging on trees subjected to drought. Additional predisposing factors reported to be important for some canker diseases include: extremes of weather such as frost and hail, and infection by other pathogens such as dwarf mistletoe. Any of these factors could lead to elevated incidence of a disease in a native forest. Obviously exotic plantations invite the possibilities of more severe and persistent problems.

The foregoing discussion implies that vigorous, healthy trees growing under conditions to which they are well-adapted should not sustain serious damage from canker causing pathogens. This generalization is reasonably well supported, but is predicated on a critical assumption: that the depredations of pathogenic fungi have been moderated through a long

history of association between host and parasite. Thus, weakly parasitic saprobes in-waiting are a part of the environment to which native trees are adapted. Such adaptations will not necessarily be applicable to foreign microbes, which can expose inherent genetic susceptibilities of native trees, with catastrophic consequences.

Long-distance movement of parasitic fungi offers an alternate path to pathogenesis for tree-associated fungi. Here too the pathogen may have originated as a weak parasite, but in this case it has been moved to a new host to which it is preadapted for virulence. Whereas in its native environment the fungus was a benign associate of its host tree, it has the potential for more aggressive growth on a related host species that has not previously been exposed to the parasite, and lacks the ability to contain its growth. It should be added here that it is probably the exception when introduced fungi become successful pathogens; the failures are likely to be far more numerous but they are difficult to document.

In the sections that follow, we will provide a detailed account of three canker diseases, all of which have become problematic due to movement of the pathogen to new areas. For the first of these diseases, chestnut blight, the historical impacts have been particularly dramatic, whereas the second, pitch canker, is a more recent development that continues to unfold. For the third disease, *Diplodia* canker, pathogen introductions have been important, but movement of the host to suboptimal growing sites can also be a critical determinant of disease severity. The three diseases differ in the extent of the damage they have caused and the manner in which the host–parasite relationships have stabilized. Although this is but a small sampling of the many canker diseases known to affect forest trees, it will serve to illustrate key features found among pathogens causing this type of disease.

Chestnut Blight

The causal agent of chestnut blight, *Cryphonectria parasitica*, infects species of chestnut (*Castanea*) as well as several species of oak (*Quercus*) and chinquapin (*Chrysolepis*). The fungus is a weak pathogen on some live oak species in the southeastern USA. It is more damaging to European and eastern US white oaks and other *Quercus* species, causing twig and branch dieback and perennial cankers on larger trunks leading to mortality in some cases. In species of *Chrysolepsis* from the eastern USA, similar symptoms accompany more extensive mortality, whereas *Chrysolepsis* species in California are unaffected by *Cryphonectria parasitica*. All species of *Castanea* are susceptible to some extent. On Asian species (*C. crenata* and *C. mollissima*), the pathogen is predominantly an opportunist, living as a saprobe and infecting pruning wounds and weather-induced injuries, causing deformations and some canker damage, but rarely death.

The manifestation of chestnut blight on American and European chestnut species (*C. dentata* and *C. sativa* respectively) is infamously more serious than on those described above. The fungus was introduced to the USA in 1904 and rapidly spread through the entire native range of American chestnut in the eastern third of the country. Presumably, the disease was introduced on infected young trees or seed from Chinese chestnut planted as an ornamental in New York. The chestnut blight pathogen isolated from those trees was also known from Asia where it had little effect on native trees; but by the mid-1950s *Cryphonectria parasitica* had devastated native stands of American chestnuts, reducing the trees to dense collections of vegetative shoots emerging from the bases of otherwise dead trees, and ruining the vast economy supported by products harvested and manufactured from this once-dominant tree species. Similarly, introduction to Europe is thought to have occurred in the 1920s when Japanese chestnut trees, resistant to a disease which had plagued the local chestnut industry, were planted among the extant European chestnut in Italy, Spain, and France. *Cryphonectria parasitica* was first reported in Italy in 1938 and quickly spread throughout European stands causing severe and sometimes total crop losses over a 25-year period.

Development of the Disease

Chestnut blight is initiated when spores are introduced into wounds on stems primarily by the actions of insects and birds. The fungus grows to gain access to the inner bark and cambium layers and eventually, sunken cankers form on the bark. The cankers are characterized by long cracks and the orange–brown color of stromata (a mixture of fungal and plant tissue) that hold spore-containing pycnidia (**Figure 1**). Orange strands of asexually produced spores ooze from pycnidia during moist weather and are again spread by insects and birds and by rainsplash. Brown mycelial mats form under the bark, and eventually callus tissue forms at the margin of the elongated canker and the bark sloughs off. Successive callus formation and sloughing sometimes leave a bull's-eye appearance to the cankers. As the disease progresses, perithecia form in the stromata and produce ascospores (i.e., sexual spores) that are forcibly discharged and dispersed by the wind. Both

Figure 1 A stem canker, caused by *Cryphonectria parasitica*, on American chestnut (*Castanea dentata*). Photograph courtesy of Swiss Federal Institute for Forest, Snow and Landscape Research. Reprinted with permission from the *Annual Review of Phytopathology*, Volume 32 © 1994 Annual Reviews.

pycnidia and perithecia can be present within the stromata at the same time. The fungus continues to grow until it ultimately girdles the stem, restricting the flow of water thus killing the affected part distal to the infection.

On American and European chestnuts, the early stage of disease reveals yellow and brown leaves that remain on the branch for the first year of infection. Bare branches appear as a result of earlier infections. The disease affects twigs, branches, and trunks but not the roots, which continue to live and send up numerous vegetative shoots. These shoots are eventually subjected to infection by the pathogen, canker development, and dieback. New shoots soon form and the cycle continues; the roots never produce a mature tree.

Genetic Resistance

There is no control for chestnut blight on *Castanea dentata* in the USA. Breeding programs are plagued by a high frequency of disease escapes in resistance trials. Furthermore, many Asian varieties and Asian hybrids that are resistant or show tolerance to the pathogen are grown as ornamentals and for nut production, but also provide potential reservoirs of inoculum that threaten extant American chestnut trees that were previously protected by isolation from the native stands. Outbreaks in small, isolated plantings located in California, Oregon, and southwestern Canada have been kept in check only through sanitation and early intervention to eradicate affected trees. Also, small plantings of *C. dentata* in Wisconsin, 200 miles west of the westernmost boundary of the native range, have remained intact until the relatively recent appearance of *Cryphonectria parasitica*. Intensive research efforts are focused on controlling the disease there.

Biological Control

Biological control efforts hold considerable promise for management of chestnut blight in Europe. These measures follow from observations of natural recovery of European chestnuts in southern Europe in 1951. Strains of the fungus isolated from cankers on recovering trees had a different appearance in culture and were not as virulent as strains recovered from active cankers. This phenomenon was termed hypovirulence, and is now known to be conferred by a mycovirus that infects the pathogen and can spread to uninfected strains, thereby allowing diseased trees to recover. Transmission of the mycovirus requires hyphal fusion, which can only occur between genetically compatible strains of the fungus. In Europe, interstrain compatibility is common, and natural spread of hypovirulence led to recovery of diseased trees in some areas (**Figure 2**). This has been augmented with artificial inoculations of active cankers with hypovirulent strains of *C. parasitica* to provide effective biological control of chestnut blight. As a result, chestnut is still profitably cultivated in Spain and Italy. Hypovirulence has not proven to be an effective control measure in the USA, where the genetic structure of fungal populations is much more diverse. This limits compatibility between strains, and thereby restricts transmission of the mycovirus. As a result, hypovirulence is not widespread in the pathogen population and the recovery observed in Europe has not occurred in North America.

Pitch Canker

Pitch canker is caused by *Fusarium circinatum*, an asexual form (anamorph) that also produces a sexual stage by the name of *Gibberella circinata*

Figure 2 Callus tissue grows over 'healed' cankers that were inoculated with hypovirulent strains of *Cryphonectria parasitica*. Photograph courtesy of Swiss Federal Institute for Forest, Snow and Landscape Research. Reprinted with permission from the *Annual Review of Phytopathology*, Volume 32 © 1994 Annual Reviews.

Figure 3 Pitch canker on *Pinus radiata*; multiple infections caused by *Fusarium circinatum* lead to death of the affected branch distal to the girdling canker. Diseased branches are identifiable as naked tips or by the associated killed (brown) needles, where these have not yet fallen.

(teleomorph). Although the sexual stage is readily produced under laboratory conditions, it is rarely, if ever, seen in nature. Pitch canker was first described in the southeastern USA where it came to be regarded as a chronic problem affecting a number of southern pine species in plantations, seed orchards, and nurseries. In 1986, the disease was discovered in California as a cause of extensive damage and mortality to planted *Pinus radiata* (common names: Monterey pine and radiata pine). Soon thereafter, pitch canker was reported in both native forests and plantations in Mexico, in pine seedling nurseries in South Africa, and among planted pines in Japan. More recently, pitch canker has been documented as a cause of seedling mortality in Chile and Spain.

The nature of the damage caused by pitch canker varies with the tree species and the circumstances under which it is growing, but commonly involves dieback in the canopy to varying extents (**Figure 3**). Death of a branch tip results from a girdling canker on the current year's growth. Disease severity increases due to multiple tip infections and independent infections on larger branches. Complete girdling of the main stem can occur as well, most often probably as a result of multiple coalescing cankers rather than a single infection. Resin accumulates in and around infection sites, this being especially pronounced on older branches and the trunk of the tree (**Figure 4**). Cones and pollen-bearing strobili may also be infected.

Although pitch canker is a relatively minor problem for plantation forestry in the southeastern USA, it represents an evolving and increasingly serious problem in other parts of the world. In California, native *P. radiata* forests have been badly affected, and damage to the extensive landscape plantings of this species is a continuing problem. Although *P. radiata* has suffered by far the most damage from this disease, most pines native to California are also susceptible, and some of these have become severely diseased where they are exposed to the pathogen. Perhaps the greatest concern about pitch canker lies in its potential to attack exotic pine plantations in the southern hemisphere, where *P. radiata* is a critically important species.

Figure 4 Resin streaming from multiple cankers on the main stem of *Pinus radiata* caused by *Fusarium circinatum*. Reprinted with permission from Gordon TR, Storer AJ, and Wood DL (2001) The pitch canker epidemic in California, *Plant Disease*, 85: 1128–1139.

Although pitch canker was first described in the southeastern USA, it is not known if this constitutes the site of origin of the pathogen, or if it was introduced there. One other possibility for the aboriginal home of the pitch canker pathogen is Mexico, which is a center of diversity for the pine genus and where the population of *F. circinatum* is genetically quite diverse. In any case, most accounts of pitch canker in the southeastern USA suggest that the disease has 'stabilized' in this area, being widespread but rarely severe. On the other hand, where the pathogen is a more recent arrival, its geographic range may still be expanding. Thus pitch canker is regarded as an endemic problem in the southeastern USA but an invasive pathogen in California, South Africa, Chile, and Spain.

Factors in Susceptibility

The potential for pitch canker to pose problems for forest trees can be assessed by considering the basic

Figure 5 A stand of *Pinus radiata* that includes many pitch canker-infected trees, one of which (large tree on the right) has been killed by the disease. An adjacent tree (left of the dead tree) has remained free of symptoms due to genetic resistance.

elements required for a pathogen to cause disease: a susceptible host and a permissive environment. This is well illustrated by *P. radiata* which, as a species, is highly susceptible but does include some resistant individuals (**Figure 5**). In addition, some trees that become infected may eventually recover from pitch canker. This results from a loss of symptomatic branch tips through breakage in the vicinity of the canker and a lack of new infections, which has been attributed to systemic induced resistance. Thus, the damage caused by pitch canker to a stand of *P. radiata* will be determined, in part, by the proportion of trees that manifests inherent and/or induced resistance to the disease.

Where trees are fully susceptible to pitch canker, disease development may be constrained by environmental limitations. The pathogen requires a wound and ambient conditions that allow wounds to remain moist long enough for infections to become established. In the southeastern USA, weather-related events are regarded as the important cause of wounds for infection, whereas in California pine-associated insects appear to be the most important wounding agents. Where aboveground damage caused by pitch canker is a problem, atmospheric moisture is plentiful during periods of moderate temperatures. Such conditions are provided by warm summer rains in the southeastern USA, and by rain and fog along the California coast. Movement of the disease to more northerly locations in North America may be limited by progressively lower temperatures. In California, drier conditions in more inland areas are likely to be restrictive. It is noteworthy, however, that most of the approximately 4 million ha planted to *P. radiata* elsewhere in the world are in regions where the climate should be conducive to development of pitch canker.

As noted earlier, pitch canker can be a problem in seedling nurseries. Under these conditions it infects roots and may kill seedlings at or before emergence or almost anytime thereafter. In very young seedlings, symptoms of the disease are not distinctive, but in older seedlings (1–3 years) tree death is associated with girdling of the main stem near the soil line, where the accumulation of resin is similar to what is seen in the aerial phase of the disease. Where *F. circinatum* is operating as a soilborne pathogen, the environmental limitations described above are not likely to pertain.

From a global perspective, management of pitch canker should emphasize prevention, by avoiding importation of the pathogen into areas where forest resources would be at risk from this disease. Although *F. circinatum* can survive in association with various infected plant materials (and in soil), seed probably represents the most efficient vehicle for dissemination. Seed can be infested externally or internally and may give rise to seedlings that die quickly or survive without symptoms for 6 months and possibly much longer. Sowing infested seed will contaminate soil, which may thereafter serve as a reservoir of the pathogen, and subsequent shipment of symptomless seedlings provides an ideal mechanism for establishment of the disease in plantations or landscapes.

Genetic Resistance

In the long term, genetic resistance to pitch canker may have a role to play in disease management. In native forests, practices that promote regeneration may facilitate natural selection for more resistant genotypes, which are known to be represented within *P. radiata* populations, for example. In managed plantings, it may be prudent to avoid highly susceptible pine species altogether, in favor of those less prone to severe damage. However, where industries are heavily committed to *P. radiata*, as in Chile, New Zealand, Australia, and Spain, it should be possible to enhance the level of resistance in improved varieties. This can be accomplished through vegetative propagation of known resistant clones, but it is as yet unknown how many different genetic combinations may confer resistance. As a consequence, it may not be possible to deploy an array of clones that is sufficiently diverse to avoid undue risk of catastrophic failure due to selection for more virulent strains of the pathogen. Likewise, the heritability of resistance has not been established so the prospects for increasing resistance through family-level selection cannot yet be assessed. Research is needed to better understand the genetic basis for resistance to pitch canker and facilitate its application to disease management.

Diplodia Shoot Blight and Canker

Diplodia shoot blight and canker, caused by *Diplodia pinea* (syn. *Sphaeropsis sapinea*) is common in temperate regions worldwide. This pathogen infects conifers in both the Pinaceae and Cupressaceae, including various species of *Cedrus* (cedar), *Juniperus* (juniper), *Picea* (spruce), *Pinus* (pine), and *Pseudotsuga* (Douglas-fir). The disease is most frequently seen on two- and three-needled pines, on which tip dieback is the most common symptom (**Figure 6**). Cankers caused by *D. pinea* are characterized by elongated, depressed areas on branches or stems, often with resin flow on the outer bark. When the bark is removed, resin-soaked wood is visible (**Figure 7**). In older infections, callus growth may be observed around the edges of the canker. Where young branches are infected, needles and shoots stop expanding and are quickly killed, later appearing stunted and necrotic. Infected stem tissues are initially water-soaked in appearance, later becoming purplish-brown and necrotic. Repeated tip and/or stem infections can lead to branch death. The seedling phase of the disease, called collar rot, is characterized by similar infections, which may girdle the stem and cause mortality of seedlings.

Figure 6 Shoot tip dieback on ponderosa pine (*Pinus ponderosa*) in California caused by *Diplodia pinea*. Photograph courtesy of M.H. Morris.

Figure 7 Resinous cankers, caused by *Diplodia pinea*, on a branch tip (a) and the main stem of pine (b). In both cases, the bark has been removed to show resin-soaking and discoloration in the underlying wood. Photograph 7b courtesy of US Department of Agriculture Forest Service, North Central Research Station.

Figure 8 Perennial canker caused by *Diplodia pinea* on trunk of radiata pine (*Pinus radiata*) on a plantation in South Africa. Photograph courtesy of M.J. Wingfield.

Economic Importance

Diplodia shoot blight and canker is seldom damaging to trees planted within their natural range and as such it is rarely important in native forests. On the other hand, exotic pine species in plantations or in landscape plantings can be severely affected. For example, this disease has caused economic damage to *Pinus patula* (Mexican weeping pine) and *P. radiata* in plantations in the southern hemisphere (**Figure 8**) and to both exotic (e.g., *P. nigra* and *P. sylvestris*) and native (*P. resinosa* and *P. banksiana*) pines in the north central USA.

Two subgroups of the pathogen have been described, morphotypes A and B, which are differentiated on the basis of colony morphology and growth rate in culture as well as by isozyme profiles and DNA polymorphisms. The two morphotypes also exhibit differential aggressiveness to pine, with A being more aggressive. Recent work indicates the two morphotypes should be considered separate species, with the A morphotype being associated with the binomial *D. pinea* and the B morphotype designated as a new species, *D. scrobiculata*.

Life Cycle

The fungus produces spores in fruiting bodies (pycnidia), visible with a hand lens, on needles, fascicle sheaths, cone scales, and bark. Even apparently symptomless trees may harbor spores formed on cone scales. Pycnidia typically form in the spring of the year following infection. Spores contained in pycnidia are transparent at first and later become brown. These are released during wet weather and are spread by rainsplash or wind-driven rain. Spores can infect expanding needles and shoots, and second-year cones, where surface wetness persists for 12 h at 12–36°C. Infection of mature current-year and older

shoots and stems can occur through fresh wounds, such as those caused by insects, hail, or pruning.

This fungus is also considered an endophyte because it can be associated with symptomless infections. Such infections are thought to become damaging when the host is predisposed by some form of abiotic or biotic stresses. Drought stress in particular has been well-documented to induce damage in *D. pinea*-infected trees. The ability of *D. pinea* to exist within its host as a latent pathogen suggests that this fungus has a long history of association with pines. The observation of more severe disease on off-site or stressed trees and on exotic species in plantations fits the pattern described above for weak parasites that become opportunistic pathogens when conditions are altered to the detriment of the host.

Management

Management of diplodia shoot blight and canker in plantations depends primarily on avoidance of the disease. Careful selection of the planting site and of a timber species appropriate for that site are important to minimize stress. High soil fertility should be avoided as this has been shown to aggravate the disease in some situations. Should the disease reach severe levels within a commercial stand, replanting or early harvest may be warranted. In nurseries, seedling beds should not be established in the vicinity of older trees as these may serve as a source of inoculum. Seedlings can be protected by frequent fungicide applications until shoots and needles are mature. On Christmas-tree farms, fungicides may also be employed to protect trees, but control depends primarily on avoiding shearing operations during wet weather.

Conclusion

The three canker diseases described above illustrate ways in which movement of plants and pathogens can lead to new or significantly altered host–parasite relationships, with devastating consequences. Chestnut blight in North America offers an extreme example of an exotic pathogen preadapted for virulence, where susceptibility of the host was complete and the result was disappearance of a dominant tree species throughout a vast forest ecosystem. Pitch canker is also caused by an exotic pathogen, but one that has not found a host as devoid of resistance as the American chestnut was to the blight pathogen. Susceptibility to pitch canker is the rule in *P. radiata* and mortality has been widespread, but a small percentage of individuals proved to be highly resistant and many others have sustained relatively minor damage. This, combined with remission in some severely diseased trees, suggests that the host–pathogen relationship will stabilize in a way that allows *P. radiata* to remain a defining feature in forests that bear its name. Likewise, chestnut blight in Europe show signs of stabilizing, not through a response of the host but because the pathogen is itself debilitated by a parasite. Thus, various mechanisms can serve to constrain the aggression of an invasive species, imparting a resilience to forested ecosystems that has no doubt been critical to their persistence.

Diplodia pinea offers a contrast to chestnut blight and pitch canker in that native forests suffer little damage from it. In this case, too, introduction of the pathogen into new areas has created problems, but primarily for exotic plantation forestry. Thus, movement of the host to suboptimal sites is the primary reason *D. pinea* is a damaging pathogen. The genetic constitution of most potential hosts prevents aggressive growth of *D. pinea*, except where trees are compromised by stress. In a broad sense, all three canker-causing pathogens are opportunists that differ in the nature of the opportunities they have exploited.

In terms of management, these diseases illustrate the importance of prevention, especially where this can be achieved on a regional scale by limiting new pathogen introductions. Unfortunately, historical barriers to the spread of plant pathogens, such as spatial gaps in host distribution, are increasingly likely to be breached through steadily increasing trade and travel. Ever-present threats include not only known major pathogens but also untold numbers of minor pathogens that await their opportunity to flower into full virulence. Thus, the implementation of any new and effective measures to limit more widespread distribution of plant-associated microbes offers great potential to reduce the risk of major new disease problems for forest resources worldwide.

See also: **Health and Protection**: Diagnosis, Monitoring and Evaluation. **Pathology**: Diseases of Forest Trees. **Tree Breeding, Practices**: Breeding for Disease and Insect Resistance.

Further Reading

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Insect Associated Tree Diseases

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Introduction

The causes of tree diseases range from the simple – with one principal damaging agent – to the complex – where a number of damaging agents interact.

A key role for insects in some simple diseases is that of vector for a pathogen. This is particularly important for certain fungal diseases. However, insects also play a vital part in the dissemination of some tree-pathogenic nematodes, phytoplasmas, xylem-limited bacteria, and viruses.

In complex tree diseases insects may play a variety of roles. At an early stage, leaf-eating insects may act as agents of stress. At a later stage it may be bark- or wood-inhabiting beetles that deliver the final death blow to the tree.

Fungal Diseases Vectored by Insects

Among the fungi, there are three main taxonomic groups: the Oomycotina, the Basidiomycotina, and the Ascomycotina (plus the closely allied Fungi Imperfecti). The majority of fungal pathogens with known insect vectors are either Ascomycotina or Fungi Imperfecti, and most of the ascomycetes fall within two genera of the family Ophiostomataceae – *Ophiostoma* and *Ceratocystis*. In addition, most of the imperfect fungi are *Chalara*, *Leptographium*, or *Verticicladiella*, genera that would be expected to have *Ophiostoma* or *Ceratocystis* perfect states. The nature of the insect–fungus relationship with this group of pathogens is discussed in some detail later in this article. Among other ascomycetes, recent attention has focused on the role of insects as vectors of *Fusarium circinatum*, the cause of pitch canker. This major pathogen, first recognized in the eastern USA, was reported from California in 1986. Studies there on *Pinus radiata* have focused on the twig-feeding bark beetles *Pityophthorus* spp., but other beetles, including the cone beetle *Conophthorus radiata*, have also been implicated. In southern Europe, a cone-feeding insect, the seed bug *Orsillus maculatus*, has been found to transmit the important canker pathogen *Seiridium cardinale* to *Cupressus sempervirens* cones. Once the cones are colonized by the fungus they become a source of inoculum for branch infection. Interestingly, it seems that when *S. cardinale* arrived in Europe, it took over a long-established relationship between the insect and a nondamaging fungus *Pestalotiopsis funerea*.

Although the Basidiomycotina include many fungal pathogens of trees, the only indubitable case of insect transmission concerns the *Amylostereum* species that are transmitted by woodwasps in the genera *Sirex* and *Urocerus*. In most parts of the world these organisms do little damage but in Australia and New Zealand, *Sirex noctilio* and *Amylostereum areolatum* can cause significant losses in *Pinus radiata* plantations, especially during periods of drought.

The fungus is carried by the adult siricids in a pair of small invaginated intersegmental sacs protruding into the body. These are connected by ducts to the anterior end of the ovipositor. During oviposition (**Figure 1**), spores of the fungus are 'injected' into the sapwood of trees and developing mycelium then invades the wood around the oviposition hole and larval tunnels. As is commonly the case with xylem pathogens, a zone of reduced moisture content develops around the tissue occupied by *Amylostereum* and this ensures that the *Sirex* eggs hatch and the larvae develop in relatively dry wood. In addition, host resinosis is reduced in colonized tissue, and this also favors larval development. Female larvae, from the second instar onwards, carry the

Figure 1 *Sirex* woodwasp, vector of basidiomycete *Amylostereum* ovipositing on pine. Courtesy of Forest Commission.