LESSON 3 Root Diseases

LESSON OVERVIEW

CONTENT

This third lesson of the forest pathology course deals with root diseases. As a group, these diseases are perhaps the most serious and damaging in western North American forests. You will encounter them almost every day as you work in the forest. Sometimes they occur as small groups of dying trees; sometimes as areas of several hundred hectares in which certain species have been killed and replaced by others; and sometimes there is little above-ground evidence of their presence, although root mortality leads to substantial decreases in increment, while decay of the lower bole also represents a significant loss.

The content of this lesson is discussed under the following main topics:

- Types of root diseases
- The soil environment
- Root structure
- The main basidiomycetous root pathogens
- Infection
- Root diseases caused by non-decay fungi

OBJECTIVES

When you have completed this lesson, you will be able:

1. to summarize the role of forest soil organisms in the spread of root diseases;
2. to describe root structure and the main infection pathways for root diseases;
3. to diagnose the major conifer root diseases of western conifers in the field;
4. to outline the major requirements for infection and spread of common root disease;
5. to describe the role of root diseases in the natural ecology of western forests; and
6. to make silvicultural prescriptions for stands infected by root disease.

LESSON STUDY INSTRUCTIONS AND ASSIGNMENT

You should start this lesson by reading Chapter 16 in Manion (1991), and the section in *Common tree diseases of British Columbia* (1996) by Allen, Morrison and Wallis that deals with root diseases (pp. 2–24). Then study the material below, including the Pest Leaflets Numbers 3, 15, 56, and 67, and two recent summaries of root disease (Thies and Sturrock, 1995. *Laminated Root Rot in Western North America.* PNW-GTR-349; and Morrison, Merler and Norris, 1992. *Detection, recognition and management of Armillaria and Phellinus root diseases in the southern interior of British Columbia.* FRDA Rep. 179), both supplied with the course manual package. Finally, complete the self-testing/review questions at the end of this lesson.

COMMENTARY

Types of Root Diseases

The major root diseases can be divided into three main groups. The first and most important of these is caused by a group of fungi belonging to the Basidiomycotina. In many ways they resemble the decay fungi, but with the added characteristic that they can invade and kill living bark and sapwood. (Manion calls these "host-dominant tissue-nonspecific diseases.") The second group consists of a set of pathogens that invade young succulent roots, although they may subsequently colonize older, larger roots as well. This second group contains many important pathogens of young seedlings, particularly in a nursery setting. (Manion: "pathogen-dominant tissue-nonspecific diseases.") Members of this group do not figure prominently in west coast natural forests, possibly because invasion by them is strongly inhibited by ectotrophic mycorrhizae, and these are virtually universal in west coast forests. This lesson will not deal with this second group; rather it will be discussed under the heading of seedling diseases in a later lesson. The last, small group consists of fungi that live in the sapwood of roots and interfere with the transpiration stream, but do not have the ability to digest wood. (Manion: "host-dominant tissue-specific diseases.") As in all classifications, there are always a few individuals that do not fit well into the established scheme. *Rhizina undulata,* the cause of the "tea break disease" is a case in point.

Before turning to a discussion of the most common and damaging root diseases, it will first be necessary to discuss the soil environment in which these pathogens have to spread, and to describe root structure. This will help us to understand why these pathogens behave the way they do.

The Soil Environment

Soils consist of two major layers, namely the forest floor made up of litter in various stages of decomposition and the mineral soil, usually made up of several distinct horizons. Each of these layers has its own peculiar properties, and its own particular micro flora and fauna. In many coniferous forests in cool, moist climates, the forest floor is well developed. Most of the processes relating to release and take-up of mineral nutrients occur in this layer. The forest floor is particularly rich in micro-organisms. Fungi, bacteria and actinomycetes abound, all feeding on and digesting the litter (including dead roots) and each other. The soil fauna, in turn, feeds not only on the dead litter, but also on the microflora. Thus there are very large numbers of species competing for the same energy and nutrient supply. Trees compete for the mineral nutrients with other plants and also with the microflora and fauna, but not for energy in the form of organic compounds.

Antagonism

In the complex soil system, various interactions between organisms are common. One of these is **antagonism**, which may be defined as the mutual inhibition of fungi (or micro-organisms) apparently caused by

toxic products. Antagonism can often be observed on agar plates where two colonies of different species may stop growing before they meet, leaving a clear zone between them. Antagonism refers to a relationship between a pair of organisms, and is not caused by competition for mineral nutrients and energy; antagonism occurs on Petri plates where nutrients and energy are in abundant supply.

Mycostatis

A more common phenomenon is **mycostasis**, which may be defined as the general inhibition of microbial activity in soils and other complex microbial communities. For instance, if one takes a core of forest floor and measures the rate of carbon dioxide evolution, then sterilizes that core, inoculates it with one of the fungi that was present in the original microbial community, and measures it again, the new rate of carbon dioxide evolution is often much higher. Similarly, spores often germinate poorly in natural soils (although they may remain alive for quite a while), but much better on the same soil after sterilization.

There are several causes of mycostasis. One is antagonism. Another is competition for mineral nutrients and carbon sources (energy). Thus, adding sugar or NPK to a forest floor core will increase carbon dioxide evolution substantially. There may also be chemical effects. It has been argued that mycorrhizal fungi decrease the rate of breakdown of litter. Where that is the case, it is probably because they have their own external energy source (the tree host) and can therefore compete strongly for the available nutrients that other micro-organisms need to grow. In mineral soil horizons mycostasis is much less pronounced.

Thus natural soil can be viewed as a hostile environment for micro-organisms. Mycostasis is effective in reducing the activity of various pathogens. Several major root diseases are restricted to growth on and in roots, and do not grow freely in soil unless they have a large energy source, usually in the form of a substantial volume of wood, available to them. Some grow on root surfaces in the mineral soil but not in the forest floor where the effect of mycostasis is much more pronounced. Several other root pathogens occur in soil as dormant spores that do not germinate until they are stimulated by root exudates which provide both minerals and energy. This latter group includes the diseases that are inhibited by vigorous mycorrhizae, possibly via antagonism and the reduction in exudates from root tips colonized by ectotrophic mycorrhizal fungi. It contains several serious diseases of bare-root nursery stock, but few that are ever serious in our native conifer forests.

A good example of the effect of mycostasis on disease development is provided by the "**tea break disease**" caused by *Rhizina undulata* Fr.:Fr. [refer to Allen, Morrison and Wallis, 1996, p. 24]. The etiology of tea break disease is interesting: First noted in Great Britain, it was observed that small groups of conifers which once surrounded small fires began to die several years after. It so happened that the fires were made by forest workers to brew tea. It was found (after the pathogen, an Ascomycete producing irregular apothecia, was identified) that the pathogen possesses some peculiar characteristics. First, the ascospores

require a heat stimulus of about 40°C to break dormancy before they can germinate; and, second, spores are very resistant to heat and can survive short periods of exposure to 70°C or more. A fire sterilizes a shallow layer of soil below it. *R. undulata* spores survive on the edge of the sterile area, receive their heat stimulus, and germinate and invade the sterile area after the fire has gone out and the soil has cooled down. From this rich food base, the pathogen can invade and kill roots of surrounding trees. Eventually full mycostasis is reestablished and further spread stops. In the meantime the fungus has produced spores which are carried into the surrounding forest, where they lie dormant in the forest floor until the next fire.

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A similar set of events occurs following slash burning. If the pathogen is present in the forest floor as dormant spores, large colonies of the active fungus may be established. Planting seedlings in such

colonies results in rapid invasion and death. A few years later, when the regeneration survey shows plantation failure, the pathogen is no longer active and difficult to identify. Replanting is usually successful. Regular slashburning of successive cutblocks within a drainage may lead to a buildup of the pathogen. Long periods without fire results in its decline and virtual disappearance.

Root Structure

The structure of roots resembles that of stems (except that roots don't have a pith). At the ends lie the root tips. These are mostly succulent tissue consisting of cortex, and enclosed in an epidermis, with a small vascular bundle in their center. Root tips don't last very long. Within a year of being formed (and on fast growing roots much sooner), a layer of cork forms around the vascular bundle, and the cortex and epidermis die. At this point the living root segment may be much smaller in diameter than the original root tip. However, starting at the same time, the vascular cambium begins to form xylem and phloem, and the root starts to grow in diameter. Each year a layer of wood is formed, and as root grow in diameter, the bark is stretched more and more. Eventually the outer bark dies, a newly formed cork layer being the boundary between the living and dead bark. This happens both in stems and roots of course, but much later in roots. The dead bark is called the rhitidome.

Roots are opportunistic. Those that find themselves in locations rich in nutrients and/or water, and in well oxygenated locations develop quickly. Such roots can grow over a meter a year. Other roots in less favourable positions may grow very slowly. Thus on a given tree some roots may be very vigorous, and other moribund. That variation in vigour becomes important in defense reactions. Vigorous roots can respond quickly and vigorously to injuries including attack by pathogens, and may be resistant; moribund roots cannot do so, and may succumb.

The Main Basidiomycetous Root Pathogens

Most of the important root diseases in west coast coniferous forests are caused by a group of fungi belonging to the Basidiomycotina that in many ways resemble the decay fungi. Large parts of the life cycle of these root disease fungi can be in a saprophytic phase, meaning that they live on dead tissues (mostly roots and stumps), and derive their energy by decay of wood and bark tissues. However, they are also able to invade living roots, killing both the living bark and sapwood. It is difficult to draw a sharp distinction between this group of organisms and "regular" decay fungi. Most decay fungi are restricted to dead heartwood and cannot invade and decay living sapwood. However, there are some exceptions. Several members of the genus *Phellinus,* for instance, can invade sapwood from the heartwood. These species are still classified as decay fungi because they develop primarily in tree boles rather than roots, and because they seldom kill trees directly by girdling the sapwood completely. In root pathogens this parasitic ability is stronger, and of course, root pathogens develop primarily on roots rather than on tree boles.

Spore Infection

Infection of new trees can occur in two ways, namely by spores, or by vegetative spread from infected adjacent trees, usually at root contacts or grafts. Spore infection is common for some root diseases and rare or virtually absent in others. Once they have been established in a tree, all of the root pathogens discussed here can spread from tree to tree via root contacts. Thus they commonly develop as infection centers. Such centers may consist of groups of dead trees in which trees near the middle have obviously been dead for some time, while trees along the periphery are in the process of dying, and may show various crown symptoms. In other cases, however, the root disease may not result in tree death, but only in decay of the center of the larger roots and the base of the trunk and some fine root mortality. In such instances one can still speak of root disease centers, but they may not be very obvious because all trees remain alive, even though they may be growing slowly.

It is important to know whether spore infection plays a significant role in the establishment of new centers. If it does, then we can expect new centers to be established in stands that were previously disease-free. Also, it will then be important to ask what conditions are necessary for the establishment of new centers, so that such conditions can be avoided. Some root pathogens, for instance, commonly infect fresh scars and stumps. Their entry can be prevented by proper treatment of stumps. If spore infection is rare, as it appears to be for some of the common root diseases, then all root disease centers in a stand can be traced back to centers that existed in a previous stand. In such cases, control will be aimed at removal of inoculum in stumps and roots or the use of resistant species. The length of time that the pathogen remains viable in stumps and roots will also be an important consideration. In root diseases in which spore infection is common, vegetative spread and survival from one generation to the next also occurs, so control measures must be directed at both prevention of infection and inoculum removal.

Much of the evidence for spore infection is based on genetic analysis of isolates taken from either single large root-disease centers or groups of small ones. Nowadays this is usually done by some form of DNA analysis. In a single mycelium resulting from vegetative spread, all the DNA will be identical, whereas in the case of spore infection, each separate infection will have different DNA. The details of how differences in DNA are detected need not concern us here: several different tests are possible.

A second, somewhat less precise way of identifying genetically different mycelia is to match isolates in Petri dishes. Isolates that are genetically identical will grow together smoothly, while genetically different isolates usually react by forming distinct boundaries. The larger the areas occupied by genetically uniform mycelia, the smaller the relative importance of new spore infections in the life cycle of the root disease.

We are now ready to consider some of the major root diseases of cool coniferous forests, including *Phellinus weirii, Armillaria ostoyae, Heterobasidion annosum, Inonotus tomentosus,* and *Phaeolus schweinitzii.* Together these comprise the major root diseases of western coniferous forests, and all of them are common and important in at least one of the major ecological zones of the forest of British Columbia.

Phellinus weirii

Phellinus weirii (Murr.) Gilbert (previously known as *Poria weirii* Murr.) causes a disease known as yellow laminated root rot. The geographic range of this pathogen coincides roughly with that of Douglas-fir. Susceptible species include spruces, true firs, hemlocks, Douglas-fir and larches. Pines and cedars are moderately resistant. Hardwoods are immune.

infection and spread

P. weirii survives from one rotation to the next in the stumps and roots of the old trees. Spore infection is apparently very rare. When a living root of a neighbouring tree grows in contact with a *Phellinus*-infected root, the fungus spreads to that root. The initial spread of the mycelium is on the root surface; this is called **ectotrophic** mycelium. The fungus forms a layer of white to gray mycelium between the soil and root. In some locations (particularly dry sites and soil horizons high in organic matter), there may also be a brown fungal sheet (an external zone line) between the normal vegetative mycelium and the soil. Once the fungus is well established, it penetrates and kills the living bark and enters the sapwood of the root. Here it causes a reddish-brown stain. The fungus continues to advance in a proximal and distal direction, both on the root surface and inside the root, killing as it goes, until it girdles and kills the base of the tree. The root surface mycelium can form only on roots in the mineral soil. Rate of growth along roots in moist mineral soil is about 50 cm per year. In roots in the forest floor, progression is limited to advance in the sapwood, and, in large old roots, also within the rhitidome. In these two zones, the rate of advance is much less, and probably no more than 25 cm per year. As decay progresses, small (1 by 2 mm) elliptical pockets appear in the earlywood part of the ring. These then coalesce and the wood breaks into sheets (lamina) that the disease is named after. Eventually all the wood is decayed, leaving a cavity. The small elliptical pockets are filled with fungal mycelium, and within that mycelium are specialized setal hyphae (thick-walled, straight, dark hyphae), that can be seen with a hand lens. These are a good diagnostic feature of *P. weirii.* Bark is very resistant to decay by *P. weirii* (and all other decay fungi). Sometimes all that is left of infected trees is hollow bark rings, all the wood having decayed. In such rings the knots are usually still present. Obviously, by the time all the wood is gone, the tree has been dead for quite a while, and other tree species or shrubs will have grown up in the old disease center.

Young trees are killed quickly (1–3 years from the time of the first crown symptoms to death); older trees (40–60 years old when first infected) may survive for decades, but such trees produce little increment. Instead, the host spends much of the available energy

producing new roots to replace those that have been killed by the pathogen. The fungus can cause some butt rot, but it seldom advances more than a few meters up the trunk. The fruiting body is a perennial, brown, resupinate conk, and is usually produced on windthrown trees.

P. weirii is best thought of as a disease of the site. Once an area has become infected, the fungus survives from one generation to the next in stumps and roots. It can survive in large stumps for as long as 80 years, and is usually still present as patches of ectotrophic mycelium on the major roots of 20-year-old stumps. So long as stands of susceptible species succeed each other on the site, the infected area slowly grows larger. If the area is occupied by a non-host species, such as red alder, for a rotation, the pathogen may die out, or it may survive in only a few large stumps, and start to spread from there again once a stand of host species is re-established. Thus, "good forest practice" in lower elevation coastal forests, consisting of rapid and complete regeneration of clear-cuts by planting Douglas-fir and other susceptible conifers, in fact promotes the survival and rapid spread of this pathogen!

identification

P. weirii can be identified in a number of ways. First one looks for infection centers. These consist of small openings in the canopy in which susceptible conifer species have been killed, and are being replaced by hardwoods or more resistant conifers such as western red cedar. The dead trees may still be standing. More commonly, they lie criss-cross in the opening (in contrast to windthrown trees which all lie in the same direction), with all the major roots broken off close to the base of the bole, forming typical "root balls" Along the periphery of such centers one will normally see symptomatic crowns on susceptible hosts. Crown symptoms develop as follows: first, height growth declines, leading, in pole-size stands, to rounded rather than sharply pointed crowns; shortly afterwards, diameter growth also slows; then crowns slowly turn chlorotic, older needles are shed, and a crop of distress cones may be produced. Crowns do not normally turn red, except when death is due to bark beetles invading infected and weakened trees. Finally, the tree dies.

However, there are several root pathogens that form similar root disease centers. To make a proper diagnosis one must look for one or more of the following:

- ectotrophic mycelium on roots of infected trees in mineral soil;
- red-brown stain in the inner sapwood just above the ground in patches directly above major infected roots (or in such roots);
- typical laminar advanced decay in the center of rotten roots and in the broken roots of windthrown trees.

Fruiting bodies may also help to identify the pathogen, but these are not produced consistently, and are often absent. The symptoms are most easily seen in pole stage even-aged stands. In plantations up to 15 or 20 years of age, affected trees may appear singly, and only ectotrophic mycelium on roots of infected trees is consistently evident. In such cases, however, evidence of the pathogen can also be found in the stumps of

the previous rotation. In stands that are much older, *Phellinus* often acts as a butt rot, and typical root rot centers are harder to see because they will now be occupied by resistant and immune species, forming a full canopy.

Recently it has been recognized that there may also be diffuse *P. weirii* infection. Diffuse infection consists of individual or small groups of infected trees in otherwise healthy stands which do not develop into normal root disease openings. Instead, in diffuse infection, the pathogen acts primarily as a butt rot. Infected trees remain alive. Some roots are killed, and slight crown symptoms may develop. The major loss in diffuse infection is butt rot and increment loss. Diffuse infection is much more difficult to diagnose because above-ground symptoms are slight. At harvest, diffuse infection will always be evident as stain and decay of the stump. It is not clear why some *P. weirii* infections develop into centers while others don't. Nor is it clear why diffuse infection occurs in some stands (usually together with normal root disease centers) while in other stands all infections develop into normal root disease centers.

assessment

In order to make appropriate prescriptions for *Phellinus*-infected areas, it is first necessary to assess disease severity. This is best done by a line intercept survey. A base line is established in the stand in question, and survey lines are then laid out at regular intervals at right angles to that base line. The proportion of the length of such survey lines that falls in *Phellinus* centers is then an estimate of the proportion of the stand area infected by the disease. Only above-ground symptoms are taken into account. The boundary of a root disease center is defined as a line halfway between trees with crown symptoms (reduced height growth; chlorotic, thin crowns) along the periphery of the center and the surrounding healthy trees. The pathogen is usually present on roots beyond that boundary, but its extent cannot be determined without a lot of digging, and that is impractical for operational surveys.

Such a root rot survey gives an indication of the amount of disease at the time of the survey. It is often necessary for planning purposes, however, to estimate the extent of the centers and the losses at the time of harvest. To do so, one must also measure the size of each of the centers encountered. One can then model the spread of the pathogen, based on an average or site-specific rate of spread, taking into account the changes in pathogen behaviour with increasing age of the stand. The process is described in some detail in the following report by Bloomberg (1983) *A ground survey method for estimating loss caused by Phellinus weirii root rot. III Simulation of disease spread and impact.* CFS, PFRC, Inf. Rep. BC-R-7.

management

After *Phellinus* root disease has been diagnosed and assessed in this fashion, a reasonable management plan can be formulated. There are several options. First, it is usually best to leave infected stands until the healthy portions reach a merchantable size. Infected stands should be considered for an early harvest. However, decisions on harvest schedules

involve many considerations, such as the age class structure of the management unit, the location of infected stands, and the requirements for various types of logs and species mixes, as well as appropriate summer and winter logging areas. Any decision should take into account that losses increase with time at a reasonably predictable rate. Sometimes it is possible to salvage individual *Phellinus*-infected trees before they die, and where that is the case, the loss in volume will be less, although logging costs will usually be high.

A plan to deal with *Phellinus* must be in place before any harvest commences. There are basically two ways of dealing with the disease: eradication of the pathogen from the site, or regeneration by resistant species. Eradication is difficult. Fire (slashburning) doesn't work — the temperature a few centimeters below the surface does not reach lethal levels. Currently available fungicides are of limited use because they cannot be applied to the places where the pathogen survives (on roots and in stumps). New, more volatile fungicides, such as chloropicrin, are being studied, and these hold some promise, although their application will probably be difficult. Biological methods are also being studied. In general they aim to inoculate stumps with fungi that are strong competitors of *Phellinus* and will replace it in wood, or at least limit it to the wood it occupied at the time of harvest, so that it dies out sooner.

Stump removal. The main technique of eradication is to remove the stumps and major roots. All that is necessary is to lift the stumps out of the soil. Phellinus will then retreat to the inner portions of such stumps, and roots of the new stand will not come in contact with it. Of course, some *Phellinus* will survive on small roots left in the soil, but when such roots are broken, the natural boundaries between *Phellinus*-occupied roots and the soil microflora are broken, and the remaining *Phellinus* dies out quickly. Stump removal can be done in various ways. Caterpillars should be avoided since they cause too much site disturbance and compaction. Backhoes are much better. Various stump pulling devices have been tried, but so far they are not capable of handling the size of stump that must be removed efficiently. **"Push-over" logging** is another possibility. It has shown some promise in the interior of B.C. with *Armillaria*-infected trees. Push-over logging consists of pushing trees over with a backhoe so that the main roots are lifted out of the soil. The tree is then shaken to remove most of the soil, and bucked either on site or at the landing.

Not all areas can be treated in this fashion. Steep slopes prohibit the safe use of machinery. Erosion and compaction are also concerns. In addition, the exposure of mineral soil creates a good seedbed for alder, and special care must be taken to avoid invasion of treated sites by this species.

Care must be taken to remove all infected stumps. It is easy to miss small, low stumps buried under slash. Each infected stump that is missed can start a new infection center, and if as few as 10% of the

infected stumps are left in the soil, the resulting disease levels can equal that of the previous stand in one rotation, thus negating any benefits.

Planting should follow stump removal immediately. Some of the newly planted trees (those that are placed directly over small, *Phellinus*-infected roots) will die, but experience has shown that such mortality stops after a few years when the remaining small colonies of *Phellinus* die out. This occurs before there is any root contact between trees; hence root disease centers do not develop.

Stump removal is an expensive treatment, with costs ranging up to $1000 or more per hectare. If properly done, however, it restores to full productivity a site that was essentially non-productive. Because the gains can be very large, stump removal should be considered a silvicultural investment, and should be compared to other possible silvicultural treatments elsewhere in the management unit. A cost benefit analysis will often show that it is at least as attractive as other silvicultural operations, such as juvenile spacing.

Use of resistant species. A second way of dealing with *Phellinus* is through the use of resistant species. All hardwoods are immune. Thus a rotation of alder will allow *Phellinus* to die out completely, and after that one can return to susceptible species such as Douglas-fir. Birch or cottonwood could also be used, depending on site characteristics, and these will have the same effect on *Phellinus*. While *Phellinus* can survive for longer than the 40 or so years of a normal hardwood rotation in very large old-growth stumps, the fungus will no longer be present on the outer surfaces of such stumps after 20 or 30 years, and hence it will not be passed on to newly planted susceptible conifers. The market for hardwood logs is variable. Quality is an important consideration. In general, however, prices for hardwoods are rising faster than those of conifers, and hardwoods can no longer be regarded as "valueless weed species." Thus hardwoods must be seriously considered as an option.

Among the conifers, pines and western red cedar are resistant. *Phellinus* will not cause significant mortality in such species, although it may survive as a minor pathogen on roots. White pine would be an excellent choice, but it has its own serious disease problem, namely white pine blister rust. Until resistant planting stock becomes available, it is not a viable option except in a few areas where the hazard of blister rust is very low. On some drier sites shore pine (*Pinus contorta*) might be considered. However, cedar is usually the best choice. It commonly regenerates naturally in *Phellinus* centers, and grows well in that situation. Sometimes stands of Douglas-fir are replaced by cedar, and it is often not recognized that small patches of cedar in Douglas-fir stands actually represent old *Phellinus* centers that now support somewhat younger cedar. One can often find the old, *Phellinus*-killed Douglas-fir stumps in such areas now reduced to hollow tubes of bark. The decade of the '80s has seen a remarkable reassessment of cedar as a useful commercial species on the coast. It is now recognized that cedar is capable of very good volume growth. Thus the cedar option is now

much more attractive than it appeared ten years ago. Some would argue that it is almost always to be preferred to the violent intrusion of stump removal.

The delineation of treatment areas always presents a problem. Infection centers are easy to recognize in mature stands. After clearcutting, however, the only useful remaining symptoms are stain and decay on the stump surface, and the stains fade quickly so that unless stumps are marked at the time of falling, they do not serve to identify the disease for long. The best approach is to ignore single isolated centers, and to map out and apply treatments (stumping or alternate species) to those parts of the clearcut area on which visible infection centers occupied more than about 20% of the area. In theory it is possible to make a map of individual centers, to re-locate and mark them after logging, and to restrict treatment to these centers (and a border of about 15 m around them), but the process, if properly done, is too expensive and time consuming to be useful.

special considerations

Phellinus presents special problems in high use areas such as urban parks, campsites, and picnic areas. Managing agencies have a legal responsibility to create safe conditions. Many such areas are located in older stands in which infected trees have extensive butt rot but only minor crown symptoms. Windthrow is common in such situations and is probably aggravated by the heavy use and partial cutting necessary to develop such areas. Thus, it is best to avoid root disease centers when establishing campsites and picnic areas. When *Phellinus* is detected in such areas, a vigorous tree removal program is mandatory, even if it destroys the major amenity value of the area. Trenching around infection centers may be warranted in such places in order to break all root contact between infected and healthy trees, thus stopping the spread of the pathogen.

While *Phellinus* is undoubtedly a destructive disease, it is native to west coast coniferous forests, and has its own role in the ecology of such forests. In this context, the pathogen is probably best seen as a major agent of diversity. Infection centers develop into patches of hardwoods or cedar in what would otherwise be uniform stands of susceptible conifers. In older stands, the snags may also represent important habitat for cavity nesting birds. Ungulates may also benefit from the herb and shrub layers that develop in infection centers. Thus it may sometimes be appropriate to set infected areas apart for non-timber uses. When that is done, however, the allowable cut must also be reduced in proportion.

So far we have discussed how *Phellinus* root disease spreads. Now it is clear that if, at any time, the disease is either spreading or at least remaining wherever it is, then it should by now (i.e., since the retreat of the glaciers and the establishment of coniferous forests) have spread to cover at least all of the low elevation coastal forests. Clearly that is not the case. So the question arises, "Does it ever retreat, and if so, how?" The answer is that it does indeed retreat. If infected sites go through a period of non-host occupancy (hardwoods, shrubs, climax stands of

cedar and hemlock), the pathogen slowly dies out. Thus the disease can disappear from large areas by natural processes. Spread and retreat are balanced in natural forests over the long term, and the balancing point for low elevation coastal forests is probably about 10–15 percent of the total forest area in infection centers. Seen in this light, it is obvious that what we call "good silviculture" (viz., early harvest and immediate replanting with susceptible species) will lead to steady increases in the root disease over time to well above "natural" levels.

Armillaria ostoyae

Armillaria ostoyae (Romagnesi) Herink is one member of a large group of closely related species which, until recently, were all called *A. mellea.* The larger group occurs worldwide, both in temperate and tropical forests. Some members of the group are mostly saprophytic, some parasitize hardwoods. Several species occur in the Pacific northwest, but *A. ostoyae* is the only one that attacks healthy conifers. Much of the early literature on *Armillaria* is confusing because it was not recognized that there were several closely related species, and hence it must be interpreted with caution. The geographic range of *A. ostoyae* in B.C. is roughly that of Douglas-fir. In the north of its range it is largely restricted to warm, south-facing slopes. On the B.C. coast, *A. ostoyae* mortality is common only on young trees, while it is much more serious in the interior, killing trees of all ages. In the interior, *A. ostoyae* can form very large, diffuse disease centers, and at lower elevations it often occurs together with *P. weirii.* Larch is moderately resistant when old (over 40 years). All the other conifers are susceptible. Hardwoods are resistant but not immune (resistant means that the though the disease may invade the host to a minor degree, no damage ensues; immune that the disease does not occur at all on the host species in question.)

The fruiting body of *Armillaria* is a honey coloured mushroom that appears in clumps at the base of infected trees in late summer. However, spores do not play a significant role in the spread of the disease. Instead, infection of new trees occurs via root contacts and **rhizomorphs** (shoestring-like structures consisting of a black outer layer surrounding a core of normal somatic hyphae — the somatic hyphae are thus protected from the hostile soil environment). Rhizomorphs extend short distances (rarely more than 30 cm) from infected tree roots. When a rhizomorph contacts a living root, the fungus penetrates to the cambium and kills the bark, producing sheets of white mycelium in a fan-shaped arrangement in the dying phloem. It also invades and kills the xylem and begins to decay it. There is no external mycelium on the root surface, and infected sapwood remains pale in colour. Advanced decay is stringy, soft and bleached. Resinosis around the infected area is a common host reaction. Together, these symptoms (standard root disease crown symptoms, basal resinosis, white mycelial fans in the phloem, and rhizomorphs extending into the soil) serve to diagnose *Armillaria* root disease. On some tree species, however, basal resinosis is minimal or absent. Thus lodgepole pine seldom exhibits basal resinosis, while on spruce and hemlock it is much less pronounced than on Douglas-fir.

Host reactions play a much more important role in *Armillaria* root disease than in *Phellinus* root rot. Vigorous and well-established trees can resist invasion by *Armillaria* or stop its spread along roots. The initial host reaction consists of the formation of periderms (layers of cork - these are discussed in detail in the next lesson dealing with bark diseases) in the phloem. Copious resin production can also play a role. Once the advance of the pathogen has been arrested, the host produces calluses around the necrotic cambial area and CODIT-like structures in the xylem, thus forming strong barriers to further spread. (In the case of *Phellinus,* such reactions may also begin to occur, but because that pathogen can grow ectotrophically, it can circumvent the barriers and invade again higher up the root.)

Furthermore, the ability of Armillaria to invade also depends on the vigour of the parasite. The term used here is **inoculum potential.** Inoculum potential is best thought of as the energy available to the parasite at the point of penetration. This will depend on the size and quality (state of deterioration; mineral nutrient content) of the woody base in which the parasite is established, and on the distance from that base to the point of attack.

The outcome of an attack by *Armillaria* will depend on the balance between host vigour and inoculum potential. If inoculum potential is high, the parasite will be able to invade all but the most vigorous hosts. If inoculum potential is low, all but the most moribund host parts will be resistant. Note the spatial dimension. Both inoculum potential and host vigour describe the situation at the point of attack (or spread). Take the example of a 10-year-old plantation. If *Armillaria* is present, it will occur as colonies in stumps and roots of the previous rotation. Since these colonies are now mostly 10 years old, they will be slowly declining in vigour as the energy available from digestion of the wood is used up. However, right next to (i.e., within 10 cm of) these colonies (or at least the larger ones), inoculum potential would still be high. As one moves away from such colonies inoculum potential drop to zero at a distance of about half a meter. Host roots will of course extent through most of the soil by age 10. As noted above, some of these roots will be vigorous, and some moribund. Resistance depends on the ability of the host to marshal energy resources at the point of attack. This requires that those resources are available in the tree, and that they can be translocated quickly to the point of attack. In a 10-year-old plantation, therefore, there will be places where *Armillaria* can successfully attack roots of the new crop (low vigour roots close to inoculum colonies) and places where roots can resist attack (more vigorous roots at some distance from inoculum colonies). So one would expect some of the trees to harbour infections. As the pathogen advances along infected roots, however, the balance between inoculum potential and host vigour can change again. The distance between the main source of energy for the pathogen (the old inoculum colony (although the newly invaded root will also serve as a smaller energy and nutrient source) and the advancing front of the pathogen in the root will increase. Also, as the pathogen advances along

the root it may encounter more vigorous roots. Thus the disease may be stopped before it reaches the root collar. The result will be a static colony of *Armillaria* on the root. Mostly damage to the tree will be minimal. On the other hand, *Armillaria* may also be able to continue to advance until the tree is killed. It will then be established in a new colony in the stump and major roots of such a tree from which it could spread to other trees.

Static colonies of *Armillaria* do survive, and if tree vigour is greatly reduced they may begin to spread again. Such reductions in tree vigour may come about because of extreme drought or other adverse environmental factors, attack by other pathogens or insects, or when the tree is cut. Large stumps live for a couple of years, surviving on stored energy. During that time, *Armillaria* can escape from static colonies and invade the whole stump, thus creating a large source of inoculum. Once the stump tissues die, and other saprophytic organisms move in, further spread of *Armillaria* is halted. Thus cutting trees in infected areas will lead to a great increase in average inoculum potential. In partial cutting situations this may result in a flare-up of *Armillaria* starting perhaps five years later. Following clearcutting the same thing happens.

On the west coast, *Armillaria* is seldom serious. It will appear in plantations, causing scattered mortality for about a decade, but it seldom kills trees older than 20 years, unless the trees are exposed to stress. (*Armillaria* often plays a role in declines.) In the B.C. interior (particularly in the ICH) and the intermountain forests of U.S.A., the situation is very different. Here *Armillaria* is almost universally present. In most of the older, undisturbed stands, however, almost all the infections will be contained or static, and there will be few above-ground symptoms. In such stands *Armillaria* is said to be **quiescent**. Elsewhere, particularly where there has been some disturbance, *Armillaria* will be killing trees in rather diffuse root rot centers. Here we speak of **active** *Armillaria*. Harvesting in both types will lead to high inoculum potential and mortality in new plantations.

The big question that remains unanswered for the moment is what will happen in such plantations as they grow older. Will *Armillaria* remain active, or will it become quiescent? Early indications are that in vigorous plantations the disease will disappear, much as it does in coastal forests. The story here is that as plantations age, the inoculum potential represented by the old stumps declines, while the food base represented by young trees that are killed is small. At the same time the vigour of the trees (and particularly their ability to marshal large amounts of energy at the point of attack) increases, thus tipping the balance in favour of quiescence. However if the plantation species is not well matched to the local site, or if some other factor either reduces tree vigour or increases inoculum potential, the disease may remain active. Precommercial thinning will undoubtedly increase inoculum potential because thinning stumps are readily invaded by *Armillaria*. Whether that increase in inoculum potential is enough to cause a switch from quiescent to active *Armillaria* is not known at this time. The probable answer is that it will on some sites and not on others.

Because *Armillaria* may be present when there are no above-ground symptoms, root rot surveys for *Armillaria* are difficult. If *Armillaria* is detected, it is certainly there, but if it is not detected, it is often nevertheless present and likely to be a problem following harvesting. One can of course do a lot of digging to determine whether *Armillaria* is present but quiescent, but the amount of work involved is prohibitive for operational surveys. Instead it is possible to use regional averages rather than conducting surveys in individual stands. As researchers obtain more information, averages for subzones and site types will become available. Nevertheless, individual stands are likely to vary widely around such averages. So far this problem has not been solved, and no solution is on the horizon.

Treatment options are similar to those for *Phellinus*. Stump removal is an option, but harder to justify on the less productive sites. Much less is known about the relative susceptibility of various species. All conifers are susceptible, although to different degrees. Also, susceptibility is a function of site. In some paces, for instance, lodgepole pine is more resistant than Douglas-fir, while in others the reverse is true. Again, however, we do not have enough information to predict with confidence. Hardwoods (birch, aspen, cottonwood) are resistant (but not immune). There is also a great deal of speculation about the use of mixed stands, particularly ones including a hardwood component, but there is little hard data to support the various contentions. Serious study of *Armillaria* in the interior has only just begun, and much remains to be learned.

Heterobasidion annosum

Heterobasidion annosum (Fr.) Bref. (formerly known as *Fomes annosus*) is found around the northern hemisphere. In B.C. it occurs mainly on hemlock, true fir and spruce on the coast, although Douglas-fir is also susceptible, and in the moist warm parts of the Interior (ICH). It has not been recorded in the extensive dry, mid- to high-elevation lodgepole pine forests of the B.C. interior, although that species is quite susceptible in Europe. The pathogen is also a common and vigorous invader of trunk wounds, causing extensive decay in the wounded tree. Hardwoods are immune.

The pathogen exists as at least two sexually incompatible strains. One of these, the P or pine strain behaves much like *Phellinus weirii*. It can grow as ectotrophic mycelium on roots, particularly in mineral soils with a high pH and on abandoned agricultural soils. The other, the S or spruce strain causes extensive butt and trunk rot. It kills smaller roots and young trees, but seldom large trees. Thus the switch from the parasitic root-killing phase to the saprophytic root and butt rotting phase occurs much sooner in the S than in the P strain. The B.C. forms all behave like the S strain. In North America, the P strain occurs in the Great Lakes area, along the Atlantic coast and in south west U.S.A.

Identification is by the appearance of the advanced decay (decayed wood is pale in colour and has long (1 by 10 mm) cavities, some of which are filled with black mycelia. At a very advanced state the wood

becomes soft and spongy) and by typical shelving fruiting bodies produced at the base of infected trees. Also infected roots often have small, dense, white or yellow mycelial pustules on their surface. This pathogen also produces a characteristic asexual stage, consisting of a small stalks tipped by a bulbous, spore-bearing head. Infected pieces of wood wrapped in moist paper and stored at room temperature for a week will produce this stage abundantly on their surface, where it can be identified by a good hand lens.

Spores play a major role in the spread of this pathogen. Infection requires fresh wood surfaces such as scars, and stumps produced by thinning and clearcutting. Stumps remain susceptible to infection by *H. annosum* for only a few weeks. After that other fungi colonize the wood surface, and *H. annosum* can no longer enter. The P strain will spread from such stumps to surrounding trees, causing a great deal of mortality, especially in calcareous soils. In areas where the pathogen is prevalent, stumps must be protected. For pine stumps this is most effectively done by inoculation with a competitor, the decay fungus *Peniophora gigantea*. *P. gigantea* produces asexual spores that can be collected and stored in pill form, and then suspended in water with some food colouring and sprayed on stumps directly after the tree is cut. For other tree species (spruces, hemlocks, Douglas-fir and true firs) such a competitor is not yet available and protection is achieved by dusting the stump surface with borax.

The S strain is not nearly so pathogenic, and does not spread from stumps to living roots very quickly, nor does it advance quickly in living roots. There is a great deal of uncertainty about the necessity of protecting stumps created by thinning or clearcutting in areas where the S strain is dominant. In these areas, a substantial proportion of such stumps are infected. It is less clear, however, to what extent and how fast the pathogen spreads from such stumps to surrounding living trees. Some studies suggest that stump protection is warranted under these conditions; others show that transfer to living trees is uncommon, and that the rate of spread proximally on living roots is quite slow, so that damage to crop trees may be minimal. In the light of these different conclusions, local experience is the best guide for selection of treatment.

Inonotus tomentosus

Inonotus tomentosus (Fr.) S. Teng. (formerly known as *Polyporus tomentosus*) is a root disease of boreal and subalpine spruce and associated conifer species. It occurs throughout northern Canada and in mountain forests elsewhere in North America. The pathogen appears to be restricted to stands with a significant spruce component, but when spruce and pine are mixed, both species are equally attacked. Subalpine fir is quite resistant. Hardwoods are immune.

The pathogen spreads in a manner similar to *Phellinus* (ectotrophically on small roots but only in the inner xylem of larger roots). In older trees (over 50 years) it acts mainly as a root and butt rot without actually killing trees outright, but often resulting in windthrow. The fruiting body is a mushroom-like polypore produced above infected roots.

Spores may play a role in establishing new infections. Infection centers are usually small and clustered together in groups: regularly shaped large openings seldom develop. Young infected trees are chlorotic and show reduced height growth, but these symptoms develop much more slowly than with *Armillaria* or *Phellinus*. Crown symptoms in older trees are not well developed and difficult to spot unless light conditions etc. are near perfect. In the early stages of decay, the wood is red to reddish brown. Identification can most readily be made by looking for the characteristic large (2 mm by 20+ mm) pits in roots with advanced decay in windthrown trees. Such advanced decay remains brittle, even though most of the wood substance has been digested. However care must be taken not to confuse *I. tomentosus* with *Phellinus pini,* a common decay fungus of spruce that produces similar though smaller pits in decayed wood. Fruiting bodies are produced in the fall, but not necessarily every year. They have disappeared by the time the snow melts the following spring.

Infection arises from inoculum surviving from previous stands and via new spore infection. Evidence for the latter consists of the appearance of infection centers in spruce stands on old agricultural sites (where there was no old inoculum) and of variation in the DNA of isolates taken from small areas. It has been suggested that under unmanaged conditions catastrophic destruction of spruce forests is often followed by a period of hardwoods (aspen, cottonwood, willows, and birch) with spruce reappearing as a late successional species after 40–50 years. If that is truly the common natural cycle, *I. tomentosus* probably dies out during the period of non-host occupancy, so that new spruce stands start out free of disease and become infected via spores. The site of spore infection is uncertain, but is thought to be small new roots lying near the soil surface. Rapid regeneration by planting spruce immediately after harvest will result in continued spread of established centers from inoculum in stumps as well as new spore infection. The relative importance of these two processes is not known. There are few spruce plantations in B.C. old enough to determine the extent to which new infection centers arise from infected stumps, and in the few stands that are old enough, the status of tomentosus infection at the time they were harvested is not known. Also, the rate of spread in plantations is unknown, but probably somewhat slower than for *Phellinus* and *Armillaria*. There appears to be a strong relationship between site and the incidence of *tomentosus* root disease. Infection is common on warmer, moist to dry sites and almost absent on wetter sites. Inoculum in stumps can probably be removed by removing stumps.

Tomentosus root disease plays a significant role in northern spruce forests. It results in small stand openings (spatial diversity) and in scattered windthrow of old trees (e.g., in one study, at a rate of approximately one tree per hectare per year). The steady supply of such trees helps to maintain spruce bark beetle populations at an endemic level.

Damage by *tomentosus* root disease consists of tree mortality and increment loss in infected trees. In older mixed stands, lodgepole pine is killed standing, while spruce is often windthrown before it dies. Centers are small, and fill in with brush and hardwoods. If subalpine fir is present, it is released and may capture a good part of the site productivity. Inoculum reduction by stump removal is possible, but it is not clear whether the losses justify such action. Also, many of the productive spruce sites are very sensitive to site disturbance and compaction. Unless stump removal is done with great care, the damage resulting from disturbance may outweigh the benefit of disease reduction.

Before ending this discussion of *tomentosus* root disease, it needs to pointed out that serious study of this pathogen did not start in B.C. until recently. Many trials have now been established, and much will be learned in the next decade. You can expect our understanding to change considerably in the near future.

Phaeolus Schweinitzii

Phaeolus Schweinitzii (Fr.) Pat. (formerly known as *Polyporus Schweinitzii*) is a common butt rotting decay fungus. It is included in this group of root pathogens because it can kill small roots, and because the extensive butt rot it produces often results in breakage of trees close to the ground. Apparently, the main means of spread is by spores — mycelia isolated from different infected trees are almost always different from each other. Many coniferous species are susceptible to some degree but the fungus is most common on spruces and Douglas-fir. Hardwoods are immune. Initial infection is via spores, and is thought to be through scars, particularly fire scars, although small unwounded roots have also been implicated. After many years, when the pathogen has developed into severe butt rot, the original scar may no longer be evident. Spread from tree to tree is minimal. *P. Schweinitzii* produces a typical cubical brown rot. The large, stalked fruiting bodies are produced above major roots and at the base of the tree in late summer and fall, and deteriorate during the winter.

Root Diseases Caused by Non-Decay Fungi

All the root diseases discussed so far are caused by fungi that obtain most of their energy by decay of wood in roots and butts of trees. There are a few root diseases caused by Ascomycetes that do not belong in this group. A good example is Black Stain Root Disease caused by *Ophiostoma wageneri* (Goheen and Cobb) Harrington (the asexual stage is known as *Leptographium wageneri*). The pathogen is apparently introduced into trees by root bark beetles which carry live spores or mycelium on their bodies. Once inoculated into a tree it produces black stain in the outer sapwood rings. The fungus interferes with the transpiration stream (it belongs to the same group of fungi that cause wilt diseases in hardwoods). The exact manner in which this happens is unclear. Both blockage of the xylem and production of toxins may be involved. Spread from tree to tree is via root grafts and possibly close root contacts. The pathogen cannot decay wood efficiently, and unlike

the other root diseases, it does not survive in stumps or dead trees for more than a few years. Diagnosis requires the development of typical root disease crown symptoms, and narrow, longitudinal bands of black stain in the outer xylem rings of such symptomatic but still-living trees. Once the tree dies, several other non-pathogenic staining fungi invade the sapwood, and diagnosis becomes virtually impossible.

The incidence of this pathogen appears to be increasing rapidly. The first records in B.C. date from the early 1970s. Now it is quite common on the lower coast, and there are isolated collections throughout the southern interior. Since the insect vector is attracted by weakened or damaged trees, black stain is often found along recently built roads and edges of clearcuts. The apparent recent build-up of *O. wageneri* may be a case in which the root bark beetles are changing their fungal symbiont from a saprophytic blue stain to one that kills trees readily. Such a change would presumably be advantageous to the beetle because it eliminates a strong host reaction.