

Topics

Introduction	1
Taxonomy	2
Incidence of fir-type	2
Species susceptibility	3
Life History	3
Spore production	5
Ecology: infection	6
Ecology: stumps	7
Ecology: secondary spread	8
Biological Control	9
Management	10
Considerations for Cedar	11
Other Reading	12
Field Guide	
Management Guide Index	

Key Points

- **One of two important species of *Heterobasidion* in North America**
- **Serious disease of true firs, Douglas-fir and cedar**
- **Spore-initiated infections directly or through wounds on roots**
- **Usually found with *Armillaria* or *Phellinus weirii***

Management Guide for**Annosus Root Disease***Heterobasidion* sp. (fir-annosum)**Hosts:**

- True fir
- Douglas-fir
- Engelmann spruce
- western redcedar
- western hemlock

In northern Rocky Mountain forests, mortality is common in Douglas-fir, grand fir and subalpine fir. Butt rot commonly develops with infection in spruce, true firs, western redcedar and western hemlock.

In the central Rockies, butt rot is common in true firs and spruce but mortality is rare

Introduction

Annosus root disease is the most common and damaging fungus disease of conifer forests in the northern hemisphere. In northern Idaho and western Montana, Annosus root disease is economically important in true firs and Douglas-fir. Further south in Idaho, Wyoming, Utah, and Nevada, it is frequently found decaying roots and butts of mature trees, and sometimes killing small trees, but the overall impact is thought to be minor.

Annosus root disease is caused by several species which, until recently, have been grouped under the names *Fomes annosus* or *Heterobasidion annosum*. They are differentiated most readily based on host preference. In the

northern and central Rocky Mountains, Douglas-fir, grand fir, and subalpine are commonly killed by the fir-disease type. Other tree species, particularly spruces, western redcedar and western hemlock develop extensive butt rot and occasionally die from infections of fir-annosus. Ponderosa pine is the only significant host for pine-annosus in this area.

Trees of all ages can die from this disease and volume losses from butt rot are substantial in some species. Establishment of new infections through root or basal stem wounds appears to be the most common means of spread of fir-annosus.

**OVERVIEW OF
FIR-ANNOSUS ROOT DISEASE MANAGEMENT**

1. **Favor resistant species.** Especially pines and western larch.
2. **Thin early and avoid partial harvests.** Precommercially thin.
3. **Avoid basal stem and root damage.** Use care when logging.
4. **Use caution when thinning western redcedar.** Cedar decline may become a problem if cedar residuals are to be managed.

Taxonomy: Awaiting a name

About Types of Annosus

Similar but Genetically-Distinct Types of Annosus

Heterobasidion annosum in western North America consists of two inter-sterile (non-interbreeding) groups. These two types, 'fir' and 'pine', have very different host specificities.

The hosts for the fir type include true firs, Douglas-fir, Engelmann spruce, western red cedar, and western hemlock. (See Table 1 for details.)

Ponderosa pine is the main host for the pine type.

The basidiomycete fungus *Heterobasidion annosum* (Fr.) Bref. has been known for many years to consist of at least two distinct types, based on host preferences. Molecular and genetic studies of European isolates have demonstrated that the former *H. annosum* consists of at least three distinct species. The pine pathogen retains the name *H. annosum*. The species formerly referred to as "s" type in Europe was recently named *Heterobasidion parviporum* Niemela & Korhonen. This species is

found primarily on *Picea abies* (Norway spruce). *Heterobasidion abietinum* Niemela & Korhonen, is the new name suggested for the pathogen of European true firs (*Abies*).

The fir pathogen of this complex in western North America has yet to be named, and is currently referred to as the "North American S group" (Korhonen and others 1998). For this publication, it will be referred to as 'fir-annosus'.

Incidence in northern Rocky Mountains

Grand fir and Douglas-fir

In northern Idaho and western Montana, fir-annosus is broadly distributed and very common in forest types that include grand fir and Douglas-fir. *Armillaria ostoyae* root disease generally co-occurs with fir-annosus, making it impossible to distinguish the individual impacts of the two diseases.

Stands without prior harvests as well as harvested stands with sufficient host composition, have abundant symptoms and signs of annosus root disease. First-entry grand fir stumps, in particular, produce abundant annosus conks, indicating pre-existing root infections in a high proportion of trees. Northern Idaho, from St. Maries to the Salmon River, appears to have an especially heavy incidence of fir-annosus. Most the annosus-related Douglas-fir mortality is reported from this geographic area as well.

Bark beetles, especially fir engraver in grand fir, frequently attack trees with fir-annosus root disease. Mortality rates of root

disease-afflicted trees are probably significantly increased by bark beetle attacks.

Subalpine fir

Fir-annosus is implicated in a widespread decline of subalpine fir in the northern Rocky Mountains. Mature trees are especially damaged, with rapid death following long periods of growth decline. Western balsam bark beetle (*Dryocoetes confusus*), fir engraver beetles, and *Armillaria ostoyae* also are involved in the decline.

Other tree species

Extensive root decay and very gradual death of western redcedar is common on drier and partially-harvested cedar sites (Hagle, unpublished data). Fir-annosus, *Armillaria ostoyae*, and *Phellinus weirii* appear to be about equally responsible for the damage. Butt rot in western redcedar, Engelmann spruce and western hemlock are also very common, particularly in mature trees.

Central Rocky Mountains

1. Annosus root and butt decay are common in older trees, with little apparent mortality.
2. Conks are common in decayed stumps and dead wood.
3. Small trees are occasionally killed.
4. Good data are lacking on the extent or impact of the disease.

Table 1. Conifer species susceptibility to fir-annosum root disease in the northern and central Rocky Mountains.

Location	Least Susceptible	Susceptible but tolerant	Highly Susceptible
Northern Rockies (Montana and Idaho north of the Salmon River)	Larch, ponderosa, lodgepole, and whitebark pines	Spruces, hemlocks, western redcedar, western white pine; butt rot and extensive root decay may develop but mortality rates are low.	True firs, Douglas-fir; butt rot is common in grand fir and mortality is common in trees of all sizes.
Central Rockies (southern Idaho, Utah, Nevada, western Wyoming)	Pines	True firs, Engelmann spruce; Butt rot and root decay, some small pockets of mortality.	Unknown Extent and severity of annosum remains unknown in many areas.

Life History

Basidiospore-initiated infections

Trees can become infected in numerous ways but it appears the most common occurrence is spore-initiated infections in live trees (Figure 1). Airborne basidiospores land on, or are rain-washed to, a freshly wounded root or lower stem. Here the spores germinate and hyphae penetrate the wood to establish either latent or active infections. Latent infections may remain viable for decades before resuming growth (Garbellotto and others 1999) while active infections begin expanding immediately. The infection expands preferentially toward the root collar. The pathogen grows nearly unhindered in root and butt heartwood of true firs. In true firs and western redcedar, particularly, annosus often produces large cavities in roots and lower stems of trees showing little or no outward symptoms of disease.

Minimal secondary spread

There is relatively little spread

of the disease from tree to tree via root contacts compared to other important root diseases such as *Armillaria* root disease and laminated root rot. Most individuals, or genets, are confined to a single tree. Occasionally groups of several trees are infected by the same genet but this appears to be uncommon.

Despite minimal tree-to-tree spread, very high rates of infection are typical of true fir, western hemlock and western redcedar stands in the interior West. The abundance of spores capable of taking advantage of wounds and high frequency of root and stem wounding in these species apparently provide ample opportunity for successful establishment of new infections.

Abundant spore production in dead trees and stumps

When infected trees die, the fungus may continue to live for many decades in the moist, underground portions of the tree.

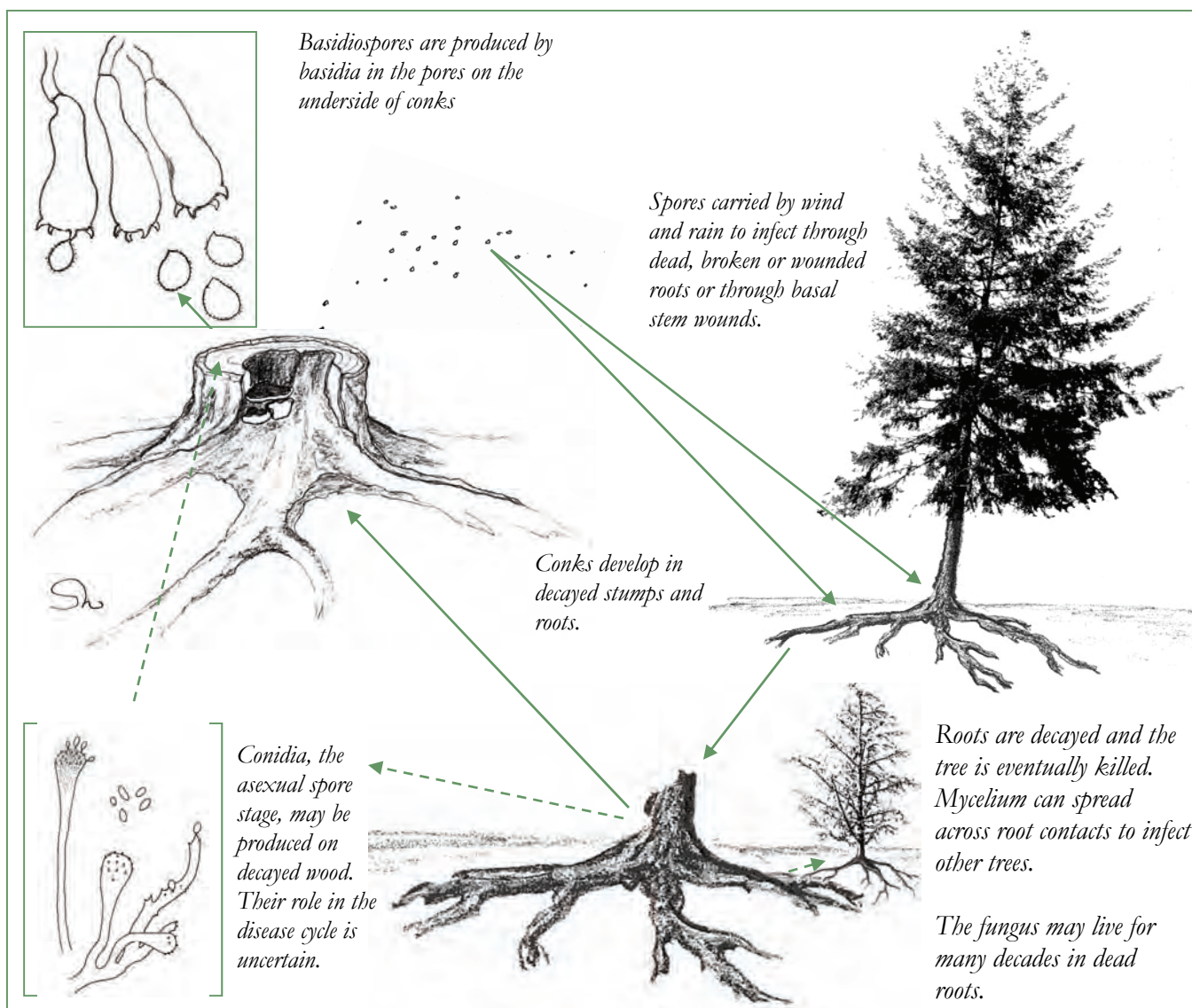
Genet

A genet is an individual derived from compatible spore pairing; a diploid mycelium.

Spore-initiated infections through tree roots or basal stem wounds may account for most of this disease.

Mortality due to *Armillaria* root disease often obscures the effects of fir-annosus when the two occur together in stands.

Figure 1. Life History of fir-annosus



Sporophore production on roots and in hollow stumps continues for many years after the host has died.

Although mycelia in stumps and dead trees infrequently spread to nearby living trees, they produce conks that are important sources of spores that spread the disease.

Sporophore production continues for many years after the tree has died and airborne basidiospores can nearly always be found in abundance in coniferous forests. These spores are able to infect freshly cut stump surfaces as well as basal and root wounds in live trees.

Asexual spores

Fir-annosus conidiospores are commonly seen in culture and on incubated specimens of infected wood but are not often found in nature. They do not appear to produce a mycelium that is vigorous enough to infect live tissue nor to compete with other fungi for dead wood substrates. Garbelloto and others (1999) suggested that they may play a sperm-like role in producing the heterokaryotic mycelia typically found in stumps. For now, their function remains a mystery.

Spore production by fir-annosum

Annosus sexual sporophores, commonly called “conks”, are often found inside decayed, hollowed stumps (Figure 2). Less often, they are found on the outside of roots of dead trees, just below the duff. Small, incompletely formed, sporophores called “button conks” occasionally appear on roots of small trees killed by annosus root disease.

Annosus conks usually are perennial, sometimes annual. They may be effused-reflexed (shelf-like) or resupinate (crust-like). The older layers are tough and woody but the newest pore layer is softer and white to cream-colored. The pore layer (hymenium) wraps over the outer edge of an effused-reflex conk producing a “sterile margin” of cream-colored tissue. This contrasts with the dark brown or gray upper surface of the conk (Figure 4) making them easy to spot in dark stump hollows. The pores of the hymenium are small, typically 0.3-0.6 mm diameter.



Figure 2 *Annosum* sporophores (conks) often are produced in abundance in decayed stumps. This grand fir stump had 14 conks lining the perimeter of the hollow, including those in Figure 4 at right. [Photo by S. Hagle]

They are mostly round or oval but can be elongate near the margins. Basidia line the interior of the pores producing basidiospores on short sterigmata (Figure 3). The spores are forcibly ejected from

the basidia, effectively launching them into the air. In conifer forests, basidiospores of annosus may be found in the air whenever the temperature is above freezing and the humidity is not too low. Basidiospores are washed into soil and along roots by rain and may remain viable for a year. Annosus is incapable of living freely in soil or duff so the spores must germinate on a suitable substrate. Clearly, few spores survive.

The basidiospores are typically dikaryotic, that is, they bear two nuclei, and these nuclei are haploid. Upon germination, basidiospores produce a homokaryotic mycelium which must mate before producing the sexual sporophores. Mating is a simple matter among fungi. Compatible mycelia that meet, can fuse and exchange nuclei. There is another reason for mating of two homokaryotic mycelia; heterokaryotic mycelia are more vigorous than homokaryons (Korkonen and Piri 1994), making them more effective pathogens.

Annosus also produces an asexual spore stage in the form of conidia. They are borne on top of club-shaped conidiophores (Figure 5). Both homokaryotic and heterokaryotic mycelia produce conidia so these spores can be either. The conidia-producing stage bears its own name, *Spiniger meinekellus* (A.J. Olson) Stalpers.

This spore stage is readily produced in culture and on incubated specimens of infected wood. However, it is not generally observed outside of the laboratory. The role of these spores in the disease cycle is not known. They do not appear to be capable of producing infections in living hosts.

Figure 3. Sexual spores of annosus

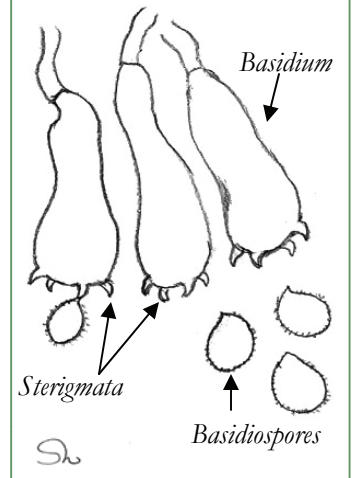
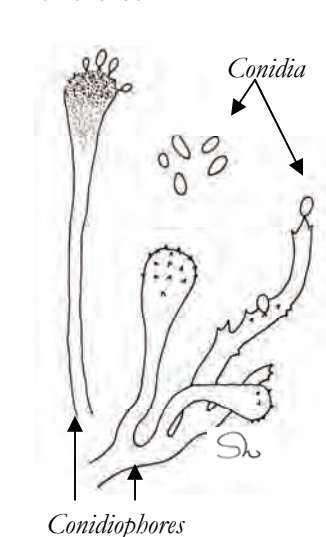


Figure 4. Effused-reflexed annosus conks growing inside a stump. [Photo by S. Hagle]

Figure 5. Asexual spores of annosus; *Spiniger meinekellus*



Ecology of fir-annosus root rot

High rates of fir-annosus infection are typical of mature true fir, western hemlock, and western redcedar stands in the interior West.

Fir-annosus infection in the northern Rockies:

- High rates of infection are typical of mature grand fir, subalpine fir, and western redcedar.
- Despite high infection rates, mortality rates usually are low except where *Armillaria ostoyae* is present as well.
- Butt rot is very common in grand fir and western redcedar.
- Most infections in live trees probably originate from basidiospores but the mechanism of infection is poorly understood.
- Root-to-root spread between trees is minimal.

Annosus root disease is the most common and damaging fungus disease of conifer forests in the northern hemisphere. It has been studied extensively in Europe, especially Sweden, Finland, Great Britain, and France, as well as in North America. Only recently, technology has provided the means to separate the species that have been known under the name *Heterobasidion annosum*. Therefore, most of the literature refers to the species complex as *H. annosum*. The following summary applies to the ecology of fir-annosus.

Modes of spread

Annosus root disease can spread in several ways. "Primary spread" is due to airborne spores produced by a fruiting body or "conk" (Hodges 1969, Hsiang et al. 1989). When spores land on a newly cut stump or fresh basal and root wounds they may germinate and colonize the wood, if conditions are favorable. "Secondary spread" results from growth of the mycelium along host roots. From a diseased tree annosus can spread to a neighboring tree by ectomycelium growing across root contacts, or internally by endomycelium expanding through root grafts.

Fine roots can be infected by mycelium from woody inoculum or by germinating spores. Dead roots are less likely to be avenues of infection because annosus is a poor competitor with saprophytic fungi that inhabit dead roots (Stenlid and Redfern 1998). Annosus is not known to have

insect vectors (Hunt and Cobb 1982)

Primary spread

Fir-annosus, at least in western North America, appears to rely more on spore-initiated infection directly in live hosts, than on stump-to-tree or root-to-root spread (Otrosina and Cobb 1989, Garbelloto and others 1993). Several studies have reported fir-annosus genets to be numerous and of limited size Lockman 1993, Garbelloto and others 1997, 1999. Garbelloto and others (1999) studied genet distribution in white fir stands in California. They found that genets of fir-annosus tended to be small, often infecting a single tree or even a single root. Multiple homokaryotic infections are commonly seen in live trees, indicating there are abundant opportunities for basidiospore infections to establish.

Wounds on the basal stem and on roots appear to be likely entry points for basidiospore infections, but there is considerable uncertainty regarding the modes of establishment of fir-annosus in live trees and other substrates. Small root wounds from insect and rodent feeding, and superficial wounds resulting from surface fires and rock abrasion may provide entry courts. Garbelloto and others (1999) postulated that a period of latent infection may occur based on isolations of pathogen from asymptomatic sapwood and roots. Wounding or a period of drought stress may trigger growth of the fungus from latent infections.

Origins of stump infections

Filip and others (1992) found that infection of fresh stump surfaces probably plays an insignificant role in initiating infections in stands in eastern Oregon. High rates (89%) of stump infection in true fir stumps were measured following clearcut harvesting but most were thought to have originated from mycelia already present in the trees before harvest. This observation is consistent with that of Garbelloto and others (1999) who also concluded that most trees already had root infections before tree harvest activity occurred. Additionally, neither harvest season nor stump size appear to influence frequency of stump infection. (Filip and others 1992).

Lockman (1993), studying fir-annosus in northern Idaho, found a possible increase in diversity of annosus from clearcut compared to uncut stands, using vegetative compatibility testing. Basidiospore infection of stumps could account for such an increase. She also found that at least 70% of the genets were limited to a single tree or stump, leading her to conclude that most infections are spore-initiated and had not spread to other trees or stumps.

Mycelium spread from stumps is uncommon

Despite high rates of infection in fir stumps (89%), Filip and others (2000) reported finding very low rates of infection of trees regenerated near stumps. Less than 0.5% of young fir trees growing near stumps died of annosus infection in the ensuing 15-19 years. If primary spread had occurred, it had caused little mortality.

Lockman (1993) found only 19% *H. annosum* infection in stumps of clearcut-harvested stands in northern Idaho. The stumps were mostly Douglas-fir and grand fir that had been harvested several decades earlier. Subsequent, naturally regenerated, 10-30 year-old Douglas-fir and grand fir trees had a 1.7% rate of infection. This was lower than the 2.6% infection in paired stands on never-harvested sites. Douglas-fir and grand fir were about equally infected. Like Filip and others (2000) Lockman commonly found both fir-annosus and *Armillaria ostoyae* in symptomatic roots.

Similarly, Slaughter and others (1991), found no significant mortality in trees surrounding infected fir stumps in California up to 6 years after harvest. This lack of spread of annosus from stumps may be partly due to loss of viable mycelia in stumps within a short time. Morrison and Johnson (1978) reported a steep decline in stump infection rates of coastal Douglas-fir and western hemlock. Initially 82% of Douglas-fir stumps and 62% of western hemlock stumps were infected, but within five years those rates dropped to 5% and 7.5%, respectively.

Small genets in live trees have consistently been found to be unrelated to stump infections (Filip and others 1992, Lockman 1993, Sullivan and others 2001, Garbelloto and others 1999). In the latter study, infected trees generally were not near stumps and isolates from stumps were heterokaryotic, while infections in live trees were homokaryotic. It is, therefore, unlikely that the live-tree infections originated from stumps.

Role of stumps:

- **Most fir-annosus in stumps was present in the roots of the tree prior to cutting.**
- **Neither harvest season nor stump size appear to influence frequency of stump infection**
- **This fungus rarely transfers from infected stumps to kill young trees.**
- **Stumps and dead trees are sites for mycelium mating and sporophore production.**

***H. annosum* is a poor competitor with saprophytic fungi that inhabit dead wood.**

Airborne basidiospores are abundant most times of the year, and individual spores may survive a year or more in bark crevices.

These factors allow annosus basidiospores to take advantage of most of the wounds that occur throughout the life of a tree.

Fir-Annosus Rate of Spread

Longitudinally in roots:

**Average —
23-58cm
(9-23 in.)
per year**

True fir in California
(Garbelloto and others
1997)

Site Factor

Acidic soils inhibit ectotrophic mycelium development— growth is slower without ectotrophic growth

The fact that live-tree infections were homokaryotic probably indicates that they were basidiospore-originated. Lockman (1993) reported finding the same genet in a tree and nearby stump only once in 53 isolates.

Mycelium mating and sporulation in stumps and dead trees

The significance of stumps and dead trees appears to be that they provide a substrate for mating of homokaryotic mycelia. The homokaryons originate either from basidiospore infection of the live tree (before it was cut or otherwise killed) or spore infection of stump surfaces. The resulting heterokaryons produce sporophores (conks) in the stumps or dead trees. This process plays a significant role in increasing spore-loading on a site. In order for the fungus to take advantage of wounds as they occur in live trees, an abundance of spores must be present continuously. Therefore, even without direct spread of mycelium, stumps and dead trees probably play an important role in increase of the disease in stands.

Basidiospores are wind transported and capable of establishing mycelium in live and dead host tissues. Stump surfaces are only infected when they are fresh (Morrison and Redfern 1994, Morrison and others 1986), before competing saprophytic fungi have become established. *Heterobasidion annosum* is a poor competitor.

Secondary spread

There is no evidence that *H. annosum* can achieve the tremendous sizes and ages that result from secondary spread in

Armillaria ostoyae genets. The largest published *H. annosum* genet was about 50 m. (Korhonen and Stenlid 1998) Assuming an average radial growth of the fungus to be 20cm per year, the age would be not much more than 100 years.

Tree-to-tree transmission

Secondary spread between live trees also appears to be minimal. Lockman (1993) found genets to be mostly confined to single trees. At least 70% of genets in stands included in her northern Idaho study were limited to a single tree or stump. She concluded that most infections are spore-initiated. However, in at least one case, the genet had clearly spread to several adjacent trees producing a mortality cluster in a stand otherwise apparently unaffected by the disease. Four mature grand fir and Douglas-fir trees were symptomatic or dead and a single genet of *H. annosum* was isolated from them. These trees were growing close together and thus tree-to-tree transmission was suspected. In both cases the trees bearing isolates of the same genet were less than 7 m (23 ft) apart.

Garbelloto and others (1999) also found that nearly all genets (86%) occupied only one tree in white fir stands in California. Multiple-tree genets colonized up to 11 trees, and extended up to 10 m diameter. Despite the low frequency of secondary spread, the few genets that had spread to multiple trees accounted for 33% of the infected basal area. All inter-tree spread was via root contacts or grafts by endotrophic mycelium. (Ectotrophic mycelium was not observed.)

Although wounding appears necessary for infection by spores, penetration of intact bark by mycelium is common in the case of spread from root contacts.

Mortality is clustered in stands

Garbelloto and others (1999) noted that, although the trees were not infected by the same genet, mortality tended to be clustered in stands. Clusters of mortality typically had several genotypes of fir-annosus. Similar clustering of mortality is often observed in the northern and central Rockies. Several factors may be involved in producing this mortality pattern. Study of fir-annosus is often confounded by the close-association of *Armillaria* root disease and laminated root rot.

These diseases occur on the same sites with fir-annosus and, commonly, in the same tree. The relative ease of diagnosis of *Armillaria* root disease also probably results in under-diagnosis of annosus root disease.

Armillaria ostoyae root disease, in particular, could be the cause of clustered mortality without regard to annosus genet distribution. Additionally, fir engraver and the western balsam bark beetle are known to frequent trees with annosus root disease (Hertert and others 1975, Lane 1976, Ferrell and Parmeter 1989). These beetles commonly cause clustered mortality as bark beetle pheromones aggregate attacking beetles.

Biological Control

Many naturally-occurring saprophytic fungi invade conifer stumps shortly after tree harvest or thinning. These organisms have the potential to limit the development of root disease fungi. Among the most common fungi seen fruiting on stumps in the northern rockies are *Fomitopsis pinicola* (Swartz:Fr.)Karst., *Trichaptum abietinum* (Dicks.:Fr.) Ryv., and *Gloeophyllum sepiarium* (Fr.) Karst. and *Antrodia heteromorpha* (Fr.) Donk. In addition, several are routinely isolated from dead roots of trees, in particular *Perenniporia subacida* (Pk.) Donk, *F. pinicola*, *Resinicium bicolor* (Alb.& Schw. ex Fr.) Parm. and *T. abietinum*. These fungi are probably natural controls for this and other root diseases by competing for woody substrates.

Another fungus that is occasionally seen fruiting in the northern Rockies, *Phlebiopsis gigantea* (Fr.) Jül, has been developed as a biological control for European species of *Heterobasidion*. Spores of this decay fungus are commercially available as Rotstop ® for inoculation of fresh stump surfaces (Nicolotti and Gonthier 2005). The current state of our knowledge of fir-annosus suggests that this or other direct methods to prevent stump infections are not warranted because 1) most stump infections arise from pre-existing root lesions rather than stump surfaces and 2) fungus does not appear to spread aggressively from stumps. However, the spore-producing role of stump-inhabiting mycelia could be important in maintaining the disease.

Clustered mortality is characteristic of fir-annosus infected stands even though studies indicate that most trees are independently infected by basidiospores rather than by mycelium spreading from adjacent trees or stumps.



Figure 6. Saprophytic fungi such as *Antrodia heteromorpha* (shown on a Douglas-fir stump above and below) quickly establish on stumps and dead trees and effectively exclude root pathogens from the resource. They probably play an important role in limiting the buildup of root diseases on harvested sites. (Photos by S. Hagle)



Management of fir-type annosus

MANAGING FIR-ANNOSUS

Resistant species

Conversion to disease resistant species is usually the most practical method to control damage.

Thin early

Density may promote mycelial spread tree-to-tree and may increase effects of drought stress including more rapid extension of annosus root lesions and attack by bark beetles.

Avoid partial harvests

Partial commercial harvests that leave a large composition of susceptible species may produce the undesirable outcome of losing most of the remaining trees within a few years.

Avoid wounding

Wounds may establish new infections and stimulate growth of existing lesions.

Use particular care with cedar

Western redcedar often goes into a long-term decline after thinning, due to rot diseases.

Silvicultural Management

Altering species composition to increase the proportion of root disease resistant species, avoiding tree wounding during thinning, and reducing tree density at an early age to minimize drought effects are recommended measures to manage fir-annosus impacts. Also an important consideration for management is the fact that other root pathogens and bark beetles usually co-occur with fir-annosus.

Alter stand composition—manage for resistant species

Pines and western larch are not significantly damaged by fir-annosus. They provide the most practical means to minimize root disease impacts. True firs and Douglas-fir are the species most likely to be killed by fir-annosus, while growth loss and slow decline is typical of annosus-infected western redcedar. Regeneration harvest, planting, and stand-tending to maintain pines and larch are efficient means of managing fir-annosus and other fir root diseases.

Thin early

Density may affect tree-to-tree spread of fir-annosus root disease, perhaps more than the other root pathogens. Early thinning may also help alleviate some of the effects of drought. If, in fact, latent infections do establish in fir, and if these infections are stimulated by factors such as drought, preventing over-crowding may forestall the lethal effects of annosus. Thinning is also thought to reduce the impacts of fir engraver beetles,

possibly also by reducing the impacts of drought. Care should be taken to avoid over-thinning western redcedar which also seems to stress the trees and possibly cause increased infection by annosus and expansion of existing infections.

Avoid partial harvests that leave susceptible residuals

This is recommended for all of the major fir root diseases. Although this method may not increase mortality rates, it is unlikely to decrease them and often renders the residual stand less manageable because the continuing mortality in the residual susceptible trees leaves little commercial value to finance site preparation and artificial regeneration. Natural regeneration is generally heavy to susceptible species and therefore unlikely to improve the overall condition.

Avoid wounding trees

Wounds may also be significant in fir-annosus development in susceptible stands. In addition to mechanical wounds, it is likely that infection also occurs directly through root bark or through minor wounds caused by insect and rodent feeding. In any case, prevention of wounding is always a good practice management practice.

About those stumps...

Harvest of grand fir stands, in particular, often reveals high rates of pre-existing butt rot from annosus. Even at the time of the first harvest entry the rates of butt rot often are very high, indicating

that a history of harvest is not necessary to incite high infection rates. However, studies in eastern Oregon indicated that incidence of annosus root disease in true firs increased with each stand entry. Wounding associated with logging may account for most of this

increase. Evidence appears strong that stump-infesting mycelia do not commonly expand to infect live trees. At this time, it appears efforts to directly control infection in fir stumps are unlikely to produce much improvement.

Special considerations for cedar

Western redcedar can be considerably damaged by fir-annosus although it is considered a moderately resistant species. In fact, on some sites cedar has a high rate of root decay and butt rot caused by fir-annosus, especially in residual cedar after a partial harvest. Several other root pathogens are involved and although mortality rates are low, growth decline and loss of crown vigor is often dramatic (Hagle, unpublished data).

Koenigs (1969) reported severe root infection of western redcedar by 20 years after thinning in Priest River Experimental Forest in northern Idaho. *Armillaria* (probably *ostoyae*), *Corticium galactinum*, *Phellinus weirii* and *H. annosum* (probably fir-annosus) were found in decayed roots. Roughly 90% of the basal area had been removed from 80-yr old stands leaving only western redcedar. The released cedar were young, understory trees; 20 years after thinning they averaged 4.4 to 5.5. inches dbh. Both thinned and unthinned stands had high rates of root infection (94% and 67%, respectively) but the rate of basal cankering was much higher in thinned stands (75% compared to 27%). These cedar also exhibited chlorosis, basal resinosis and growth decline 20

years after thinning (Koenigs 1969).

Recent excavations on the Clearwater National Forest in north Idaho revealed that at least 80% of the lateral roots of western redcedar (trees at least 5 inches dbh) were infected by fir-annosus, *Armillaria ostoyae* or *Phellinus weirii* whether stands had been disturbed by harvest or not. However, extent of root and basal stem girdle was significantly greater on sites with evidence of harvest activity several years prior (average 63% compared to 21%). Also, the cause of root decay and basal stem girdle was more often *Armillaria ostoyae* or fir-annosus on harvested sites, and more often *Phellinus weirii* on unharvested sites.

The amount of root girdle near the root collar correlates with decline symptoms (Figure 7) in the crowns of mature cedar. Although mortality rates are low, decline rates indicate substantial cumulative effect of these infections.

Although research on this continues, it is clear that annosus and other root diseases cause significant damage in cedar stands and that severe thinning that leaves residual western redcedar promotes development of long-term, root-disease caused, decline.



Figure 7. Crown thinning, chlorosis and poor growth are typical symptoms of cedar decline.

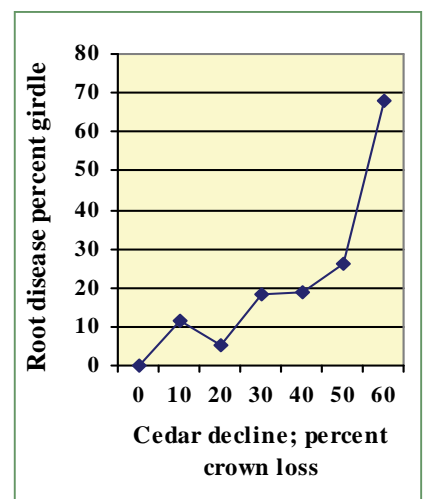


Figure 8. Relationship between root collar girdling by basal cankers caused by root disease and visible crown loss in western redcedar at least 5 inches dbh.

Other Reading

- Ferrell, G. T. and J. R. Parmeter, Jr. 1989. Interactions of root disease and bark beetles. *In* Proceedings on the Symposium on Research and Management of Annosus Root Disease (*H. annosum*) in western North America. April, 1989. Monterey, California. USDA For.Serv., Gen. Tech. Rep. PSW-116. Pp. 105-108
- Filip, G. M., C. L. Schmitt, K. P. Hosman 1992. Effect of harvesting season and stump size on the incidence of annosus root disease of true fir. WJAF 7(2): 54-56.
- Filip, G. M., C. L. Schmitt, C. G. Parks 2000. Mortality of mixed-conifer regeneration surrounding stumps infected by *Heterobasidion annosum* 15-19 years after harvesting in northeastern Oregon. WJAF 15(4): 189-194.
- Garboloto, M., F. Cobb, T. Bruns, W. Otrrosina, G. Slaughter, and T. Popenuck. 1993. Preliminary results on the genetic structure of *Heterobasidion annosum* in white fir (*Abies concolor*) root decay centers. *In* Proceedings of the 8th IUFRO Conference on Root and Butt Rots, Sweden/Finland. August 1993 Swedish University of Agricultural Studies, Uppsala, Sweden, Pp. 227-232.
- Garboloto, M., G. Slaughter, T. Popenuck, F. W. Cobb, T.D. Bruns 1997. Secondary spread of *Heterobasidion annosum* in white fir root disease centers. Can. J. For. Res. 27: 766– 773.
- Garboloto, M., F. W. Cobb, T. D. Bruns, W. J. Otrrosina, T. Popenuck, and G. Slaughter. 1999. Genetic structure of *Heterobasidion annosum* in white fir mortality centers in California. Phytopathology 89: 546-554.
- Hadfield, J. S., D. J. Goheen, G. M. Filip, C. L. Schmitt and R. D. Harvey. 1986. Root diseases in Oregon and Washington conifers. USDA Forest Service, Pacific Northwest Region, Portland, Oregon. 27 p.
- Hertert, H. D., D. L. Miller and A. D. Partridge. 1975. Interaction of bark beetles (Coleoptera: Scolytidae) and root rot pathogens in grand fir in northern Idaho. Can. Entomol. 107: 899-904.
- Hodges, C.S. 1969. Modes of infection and spread of *Fomes annosus*. Annual Review of Phytopathology 7: 247-265.
- Hsiang, T., R.L. Edmonds and C.H. Driver. 1989. Detecting conidia of *Heterobasidion annosum* in western hemlock forests of western Washington. *In* Proceedings of the Seventh International Conference on Root and Butt Rots. August, 1988. Vernon and Victoria, British Columbia, Canada. Pp. 417-426
- Hunt, R. S. and F. W. Cobb, Jr. 1982. Potential arthropod vectors and competing fungi of *Fomes annosus* in pine stumps. Can. J. Plant Path. 4: 247-253.
- Kliejunas, J.T. 1986. Frequency of *Fomes annosus* spread from true fir stumps to adjacent planted pines. USDA Forest Service, Pacific Southwest Region. Report No. 86-4. 4 p.
- Koenigs, J. W. 1969. Root rot and chlorosis of released and thinned western redcedar. J. For. 67: 312-315.
- Korhonen, K. and T. Piri. 1994. The main hosts and distribution of the S and P groups of *Heterobasidion annosum* in Finland. *In* Proceedings of the 8th IUFRO Conference on Root and Butt Rots, Sweden/Finland. August 1993 Swedish University of Agricultural Studies, Uppsala, Sweden. Pp. 260-267.
- Korhonen, K., P. Capretti, R. Karjalainen and J. Stenlid. 1998. Distribution of *Heterobasidion annosum* Intersterility groups in Europe. Pp 93-104.
- Korhonen, K. and J. Stenlid. 1998. Biology of *Heterobasidion annosum*. Pp. 43-70. *In* *Heterobasidion annosum*; biology, Ecology, Impact and Control. Ed. by S. Woodward and others. CAB International, New York, NY. 589 p.

- Lane, B. B. and D. J. Goheen. 1979. Incidence of root disease in bark beetle infested eastern Oregon and Washington true firs. *Plant Dis. Repr.* 63(4): 262-266.
- Lockman, I.B. 1993. Population structure and incidence of *Heterobasidion annosum* in Grand Fir and Douglas-Fir. M.S. Thesis, Oregon State University, Corvallis, Oregon. 78 p.
- Morrison, D. J. and A. L. S. Johnson. 1978. Stump colonization and spread of *Fomes annosus* 5 years after thinning. *Can. J. For. Res.* 8: 177-180.
- Morrison, D. J., M. D. Larock and A. J. Waters. 1986. Stump infection by *Fomes annosus* in spaced stands in the Prince Rupert forest region of British Columbia. Inform. Rep. BC-X-285. Can. For. Serv., Pac. For. Cen., Victoria, B.C. 12 p.
- Morrison, D. J. and D. B. 1994. Long-term development of *Heterobasidion annosum* in basidiospore-infected Sitka spruce stumps. *Plant Pathol.* 43: 897-906.
- Nicolleti, G. and P. Gonthier. 2005. Stump treatment against *Heterobasidion* with *Phlebiopsis gigantea* and some chemicals in *Picea abies* stands in the western Alps. *For. Path.* 35: 365-374.
- Otrosina, W.J. and F.W. Cobb, Jr. 1989. Biology, ecology, and epidemiology of *Heterobasidion annosum*. Pp. 26-33. In *Proceedings on the Symposium on Research and Management of Annosus Root Disease (H. annosum) in western North America*. Monterey, California. April 1989. USDA For. Serv., Gen. Tech. Rep. PSW-116.
- Schmitt, C.L., D.J. Goheen, E.M. Goheen, and S.J. Frankel. 1984. Effects of management activities and dominant species type on pest-caused mortality losses in true fir on the Fremont and Ochoco National Forests. USDA Forest Service, Pacific Northwest Region, State and Private Forestry. Portland, Oregon. 34 p.
- Slaughter, G. W., J. R. Parmeter, and J. T. Kliejunas 1991. Survival of sapling and pole-sized conifers near true fir stumps with annosus root disease in northern California. *WJAF* 6(4): Pp. 102-105.
- Stenlid, J., and D. B. Redfern. 1998. Spread within tree and stand. In *Heterobasidion annosum: Biology, Ecology, Impact and Control*. Ed. By S. Woodward, J. Stenlid, R. Karjalainen and A. Huttermann. CAB: International. Pp. 125-141.

**Forest Health
Protection and
State Forestry
Organizations****Assistance on State
And Private Lands**

Montana: (406) 542-4300

Idaho: (208) 769-1525

Utah: (801) 538-5530

Nevada: (775) 684-2500

Wyoming: (307) 777-5659

**Assistance on
Federal Lands**US Forest Service
Region OneMissoula: (406) 329-3605
Coeur d'Alene: (208) 765-7342US Forest Service
Region FourOgden: (801) 476-9720
Boise: (208) 373-4227