LESSON 5 The Rusts

LESSON OVERVIEW

CONTENT

This lesson deals with the rust fungi, a special group of obligate parasites of leaves, bark and flowers, that often require two hosts to complete their life cycle. They have up to five separate spore stages of these hosts.

The lesson starts by describing various rust life cycles. Then it discusses some major diseases caused by rusts, paying particular attention to the various ways in which forest managers can intervene to reduce losses.

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

- Life cycles of rusts
- Classification of rusts
- Common tree rusts in North America
- White pine blister rust
- Management of other rust diseases

OBJECTIVES

This lesson serves two purposes. First, it should familiarize you with the common rusts and their effects on various forest trees. Second, this lesson contains the second of the detailed discussions of a single pathogen, *Cronartium ribicola* (the other being *Phellinus* root rot). These discussions, though not exhaustive, serve as examples of the types of issues that you need to consider when dealing with a disease that is prevalent in a stand or larger area for which you are responsible.

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

1. to recognize rusts on various plant species;
2. to identify the stages of the life cycle;
3. to describe the signs and symptoms of the major rust pathogens of North American forests;
4. to assess the effect of various levels of infection on crop yield and other purposes of management; and
5. to design appropriate management strategies to deal with rusts and where, when, and how to prescribe treatments to put these strategies into effect.

LESSON STUDY INSTRUCTIONS AND ASSIGNMENT

You should start this lesson by reading Chapter 11 in Manion (1991). Then study the material in this commentary and Pest Leaflets 26, 37, 48, 49 and 50.

As you study the material, refer to the illustrations of rusts in the *Field Guide to the Pests of Managed Forests in British Columbia* (Finck, Humphreys & Hawkins, 1990) and in *Tree Diseases of the Canadian Prairies* (Hiratsuka, 1987). Another very good reference is *The Tree Rusts*

of Western Canada by W.G. Ziller (a 1974 Canada Forestry Service publication, No. 1329, available from the Extension Library).

After covering the material in this lesson, complete the self-testing/review questions at the end. There is no assignment to be submitted for marking at the end of this lesson, but Assignment #3 at the end of Lesson 8 includes questions on this material.

COMMENTARY

LIFE CYCLE OF RUSTS

Rusts belong to the class Uredinomycetes of the subdivision Basidiomycotina. They are obligate parasites, partly because they require special chemicals from their host, but mainly because the internal plant environment represents a well protected niche in which these organisms can develop free from competition by other micro-organisms. The obligate parasitic habit requires that these fungi penetrate hosts without eliciting a host defensive response. This is achieved in part by a manner of colonization of the host that does not involve killing of host cells. Unlike most diseases caused by facultative parasites, rusts thrive best on vigorous hosts. The effect of host vigour is to increase the rate of growth of the rust mycelium within the host and to increase spore production. The infection process itself is not affected by host vigour.

Infective spores land on host surfaces (leaves, flowers, young bark, and occasionally wounds), germinate and penetrate either directly through a cuticle and epidermis (never a periderm) or, more commonly, through stomata. Inside the host tissue they develop an intercellular mycelium (i.e., a mycelium that lies wholly between the host cells) with special cell-penetrating structures called **haustoria**. Infected host tissues may be altered by hypertrophy (abnormal increase in cell size) or hyperplasia (abnormal cell division), but the cells remain alive and functional for weeks in the case of leaf tissues and years or decades in the case of bark (phloem). Death of infected tissues is usually caused by secondary organisms (often fungi) which invade and kill rust-infected tissues. Also, various rodents and some insects feed preferentially on rust-infected bark, thus girdling stems. To say that white pine blister rust kills white pine is, strictly speaking, incorrect; death of infected bark is caused by secondary organisms. However since invasion by such secondary organisms always follows rust infection, the critical event that leads to host death is the original rust infection.

Rusts have complex life cycles, an example of which is shown in Figure 11-7 in Manion (1991). Basidiospores infect the **aecial** (i.e., primary) host and produce a haploid (1n) intercellular mycelium. After a period of weeks to years (depending on the rust species), this mycelium produces **spermogonia** (also known as **pycnia**) which produce small, water-borne spores called **spermatia** (equivalent to **pycniospores**). These spermatia do not infect new tissues. Rather, they act as sex spores to transfer genetic information from one mycelium to another. Most rusts are **heterothallic**, having two sexes which are morphologically indistinguishable, and which may be designated as positive (+) and negative (–). Of the four basidiospores produced by a single basidium, two will be + and two –. The haploid mycelia derived from such basidiospores will also be + or –. The function of spermatia is to carry + nuclei to – mycelia and vice versa. Until this is accomplished, the next spore stage will not develop. In many species of rusts, the spermogonium consists of a flask-shaped structure embedded in host tissue. A droplet of sweet exudate containing spermatia oozes out of the narrow neck.

Each spermogonium also has some receptive hyphae, which are long hyphae sticking out of the neck of the spermogonia. Transfer of spermatia is achieved by insects which feed on the liquid, and transfer spermatia from + to – spermogonia (and vice versa) in the process. Rain splashing or other forms of mechanical transfer can also occur. Spermatia of the opposite sex type fuse with receptive hyphae, and their nucleus passes into the mycelium.

The next spore stage consists of **aecia** (singular is **aecium**) which produce **aeciospores**. An aecium consists of an outer skin, the peridium, forming either a blister-like structure or a small tube. Aeciospores are produced in chains within the aecium from a layer of aeciospore mother cells which have become dikaryotic following spermatial transfer. All aeciospores are also dikaryotic. Aecia erupt through the lower epidermis of leaves or needles, or, in the case of infected bark, through the outer periderm. Aeciospores are large, thick-walled spores that carry a large energy reserve in the form of lipids. They can spread long distances, exceeding 100 km.

At this stage of the life cycle, **host alternation** occurs. Aeciospores produced on one host species cannot infect that species, but instead they must land on a different, quite unrelated plant species, known as the **telial host**. Given the right conditions (usually warm but not hot temperatures and liquid water on the plant surface), they germinate and penetrate, giving rise to an intercellular mycelium with haustoria-penetrating cells. The mycelium resembles that of the primary or aecial host, but it is dikaryotic (i.e., it has a pair of nuclei of opposite sex type in each fungal cell).

Within weeks of infection of the telial host a third spore stage is produced, namely the **urediniospore** stage. Urediniospores are produced in small (about 1 mm diameter) orange pustules called **uredinia**, usually on the leaves of the telial host. The urediniospores are as large as the aeciospores, but they are typically thin-walled. Urediniospores infect the host species on which they are produced. The cycling time (the time from infection to production of the first new urediniospores) is as brief as a week, leading to a rapid buildup of the disease on the telial host during the growing season. In many agricultural crops, such as wheat and maize, the urediniospore stage is the damaging stage. The name "rust" derives from the colour of these crops when they are heavily infected. In forest trees, either of the hosts may be the commercially important one.

Toward the end of the growing season, as days shorten, temperatures drop, and leaves begin to senesce, the dikaryotic mycelium in the telial host begins to produce a fourth spore stage, namely the telial stage, often on the same leaves on which uredinial are being produced. **Telia** are varied in appearance, but consist of masses of large, dikaryotic teliospores. For instance, in *Cronartium* they are arranged in bristle-like structures; in *Melampsora* as dark waxy crusts with the teliospores arranged side by side in a single layer; and in *Puccinia* singly on short

stalks. Teliospores do not infect new tissues. Rather they serve (in most rusts) as the overwintering stage. When teliospores are ready to germinate, the two haploid nuclei in each spore fuse to form a true diploid nucleus. This is followed immediately by meiosis, resulting in four haploid nuclei. During this time, a single basidium has formed either on the outside or within the old teliospore wall, and the four products of meiosis move into four basidiospores, which are then released to complete the life cycle.

CLASSIFICATION OF RUSTS

Classification of rusts is based largely on the shape of teliospores and their arrangement into telia. The earlier spore stages in the life cycle are nearly identical for all rusts, and cannot be used to define species. However, rusts are usually very host-specific. Therefore, the appearance of a rust spore stage such as aecia or uredinia on a particular host is usually sufficient to identify the rust.

Various reductions occur in the full heteroecious (two host) eu-type life cycle described above. In some rusts, all five spore stages occur on the same host (autoecious). In such cases, the host is usually closely related to the aecial (rather than the telial) host of related rust species with a heteroecious life cycle. In other rusts, certain spore stages may be missing. For instance, in the genus *Gymnosporangium*, there are no urediniospores. In other rust species, such as some species of *Cronartium*, urediniospores are rare. Sometimes spermogonia and telia are the only spore stages. A few species of pine stem rust produce only aecia (and sometimes spermogonia at irregular intervals). In this last case, the nuclear cycle is still a matter of dispute. All these appear to be reductions from the full, five-spore stage two-host life cycle, and not steps leading to the original evolution of that life cycle.

There is also a good deal of variation in the time of year that the various spore stages are produced. This information is most easily conveyed by life cycle diagrams of the type proposed by Ziller, and shown in Figure 5.1. In these diagrams, the months of the year follow each other clock-wise around the circle. The aecial host is represented by the inner, and the telial host by the outer spiral (solid shading represents dead host material such as dead leaves in winter). Three types of arrows represent the three infective spore stages. The time of year that the various spore stages are produced, the length of time between infection and spore production, and the manner in which the rust overwinters can then be readily seen. Two examples are shown in the figure.

For *Melampsora*, the leaf rusts of members of the Salicaceae, the main aecial host is Douglas-fir, although pines, larch, hemlock, true firs, and spruce can also serve. Infection by basidiospores (the half-arrow heads) occurs in spring. The susceptible tissue consists of newly flushed needles. Spermogonia are produced within a week, and aecia within two weeks; the aeciospores infect cottonwood and willow leaves (solid arrows). On these leaves, the rust produces urediniospores which spread infection on the telial host all summer long (looping open arrows). Then

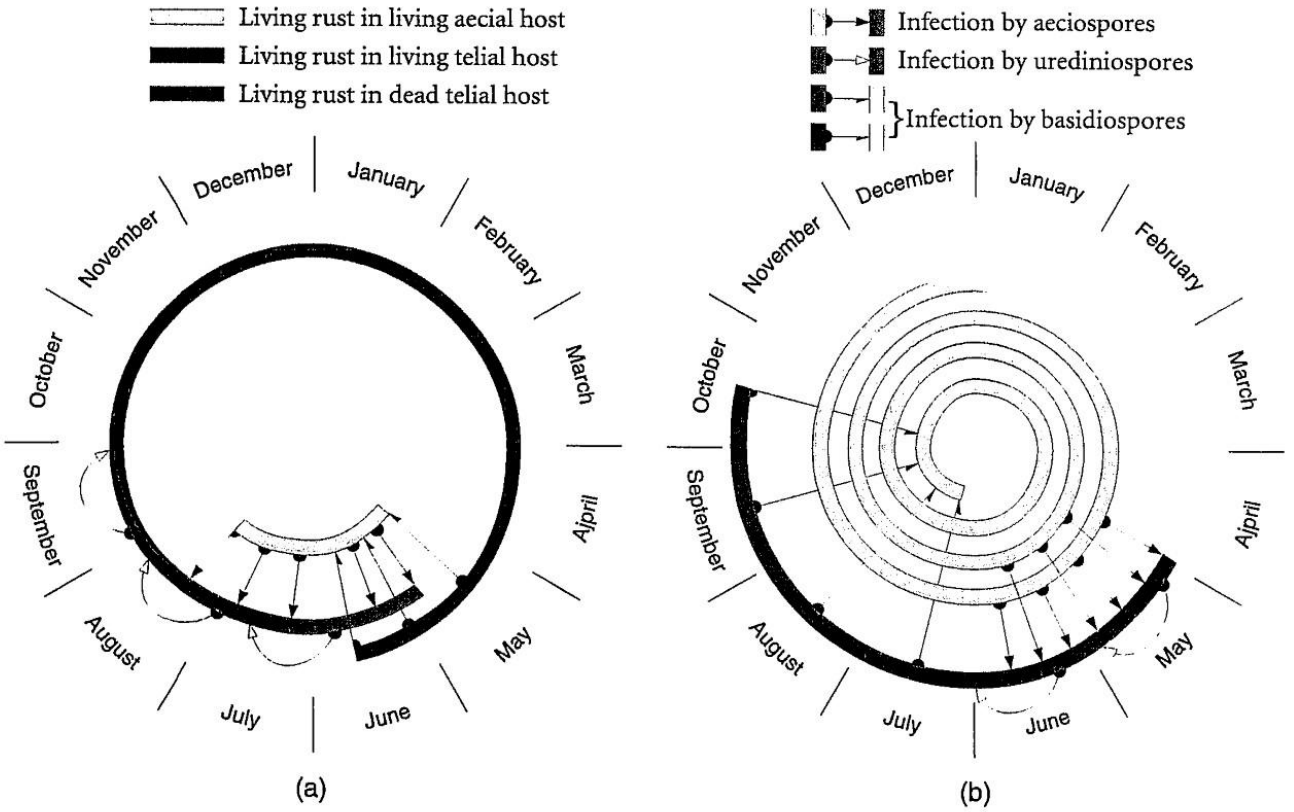

FIGURE 5.1 Life cycles of (a) *Melampsora* and (b) *Cronartium*. [Adapted from W.G. Ziller, 1974, *The Tree Rusts of Western Canada*.]

the rust produces telia which overwinter on dead leaves on the ground and germinate in spring to give rise to basidiospores again.

The second example is for *Cronartium* rust of pines. Here the aecial host is pine (various species for different species of *Cronartium*) and the telial host is various shrub and herb species, again depending on the species of rust. Infection of the aecial host occurs in late summer and fall from basidiospores produced by telia on the leaves of the telial host. These basidiospores land on and infect one- and two-year-old (or even older) needles of the aecial host. The rust mycelium grows down the needle to the bark, and after about two years produces spermogonia and then aecia on the bark. The rust is perennial on pine, and once established, it produces a new crop of aeciospores every spring. Aeciospores spread infection to the telial host, and urediniospores intensify infection on that host all summer long. So, in *Cronartium*, overwintering occurs as mycelium in the aecial host.

Common Tree Rusts in North America

As you study the various species discussed below, examine the illustrations in the supplementary materials. They will help you to visualize the signs and symptoms produced by the various rust species.

There are several native *Cronartium* species. All start as infection of pine needles, with the mycelium growing along the needle to the stem or branch, and then developing in the phloem and cortex of the bark. Aeciospores are produced on bark several years after infection. *C. coleosporioides* (telial host: *Castilleja* spp. and members of a few closely related genera), *C. comandrae* (common telial host: *Geocaulon lividum*) are common stem rusts of northern and western hard pines. *C. comptoniae* (alternate host: *Myrica gale*) is less common and also occurs on hard pines. *C. quercuum* is an eastern species that cycles between hard pines and oaks. It consists of several forms that parasitize different pine species. One form, *C. quercuum* f. sp. *fusiforme* is particularly serious on southern pines, largely because of the cultural practices adopted in that area (large areas of young, fast growing, uniform pine plantations in areas where the rust hazard is high). Most of the native *Cronartium* rusts cause limited damage in their native habitat, and they certainly do not represent a threat to the survival of their hosts. All of them can occasionally be troublesome. The lesson provided by fusiform rust in the southeast should be carefully considered; some silvicultural practices may promote these rusts and lead to severe damage.

The most serious *Cronartium* rust is *C. ribicola,* the cause of white pine blister rust. Unlike the other *Cronartium* rusts, which are all native to North America, *C. ribicola* was introduced from Europe (following an earlier introduction to Europe from Asia). The introduction early this century of this rust to North America occurred separately in eastern and western regions. The rust has spread very rapidly, causing extensive mortality in all five-needled pines.

Endocronartium harknessii, the cause of western gall rust, induces woody galls on hard pines in the western and northern forests. The species is clearly related to the *Cronartium* rusts, but has a greatly reduced life cycle. Only aeciospores are produced, and these infect pine directly through the epidermis of new shoots in spring. There is no alternate host.

Melampsora species cycle between conifer needles (the aecial host) and leaves of various members of the Salicaceae. They can be troublesome in *Populus* culture. In B.C., *M. occidentalis* occurs on cottonwoods, while *M. medusae* is found on aspen. Other species occur on willows.

Chrysomyxa species cycle between spruce and Ericacious plants. On spruce, most species infect needles. One species, *C. artostastaphyli,* forms pronounced yellowish brooms with deciduous needles. A few (*C. pirolata* and *C. monesis*) infect spruce cones, and these can destroy complete cone crops.

Many rusts of conifers infect only needles. On rare occasions rusts cause severe defoliation of either current or one-year-old leaves. Seedlings may be killed; larger trees usually recover. These rusts should not be confused with foliage diseases caused by Ascomycetes.

White Pine Blister Rust

Having reviewed various common rust diseases of western forest trees, we will now consider one of them, namely white pine blister rust (WPBR), in detail. In the discussion below, material related to the biology and behaviour of the rust is integrated with the various options for managing this disease.

Introduction of WPBR to North America

Cronartium ribicola, the cause of WPBR, is native on Asian white pines, particularly *P. griffithii* in the Himalayans and some five-needled pines in China and Japan. It may also be native on the European stone pines in the Alps and Balkan Mountains. Early in the 19th century, Germans started to plant *P. strobus* from North America in their lowland plains. WPBR moved down from the mountains and destroyed these plantations. From that time on, the rust has survived on specimen plantings in gardens and arborita throughout Europe. Records are hard to come by because rust life cycles were first figured out near the end of the 19th century.

In 1910 the rust was introduced into North America on a shipment of *P. strobus* from France to Vancouver (Point Grey). The rust wasn't noticed until several years later, and by that time it had spread more than 100 km in all directions. All North American five-needled pines, including the soft, stone, and foxtail pines, proved to be very susceptible. West coast forest pathologists tried to stem the spread of the rust, but nothing stopped its advance. By the 1960s, it had spread throughout the range of all white pines, reaching all but a few isolated populations in the Californian mountains. Apparently, in 1905 there was a separate introduction of the rust at Geneva in the state of New York, which allowed it to spread throughout the range of eastern white pine.

Introductions of foreign pathogens into North America are not uncommon. While most have difficulty surviving, or may develop into minor diseases, a few, such as WPBR, chestnut blight, and Dutch elm disease, have proven to be major destructive diseases. It seems that not all introduced diseases are necessarily serious. Considering that the native *Cronartium* rusts are not very harmful to their hosts, the hard pines, why is white pine blister rust so damaging to white pines? The answer is that North American white pines may well have been the only group of pines in the world without a native *Cronartium* rust. Perhaps such a rust existed in North America long ago, but separation of the two hosts during glaciation may have led to its disappearance here. Since the separation, the natural resistance mechanisms to *Cronartium* were no longer an advantage to the individuals possessing them, and thus resistance disappeared through the mechanisms of genetic drift and selection against individuals carrying such momentarily useless mechanisms. This resulted in a population of hosts that was very susceptible to the rust. In time (possibly centuries), the species will undoubtedly develop considerable resistance to the rust, and the pathogen will begin to have the same effect as the native *Cronartium* rusts. In the meantime, however, several white pine species (e.g., *P. strobus, P. monticola,* and *P. lambertiana*) are no longer useable as

commercial species in most of their range. This represents a considerable loss, because white pines are easily regenerated and fast growing, and produce valuable high quality lumber.

The loss of white pine as a major component of western forests has further implications. White pine has been replaced by other conifers such as Douglas-fir and true firs which are much more susceptible to *Phellinus* and other root diseases. Thus the loss in forest productivity is far greater than might be expected from a change merely in species composition because of the greater prevalence of root disease.

Development of WPBR on the pine host

The life cycle of WPBR starts with infection of white pine needles by basidiospores produced in late summer and fall. Needles of all ages may be infected. Primary needles on seedlings are particularly susceptible, and two-year-old secondary needles are more susceptible than the one-year-old needles, apparently because their waxy cuticles have worn down so that they can be wetted evenly. The germ tube enters through needle stomata, and the rust mycelium develops in the needle mesophyll, causing a pale yellow or red spot on the needle. It then enters the needle vascular bundle and grows along the needle phloem to the short shoot and the bark. It takes about a year from infection to entry of the bark. If the infected needle is shed during that time, the infection is lost. After reaching the stem (or more commonly the branch), the mycelium develops in the bark and between living xylem ray cells, growing both proximally and distally from the point of entry at about 10 cm per year longitudinally and 1 to 3 cm tangentially, but much slower in low vigour branches. Infected bark is often somewhat orange and there may be a slight fusiform swelling at this stage. Two or three years after reaching the bark, the rust produces spermogonia, and after transfer of spermatia, the first crop of aecia. Aecial initials form within the living bark, and as aecia mature, they rupture the bark surface. The rust mycelium continues to extend in the pine host as long as the host remains alive. Each year more spermatia and aeciospores are produced. After a patch of bark has produced three or so crops of spores it usually dies. Strictly speaking, death is caused by secondary organisms which invade through the wound (the break in the outer periderm) caused by the emerging aecium. The gnawing of rodents on infected bark (they prefer such bark because it is rich in nutrients and oils) is another major cause of death. Stem infections that haven't yet girdled the bole can be stopped by gouging a strip of bark around the infected area ahead of the advancing mycelium. While this procedure has limited application in the forest, it is valuable for saving specimen and landscape trees.

Most infections occur on branches, and the loss of these branches may represent a small loss to the tree. Major damage does not occur until the rust reaches and girdles the main bole and either kills the tree (if the girdle occurs at the base of the live crown), or the top (if the rust reaches the bole via one of the upper branches). In the latter case, the rust continues to move down the stem, and the tree soon finds itself in an intermediate or suppressed crown position, and dies. Thus infections

can be divided into two groups. Lethal infections are located close to the main stem and in the upper crown. Such infections have a high probability of reaching the main stem and eventually killing the tree. Non-lethal infections occur in the outer and lower crown. Such infections may girdle and kill branches, but they are unlikely to reach the stem before the tree is harvested or before the branch on which they occur dies by natural branch suppression.

White pine trees that were already large and old when the rust first arrived, are mostly still alive. Such trees have no needles close to the main stem in the lower crown, and hence most infections on them are of the non-lethal type. Many of these trees, however, do have dead tops.

Because of the manner in which WPBR develops in the host, there is a considerable time lapse between infection of needles and girdling of the bole. The older and larger the tree at the time of infection, the longer that process will take. In trees older than about 40 years, the lower bole will be free of needle bearing branches and thus it will no longer be in danger of becoming infected. Infection in the crowns of such trees will often take as much as 20 to 40 years to girdle the main stem, and that girdle will be near the top of the tree, leaving the valuable lower bole intact. Thus if pine is to be harvested at 80 to 100 years of age, the danger of major threatening infections is largely past at age 40. The first decades, and particularly the first 15 or so years are the critical time for infection, and for various treatments that reduce infection. After that it is either too late, or the tree will probably survive any threat from WPBR.

Foliage on young trees (up to 15 or so years) may be more susceptible than that on older trees (a "juvenile effect"). More important, the microclimate within one or two meters of the ground often (but not always) is much more conducive for the tree to become infected there than at greater heights. Much data show that in open-grown trees about 10 meters tall, as much as 90 percent of the infections occur within two meters of the ground. Part of the phenomenon is undoubtedly attributable to the fact that the target area (the number of needles) is also much greater near the ground, but some of the effect relates to the microclimate. A further reason for high infection near the ground may be that basidiospores are produced on shrubs, and that their concentration is much greater near the ground. Since most of the infections occur near the ground, if the pine can live through the first twenty or so years without serious infection, the worst is past.

Reducing Rust Infection Risk through Pruning

One way of reducing infection risk is by pruning the tree when it is young. Pruning all lower branches reduces the probability of stem infections. Particular care must be taken to remove the lowest small branches that are hidden in the shrub layer and partly covered by litter, because these branches are often infected. The idea is to remove infected branches before the rust mycelium has reached the main bole, and to eliminate potential infection sites in the susceptible lower crown. Pruning all lower branches is usually quicker than inspecting each branch and removing only those that are infected.

Pruning is a worthwhile technique if the following conditions hold:

- the microclimate is such that few infections occur above one to two meters;
- the pine stand is young and even-aged, with few stem infections; and
- the severity of infection is low to moderate, so that at the time of pruning most trees are still free of stem infections.

In many naturally regenerated stands, the white pine component is established over a period of 10 to 15 years. Pruning of these stands is much less effective than it is for trees in plantations, because in natural stands different trees reach the optimum pruning age at different times. Even at the single best time for pruning, some trees will be so old that infections in the lower branches will have already spread to the lower bole, and at the same time, other trees will be too small to prune all the branches in the danger zone one to two meters above ground.

WPBR on *Ribes*

Aeciospores infect all species of *Ribes,* although some species are much more susceptible than others. For infection, moist weather is required. On *Ribes* the rust produces uredinia and later in the season, telia. Infected *Ribes* leaf spots live for about five weeks. During that time the urediniopores produced on that spot must cause new infections or the rust dies out. Hence long periods of dry weather during the summer may greatly reduce the amount of infection on *Ribes* and hence the inoculum to which the pine is exposed. This may be a major factor in low-risk sites, as areas with frequent long rainless periods in summer have light to moderate infection on pine. Of course, such areas are also at the dry end of the range of white pine.

Basidiospores are produced in the fall. They are small spores that do not normally travel very far. Most studies suggest that few spores travel more than 200 to 500 meters. Local air flow patterns during times of spore release are important, and often explain why certain topographic locations such as creek beds and gullies are associated with much greater hazard than other locations.

WPBR Control through *Ribes* Removal

Ribes removal to control the rust has been attempted on a large scale starting in the 1930s and continuing until the mid 1960s. The results have been disappointing. There are several reasons for this. First, *Ribes* is difficult to remove by mechanical means, since bits of roots left in the ground will sprout again. Herbicides are somewhat more efficient, but still do not give full control. Furthermore, there is a large seed bank of dormant *Ribes* seed in the soil, and that seed germinates in response to various types of disturbances such as partial or clearcut logging. Also most forests are subject to a constant *Ribes* seed rain via bird droppings. So it is nearly impossible to ensure the absence of *Ribes* at the time that white pine is at its very susceptible seedling stage. Also, there is some long distance spread by basidiospores from nearby untreated areas such as stands without a significant white pine component. Thus *Ribes* removal programs can reduce the amount of infection, but they cannot

eliminate the disease altogether. The maximum attainable reduction in infection is about 90–95 percent.

It is clear from our understanding of the nature of the disease that a single, well placed "lethal" infection at some time during the first forty or so years of a tree's life, is sufficient to kill the tree. In the main white pine areas of the Inland Empire (Northern Idaho and the Kootenays), infection rates are generally high. During the critical first forty years, there is an average of 60 or more infections per tree, a quarter of which (15 infections per tree) may be lethal. (Of course, many trees are killed by the rust before they reach 40 years of age and before they have sustained a large number of infections.) A good *Ribes* removal program might reduce infection by 90 percent to an average of 1.5 lethal infections per tree, which is still sufficient to kill almost every tree! The lesson is that *Ribes* removal may be useful in low- to medium-hazard areas, or in conjunction with other methods that reduce the rate of infection, but that by itself, it cannot be successful in high-hazard areas.

The calculation above is somewhat simplistic because it is based on averages and does not take into account variation in resistance between trees. If the stand has an average infection severity of 15 lethal infections per tree, as in the above example, then the most susceptible trees may well have over 100 lethal infections, while the more resistant trees may have only a few. In such a case, the susceptible trees are inevitably lost, but many of the moderately resistant trees can be saved by the *Ribes* removal program. Detailed mortality calculations become much more difficult, and require that we know something about the distribution of infection in natural pine populations.

Ribes removal has a major role around nurseries. Nurseries should be located in ecological zones where the natural population of *Ribes* is low. Because conditions in nurseries are ideal for infection (young, vigorous trees and plenty of moisture) and because it takes several years from infection to appearance of obvious symptoms, it is very easy to ship infected but asymptomatic seedlings. Clearly this has happened in many experimental plantings of white pine. A less desirable alternative for nurseries is the application of an intensive fungicide spray program.

There are some natural phenomena that reduce infection. For instance, in swamps the host may carry needles for only two years or less, resulting in the loss of many infected needles before the mycelium has reached the bark, resulting in low levels of infection. There is also a naturally occurring hyperparasite, *Tuberculina maxima,* which attacks *Cronartium* infections on pine and stops aeciospore production. In areas of heavy infection, this fungus can be common and can substantially reduce the number of aeciospores. Depending on summer weather, this may in turn result in a reduction in basidiospore production on *Ribes.*

The common *Ribes* species are shade intolerant. As even-aged stands grow up, and crowns close, the amount of *Ribes* decreases markedly, and with that, the number of basidiospores. However, particularly in mountainous white pine areas, there are frequent stand openings such as

rock bluffs, creeks, and swamps — areas in which *Ribes* flourishes. Nevertheless, part of the apparent decrease in infection rates with increasing age may be due to the concomitant reduction in the density of *Ribes*.

It should also be evident from the above discussion that one way to reduce infection is to keep stands relatively dense. Dense stands provide several beneficial effects: first, because there is a reduction in the life span of the lower branches, and they have less foliage on them, the rate of growth of the rust mycelium in the bark of such branches is slowed, thus reducing both the number of infections and the likelihood that such infections will be lethal (i.e., that they will reach the bole). Another benefit is that early crown closure will result in a more rapid decline of *Ribes*. Finally, if the number of stems is kept high at an early age, considerable mortality can be tolerated before stocking declines to a point that productivity begins to suffer. In this regard, it is also wise to regenerate white pine in mixtures with other species, so that one can always fall back on the other species to provide the necessary stocking if the white pine should fail.

Tree Breeding to Increase WPBR Resistance

There is considerable variation in resistance between individual trees. A few trees in the population are resistant enough to be useful for breeding. Natural selection would no doubt eventually result in a moderately resistant white pine population, but selection and breeding can speed this process substantially. The first of such programs, initiated in Idaho, is now producing considerable quantities of resistant stock. Lack of strong provenance development in white pine means that only a few (perhaps just coast and interior) seed zones need to be recognized. (Every pine provenance needs its own breeding program, so the lack of strong provenance development in white pine is very fortuitous.) Breeding trees for resistance and stability of the resulting resistance will be discussed in Lesson 7. Here it is sufficient to note that the rate of infection on the best resistant pine stock is a fraction of that of wild stock, perhaps as low as 10–20 percent (final data are not yet available). Thus breeding for WPBR resistance has become a major tool for reestablishing white pine as a commercial species.

The key to the management of white pine is the recognition of site hazard. A good measure of hazard would be the average number of lethal infections per tree that accumulate over the life of a stand. In those terms, the number of lethal infections per tree in a high hazard site might be greater than 10; medium hazard, 1 to 10; and low hazard, less than 1. Such a definition is a little simplistic because it ignores variation in infection with tree height, and that has a major impact on the efficacy of treatments such as pruning. Hazard is determined by a number of factors such as the species of *Ribes*, the number of bushes and their spatial distribution on a site (and its pattern of change over time); the summer climate, as it affects the success of urediniospores; the fall microclimate, as it determines the success of basidiospore infection on pine and the vertical distribution of pine infection.

There is considerable variation in hazard between regions and between stands within regions. In general, the drier parts of the coastal range of white pine represent low-risk areas, whereas the Kootenays represent the highest risk. This variation arises in several ways. The major *Ribes* species vary in the degree to which they produce basidiospores. For instance, *R. lacustre* is a poor host while *R. bracteosum* is a good one. In addition, summer drought plays a role as described above. Also, microclimate at the time of pine infection is critical and determines the amount as well as the average height of infection. In many areas there is little infection of pine above two or three meters; elsewhere even tall trees are readily infected.

As we have seen above in the discussion of the efficacy of *Ribes* removal programs, none of the available treatments (pruning, *Ribes* removal, or breeding for resistance) are likely to be successful if applied by themselves in high-hazard areas. On the other hand, when used together, particularly in medium-hazard areas, they can result in a great reduction in white pine blister rust mortality.

Management of Other Rust Diseases

The native *Cronartium* rusts resemble *C. ribicola* in many ways. The major difference is that the hard pine hosts show a great deal more resistance to the rust. As a result damage is insignificant in many areas. For these diseases, another factor, not discussed above, which becomes very significant is the variation in climate from year to year. Rust spores are dispersed over periods of weeks or months (depending on the spore stage), but the bulk of the spores are dispersed during a few peak dispersal events during that time. Infection occurs only during the brief times that conditions on the leaf or needle surface are suitable for germination and penetration. Thus, heavy infection requires the coincidence in time of optimum dispersal and infection conditions. Such coincidences are infrequent, and in many years may not occur at all. This leads to what is known as the "wave year" phenomenon (a "wave year" being a year of heavy infection). In the case of the native *Cronartium* rusts (including *Endocronartium*), years of significant infection are often as infrequent as once every ten to twenty years. The size of the area over which such wave years occur also varies. Sometimes such areas stretch over many thousands of square kilometers, but at other times they are restricted to small microsites no more than a hectare in size.

The relationship between tree height (and age) and susceptibility to the native pine stem rusts is even more pronounced than it is for white pine blister rust. Infection during juvenile stages is most critical. Above two or three meters, there is little infection and most of these infections are not lethal. Whether or not a particular stand sustains significant damage depends on the number of wave years that occur during the critical first fifteen or so years. Some stands make it through that critical period without a single major infection event, and as a result escape damage. Many more stands experience a single major infection event during the critical period, and some damage ensues. A few stands are

exposed to several wave years during the critical period, and in such stands damage can be severe. It has proven to be very difficult to predict where wave year events are likely to be frequent: most of the variation seems to be random.

The number of wave years is the major determinant of the amount of infection. It masks the effects of other factors, such as stand density, species mixtures, fertilization, and various other silvicultural operations. The native *Cronartium* rusts are most commonly encountered on lodgepole pine. Whether or not damage occurs depends on the extent of mortality and the original stocking. In many stands the number of stems per hectare is well above the minimum required to capture site productivity. In such stands these rusts act as thinning agents, and only very seldom do they result in unacceptably low stocking. Almost all the problems arise from inappropriate precommercial thinning. At the time of precommercial thinning, the period of high infection is almost over. Both infected and healthy trees will have normal looking crowns at this time, but of course many of the infected trees will die over the next twenty or so years. What has happened again and again around the province is that heavily infected stands are thinned, taking the stocking down to a low level, without attention to rust infection. Very often in such cases the percentage of trees infected is higher after than before precommercial thinning, mostly because larger trees are more likely to be infected than smaller trees. The result is further mortality leading to unacceptably low stocking. The proper procedure is to inspect young lodgepole pine stands for rust infection, and to either (1) make sure that rust infected trees are all removed (difficult to do in operational precommercial thinning) or (2) increase the target density so that even after the expected rust mortality has occurred, stocking will still be acceptable.

Western gall rust, caused by *Endocronartium harknessii,* needs a special mention. This rust differs from the other pine stem rusts in that it forms a local woody gall at the point of infection, and does not grow along branches to the bole. Branch infections may eventually result in branch mortality, but only stem infections lead to tree death. Infection must occur through immature shoots in spring. Hence the only part of the stem that is susceptible to infection is the new leader. Since, on most sites, the rate of infection falls off quickly with height above the ground, the susceptible period is very short, perhaps only ten years, and unless a wave year occurs during that short period, there is little or no long-term damage, even though the lower crown may be heavily infected.

The *Melampsora* rusts of cottonwood and aspen damage their hosts by reducing the photosynthetic area of leaves and by the fact that infected parts of leaves act as energy sinks. The exact impact of these rusts has not been quantified in detail but they may well cause average increment reductions in the order of 20–40 percent. Fortunately, the hybrid poplars that are used in most commercial *Populus* plantations are quite resistant to *Melampsora.* If native hosts are to be used, the most promising approach is to search for resistant clones. Chapter 7 deals

with stability of resistance, and the likelihood that currently resistant clones will lose that resistance.

The needle and broom rusts caused by *Chrysomyxa* species apparently do not cause a great deal of damage, but the two cone rusts do. Again, infection varies considerably from year to year. In some years and locations, the whole spruce cone crop may be destroyed. Thus the cone rusts are important in seed collection areas. Cones that are infected turn brown and begin to open two to three weeks before normal cones. The brown, partly opened cone scales will be bearing the aecia of these rusts. Indications of the likelihood of infection can also be obtained earlier in the season by inspecting the telial hosts for their abundance and the level of infection on them. These telial hosts are *Moneses uniflora* for *Chrysomyxa monesis,* and all common species of the genus Pyrola as well as *M. uniflora* for *Chrysomyxa pirolata.* All these telial hosts have evergreen leaves. This allows these rusts to overwinter as dikaryotic mycelium in these leaves and continue to reproduce by urediniospores the following year. (They can also overwinter in the more normal way as dormant telia.) Thus these rusts can survive on the telial host in the absence of the aecial host, and in fact they have often been reported from areas where spruce is absent. Plans for spruce cone collection from areas where the telial hosts are common should include checks for the rust on the alternate hosts as well as on spruce cones.

SECTION ASSIGNMENT

SELF-TESTING/REVIEW QUESTIONS

Test your understanding of the material in this lesson by attempting to answer these questions. Do not proceed to the next section until you are satisfied with your proficiency in this section.
Do *not* send your answers to the tutor for marking. If you continue to have difficulty with a question after you review the relevant material, you may wish to discuss it with your tutor.

1. Given the information about *Chrysomyxa pirolata* presented earlier, draw a life cycle diagram of the form illustrated in Figure 5.1 that shows when the various spore stages are produced, and when infection of the two hosts occurs.
2. What stand and disease parameters should you assess in order to decide whether or not to prune white pine for control of blister rust?
3. What factors determine site hazard for white pine blister rust?
4. How do various rust species overwinter?
5. In which of the common rust species studied in this lesson is the main damage done to the telial host? In which to the aecial host?
6. Why is *Ribes* eradication for the control of white pine blister rust unlikely to be successful in areas of high hazard?
7. Compare rusts and canker diseases with respect to the relationship between host vigour and susceptibility to disease.