SECTION 2

FOLIAGE DISEASES

reading

Read Chapter 10 of Manion (1991), then the commentary in this section, and Pest Leaflets 27, 32, 43 and 52.

Then study the illustrations and descriptions of foliage diseases in the following: *Field Guide to the Pests of Managed Forests in British Columbia* (Finck et al., 1990); and *Tree Diseases of the Canadian Prairies* (Hiratsuka, 1987).

You need not memorize all the detail in these materials, but you should become familiar with the variety of symptoms associated with various foliage diseases.

Another good reference is *Foliar Fungi of Western Trees* (Funk, 1985; Canadian Forestry Service, Pacific Forest Research Centre. BC-X-265). This book gives a complete description of all B.C. tree foliage pathogens.

Foliage diseases can be caused by a variety of biotic and abiotic agents, including air pollution, nutrient deficiencies or excesses, insects, mites, viruses, bacteria, and fungi. Frosts can also result in foliage symptoms that could easily be misidentified. Thus careful attention to signs and symptoms and their development over time is required for correct identification. This section is concerned mainly with the foliage diseases of conifers caused by fungi, although some fungal diseases of hardwoods will also be discussed.

Foliage diseases have a more serious effect on dry matter accumulation in evergreens than in deciduous trees because in evergreens the leaves would normally function for several years, and that productive potential is lost.

Most foliage diseases belong to the Ascomycotina; a few to the Deuteromycotina. There are also several foliage diseases caused by rusts, but these will be discussed later. Several types of life cycles occur. On conifer needles the common life cycle consists of the production of ascospores on dead or dying needles, either on the ground or still on the tree, during a relatively short spore release period some time during the growing season, leading to a simple annual (or sometimes biannual) cycle. In a few cases the dominant spore stage consists of asexual conidia, while the sexual stage may be absent or rare. In such cases, the period from infection to spore production can be much shorter, and spores are produced whenever conditions are favourable rather than at specific times.

Most foliage diseases of broadleaved trees have a more complex life cycle. Here the pathogen usually overwinters on dead leaves on the ground and produces its sexual ascospores early in the spring, to coincide with bud burst. Infected leaves then begin to produce asexual conidia which are released and spread to other leaves throughout the growing season, whenever weather conditions are suitable. The asexual

cycle is very short, often less than two weeks, and, if the weather conditions are right, very high levels of infection can result.

The incidence of foliage diseases fluctuates remarkably from year to year. Some years infection is so intense that all susceptible foliage is killed by early summer; in other years the same pathogen may be hard to find. In the case of angiosperms, it is common for trees to produce a second set of leaves early in the summer if the first set has been destroyed by a pathogen. Such late season leaves usually remain uninfected, often because climatic conditions suitable for infection are uncommon in mid and late summer.

Infection requires living spores, susceptible tissue, and suitable environmental conditions. The bulk of the ascospore release usually occurs as a few short-duration (a few hours to no more than a day) events triggered by the right temperature and moisture conditions. Such events typically occur over a total period of about six to eight weeks. For most foliage diseases, susceptible tissue consists of newly formed foliage. Such tissues are more susceptible for at least two reasons. First, all exposed plant surfaces are quickly colonized by a whole range of microorganisms including fungi, bacteria, algae, and small lichens, forming what is known as the **phytoplane community**. This community may serve to inhibit spore germination via antagonism. It takes some time to develop this phytoplane community, and the rate is dependent on such factors as weather, dust and debris deposition, and leachates in canopy throughfall, as well as the degree of exposure to direct sunlight. (A second group of foliage diseases of conifers invades primarily old, senescing needles. These are not affected by the phytoplane community. Their effect is to hasten needle shedding, and it is unclear whether they cause any significant damage, because old needles do not contribute much to total tree photosynthesis.)

The second factor governing susceptibility is tissue maturation. Here the mode of entry is important. Some foliage pathogens penetrate directly through the cuticle and epidermis; other enter through stomata. For the former, penetration of the outer protective layers (epidermis, cuticle) is easier before these layers have fully matured.

The coincidence in time of a major spore release with the availability of such susceptible tissues and weather conditions that allow spore germination and penetration can result in severe infection. In most years however, these three do not coincide perfectly, leading to low infection.

Variation in flushing time ensures that individual trees in a population reach maximum susceptibility at different times. Hence some years the early flushing trees may be infected while the late flushing trees escape because their foliage was still protected by the bud scales at the time of the major infection event, while in other years the late flushing trees are hit while the early flushing trees are only lightly infected because their new foliage was too mature to allow heavy infection at the time that the first major infection event occurred.

Trees with indeterminate growth have some newly formed, susceptible tissue at all times. Pines are a good example. Pine needles continue to elongate for most of the summer, so that there is some susceptible needle tissue just emerging from the needle fascicle sheath at all times. In fact, major infection events in pine can often be dated by the position on the needle of the infection: early infection occurs near the tip; later infection lower down. There are a large number of pine needle parasites, and many of these produce their ascospores mid to late summer, much later for instance than related parasites of the spruces and true firs.

Many needle pathogens of conifers are naturally controlled by a second phenomenon. It so happens that needles that have been infected by such (primary) pathogens become rather susceptible to certain specific secondary fungi that cannot readily infect healthy foliage. Many of these secondary pathogens have an asexual stage with a spore-to-spore cycling time of one or two weeks. Invasion of diseased needles by the secondary pathogen results in a substantial reduction in spore production by the primary parasite. A good example is provided by *Lophodermella concolor,* a virulent pathogen of lodgepole pine foliage and *Hendersonia pinicola,* a secondary parasite that is almost wholly restricted to needles that have already been infected by *L. concolor.* In years of low infection by *L. concolor, H. pinicola* barely survives. The secondary parasite produces many spores, but since these are randomly dispersed, most land on healthy needles which they are unable to colonize. It may be that of all the spores produced by *H. pinicola* on a single needle in a year, fewer than one (on average) lands on and infects a *L. concolor*-infected needle. In such a case the population of *H. pinicola* will decline. Every now and then however, environmental conditions will be such that there is a severe infection by *L. concolor.* These outbreaks can be spectacular — over large forest areas, all the one-year-old needles turn reddish brown. The next year, spore production of *L. concolor* will be very high, and hence, even if conditions for infection are sub-optimal, the outbreak will continue. During the outbreak, a large proportion of the randomly dispersed spores of *H. pinicola* land on *L. concolor*-infected needles, and the secondary parasite rapidly infects all such needles (remember the short, asexual life cycle). Two or more years later, there are virtually no *L. concolor* spores produced (all infected needles produce *H. pinicola* instead), and *L. concolor* declines rapidly, followed by a decline in *H. pinicola.* In B.C. at least 19 such pairs of primary and secondary needle parasites have been described or are suspected.

A third natural phenomenon that limits damage by foliage diseases is a marked, genetically conditioned variation in susceptibility. That variation occurs at the provenance level (provenances from areas that are climatically suitable for the pathogen show greater resistance than other provenances when they are compared under standard conditions), and at the stand level with considerable variation from tree to tree. Some evidence suggests that different races of a particular foliage pathogen are virulent (i.e., cause disease) on different and overlapping subsets of the

host population. This will be discussed further in the section dealing with the genetics of resistance.

Some foliage diseases become systemic in the branch. Thus *Hypodermella laricis* survives in the short shoots of larch, and all needles produced by infected short shoots are already infected as they emerge in spring. Similarly, the serious needle parasite *Elytroderma deformans* enters the cortex and terminal branch meristem of several hard pine, and induces such meristems to produce brooms (e.g., in ponderosa pine) or an abnormal growth pattern (e.g., in lodgepole pine). *E. deformans* produces spores on only a few of the needles borne on such systemically infected branches or brooms.

A special group of foliage diseases develops under snow. These snow molds have the ability to grow at low temperatures, and to grow from needle to needle as external hyphae in the high humidity snow environment. Whole groups of trees may be green when they are covered by snow in the fall, and dead and covered by a mat of dark mycelium when the snow melts. These fungi play an important role in maintaining the patch-like structure of higher subalpine forest, and in general lower the tree line. A good example is the brown felt blight caused by *Herpotrichia juniperi.* Snow molds are also occasionally troublesome in nurseries where dense, continuous stands of succulent seedlings promote their growth and spread. The pathogen *Phacidium abietis,* the cause of snow-blight, is best known in that situation. It can be controlled by a fungicide application late in the season, just before snowfall.

Foliage diseases of conifers with an annual life cycle do not develop obvious symptoms such as browning and necrosis of needles until a month or two before the pathogen produces its reproductive structures. Shortly after spore dispersal the needles are shed. This means that those with an annual life cycle remain asymptomatic for a ten-month period from infection until just before spore production the next year. During this period the pathogen behaves as an endophyte, living and spreading within the needle without producing obvious symptoms. For foliage diseases with a two-year life cycle, this period can be over a year and a half. This feature can make diagnosis difficult. During early summer, when the needles infected the previous year turn red or brown, whole landscapes can be discoloured. Once the old infected needles are shed, however, all the remaining foliage looks normal until the next spring. While old fruiting structures can sometimes be found on old needles on the ground, unequivocal diagnosis is usually difficult. One feature of foliage diseases helps: since infection is limited to new foliage, all needles that were mature at the start of the outbreak will be retained on the tree. So, during a foliage disease outbreak, the current year's foliage will all be present on the branch tips. During most of the year it will look normal, and it will not be possible to tell whether the outbreak is over, or whether most of these needles are infected and asymptomatic until symptoms do develop just before spore production. The next several internodes (depending on the number of years the outbreak has lasted)

will be bare, while needles that were mature before the outbreak started will still be on the tree (except for very long outbreaks).

Damage resulting from foliage parasites consists largely of reduced increment during outbreak years and the years immediately following such outbreaks. Foliage diseases can be particularly important on seedlings. Here the additional drain on the energy resources of the small plant can make the difference between survival and death. Conditions in the nursery often favour infection, and unless precautions are taken to remove the inoculum or to prevent infection by fungicides, losses can be substantial. Many foliage diseases do not develop obvious symptoms until several months after infection. As a result, there are occasions when badly infected but asymptomatic seedlings are lifted and shipped from nurseries.

Apart from nursery situations where sanitation combined with judicious use of fungicides may control serious outbreaks, there isn't much that can be done about foliage diseases in the forest. The loss of increment that results from these diseases has never been well defined, and such losses tend to be ignored or accepted as inevitable. Recent work suggests that losses may be substantial. The death of foliage represents not only a loss of photosynthetic capacity, but infected and dying leaves or needles act as substantial sinks of sugar, largely because the rate of respiration in such infected tissues is much higher than normal. Thus the total loss of carbohydrate to the tree is greater (percentage-wise) than the percent of leaves or needles infected. The remaining carbohydrate reserves are preferentially allocated to stem growth rather than to root growth and storage. As a result, the loss of foliage in one year leads to slower growth over the next several years.

One final observation: While foliage diseases undoubtedly harm the hosts on which they occur, other species may benefit. During outbreaks, the amount of light that reaches the forest floor is greatly increased. At the same time there will be an increase in nutrient availability resulting from the heavy litter fall. Many perennial plants living under dense tree canopies to not produce flowers regularly because they simply cannot produce the required energy in the dense shade of full canopies. However, during years of foliage disease outbreaks (and other defoliation events such as by insects), light increases, nutrient availability is improved, and competition from the tree canopy is reduced. For some species these are the only times that they produce seed. Other possible benefits include improved forage production for grazing animals, and establishment of a tree regeneration layer.

SECTION ASSIGNMENT

SELF-TESTING/REVIEW QUESTIONS

Test your understanding of the material in this section 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. Illustrate and describe the three basic types of life cycles of foliage diseases.
2. How does variation in flushing time influence the severity of foliage disease?
3. How do secondary needle parasites control foliage disease?
4. What are the major reasons for severe fluctuations in the severity of foliage diseases from year to year?
5. What are snow molds and snow blights?
6. If our current suspicion that foliage disease causes a substantial increment loss proves to be correct, what approaches do you suggest to reduce the incidence of these diseases to levels lower than those that occur naturally in unmanaged forests?