SECTION 3

CANKERS

reading

This section deals with canker fungi. First read Chapter 12 in Manion (1991), then the commentary below, and Pest Leaflet No. 25.

Also examine the illustrations in *Field Guide to the Pests of Managed Forests in British Columbia* (by Finck, Humphreys & Hawkins, 1990) and *Tree Diseases of the Canadian Prairies* (by Hiratsuka, 1987).

Parasitic Microfungi of Western Trees, a publication of the Canadian Forestry Service, Pacific Forest Research Centre, BC-X-222 (by Funk, 1981) gives a detailed taxonomic description of the canker pathogens found in western forests.

WHAT IS A CANKER?

A canker is a disease of bark resulting in sharply delineated, usually elliptical patches of necrotic phloem or cortex tissue. These patches may or may not extend to the cambium, and they may or may not girdle the stem or branch on which they occur. Their extension may occur continuously or only during certain seasons, usually winter and early spring.

Most canker pathogens belong to the Ascomycotina. Infection may be by ascospores or conidia produced on the dead bark of cankers. Such spores, when they land on uninjured bark, encounter the phytoplane microbial community which inhibits their germination. In addition, the outer plant defensive barrier, the periderm, is too thick and effective to allow penetration by a spore germ tube. Thus special infection pathways are required. A special group of bark diseases which result in canker-like symptoms is caused by rusts; these will be discussed in the Lesson 5.

CANKER INFECTION PROCESSES

Little is known about the infection process of most canker diseases, and most of what we surmise comes from indirect evidence. The reason for this lack of knowledge is quite simple. The number of cankers on a single tree is usually quite small; one or two may suffice to kill a tree if they happen to be in the right location. Thus the total number of successful penetrations occurring over many years anywhere on the bark surface is usually only a few, and the likelihood of actually observing the process is infinitesimally small. The first symptoms often don't become obvious until a year or more after infection, and at that time it is no longer possible to determine the exact time of infection nor to reconstruct the climatic conditions. Hence infection by canker fungi may be thought of as a rare event. It is of course possible to create cankers by artificial inoculation, and then the process can be observed and described in detail. However, it is uncertain that the events accompanying artificial inoculation are the same as the ones that lead to natural infection. Nevertheless, studies using artificial inoculation have shown that wounds of some sort are usually necessary, and a number of likely pathways have been worked out. The common ones are:

- insect wounds, in which the insects may also act as vectors, with the emerging insect brood carrying the spores;
- mechanical bark wounds, including, occasionally, pruning wounds;
- lenticels, or leaf traces shortly after leaf drop, or leaves, with the pathogen growing into the stem from the petiole;
- invasion of dead branches or twigs and subsequent spread into the living bark.

HOST RESISTANCE REACTION

The main host resistance reaction is the formation of a periderm. To understand canker formation and healing, you must be familiar with the different kinds of periderm. Also, you will remember that periderm formation plays a role in some root diseases. Our discussion of periderms also applies to that situation.

Periderms originate from a layer of meristematic cells that is derived in turn by a process of de-differentiation from cortex or phloem parenchyma cells. This layer is known as the **phellogen** or **cork cambium**. The phellogen produces cork or **phellem** cells to the outside and **phelloderm** cells to the inside. Cork cells die soon after they are formed; phelloderm cells stay alive. The maintenance of periderms by annual growth may happen in two ways: the same phellogen may become active early in the growing season every year and produce more cork and phelloderm; or a new phellogen may differentiate directly below last year's periderm and form a new layer of cork and phelloderm.

The whole of the outer surface of stems, branches and roots (but not leaves) of trees is protected by a periderm. The first periderm is formed directly beneath the epidermis, usually during the growing season when the tissue is formed. This first periderm, known as the **exophylactic periderm**, can renew itself for many years and "stretch" more than a hundred-fold. Bark covered by an exophylactic periderm is smooth and often it is green because the cortex tissues lying below it have some chlorophyll. The layers of cork on the outside usually peel off as the stem increases in circumference. A good example is the arbutus tree, which annually sheds a layer of cork. Sometimes the cork stretches and is retained, as in the case of birch and cherry, forming a tough outer layer.

The formation of a secondary or wound periderm (called the **necrophylactic periderm**) follows injury of any sort to the bark. It may also result from growth in circumference of the bole to the point where the exophylactic periderm can no longer "stretch" without breaking. The stages of formation are:

- formation, apparently by heavy lignification of cell walls, of a layer of cortex or phloem parenchyma cells that surround the wound and is impermeable to liquids and gasses;
- de-differentiation of a layer of parenchyma cells directly below that impermeable layer into a cork cambium (phellogen);
- formation of the periderm by the production of phellem and phelloderm tissues by the phellogen.

Both the phellem and phelloderm cells of a necrophylactic periderm differ in structure and chemistry from their counterparts in the primary or exophylactic periderm. Necrophylactic phelloderm cells often contain toxins in their vacuoles. Some have pigments, such as the red-purple pigments in *Tsuga, Abies,* and *Thuja.* The whole process of formation, including the initial impermeable layer, occurs only during the growing season. The speed of the process depends on host vigour and complete necrophylactic periderm formation may take from a few weeks to a whole season. Necrophylactic periderms are formed deep within the living bark, and the bark tissues to the outside die. These tissues together with old periderms within them form the **rhitidome**, the layer of dead bark on the outside of old stems. On some species, the older parts of the rhitidome are shed, and the bark remains relatively thin. In others, such as Douglas-fir, the rhitidome is retained, and may eventually become 25 cm thick. The appearance of old bark (scaly, fissured, or stringy) is attributable to the location and nature of necrophylactic periderms.

Necrophylactic periderm formation is the standard tree response to bark injuries of all types. The trigger appears to be the death or injury of phloem or cortex cells. Thus, trees react to cankers by forming periderm around the injured area, in response both to the wound that allowed the canker fungus to become established, and to the further death of bark cells as the pathogen spreads.

Types of Cankers

Cankers occur as three types, namely annual, perennial and diffuse. The type of canker that develops depends on the pathogen and host involved and the vigour of the host. Trees under stress are more susceptible to cankers, mostly because the process of periderm formation is much slower on such trees. Repeated light frosts during the growing season, nutrient deficiencies, and drought are common stress factors. Hence it is not surprising that cankers play a role in most decline diseases. The pathogens involved in those situations are typically weak parasites that are not found on healthy, vigorous trees, except on dying branches or twigs in the lower crown.

Annual Cankers

The pathogen penetrates and spreads for one dormant season. Then the host forms a periderm, which effectively isolates the infected tissues, so that there is no further spread. Annual cankers may result in some girdling of small twigs, but seldom do serious damage. Some remain superficial in the outer phloem or cortex without killing cambial cells; others penetrate to the cambium. In the latter case, a barrier also forms in the sapwood to isolate the wound. That type of barrier has been discussed in Lesson 2 on decay.

Perennial Cankers

Perennial cankers start like annual cankers, but during the second and subsequent dormant seasons, the pathogen is able to circumvent (through the sapwood) or penetrate the periderm barrier that has been formed during the preceding growing season and invade new bark tissue. The outcome is a slowly expanding patch of dead bark, often with

raised edges that result from a host callus produced every growing season. In some perennial cankers the boundary between live and dead tissue is not very distinct. Here the canker pathogen may live in the outer bark and/or in the sapwood directly beneath the vascular cambium, causing the cambium to become moribund, so that the annual rings produced by it are narrow, leading eventually to death of the vascular cambium.

Diffuse Cankers

In diffuse cankers the pathogen invades so fast that the place where the periderm is being initiated during the growing season is invaded and killed by the pathogen before the periderm is fully formed. Such cankers continue to expand during both the growing and the dormant seasons until they have girdled and killed their host.

Damage by Cankers

Types of damage caused by cankers consist of:

- bole deformation, leading to markedly reduced lumber recovery in the sawmill;
- stain and resin soaking of wood and bark at the canker (pulp chips produced from such material contain a great deal of resin-soaked bark);
- branch flagging caused by girdling of branches, which may lead to a general decline and dieback of the crown as in old red alder on the B.C. coast;
- decay entering through canker-killed bark;
- top kill resulting from girdling of upper branches and stems; and
- tree death.

Various Canker Diseases

In North America there are several serious canker diseases of hardwoods in the eastern hardwood forests. Prairie shelterbelts also suffer from several canker parasites. In western forests, however, there are only a few pathogens that cause serious canker problems. Aspen is particularly prone to canker diseases, with the most common cankers caused by *Hypoxylon mammatum* and *Nectria galligena.* Wounds on the lower bole, such as those caused by cattle, commonly become infected; the resulting cankers lead to deformation, staining, decay and death.

Damage caused by repeated growing season frosts, especially late or early frosts, often leads to canker development. Trees in frost pockets are attacked by weak canker parasites, such as *Cytospora kunzei,* acting as perennial and diffuse cankers. These pathogens enter through frost-killed tissues, and periderm formation in frost-damaged bark is slow and incomplete. The same species of pathogens occur in surrounding healthy stands, but only as insignificant annual cankers.

Drought stress and nutrient deficiencies can also lead to canker damage. The immediate cause of damage and decline in "off-site" trees (i.e., trees growing in an unsuitable site or provenance) is often a canker pathogen. Also, the kind of environmental stress that is predicted by the greenhouse effect will lead to an increase in canker diseases.

The most serious canker pathogen in North America is *Cryphonectria parasitica,* the cause of chestnut blight. This fungus was introduced from Asia via Europe around the turn of the century, and has destroyed completely the extensive and very productive eastern chestnut forests. This has resulted in a major change in the ecology of such hardwood forests, with the land now occupied by less productive hardwoods. Recently, a virus disease of the chestnut blight fungus has appeared. When *C. parasitica* becomes infected by that virus (a double-stranded RNA virus), it loses much of its pathogenicity and begins to behave like a normal perennial canker. Unfortunately, there is no efficient way of inoculating chestnut blight cankers with the virus, and natural spread of the virus is slow.

In B.C. the most serious canker pathogen is *Atropellis piniphylla* of lodgepole pine. The fungus causes long, narrow, sunken, resinous lesions on pine stems, usually below the living crown. The main damage is substantial reduction in lumber recovery and the inclusion of resin-soaked bark in pulp chips. The disease occurs most frequently in areas subject to occasional drought stress.

In forests on the North American east coast, Scleroderris canker of hard pines caused by *Gremmeniella abietina* is of considerable concern. This pathogen is often restricted to frost hollows, but more general infection is common in red pine plantations. There is also evidence that new, more virulent strains of this pathogen, possibly from Europe, have recently appeared. This pathogen also sets the northern limit for plantations of Scots and lodgepole pine in Scandinavia.

The general rule for avoiding canker damage is to keep plantations and stands in a vigorous condition. Careful provenance selection and avoidance of frosty locations are important. Tree species vary considerably in their ability to tolerate growing season frosts. For instance, in the B.C. interior, lodgepole pine is much more tolerant of such frosts than Douglas-fir, interior spruce, and subalpine fir, and may be regenerated in frost hollows where the other species would be seriously damaged. Overly dense stands may also be more susceptible. It may also be possible to increase resistance by genetic selection, but, for most species, progress along these lines has been very slow because many species do not become susceptible to cankers until they are quite old, and hence, testing for resistance is very time consuming.

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. Describe the steps in formation of an exophylactic periderm. If you wanted to examine such a periderm in the process of formation, where on a tree and at which time of year would you look?
2. Distinguish between the steps in the formation and the final structures of exo- and necrophylactic periderms.
3. List and distinguish between the three types of cankers.
4. How is stress related to resistance to cankers?
5. What are the main ways of preventing damage by cankers?