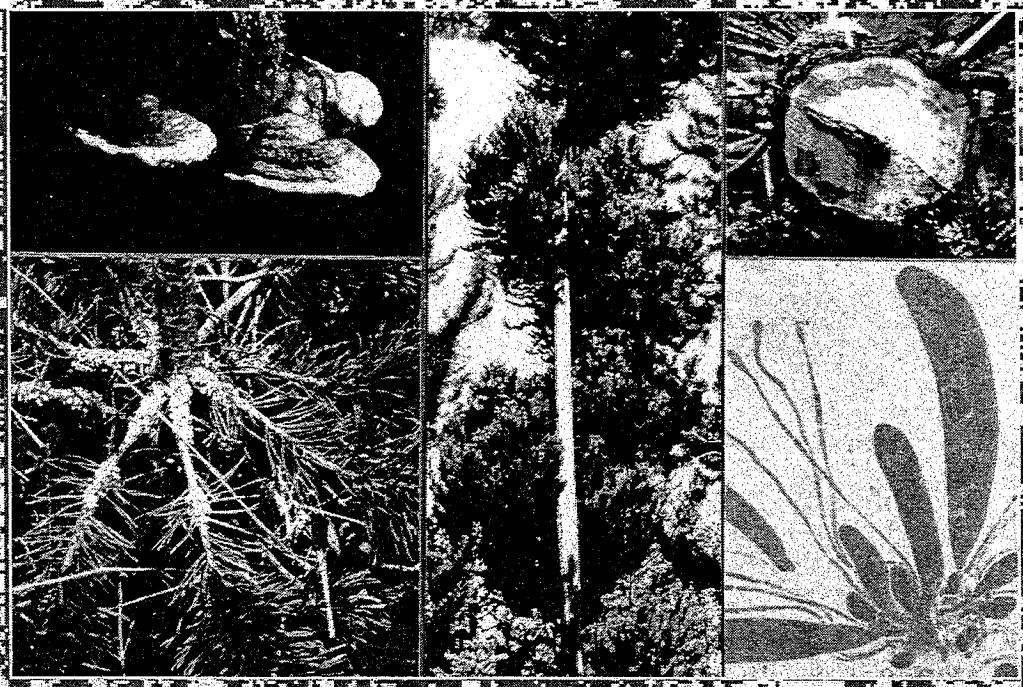
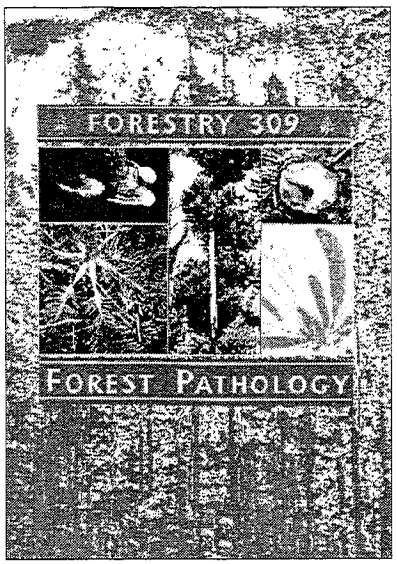
FORESTRY 309



FOREST PATHOLOGY

Distance Education and Technology
Continuing Studies
University of British Columbia



Forestry 309

Forest Pathology

FRST 309 Course Manual

B. J. van der Kamp

Distance Education and Technology
Continuing Studies
The University of British Columbia

FRST 309
COURSE MANUAL AUTHOR

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PRODUCTION CREDITS

Lorne E. Koroluk - Manual Design, Editing, Graphics and Cover

COVER PHOTOGRAPHS

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1. *Fomitopsis pinicola* fruiting bodies (photo by Lorne Koroluk)
2. Dwarf Mistletoe brooms on lodgepole pine (photo by B. van der Kamp)
3. *Phellinus weirii* stain on Douglas-fir (photo by B. van der Kamp)
4. Immature asci of *Rhabdocline pseudotsugae* (× 500) (photo by B. van der Kamp)
5. Multiple infections of *Cronartium comandrae* on lodgepole pine (photo by B. van der Kamp)

INTRODUCTION

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INTRODUCTION

Information about the Course

COURSE OVERVIEW

COURSE DESCRIPTION

FRST 309 (2.0 credits) Forest Pathology - Biology and management of forest tree diseases.

Course Prerequisites

Forestry 204 (or equivalent)

INTENDED STUDENT

The prospective student must have completed a course in forest ecology. That prerequisite, in turn, implies the further requirement of courses in soil science and dendrology. In addition, a course in climatology, with particular emphasis on microclimate, is highly recommended. Field experience in the coniferous forests of western North America will also be an invaluable asset.

Forest tree diseases affect both above- and below-ground parts of trees. Since diseases are always greatly influenced by the immediate environment, a good grasp of the soil environment and of microclimate is important for the understanding of diseases. A good grasp of normal tree form and function, such as presented in a dendrology course, is also essential before abnormalities resulting from disease can be understood. Finally, diseases play a major role in virtually all forest ecosystems, and their control is almost wholly achieved by ecosystem manipulation. Thus familiarity with ecosystem classification, succession, and ecosystem processes is essential both to understand how diseases affect these, and how, by appropriate manipulation, the impact of diseases on management goals can be minimized.

This course is designed for:

- students who wish to complete part of the requirements for a B.S.F. (Forest Resource Management or Forest Harvesting) or B.Sc. (Forest Sciences) degree through independent study;
- pupils within the ABCPF program who want to complete the course requirement in forest pathology; and
- any person who wants to learn more about forest pathology.

COURSE CONTENT

This course is divided into eight lessons. The first and the last two deal with general disease principles and concepts; the second through sixth deal in turn with the major classes of tree disease.

Lesson 1

Introduction to forest pathology - an overview of the discipline; a description of the major groups of disease causing organisms and their requirements, with special emphasis on the fungi; and a review of various climatic and other abiotic agents that can cause tree damage, and that predispose trees to disease.

Lesson 2 *Decay* - The role of decay in the carbon cycle; infection; factors that determine the rate of decay in living trees; white and brown rot decay at the chemical and microscopic level and their effect on wood properties; the role of decay in the creation of habitat for various organisms, and the implications of decay for forest management.

Lesson 3 *Root diseases* - The soil microbial environment; groups of root diseases; infection, spread, survival, identification, measurement, and management options for the major root diseases: *Phellinus weirii, Armillaria ostoyae, Inonotus tomentosus,* and others.

Lesson 4 *Wilt, foliage, bark, and seedling diseases* - Life cycles; vectors; requirements for infection and development; the phytoplane community; identification; epidemics; impact on trees and stands; management and control.

Lesson 5 *The rusts* - Life cycles and spore stages; identification of rusts; the white pine blister rust introduction and ways of dealing with that pathogen; the native pine stem rusts; foliage rusts; the cone rusts of spruce.

Lesson 6 *The dwarf mistletoes* - Life cycles; effect on hosts; species hosts, and distribution; mistletoe stand dynamics; dealing with dwarf mistletoe at harvest and in immature stands.

Lesson 7 *Inheritance of resistance* - Qualitative and quantitative resistance; passive and active resistance; selection and breeding strategies to produce stable resistance.

Lesson 8 *Forest pathology in the context of forest management* - Assessment of damage; the role of forest pathology in silviculture and management.

Course Objectives

This course is designed primarily for students who want to prepare for a career in forestry. It differs from a normal first course in plant pathology in that it does not attempt to give a complete survey of the field. Rather, the material selected for study consists of those aspects of tree disease that are most likely to be relevant to foresters making silvicultural decisions about stands of trees. Furthermore the course emphasizes diseases of the Western North American mountain and coastal coniferous forests.

Upon completion of this course you will:

1. be able to recognize and identify the common diseases of the Pacific coast forests;
2. be able to recognize and interpret disease signs and symptoms on forest and shade trees around the world; and
3. know enough about the biology and epidemiology of the common and damaging diseases of Pacific coast conifers to deal with them effectively by appropriate silvicultural prescriptions.

COURSE MATERIALS

The course materials consist of this manual, a required text book, and various supplementary reading and reference materials. Some of the supplementary reading materials are included in this manual, and others are available from the UBC Extension Library.

Textbook

The text book, available from the UBC Bookstore, is:

- MANION, PAUL D. 1991. ***Tree disease concepts.*** 2nd ed. Englewood Cliffs, NJ: Prentice-Hall.

Extension Library

Other useful reference materials are available from the Extension Library. Refer to your Student Handbook for directions on how to take advantage of the free Dial-a-Book service. Books available include:

- AGRIOS, GEORGE N. 1988. ***Plant pathology.*** 3rd ed. San Diego, CA: Academic Press.

  The standard North American plant pathology text. The emphasis is on agricultural crops. Presents the current understanding of all aspects of plant diseases. Almost all that is known about the physiology, biochemistry and genetics of host-pathogen interactions is based on studies of agricultural crops.

- BOYCE, J.S. 1967. ***Forest pathology.*** 3rd ed. New York: McGraw-Hill.

  This is the last edition of the old standard forest pathology text, with good descriptions of the diseases of western coniferous forests.

- SINCLAIR, W.A., LYON, H.H., & JOHNSON, W.T. 1987. ***Diseases of trees and shrubs.*** Ithaca, NY: Cornell University Press.

  This 574 page book is a complete compendium of North American tree diseases, providing good illustrations, short descriptions, and relevant references.

- ZILLER, W.G. 1974. ***The tree rusts of western Canada.*** Victoria, B.C.: Department of the Environment, Canadian Forestry Service Publication No. 1329.

  An exhaustive description of all the rust diseases of trees found in western Canada.

Supplementary Reading and References

The supplementary reference materials consist of several handbooks that contain descriptive and pictorial materials about tree diseases. You can obtain these by writing to the publishing agencies. When you do so, ask to be placed on their mailing lists, so that you will automatically receive updates and similar materials for the rest of your career (be sure to keep your address up to date).

The following contain essential study material for this course:

- ALLEN, E.A., MORRISON, D. & WALLIS, G. 1996. ***Common tree diseases of British Columbia.*** 3rd ed. Canadian Forestry Service, Pacific Forestry Centre

[*Address:* 506 West Burnside Road, Victoria, BC, V8Z 1M5]
This is the main source of illustrations for the course and an essential reference.
The address above for the Canadian Forestry Service - Pacific Forest Research Centre, applies also to the next three references.

- FUNK, A. 1981. ***Parasitic microfungi of western trees.*** Publication BC-X-222. Victoria, B.C.: Canadian Forestry Service, Pacific Forest Research Centre.

- FUNK, A. 1985. ***Foliar fungi of western trees.*** Publication BC-X-222. Victoria, B.C.: Canadian Forestry Service, Pacific Forest Research Centre.

- WOOD, C. 1986. ***Distribution maps of common tree diseases of British Columbia.*** Inf. Rep. BC-X-281. Victoria, B.C.: Canadian Forestry Service, Pacific Forest Research Centre. (68 pp)

- HIRATSUKA, Y. 1987. ***Tree diseases of the Canadian prairies.*** Information Report NOR-X-286. Canadian Forestry Service, Northern Forestry Centre.
[*Address:* 5320 - 122 Street, Edmonton, AB, T6H 3S5]

Selected pest leaflets are included in your course package. To remain up-to-date, you should get your name on the mailing list to receive notice of new leaflets and other publications:

- Canadian Forestry Service, Forest Insect and Disease Survey, Pacific Forestry Centre, 506 West Burnside Road, Victoria, BC, V8Z 1M5.

How to Proceed Through the Course

The lessons should be completed in sequence, finishing all the assignments in each before proceeding to the next. Directions are given for you to read and study the material in the manual and the relevant parts of the textbook. Some lessons require that you collect samples of plant diseases locally and describe them. All require that you examine the relevant illustrations in the text and supplementary materials carefully. When you have completed these activities, answer the self-testing/review questions. Finally complete the assignments which are required at the end of the second, fourth, and last lesson, and submit them for marking. Proceed to the next lesson as soon as each of these assignments is mailed.

Independent study requires self-discipline. You will find it helpful to progress at a steady pace in the course. If your personal circumstances allow, set aside a regular time and place to study. Monitor your course schedule, not just for assignment submission deadlines, but also as a check for steady progress. Note the date for sending in the application form for the laboratory session.

Course Requirements

Course requirements include three assignments that will be marked and graded, a collection of disease specimens from your local area, a two-day

laboratory session at UBC and a follow up assignment that will be graded, and a final examination. The session at UBC will consist of one day in the lab and one day in the field. That session will be scheduled about four weeks before the final examination, at a time when all lessons have been completed. Depending on class enrollment, it may be possible to hold the lab session at a location other than at UBC.

Marked Assignments

Directions on how to complete each of the four marked assignments are given in Appendix A. These assignments must be submitted to your tutor by the dates indicated on your course schedule. Don't forget to include an assignment comment sheet (on pink paper, supplied in your course package) for each submission. The first three assignments must be completed before you can take the laboratory session.

Specimen Collection

You will be required to prepare a collection of at least 20 samples of different diseases. The samples should be air-dried and stored in marked paper bags, or pressed and stored in envelopes. (Avoid using plastic bags because damp specimens may mould.) It should include at least one example of each of the following groups of diseases:

- a dwarf mistletoe
- a root disease
- a decay fungus
- a foliage disease caused by an Ascomycetous fungus
- a rust on bark
- a foliage rust
- a canker

Each specimen should be labeled with a tentative identification of the pathogen, host, name of collector, location and date of collection, and a brief ecological description of the collection site. ***The collection should be submitted at the start of the laboratory session.***

A sample layout of a 5" × 8" file card that shows the required specimen information appears on the next page. Feel free to make changes in the design of the card to make it more useful for your purposes.

Laboratory

A two-day laboratory session will be held about four weeks before the end of the course. The purpose of this session is to give you practice in recognition of diseases and in making correct diagnoses and appropriate prescriptions. One day will be spent in the field, examining a variety of diseases; there will be both demonstrations and exercises for you to work on. The other day will be spent in the lab looking at preserved specimens of many disease types. The final assignment consists of a report based on three of the diseases studied in the field part of the laboratory.

The exact location of the lab will be determined once enrollment is known. UBC is a likely place, but some Interior locations are also possible, depending on concentrations of student enrollment and the capability of a site to support the necessary lab activities.

Final Examination

The final examination will include questions based on all parts of the course, including the laboratory. A sample exam is illustrated in Appendix B. You will notice that questions range from quite specific to

PATHOGEN: NO.

HOST: DISEASE:

LOCALITY:

COLLECTOR: DATE:

REMARKS: CULTURE:

PHOTO:

DETERM. BY:

very general. The latter are designed to test whether you are able to select and synthesize, from among all the things you have learned in this course, those bits of information that are relevant to a problem.

GRADING

Your grade for this course will be based on the various assignments and the final exam as follows:

- Assignments (3 at 10%) 30%
- Specimen collection 10%
- Assignment 4 ..... 10%
- Final exam 50%

In order to pass the course, you must:

1. complete all four assignments;
2. submit a specimen collection;
3. attend the lab session;
4. obtain at least 50% on the final exam; and
5. obtain an overall average of at least 50%.

STUDENT-TUTOR COMMUNICATION

Upon registration, you will be assigned a tutor. The tutor will be available for telephone communication at specific times each week. You can call at those times free of charge. Please see instructions on the free telephone service in the Student Handbook. The tutor may also phone you, usually to discuss an assignment you have submitted (please send the tutor a completed student telephone form from the Student Handbook). You may also communicate by mail and by e-mail, and, if convenient, you may make an appointment to see the tutor in person at UBC. Your assignments will have comments by the tutor written on them when they are returned.

STUDENT EVALUATION OF THE COURSE

At the end of the course, a course evaluation form will be sent to you for completion. This is an opportunity for you to express your opinions on course content and presentation, administrative procedures, and delivery method. Your responses will be of great value in improving the course for future students.

LESSON 1 Introduction to Forest Pathology

LESSON OVERVIEW

CONTENT

This lesson serves to introduce you to many aspects of the study of forest tree diseases. It is organized into three sections. The first gives a brief overview of the discipline of forest pathology. We start by asking why you would want to know about tree diseases. The answer to that question determines to a large extent how the whole field is approached. We then introduce some of the terminology that is used when speaking about diseases, and explain how we can determine that a certain condition is in fact a disease.

In the second section we deal with the fungi, the group of micro-organisms that is responsible for most tree diseases. Habitat requirements, reproduction, and fungal classification are discussed. We also discuss bacteria and viruses as disease-causing agents.

In the last section we deal with the major abiotic causes of tree damage and disease. These include frost, drought, heat, and flooding. This list could be extended to include air pollution, pesticide damage, and problems in tree nutrition. These subjects, however, are dealt with in other courses, although you will see that they are very relevant to a group of diseases known as "declines," the last topic in this section.

READING

Specific reading assignments are suggested in each section.

LESSON STUDY INSTRUCTIONS AND ASSIGNMENT

Complete each section in turn, answering the self-testing/review questions associated with each before proceeding to the next.

In this lesson there is no assignment to be submitted for marking.

COMMENTARY

SECTION 1

FOREST TREE DISEASES

reading

Start by reading Chapter 1 of Manion (1991) "Tree Disease Concepts," then study the material that follows in this manual. To test your understanding of the material in this section, answer the self-testing/review questions before proceeding to the next section.

WHY STUDY TREE DISEASES?

The most common reason that we study tree diseases is that they are often very damaging. You have already read in Manion how certain valuable species such as the American chestnut and several white pines are no longer commercially used in many areas because of introduced diseases. Many of our native diseases are also responsible for very large losses of wood. Hence it is not surprising that the most common definition of forest pathology is: "*The study of forest tree diseases for the purpose of predicting, and preventing or minimizing damage done by such diseases.*" Notice the practical slant in the definition: the interest lies in prevention or reduction of damage, and that will be the emphasis in this course.

Two important points, however, need to be considered before proceeding. First, the type and degree of damage caused by a disease is determined largely by the *purpose of management:* a disease that is detrimental in one situation may be neutral or even beneficial in another. For instance, dwarf mistletoes result in a marked reduction in growth of infected stands, and such infection is very common in western coniferous forests. In parks or areas used for watersheds, however, that may not matter in the least. The landscape remains forested, and the purpose for which the area has been set aside is well served, and hence there is no damage in such situations.

The second point is that most pathogens are integral members of our forest ecosystems. They play their own particular and, sometimes, important role in ecosystem processes, such as nutrient cycling. Sometimes they direct the course of succession. In fact, it is slowly being recognized that pathogens may produce substantial benefits, largely because they act as major agents or causes of diversity in forested landscapes. In order to make the best forest management decisions then, both the damage and possible potential benefits must be considered.

Nevertheless, mitigation of damage remains an important reason for the study of tree diseases. Reduction of damage, however, does not necessarily imply that pathogens must be eliminated. The presence of pathogens does not matter so long as damage is avoided.

The techniques used to reduce damage consist, in the main, of making the environment unsuitable for disease, or of avoiding the use of tree species in ecological situations in which they are susceptible to disease. Hence forest pathology must be understood as a *branch of*

silviculture. Mitigation of damage by disease must be integrated with all the other objectives of silviculture, or else it will not succeed.

Notice that this course is called *forest* pathology, not *tree* pathology — this underscores our focus on the role and management of diseases at the level of forest stands or ecosystems. An understanding of how diseases develop on individual trees is necessary, but we extend this interest in a larger perspective. This course will not deal much with diseases of shade trees for two reasons: first, the value of individual shade trees greatly exceeds that of forest trees, and hence it is possible to use control techniques that are not economically viable in forests; second, many shade trees are planted well outside their native range, and all of them occur in greatly altered environments — hence environmental stress plays a much greater role in shade tree pathology than in forest pathology.

If forest pathology is concerned with preventing damage, and if damage occurs only if goals of management are not met because of disease, then the goals of management need to be clearly stated, or it will not be possible to design and implement useful disease prevention and control measures. If we don't know what we are trying to achieve by our interventions in the forest (i.e., if goals are not clearly stated), it will not be possible to determine whether any particular intervention is good, bad, or indifferent.

In discussing goals, though, we touch on a central problem in North American forest management: in the past, the goal was simply to produce quality timber without loss of the long-term productive capacity of the land. Elsewhere in the world (for instance in China or in the subtropical plantation forests of Eucalypts and radiata pine) that goal is still overriding, although in these areas of the world, as well as in North America, concerns are being expressed about the environment and other forest values. Debate rages on about what can, might, or should be the goals of temperate and boreal forest management, and I won't add my opinions on the matter (after all, this is a course in forest pathology), but we must recognize that the outcome will also define the practice of forest pest management

In the course of those debates, the issue of forest health in natural and managed forests is often raised. Some have said that all disease troubles arise from human intervention, and that in perfectly natural forests trees are vigorous enough to withstand the onslaught of disease. In this view, disease is seen as a symptom of inappropriate human intervention in natural forest ecosystems. As you study the material in this course, you will see that this is an overly simplistic view, and, in fact, quite mistaken.

A more subtle issue arises from the use of the word "health" (as in "healthy forests"). A decade ago, the term denoted a forest that was essentially free of diseases and insects, or at least one in which diseases were not damaging. Since that time it has been widely recognized that diseases play a role in natural ecosystems. The term "healthy forest" is

now sometimes used in its old way, but at other times is used to denote a forest in which diseases play their natural role. That natural role can include significant losses in volume and value. If the definition of a healthy forest is simply a forest in which diseases play their natural role, then "natural" and "healthy" are synonymous, and the word "healthy" is devoid of any specific meaning. I believe that debates about the proper use of forests are poorly served by such an ambiguous use of the term "forest health," and that it is best to avoid the term altogether.

Definitions of Terms

In preparation for discussion of the course material, keep in mind the following definitions of terms commonly used in the study of tree diseases:

pathogen — An organism able to cause disease. Hence *pathogenicity* – the ability to cause disease, and *pathogenic* – disease causing.

disease — A chronic or fatal malfunctioning of one or more parts of a plant as a consequence of invasion by foreign organisms or from unusual environmental conditions, resulting in reduced growth, death, malformation, or loss of quality. Note the distinction between *pathogen* and *disease*: the former is the cause, the latter is the result.

etiology — The development of a disease over time, from the viewpoint of the host. Thus the etiology of a disease is a description of how the various symptoms and signs develop over time.

saprophyte — A micro-organism (e.g., decay fungus) that lives on dead organic matter.

parasite — An organism that lives on a live host for at least part of its life cycle. Two types to note are obligate and facultative.

obligate parasite — A parasite that is confined to living tissues except for inactive resting stages. These parasites include rusts, dwarf mistletoes, mildews and viruses.

facultative parasite — A parasite that is able to survive, grow and reproduce on both living and dead tissue. Sometimes the host tissue is killed just ahead of the advancing pathogen (as in the case of many of the bark and root pathogens); in other cases, the host tissues die some time (weeks to months) after invasion, either at a time of stress for the host or when the pathogen begins to reproduce (as with many needle diseases).

sign — Visible (macro or micro) evidence of the pathogen itself, usually its reproductive structures.

necrosis — Death of tissue.

symptom — An abnormal host condition resulting from the attack by the pathogen. Common symptoms include resinosis, chlorosis, hypertrophy, hyperplasia and brooming. Many other symptoms, some obvious and some subtle, might present. Note that symptoms can refer both to conditions on individual trees and also to stand level phenomena, such as group dying in root disease openings.

resinosis — A symptom wherein excessive resin production results in resin soaking tree tissues as well as resin exudation.

chlorosis — A symptom that presents as yellowing of foliage.

hypertrophy — A symptom in which there is swelling or gall formation that occurs because of abnormal increase in cell size.

hyperplasia — A symptom that presents as swelling or gall formation as a result of an abnormal increase in cell number.

brooming — A symptom that shows up as a production of abnormal clumps of branches.

The parasitic habit of pathogens needs some comment. For almost all of them, some time elapses between initial invasion and the appearance of the first signs or symptoms. This is called the **latent period.** Those that can live for long periods within their host without causing any symptoms are called **endophytes.** Many facultative parasites invade and spread in their host until that host is killed. At that time, newly dead host tissues that are not already occupied by the parasite are quickly invaded by a large array of saprophytes. These saprophytes are usually strong competitors that prevent further spread of the original facultative parasite. Sometimes the original facultative parasite is replaced quickly. More often, it builds a protective layer around the domain that it occupied at the time of death of the host and survives within that domain for many years

THREE GROUPS OF DISEASES

Tree diseases can be divided into three broad groups. The first of these consists of diseases caused by **abiotic agencies,** such as frosts or toxic substances. The damage done by such agents may result in subsequent invasion and further damage by various weak pathogens, but plants usually recover if the abiotic cause is removed. The resulting disease, that is, the characteristic set of signs and symptoms, usually consists of the damage done by the abiotic agent, modified by subsequent pathogen invasion.

A second group of diseases is caused by the **invasion of pathogens** which disrupt normal plant processes. Particular environmental conditions are usually required for infection and/or disease development, but such environmental conditions are not, by themselves, damaging.

The last group of diseases is known as **declines.** Symptoms of declines usually include reduction in leaf area and reduced growth, developing into dieback of the crown and eventually tree death. Typically, a single biotic or abiotic cause cannot be identified. Rather, declines appear to result from a combination of chronic stress factors such as drought, nutrient stress, atmospheric pollution, all persisting over several years. If trees under such stress are then exposed to an acute stress factor such as frost or other severe climatic condition, they are unable to respond in the normal fashion, and begin to decline. Various weak fungal parasites, which normally do not affect the tree species in question, can then invade and speed the decline. Various combinations of such adverse events, in different orders, can all lead to the same decline condition in a particular host species.

Declines and common abiotic agents of disease are discussed in Section 3 of this lesson. The common biotic agents that cause disease include viruses, bacteria, fungi, nematodes, and vascular plants. Of all these, the fungi constitute by far the largest group of tree pathogens. Section 2 deals mainly with the fungi, although bacteria and viruses are also discussed.

There are literally thousands of species of fungi that occur naturally in forests. Some play essential roles, such as the mycorrhizal fungi. Others colonize and digest all kinds of dead organic materials. Living plant tissues are seldom sterile. The external surfaces (the **phytoplane**) are colonized by communities of micro-organisms shortly after they are formed. One can also isolate micro-organisms from within healthy, living tissues. Diseased tissues usually harbour large numbers of micro-organisms. Hence, very important questions arise: How do we distinguish between pathogens and other micro-organisms? How do we determine that a particular micro-organism is responsible for the disease being considered? The latter question can be answered by following a series of four experimental steps, which together are known as "**Koch's postulates.**"

Koch's Postulates

1. The suspected pathogen must be present whenever the disease appears. (Remember that a disease is defined by a set of symptoms and signs.)
2. The pathogen must be isolated in pure culture, identified and characterized.
3. The pathogen must be inoculated on healthy plants of the same species and variety, and produce the typical disease symptoms on these. Controls are very important here.
4. The pathogen must be re-isolated from such inoculated plants and shown to be the same one that was used to inoculate them.

These simple rules suffice in many situations to identify the pathogen responsible for a particular disease. Things will often be more complex, however. For instance, obligate parasites cannot be isolated in pure culture, thus requiring a modification of the rules. Even more important

is the fact that Koch's postulates ignore the role of the environment. Thus a disease may be "caused" by a certain pathogen, but only under special environmental conditions (such as growing season frosts). What then is the cause?

Most pathogens have a tremendous reproductive potential. Fungal spores are produced in large numbers and deposited on all plant surfaces. The mere presence of a pathogen, however, does not mean that a disease will occur. Usually conditions necessary for infection are limiting. Since most pathogens are present all the time and everywhere in the forest, management of diseases is usually aimed at creating an unsuitable environment for them. In the case where vectors play a role, control can sometimes be achieved by making the environment unsuitable for the vector.

All this can be summarized by the "disease triangle" (see Fig. 1.1). The three vertices of the triangle all interact, and that interaction must be understood as having a time dimension. Thus the environment affects the condition of the host in both the long and the short run. The long run determines such things as tree vigour and carbohydrate reserves, and these in turn affect the way that the host reacts to short term conditions such as droughts, as well as its ability to mount effective defense reactions. The environment affects the pathogen in similar ways and, in addition, very specific conditions are usually required for spore release, dispersal and infection. Since the pathogen normally lives on the host, the condition of the host may affect the spore-producing ability of the parasite. Much of this course consists of elaborating the important interactions for specific diseases among host, pathogen and environment.

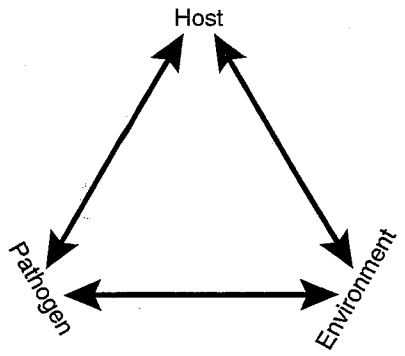

FIGURE 1.1
The disease triangle.

It is helpful to expand the disease triangle to a three sided pyramid, in which the fourth vertex represents humans (see Fig. 1.2). This calls attention to the fact that human actions have at times had a profound influence on host, pathogen and the environment in ways that change the interactions between them drastically. For instance, the creation of fully stocked, single species plantations shortly after clearcutting/stand destruction promotes survival and spread of certain root diseases, and the consequent damage they cause. At the same time, of course, disease epidemics have played an important role in human civilization, showing that the relationships operate both ways.

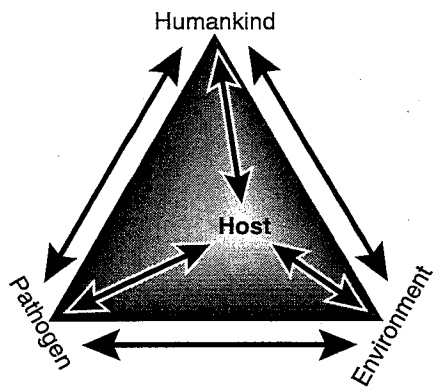

FIGURE 1.2
The disease pyramid.

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. What is the difference between a disease and a pathogen? Does the first always require the second?
2. What, approximately, is the loss in wood volume in Canada attributable to diseases in relation to the annual harvest?
3. What is the relationship between control of diseases and silviculture?
4. Give some examples of diseases that are damaging in some management contexts, but not in others.
5. List some essential functions of fungi in forest ecosystems.
6. List Koch's postulates. Why are they necessary to establish pathogenicity of an organism?
7. Give an example of relationships along each of the six edges of the disease pyramid.

SECTION 2 PATHOGENS: FUNGI, BACTERIA AND VIRUSES

reading

Read Chapters 6, 7 and 8 of Manion (1991) that deal with viruses, bacteria and fungi, respectively.

Because most pathogens of western forest trees are fungi, you need to be much more familiar with these organisms. As you read Chapter 8, examine the various illustrations carefully, especially those dealing with reproductive structures. Make sure you can relate the photographs to the diagrams for the various groups.

Study the material in the text, then read the discussion that follows in this manual. To test your understanding of the material in this section, answer the self-testing/review questions before proceeding to the next section.

VIRUSES AND BACTERIA

The two groups of pathogens, viruses and bacteria, cause many serious diseases in various agricultural crops, but they are not known to cause serious diseases in west coast coniferous forests. However, as you study the chapters in Manion, you will discover that there is considerable uncertainty about this. Particularly for the viruses, the problem may be that no one has actually looked carefully enough in coniferous trees to detect them.

FUNGI

Structure

Because of the relatively greater importance of fungi in forest tree diseases, we will discuss them in more detail. The basic body structure of most fungi is the **hypha** (plural, **hyphae**); a microscopic tube consisting of a wall of chitin (usually), surrounding a living cell. Hyphal diameter varies, commonly within the range of 1 to 10 microns (average of 3). But for some species or stages, hyphal diameter can be larger or smaller. Hyphae are often divided into segments (cells) by cross walls called **septa** (singular, **septum**). These septa may have special pores in their centers that allow movement of materials from segment to segment. Hyphae grow only at their tips; they do not "stretch" once formed. Branches may arise at the tips or, more commonly, at septa. The collective term for the mass of hyphae constituting a fungus is **mycelium** (plural, **mycelia**). Individual hyphae cannot usually be seen with the naked eye. Masses of hyphae may form white or variously coloured sheets or strands.

Most of the cytoplasm (and hence most of the metabolic activity) occurs at the growing tip. Further back, hyphal cells consist of large vacuoles. Thus the description of fungi as "simple animals in a tube" helps to visualize the manner in which fungi function. However, some fungi (e.g., yeasts) do not produce hyphae, but grow as single cells surrounded by a cell wall.

Requirements for Growth

All fungi are heterotrophs; since they lack chlorophyll, they must obtain their energy from organic materials, such as carbohydrates, fats and oils, produced by green plants. Most fungi require oxygen, though not neces-

sarily in atmospheric concentrations. Some, such as yeasts, can grow anaerobically. All the normal mineral elements (N, P, K, Ca, Mg, Mn, S, Fe, Cu, B, Mo, Zn, and perhaps others) required by living organisms are also required by fungi, mostly in simple inorganic soluble form. Most fungi can grow provided these minimal requirements are supplied in a suitable aquatic environment. Some fungal species have special requirements for certain amino acids, vitamins, and other growth factors.

Habitat

Hyphal fungi cannot ingest solid materials. All the above requirements must be met by absorption through the fungal cell wall and the outer plasma membrane. Hence all such materials must be dissolved in water. Also, wastes are eliminated in the surrounding aquatic medium. Thus fungi are essentially aquatic although parts of a mycelium, particularly reproductive structures, may grow in air. Such parts get their nutrition by translocation along hyphae from parts of the mycelium that are in contact with water.

Since the substrate (e.g., wood) is often insoluble, fungi must produce extracellular enzymes to digest such materials. Enzymatic function is strongly affected by pH. The three-dimensional structure of proteins changes with pH, so that enzymes are active over a relatively narrow pH range, (reversibly) inactivated over a wider pH range, and permanently denatured at extreme pH levels. Hence there is a requirement for a fairly narrow pH range (about 4 to 6.5 for many fungi, but either higher or lower for certain specialized ones). Of course, if energy sources and minerals are supplied in soluble form, a fungus is much less sensitive to the pH of the aquatic medium. The pH within the cytoplasm is very closely controlled by the fungus, and so long as energy sources are sufficient, it can be maintained even if the pH of the immediate environment is different.

Most fungi occurring in the forest are low-temperature organisms. Many become active just a few degrees above 0°C, and reach their maximum rate of growth between 20 and 30°C. Temperatures in excess of 40°C are often lethal. Not surprisingly, different fungi have different optimum temperature ranges; different species, and even different isolates of the same species, are often adapted to the conditions (warm or cold) from which they have been isolated.

Ultra-violet light in sunlight is destructive to all living things, and those that are exposed to sunlight have various protective devices. Vegetative fungal hyphae, as a rule, do not have sunlight protection (they are too small to have the required pigments in sufficient quantities), and hence they cannot grow in locations exposed to full sunlight.

To summarize, fungi have the following requirements for vegetative growth:

- an energy source;
- a supply of mineral nutrients;
- oxygen;
- a suitable temperature;

- protection from ultraviolet light; and
- liquid water at a suitable pH.

Some have additional requirements for special organic compounds, such as amino acids or vitamins.

Reproduction

Fungi reproduce both sexually and asexually. Asexual reproduction can occur by fragmentation of hyphae and by spores. Living hyphal fragments can be dispersed in soil particles or organic materials and establish new fungal colonies at distant locations. This is the only known method of reproduction for a few fungi. Asexual reproduction by spores is much more common. Asexual spores that are produced at the tips of hyphae, either singly or within specialized structures, are known as **conidia.** In some groups of fungi, asexual spores are formed within a container-like structure (the sporangium) and released at maturity. Such spores are called **sporangiospores.** Asexual reproduction can be very quick. A conidium landing on a suitable substrate can germinate, penetrate, and establish a new fungal culture that begins to produce more conidia, all within a week. Thus a single spore can lead to the production of millions of new spores within a month. This tremendous reproductive potential makes a mockery of attempts to control many diseases by sanitation (although, as we shall see, there are some exceptions). Generally, control is most often achieved by manipulating the environment so that it becomes less suitable for the pathogen in question.

Many species of fungi reproduce only asexually. Others reproduce both sexually and asexually, or only sexually. A discussion of sexual reproduction requires a look at nuclear cycles. In most fungi the nuclei in **somatic** (i.e., vegetative) hyphae are haploid. In some groups these haploid nuclei may occur in pairs, called **dikaryons,** consisting of nuclei of compatible sex types. **Karyogamy** (fusion of nuclei to form a diploid) and meiosis usually occur in rapid succession within specialized structures, but sometimes there may be a significant diploid stage. Thus there are three separate phases in a fungal life cycle, namely the haplophase, the dikaryophase, and the diplophase. These stages are separated by the processes of **somatogamy** (fusion of haploid hyphae to form a dikaryon), karyogamy, and meiosis, respectively.

Sexual reproduction results, among other things, in the recombination of genetic material. However, that recombination can also occur within somatic hyphae via what are known as **parasexual processes.**

In those fungal species that exhibit regular sexual stages involving meiosis, there may be one sex (so that karyogamy can occur with identical nuclei, although most often it will in fact involve different nuclei); two sexes (in which case there may or may not be morphologically distinguishable sexes — if not, the sexes are called + and –); or more than two sexes. In some decay fungi for instance, if one starts with four different haploid mycelia A, B, C and D, dikaryons can form in all combinations: AB, AC, AD, BC, BD and CD, but not as AA and so on. (If

A is male and B female, what then is the sex of C and D?) The genetic explanation is that the locus that determines sexual compatibility has many alleles, and that compatibility requires only that the two alleles be different. This is known as bipolar sexuality. In tetrapolar sexuality there are two loci, and again several to many possible alleles at each of these. Compatibility occurs when the alleles at both loci differ. The advantage of this arrangement is that almost any pair of randomly selected haploid mycelia can fuse, form dikaryons, and eventually reproduce sexually, rather than only half (or, in the case of tetrapolar species, one quarter) of such pairs.

Taxonomy and Nomenclature of Fungi

Taxonomy is the process of dividing living organisms (in this case the fungi) into a hierarchical system of groups, the basic group being the species. Species are then grouped into genera, genera into families, and so forth. **Nomenclature** is the process of naming such groups. A valid Latin name of a species consists of three parts: the **genus**, the **specific epithet** (the species name), and the **author** who described and named the species and placed it in that genus. There are strict international rules of nomenclature. These include rules of priority (the name given by the first person to describe the species is the valid one), rules about the allowable form of names, and so on. Sometimes changes in Latin names are mandated by the rules of nomenclature. It is not uncommon, for instance, that two fungi, described by different names in different parts of the world turn out to be one and the same species. When that is discovered, the earlier name applies, and the later name becomes invalid.

Taxonomy, however is not governed by a set of explicit rules. Rather, it follows to some extent current scientific fashions, but more important, as our understanding of fungal structures and function improves, it is often realized that old groupings are artificial and that new, more meaningful arrangements are possible. There is no doubt that there are natural groupings of fungi (taxonomy isn't an arbitrary process), but no taxonomic system captures that grouping completely, in part because we don't know enough about the fungi to see all the relationships. And so situations commonly arise where there are two or more valid Latin names for the same species. A good example is found on pages 261–262 of the text which discusses classification of decay fungi. Sometimes two different taxonomists working on the same group of fungi end up with quite different ways of dividing them into genera and species. When this happens, there are two valid Latin names for each of the species involved. Confusion is avoided by listing the author. When one uses such a name, one in fact says: "The fungus I am speaking of is the one named *abc* by *xyz*." Such a situation is not a case of one being right and the other wrong; the differences arise because the criteria to be used to define species, and to group species into genera are not (and probably cannot be) unambiguously specified. Different taxonomists use such criteria in somewhat different ways, and consequently end with different names. The issue is usually decided by popular demand — the majority

of scientist will use one of the two possible names, and eventually the other is more or less forgotten.

How does all this affect the forest practitioner? First, we have to live with the situation, and (hopefully) understand why some species have synonyms (two or more valid names). Since practitioners are seldom in a position to make a judgment about the issues involved, they will typically use the name that is commonly used in their region. However, when accessing the literature on a species, it is important to search under all the names, both old and new, popular and unpopular. For instance, the pathogen that causes Annosus root and butt rot was first described by Fries as *Polyporus annosus* Fr. in 1821. In 1881 the Finnish mycologist Karsten placed the species in his new genus *Fomitopsis*, and the name became *Fomitopsis annosa* (Fr.) Karst. However that name (and that system of classifying the Polypores) never become popular and was seldom used. Instead the name *Fomes annosus* (Fr.) Cke., first published in 1885, was almost universally adopted and used for a century. More recently the whole group of Polypores has been re-examined and divided into new genera, and that recent revision has now been widely accepted. Hence the name in common usage today is *Heterobasidion annosum* (Fr.) Bref.

Classification of fungi is based largely on the microscopic structures associated with sexual reproduction. For those that do not form sexual stages, the asexual stages are used. Fungi that produce both sexual and asexual stages are therefore sometimes known by two names, one for each, although in such cases, the name for the sexual stage is always the valid name for the species. For such fungi, the sexual stage is known as the **teleomorph** and the asexual stage as the **anamorph**. For the purpose of this course we will recognize five major subdivisions in the Kingdom Fungi. Below is a simple key to these; a more detailed one is presented on pages 121–122 of Manion (1991).

I Hyphae aseptate, asexual spores borne in endogenously in sporangia.

A. Male and female sexual structures dissimilar; asexual spores have flagella, and are dispersed in water.

(Class) Oomycetes

B. Male and female sexual structures indistinguishable; asexual spores nonflagellate and usually dispersed in air.

(Class) Zygomycetes

II Hyphae septate, asexual spores borne on hyphal tips

A. Sexual reproduction involves the formation of a basidium bearing haploid basidiospores.

(Subdivision) Basidiomycotina

B. Sexual reproduction involves the formation of an ascus containing (usually) eight haploid ascospores.

(Subdivision) Ascomycotina

C. Sexual reproduction does not occur (or is as yet undescribed)

(Subdivision) Deuteromycotina or **Fungi Imperfecti**

I A Oomycetes

This group contains several parasites of young roots and leaves of trees or agricultural plants. Life cycles usually consist of a brief asexual cycle (10 days from infection to spore production) and an annual sexual cycle usually involving a resting stage to tide the fungus over unsuitable conditions.

I B Zygomycetes

This group, mostly saprophytes, contains the vesicular arbuscular (VA) mycorrhizae.

II A Basidiomycotina

This is a homogenous group characterized by the production of a **basidium** (plural, **basidia**) bearing four external **basidiospores** at maturity. These basidia are usually organized in a layer called the hymenium which lines the gills of mushrooms or the pores of bracket fungi. This class contains most of the decay fungi, most of the important root diseases, and a special group of obligate parasites, the rusts. Most ectotrophic mycorrhizal fungi also belong to this group. Basidiospores are usually small, thin-walled, and therefore short-lived and easily killed by adverse conditions. On the other hand, they are produced in very large numbers.

II B Ascomycotina

This is a homogenous group characterized by the production of an ascus (plural, **asci**) containing (mostly) eight **ascospores.** These asci are arranged in various macroscopic structures. Most pathogens of foliage and bark, wilt diseases, and a few decay fungi belong to this group. Some species reproduce only sexually, and those usually have a short spore-producing period each year; others produce both sexual and asexual spores, in which case the latter usually consists of a brief cycle summer stage while the former is produced early in the season following a dormant over-wintering period. Ascospores are usually large and thick-walled. They can be carried long distances under adverse conditions.

II C Deuteromycetes

This group contains all the fungi in which sexual reproduction does not occur or is unknown. Thus the Deuteromycetes are an artificial group in the sense that it contains many species that clearly are closely related to species in the Ascomycotina in terms of their asexual reproductive structures as well as their physiology. It contains several pathogens of foliage, bark, and a variety of other tree tissues.

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.

A. Viruses

1. What are the basic components of a virus particle?
2. How are viruses classified?
3. What is the host specificity of viruses?
4. What are the common symptoms of virus infection?
5. Distinguish between local and systemic infection.
6. How are viruses transmitted from plant to plant?
7. Describe the ELISA technique as a sensitive assay for viruses.

B. Bacteria

1. List the major differences between the prokaryotic and the eukaryotic cell.
2. What types of plant tissue are most easily invaded by bacteria?
3. What are FXLBs and MLOs?
4. How are bacterial diseases transmitted?

C. Fungi

1. Fungi are essentially aquatic organisms. Explain.
2. Distinguish between taxonomy and nomenclature, and explain why a particular fungal species may have more than one valid Latin name.
3. How do fungi obtain their energy supply? Their essential nutrients?
4. Define and explain the following terms: *somatogamy, karyogamy, meiosis, diplophase.*
5. Sketch a typical ascus, basidium, apothecium, cleistothecium, perithecium, conidiophore bearing conidia, and a pycnidium.

SECTION 3

DAMAGING INORGANIC AGENTS

Trees can be damaged or killed by insects, pathogens, and other living or inorganic agents. Causes of damage or death can be confused easily — dead trees don't bear signs labelled "insect kill." Careful attention to signs and symptoms is often required to establish the cause of death or damage to trees. A discussion of inorganic agents is appropriate here because: (1) damage caused by such agents is often mistaken for diseases and vice versa; and (2) inorganic agents often predispose trees to attack by pathogens and insects.

reading

Start by reading Chapters 2 and 3 of Manion (1991). Following that, read the discussion that follows in this manual, then the article by Houston (beginning on p. 23). To test your understanding of the material in this section, answer the self-testing/review questions before proceeding to the next lesson.

FROST

Frost plays a major role in northern forests, particularly during regeneration stages. Frost damage is manifested in a variety of symptoms. Frost hardiness of trees varies. Boreal forest tree species can withstand much lower temperatures than those from farther south, and within a species, northern provenances show greater tolerance of low temperatures than provenances from warmer parts of the range of the species.

It is important to distinguish between frost tolerance of dormant trees and that of actively growing trees. Dormant trees may become very frost hardy. The same trees in active growth may be severely damaged by rather light frosts. In fact, trees in active growth are always susceptible to frosts. So, there are two ways in which trees avoid frost damage. One applies in winter, and involves the development of deep frost hardiness while the tree is dormant. The other involves the timing of bud flush in the spring and dormancy in the fall.

Frost Cracks

Mature trees in frosty locations often bear radial cracks which, by growth ring analysis, can be shown to have occurred during the dormant season. Rapid cooling of the outer portions of tree boles that has resulted in shrinkage around a warmer core has been implicated. Alternatively, during freezing weather, water may be withdrawn from cell walls and frozen onto ice crystals in the tracheid lumens, resulting in greater tangential than radial shrinkage (analogous to drying) and cracking. Once a crack is formed, a tree will attempt to heal it over by callus formation, but the weakness in the wood will result in regular opening of the crack during subsequent cold periods. Frost cracks provide entry to the heartwood for decay fungi, and the resulting decay may represent the greatest damage. However, lumber recovery from trees bearing several frost cracks is also greatly reduced.

Frost and Living Tissues

When living, dormant and frost hardy tissues are cooled below their freezing point (a few degrees below 0°C because of dissolved materials), ice crystals begin to form. The inter-cellular water freezes first because it is less concentrated than intra-cellular liquids and thus has a higher freezing point. If the temperature continues to drop slowly (i.e., at less than about 6°C to 10°C per hour), water moves out of the living cells to freeze onto these ice crystals. As water moves out, the concentration of various dissolved compounds within the cell rises, and the internal cell solution remains liquid. As temperatures continue to drop below –10°C, various chemical changes to membranes and proteins occur to allow them to retain their structural integrity, and antifreeze-like chemicals accumulate in the cell solution, preventing the formation of intracellular ice crystals. Actively growing tissues cannot respond in this way, and they are therefore killed by frosts of a few degrees below zero.

Rapid freezing leads to the formation of intracellular ice crystals, and kills even frost hardy plant tissues because such ice crystals destroy cell membrane structures. Bark and meristem tissues of many northern woody plants in their dormant state can tolerate temperatures as low as that of liquid nitrogen (–196°C).

In some tissues, such as xylem ray cells, liquid water may supercool (drop below its freezing point without ice formation) to temperatures as low as –40°C. When sapwood xylem ray cells do freeze, they are killed, and subsequent transformation of that sapwood into heartwood does not occur, resulting in **target ring** (bands of included sapwood within the heartwood, as commonly seen in interior Douglas-fir and red cedar).

Dormant above-ground tissues of northern trees growing in their natural range can tolerate very low temperatures so long as the rate of cooling is slow. Roots do not acquire great frost hardiness and may be killed at temperatures ranging from –10°C to –20°C depending on species and provenance. Severe fall frosts at a time when there is no adequate snow cover may sometimes kill fine roots near the soil surface. Container grown nursery stock that is not adequately protected by placement of styroblocks on the ground has also been killed in this way. Tops may remain green for a while, but the roots are dead.

Dormancy and frost hardiness are not identical. Plants go dormant in fall before they acquire full frost hardiness. Furthermore, the term "dormant" is misleading if it is used to indicate lack of activity. Dormant plants do not grow (there is no cell division), but they are metabolically active, particularly with regard to protein synthesis, presumably to allow for repair of damage.

Most damaging are the early (fall) and late (spring) frosts which occur when trees are not yet or no longer frost hardy. In the case of early frosts, the stem segments formed that year may be killed while older parts of the tree survive. This may not be evident until the following spring when buds fail to flush. Late frosts kill the flushing shoots. Growing season frosts usually occur during clear windless nights via radiation cooling and cold air pooling in low lying areas. Such frost

pockets can often be identified from topography, and need special consideration. Variation in soil surface temperature between the highest and lowest points in mid- and high-elevation clearcuts during clear, windless growing season nights can be as much as 15°C. Thus, in central interior locations, the soil or plant surface temperatures in low-lying parts of clearcuts drop below freezing on a few nights during every month of the growing season. Tree species that do well in elevated parts of such clearcuts may fail in these frost pockets. To counter this problem, one may plant lodgepole pine (and other hard pines) that are very resistant to growing season frosts and are seldom damaged. Alternatively, late flushing trees or provenances from farther north can also be used. More maritime provenances with their greater heat sum requirements may escape late frosts, but they do not become frost hardy early enough in the fall to escape early frost damage.

Frost problems are most severe on bare or nearly bare ground. As stands grow up, the layer of air that is cooled is much thicker, and the same heat loss will result in a smaller temperature drop. Also, two processes are involved: radiation cooling and movement of cold air down hill into frost pockets. Such cool air behaves like molasses; it will flow over bare ground but not through a canopy. Once a stand of trees grows up to several meters in height, and crowns begin to close, the greatest danger of growing season frosts is past.

Late frosts can also damage the cambium. Typically such damage results in the production of several layers of parenchyma cells rather than normal tracheids and sieve cells. The **frost ring** so formed represents a weak layer that breaks and becomes filled with resin. The phenomenon (known as **ring shake**) almost certainly originates from frost rings. Frost rings in the xylem of trees record the history of major frost events to which such trees have been subjected.

Frost damage may result in death or weakening of plant tissues. In either case the plant becomes susceptible to pathogenic fungi which invade primarily the bark. The same fungi are unable to parasitize undamaged plants. Trees planted in locations where they are subjected to frequent cold stress are often killed by such fungi, and sometimes the original cause is not clearly recognized.

Low temperature stress is not restricted to temperatures below the freezing point. In mountain valleys and frost pockets, cold air drainage and accumulation may result in a local climate in which night time temperatures are well below that of ridges and mid-slopes. Some tree species (e.g., Douglas-fir) experience considerable stress under such conditions, and that is manifested in slow growth and increased susceptibility to needle and bark pathogens. Frequent cold stress can be attributable to topographic features, but can also arise when the wrong provenance is used.

In major mountain valleys a common phenomenon called **red belt** is associated with chinook winds. Red belt consists of narrow bands of trees at mid elevations along such valleys in which the foliage has been

killed (buds usually survive). It has been suggested that such injury could result from drying of foliage exposed to the warm, dry, high windspeed air of the chinook while soils are cold and boles are frozen so that water cannot be replaced. However, rapid cooling at the fluctuating border between the warm chinook air and the cold air in such valley bottoms is a better explanation of the phenomenon.

Valleys in the coastal mountains are subjected regularly to severe winter conditions. Sometimes high-speed, cold, dry outflow winds cause death of foliage and trees. In some locations crowns of trees tend to become one-sided, the side facing the wind being regularly killed back (e.g., Douglas-fir in the eastern part of the lower Fraser valley). Ice storms occur when moist, warm Pacific air overrides cold continental air. Rain falling through the cold air layer supercools and freezes on contact. Heavy ice accumulations may result, leading to severe crown damage.

Finally, moist, silty soils without snow cover may lead to frost heaving. The soil surface freezes to seedlings. Subsequent ice crystal formation below this surface can lift the surface and pull the seedling out of the ground, particularly if the root system isn't well established.

DROUGHT

The effects of acute drought are well known. Chronic droughts are more subtle. Such droughts may result from long term climatic cycles (on the scale of decades), or from increasing water demand as stands grow in situations of limited soil storage and a summer water deficit. Trees are seldom killed outright by chronic droughts. The stress, however, predisposes trees to various diseases and insects. Chronic drought is also a common contributing factor to declines.

HEAT AND SUNLIGHT

Soil surfaces of a low albido that are exposed to full sunlight can reach temperatures that are lethal to succulent tissues. Seedlings can be killed in such situations by girdling at the root collar. The symptoms may resemble damping off, a common disease of succulent seedlings caused by pathogens.

The short wave, ultraviolet component of sunlight is very damaging to unprotected living tissues. **Sunscald** occurs when thin bark tissues that have not developed ultraviolet resistance are exposed to sunlight following thinning or spacing. Winter sun (with rays more nearly perpendicular to the bole and with less shading from the crown) appears to be most damaging. Alternate freezing and thawing of such injured bark may aggravate the problem. It is not uncommon to find the exposed bark of young trees killed on the south side of the tree for about a quarter of the circumference. Similar damage is often found on the edge of a clearcut and along a right of way, and can occur on older trees that carry a well developed layer of dead bark. In such cases the main cause is probably the rapid freezing and thawing experienced by the bole as it is exposed to sunlight and then is shaded on clear cold days. Sunscald is often followed by decay.

FLOODING Tree roots require oxygen. Temporary flooding associated with beaver activity or road construction can kill roots through lack of oxygen. By the time the trees die, the original cause may no longer be very evident, and the cause of death may be misidentified. Some species, such as western black cottonwood, can tolerate regular flooding.

DECLINES A special group of diseases are known as declines. Typical symptoms of declines include reduction in height and diameter growth, small, sparse foliage, leading to dieback of the outer and upper crown (sometimes combined with the production of sprouts from dormant buds on the lower bole), and, eventually, death. Early in the development of these symptoms, the root system also changes. Small roots die and storage of carbohydrates declines sharply. It is seldom possible to demonstrate a single cause of such a decline, although the temptation to do so is often strong. Thus air pollution, acid rain, climatic warming, and so on, have often been implicated. No doubt these phenomena play a role, but the spatial distribution of symptoms and their development over time is usually poorly correlated with such factors, making the direct assignment of cause difficult.

Declines are perhaps best understood in terms of the cumulative effect of a number of stress factors. A simple paradigm of tree growth states that the energy captured by photosynthesis over the course of a year must exceed the energy needed for essential processes, such as basic metabolism of all living tree tissues, nutrient absorption, repair, replacement of foliage, and some growth of the bole and root systems. Various stress factors, such as cold stress, chronic drought, chemical pollution, and soil problems, can both reduce the total photosynthate produced, and increase the requirements for energy. These stress factors typically act in a chronic fashion. They do not by themselves result in decline symptoms, but they result in the depletion of tree energy reserves, and compromise the ability of trees to respond to short term but acute stresses. Thus, when such trees are then exposed to a severe frost, an insect defoliation, or a mechanical injury, decline sets in. As the various decline symptoms develop, the ability of the tree to produce energy is reduced, and so symptoms become more severe over a period of several years. As the decline proceeds, the resistance mechanisms of the tree against insect and pathogen attack are compromised, and, in the end, death may be attributable to such attacks.

Typical decline symptoms may develop in certain species over large geographic areas although not to the same degree on different sites within that area. They may also occur in individual trees, particularly if the local environment is severely altered, as, for instance, along a newly constructed road, while nearby trees not subjected to the same change remain unaffected. Decline symptoms can be reversed if the original agents of stress are removed. However, the farther decline has progressed, the more difficult it is to reverse.

Tree age is a factor. Large, old trees cannot respond readily to changed conditions, and often exhibit decline symptoms while nearby younger

trees of the same species remain unaffected. Genetics also plays a role. Within a population of trees of the same age, exposed to similar stresses, some individuals are much more prone to develop declines than others.

Chronic droughts are a common stress factor. For instance, a condition known as White pine pole blight was common in the Kootenays and Idaho during the 1940s and 50s. Symptoms included short, chlorotic needles, reduced height growth, and long narrow necrotic (dead) lesions on the bole and eventually dieback of the crown. Although several pathogens and insects were associated with the condition, none of these could be shown to be the primary cause. Eventually the typical symptoms were produced by artificial induction of chronic drought over several years. Thus the condition was attributed to the long drought period of the 1930s and 40s. When precipitation returned to more normal levels in the 1950s, it took more than a decade for trees with decline symptoms to recover, and many trees were in such advanced stages of decline that they could not recover. So mortality continued well after the major causal agent was removed.

Examples of declines are plentiful. For instance maple decline, a dieback condition of sugar maple in eastern Canada became a major concern in the mid-1980s. Maple declines had been described before, but this one was quite severe. At first it was thought to be caused by air pollution. It is now clear that the problem arose from a severe early frost without snow on the ground, which resulted in the death of fine roots. Recovery is now almost complete. It is important to recognize, however, that frost, by itself, would probably not have caused the dieback symptom. Maples were already under stress, partly from pollution, and partly from cultural practices associated with maple sugar collection. This meant that trees had only limited energy reserves to replace lost roots, and so decline set in.

One of the best known and intensively studied declines is the European syndrome known as **Waldsterben**. This is a decline of both conifers and hardwoods at mid- and high-elevation central European forests. Air pollution very likely plays a major role, but is probably not the sole agent.

Other well known declines include yellow birch dieback in eastern Canada and the New England states; oak declines in southeast United States, in which the oak wilt fungus plays a role; yellow cedar dieback in Alaska; spruce declines in the mountainous areas of northeast United States; and littleleaf disease of southern pines in southeast United States, in which a pathogen of small roots, *Phytophthora cinnamomi,* plays a role. In all these cases, various possible causal agents have been identified, but none seems sufficient to account for the spatial distribution of the condition or its development over time.

Diebacks will undoubtedly become more frequent and widespread if the greenhouse effect becomes more prominent.

SECTION READINGS

READING 1 *[pp. 23–34]*

Houston, D.R. 1987. Forest tree declines of past and present: current understanding. *Can. J. Plant Path., 9:* 349–360.

CANADIAN JOURNAL OF PLANT PATHOLOGY 9: 349–360, 1987

Forest tree declines of past and present: current understanding

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Comparison of many dieback/declines indicates a common etiology: "natural" stresses physiologically alter tissues that are then attacked and killed by facultative parasites. Because these attacks succeed only after stress, and because mortality is a common consequence, stress is considered predispositional. Recent attention has focused on declines in Western Europe. Temporal correlation of damage on a variety of hosts with increases in air pollutants has fostered a hypothesis that anthropogenic air pollution (AAP) has triggered a general forest decline or "Waldsterben." This hypothesis has fueled speculation that AAP is responsible for recent declines of *Picea rubens* or *Acer saccharum* in Northeastern USA and Canada and for synchronous growth declines in many species. Research is underway in Canada and USA to determine if AAP is associated with growth loss, mortality, or changes in forest structure and function. Research in Europe and North America has yet to provide direct linkage of cause-effect. Studies are complicated, and results confounded, because natural stress — especially defoliation, drought, and snowless, cold winters — has occurred along with suspected and monitored increases in AAP. Clarifying how AAP effects contribute to, or are exacerbated by, natural stress effects is a demanding but necessary task.

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Une étiologie commune se dégage d'une étude comparative des diverges manifestations du dépérissement forestier: les facteurs naturels traumatisent les tissue qui vent ensuite attaqués et tués par des parasites facultatifs. Les attaques de ces derniers ne donnant des résultats qu'après la manifestation des traumatismes, et la mortalité consécutive étant une conséquence commune, on considère que l'hôte est prédisposé aux traumatismes. Récemment, l'attention s'est portée sur les manifestations du dépérissement forestier dans l'ouest du l'Europe. La corrélation temporelle des dommages subis par divers hôtes et l'accroissement des concentrations de polluants atmosphériques ont donné naissance a l'hypothèse que la pollution atmosphérique d'origine anthropique a déclenché la phénomène général du dépérissement forestier. Cette hypothèse a nourri les spéculations selon lesquelles cette forme de pollution est en cause dans les dépérissements récents des essences *Picea rubens* et *Acer saccharum*, dans le nord-est des États-Unis et au Canada ainsi que dans le ralentissement observé, en même temps, de la croissance chez de nombreuses autres essences. Au Canada et aux États- Unis, des travaux de recherche visent à déterminer si la pollution atmosphérique anthropique est à l'origine du ralentissement de la croissance, de la mortalité ou des modifications de la structure et des fonctions forestières. Tant en Europe qu'en Amérique du Nord, les chercheurs n'ont pas encore établi de relations directes de cause à effet. Ces études sont compliquées, et les résultats pas toujours convaincants, car les facteurs naturels de traumatisme, notamment la défoliation, la sécheresse ainsi que l'absence de neige au cours d'hivers rigoureux se vent manifestés en même temps que les augmentations soupçonnées et observées de la pollution. La tâche de déterminer de quelle façon la pollution anthropique s'ajoute aux facteurs naturels ou est exacerbée par eux constitue une tâche exigeante mais nécessaire.

Birch dieback, ash dieback, eucalyptus diebacks, maple dieback and decline, oak decline, ohia decline, sweet gum blight, maple blight, pole blight, pitch streak, beech bark disease, littleleaf disease are names given to tree disease problems that, while often similar with respect to symptoms, are mysterious, at least at the outset, with respect to cause. The syndrome common to all, and reflected in many of their epithets, is the "dying back" or death of meristem tissues of buds, branches, or stems, which, if continued or repeated, results in the decline and death of the tree.

The etiologies of these diseases vary markedly in their specifics. What is clear, however, is that all share a causal complex that begins when tissues of healthy trees are altered by stress and culminates when those tissues are invaded and killed by facultative parasites (saprogens with the ability to attack weakened tissues).

Manion (1981) presented a concept of decline diseases that entails the interaction of a number of interchangeable abiotic and biotic factors that results in the gradual and general deterioration and, often, death of trees. Manion classifies these factors as predisposing, inciting, or contributing, depending on their chronology and whether their effect is long or short term. Long-term predisposing factors include host genotype and soils that serve to render trees susceptible to such stress factors as drought or insect defoliation. Organisms able to attack weakened trees are included among factors contributing to the decline.

Houston (1973, 1982, 1984) has proposed a similar concept that indicates that diebacks/

declines are diseases initiated by the predisposing effects of abiotic/ biotic environmental stresses that culminate in attacks, often lethal, by organisms of secondary action. In Houston's definition, the predisposing factors are the stresses that by their effects render healthy, normally resistant tissues susceptible to weakly pathogenic organisms. Because these attacks are unsuccessful in the absence of stress, and because in the absence of these organisms trees usually recover with abatement of stress, organisms of secondary action are considered more than contributory. This does not imply that severe stress alone cannot kill trees. Indeed, because environmental stresses generally interfere directly or indirectly with the production, transport, and storage of sugars and biochemical catalysts, and with the absorption and movement of water and minerals, they, by themselves, if of sufficient intensity, duration or frequency, can result in decline and mortality. More often, however, mortality can be ascribed to the attacks by one or more ubiquitous, naturally occurring opportunistic organisms.

Houston (1982) recognized that two phases occur in many of these diseases. The dieback phase, frequently manifested by a progressive dying back of buds, twigs, branches, and rootlets, often results from the effects of the stress(es) alone. Dieback can be viewed as a survival mechanism, and trees often recover when stress abates. The decline phase, wherein the vitality of the entire tree lessens, usually results from attack by secondary-action organisms and often culminates in death. Recovery from this phase is less likely to occur after abatement of the stress factor. Partitioning the developmental stages of dieback-declines into separate phases emphasizes the dynamic nature of the stress-host-organism relationship and provides a framework for diagnosis and study of these problems. It should be clear, however, that in reality these relationships are a continuum of interactions manifested as physiological and morphological responses of the host tree to the effects of variable and shifting environmental stresses exacerbated to varying degrees by the invasions of opportunistic organism complexes.

The "mystery" of decline etiology is largely attributable to its complexity. Declines are difficult to diagnose, especially after the fact. Triggering stress factors are often ephemeral, and they frequently occur many months or even years prior to tree mortality. The decline episode itself is often ephemeral and trees are either dead or in an active stage of recovery when the pathologist appears on the scene.

For the last 10 years or so, much attention has been focused on declines of several European species, especially silver fir, Norway spruce, Scots pine, and beech (Schutt & Cowling 1985). The widely held belief that airborne pollutants are at the heart of this situation has led to speculation in North America that anthropogenic air pollutants (AAP) are responsible for decline and mortality of red spruce in high-elevation forests of the northeastern USA (Johnson & Siccama 1983); of sugar maple in scattered forests of New England, New York, and eastern Canada (Bernier & Brazeau 1986); and for seemingly synchronous decreases in growth of a number of eastern conifers and hardwoods (Raynal et al. 1980, Johnson et al. 1981, McLaughlin et al. 1983b).

In this paper I will discuss some of the important tree declines of the past and present and also address the current emphasis on AAP as triggers of decline.

Some declines of past and present

The following discussion of several diebacks/ declines illustrates the diversity of known or suspected causal relationships and the influence of those relationships on the temporal/ spatial patterns of disease occurrence. The triggering stress factor(s) and general times and places of occurrence for the important decline diseases of North American tree species are presented in Table 1.

Birch dieback. This major decline of paper (*Betula papyrifera*) and especially yellow (*B. alleghaniensis*) birches occurred from the early 1930s to the late 1950s. First reported from Nova Scotia, the disease was successively noted and seemed to 'spread' westward through the Canadian Maritime Provinces into Quebec and Ontario and through New England and into New York during the next 15-20 years. Yellow birch stands over thousands of square miles were destroyed, with the greatest damage occuring in New Brunswick, Maine, and upper elevation forests in New Hampshire. The disease syndrome included thinning foliage, chlorotic or curled leaves, dieback of upper crown branches, and foliage produced in clumps in the lower crown. Dieback usually was preceded by reduction in radial increment (Barter 1953). Trees often died within 3 to 5 years after onset of symptoms. The cause of birch dieback proved quite intractable and after two decades of intensive research, no single causal factor was determined (Clark & Barter 1958). However, several stress factors were found to be associated with the disease in different areas. For example, average increases in soil temperature of about 1°C, which occurred over a period of 10 to 20 years, were shown to cause rootlet mortality (Redmond 1955). Redmond showed that trees whose roots were

Table 1. Causal factors and approximate time frames for some representative declines

Diseases and primary hosts	Stress factors	Secondary organisms	Time frame	Location
Birch dieback (*Betula alleghaniensis*)	a,d,e,f	w,y,z	mid 1930s — late 50s	Can. maritimes to NY
Beech bark disease (*Fagus grandifolia*)	h	x	late 1910s — present	Can. Maritimes, Que., Ont, New Eng., NY, PA, WV
Sugar maple declines (*Acer saccharum*)	a,f	w,y	1950's	New Eng., NY
	f	w,y	mid 1960s — mid 70s	VT,NH,NY
	f	w	early 1980s — present	VT, NY
	f	w	mid-late 1950s	WI
	f,d?,e	w	mid 1960s — late 70s	NY, New Eng., Eastern Can.
			early 1980s — present	NY, New Eng., Eastern Can.
Oak declines (some) (*Quercus* spp.)	c,a	w,z	mid 1920s	NC
	f	w,z	early-late 1950s	PA, NY, WV
	f	w,z	early-late 1960s	NY, New Eng.
	f	w,z	early-late 1970s	PA
	f	w,z	early 1980s — present	New Eng., WV
	a	?	mid 1970s	NC
	a	?	early 1980s — present	Southern USA
	a	x	late 1970s — early 80s	SC
	b	w,z	early-mid 1970s	IL
Sweetgum blight (*Liquidambar styraciflua*)	a	?	late 1940s — early 60s	Mid-Atlantic, Southeast. USA
Littleleaf disease (*Pinus echinata*)	b,i	w	early 1920 — present (esp. 1930 — mid 1960s)	Southeast. USA
Ohia decline (*Metrosideros polymorpha*)	b,i	w,z	mid 1870s — present (esp. early–late 1900s and mid 50s—early 70s)	Hawaii
Ash dieback (*Fraxinus americana*)	a	x	1930s — mid 40s and mid 1950s — mid 60s	New Eng., NY
	g	—	mid-late 1950s and late 1970s — mid 80s	New Eng.
	MLO, a?		1950 — present (esp. early 70s — present)	NY, New Eng., Mid-western US

Key:

Stress factors
- a. water deficit/high temperature
- b. water excess
- c. spring frost
- d. soil/root freeze
- e. logging disturbance
- f. defoliation by insects
- g. defoliation by fungi
- h. sucking insects
- i. nutrient deficiency
- ? suspected, not proven
- MLO mycoplasma-like-organism

Secondary organisms
- w. root fungi
- x. bark fungi
- y. twig and bud fungi
- z. insect borers

killed by high soil temperatures responded by forming new ones at deeper levels. In much of the region where yellow birch grows, slightly deeper rooting depths would place roots in soil horizons where aluminum has accumulated to high levels (Hoyle 1965, 1969). In light of our current and emerging understanding of the effect of high soil aluminum content on rootlet development and mortality, this relationship warrants further investigation. Another factor affecting root mortality is deep soil freezing. Pomerleau proposed the concept, as reported by Hepting (1971), that the freezing of shallow birch roots, which probably occurred (in the Quebec area) in 1932, 1938, 1942, and 1943, resulted in both direct root breakage and poor uptake of moisture.

The search for causal agents disclosed the presence of several viruses in birch (Hansborough

& Stout 1947). Because some of the symptoms of birch dieback could be transmitted from diseased to healthy trees by grafting (Berbee 1957), there has been speculation, not yet demonstrated, that virus infection might predispose trees to other agents. Repeated defoliation by a complex of leaf mining and skeletonizing insects seemed to trigger birch dieback in some areas (Clark & Barter 1958).

The effect of these stresses, singly or together, appeared to render trees susceptible to attacks by many secondary insects and fungi. The bronze birch borer, *Agrilus anxius* (Barter 1957), the shoestring root rot fungus, *Armillaria* sp., and the bud and twig blighting fungus, *Diaporthe alleghaniensis* (Arnold 1967), were associated in varying degrees with stressed, declining birch trees.

Birch dieback essentially disappeared in the early to late 1950's as mysteriously as it had appeared. Numerous localized episodes of dieback of yellow and white birches have occurred since, many of which have been associated with periods of exceptionally heavy seed crops (Gross 1972).

Birch dieback demonstrates the great complexity of many decline diseases and the perplexing chore facing researchers attempting to decipher cause/effect relationships. Whether the etiology of birch dieback would prove as intractable today as it did 40 years ago is difficult to know. While it is true that there is today a generally greater appreciation for and understanding of stress / host / organism relationships, proving such relationships still remains a formidable task.

Beech bark disease. This disease of American (*Fagus grandifolia*) and European (*F. sylvatica*) beech occurs when bark, attacked and altered by the feeding action of the beech scale, *Cryptococcus fagisuga*, is invaded and killed by several fungi of the genus *Nectria* (Ehrlich 1934). The scale was accidentally introduced to Nova Scotia on ornamental *F. sylvatica* about 1890. Around 1920, the first scale outbreaks on *F. grandifolia* were reported, and by 1932, the disease complex occurred throughout the beech areas of the Maritimes and in scattered areas of Maine. By 1944, most of the mature beech in Nova Scotia and Prince Edward Island was dead (Belch 1944). The scale has since spread through New England and New York into Quebec, Ontario, and Pennsylvania (Houston et al. 1979). Significant outlying infestations occur in West Virginia and Ohio (Mielke et al. 1982, 1985).

The symptoms of beech bark disease differ in several respects from those of other declines (Houston & O'Brien 1981). Thus, while heavy scale infestations can result in some growth reduction and in death of some outermost bark cells, dieback of twigs and branches does not usually occur until extensive areas of cambium are killed by one or more *Nectria* spp. Leaves of larger trees severely affected by scale / *Nectria*, are often small, sparse, and chlorotic. Mortality of large trees can be high in stands affected for the first time. Small trees, frequently of root sprout origin, that emerge in the aftermath of heavy tree mortality or disease salvage, rarely die, as did their parents, from girdling cankers (Houston 1975). Rather, they gradually accumulate defects in the form of isolated, discrete cankers. As years go by, severely affected trees gradually slow in growth, lose vigor and decline, and eventually may die.

Beech bark disease demonstrates very clearly the truly predispositional role of the initiating stress factor. In North America and in Europe, the reported secondary organisms are all members of the genus *Nectria*, which, while closely related, differ in their abilities to cause disease in the absence of beech scale. *Nectria galligena* (N. America and Europe), causes perennial cankers on many hardwoods, rarely on beech; *N. coccinea* (Europe) causes annual cankers following many stresses on a variety of hardwoods including beech; *N. coccinea* var. *faginata* (N. America) is not known to attack beech or any other species. In the presence of beech scale, however, each of these organisms readily and rapidly invades and kills bark. Although the biochemical basis for this stress-host-fungus interaction is unknown, it probably lies in the ability of these fungi to colonize tissues whose normally effective responses to wounding or to invasion have been rendered ineffective by substances secreted by the insect.

Sugar maple declines. Several dieback / declines of sugar maple (*Acer saccharum*) are recognized, and a number have been studied intensively. The decline of thousands of roadside trees has been attributed to cumulative effects of deicing salts and to adverse changes in root environments, especially of water tables (Baker 1965, Button 1965, Holmes 1961, Holmes & Baker 1966, Lacasse & Rich 1964, Shortle 1972). In the sugarbush and forest, trees suffer sometimes from drought and frequently from internal decays associated with tapping or injuries created by cattle, heavy machinery, or freezing of roots during open (snow-free) winters. While the effects of these insults, which are often cumulative, can lead to decline, the most important stress factor by far in both sugarbush and forest is insect defoliation.

Insects triggering dieback, decline, and significant mortality of sugar maple have included a leaf roller-webworm defoliator complex in Wisconsin in the 1950's (Giese et al. 1964); the saddled

prominent (*Heterocampa guttivitta*) in New York and New England in the 1960s and early 1970s and 1980s; and the forest tent caterpillar (*Malacosoma disstria*) in New York, New England, and Canada in the 1950s and again in the 1970s and 1980s. The most recent outbreaks are among the worst on record because of the great acreages involved and the high mortality that ensued (Allen 1986).

Affected trees show a progressive dying back from the tips of upper crown branches. Chlorotic, dwarfed foliage is sparsely produced on sprouts. Spring flush may be delayed in years immediately following defoliation. Starch reserves, especially in roots, are markedly reduced or depleted and growth is slowed.

The most thoroughly investigated decline of forest maple occurred in northeastern Wisconsin in the mid to late 1950s. A series of intensive studies on this problem, then termed maple blight, revealed for the first time some of the cause-effect relationships of a biotically initiated dieback-decline disease (Giese et al. 1964). Maple blight was triggered by a complex of defoliating insects, including several species of leafrollers and the maple webworm (*Tetralopha asperatella*), that severely defoliated sugar maple for up to 3 successive years. Mortality of defoliated trees, associated in many cases with attacks of roots and root collars by *Armillaria* (Houston & Kuntz 1964), was extreme in localized pockets scattered over 10 000 acres.

Subsequent inoculation trials with *Armillaria* of both artificially and naturally defoliated sugar maples demonstrated conclusively that mortality of defoliated trees was usually a consequence of successful invasion of roots by *Armillaria*, and that twigs of weakened trees also were often invaded and killed by *Steganosporum ovatum* (Wargo & Houston 1974).

Studies have shown that predisposition to *Armillaria* has its basis in several stress-induced biochemical changes, especially of amino acids and carbohydrates. For example, severe defoliation can trigger hydrolysis of stored starch to unusually large quantities of reducing sugars (Parker 1970, Parker & Houston 1971). These simple sugars are especially favorable energy sources of *Armillaria* (Wargo 1972). Because similar changes in carbohydrate regime occur when sugar maple is subjected to drought (Parker 1970), it is likely that similar decreases in host resistance to *Armillaria* follow severe water stress. Drought has been associated with a number of significant decline episodes of sugar maple (Hibben 1964) and other species (Toole & Broadfoot 1959, Tainter et al. 1983). When defoliation and drought occur simultaneously, high levels of mortality can result.

Defoliation-initiated sugar maple decline demonstrates how physiological response to severe, "whole-tree" stresses can predispose trees to organisms whose ecological niches are favored by the biochemical consequences of these responses.

Oak declines. While similar in many respects to maple decline, oak declines are considerably more complex as there are more hosts, more initiating stress factors, and more important secondary organisms. Episodes of oak decline have occurred somewhere in the East during nearly every decade of this century (Table 1).

Which host species are affected during any given decline episode depends on the host range of the biotic stress agents involved and / or host susceptibility to the abiotic stress factors. Regardless of which hosts and which triggering stresses are involved (stress factors are often — indeed, usually — confounded), tree mortality invariably results from attacks by secondary-action organisms, especially by the twolined chestnut borer, *Agrilus bilineatus,* by *Armillaria,* or both (Hursh & Haasis 1931, Staley 1965, Dunbar & Stephens 1975, Wargo 1977). In oak declines, the progression of crown symptoms and the reduction in starch reserves and tree growth are similar to those observed in maple declines (Wargo et al. 1983). The girdling attacks by both *Agrilus* and *Armillaria* sometimes result in the rapid collapse and death of the trees.

Because oaks vary widely in their geographic and ecologic adaptations, they are potentially subject to a plethora of stress relationships, a situation remarkably attested to by a series of events that occurred in North Carolina in the 1920s. In 1926, over 3 million board feet of white oak (*Quercus alba*) died in the valleys and hollows, a consequence of a severe frost in May 1925 (Bear 1926). The young foliage of the white oak was more susceptible than the fully developed foliage of the red oak group. Then, in July-August 1925, severe drought caused significant damage to many of the black (*Q. velutina*), red (*Q. rubra*), and scarlet (*Q. coccinea*) oaks on ridges and upper slopes (Hursh & Haasis 1931). And, finally, in April 1927, a severe late frost probably damaged further these red oaks. Trees with severe foliage damage in 1925 were dead by 1929. Stressed trees were predisposed to killing attacks by *Armillaria, Agrilus bilineatus,* and long-horned beetles.

In the northeastern USA the major stress factor triggering oak decline is insect defoliation (Hepting 1971, Staley 1965). In spite of the wide variety of native oak defoliators associated with oak declines, greatest attention has been paid to the introduced gypsy moth, *Lymantria dispar,* to its effects, and to

ways of managing it (Doane & McManus 1981). Major gypsy moth outbreaks occurred in the mid-1950s, mid-1960s, early to mid-1970s, and early 1980s — periods also characterized as times of water shortage.

In the south and southeastern USA, the most important stress factor associated with oak decline seems to be drought. On the Nantahela National Forest in North Carolina, a decline of northern red oak (*Q. rubra*) culminated in severe losses in 1979. Growth reductions in these trees were related to water shortages that began in 1973, 1974, increased in 1975, 1976, and 1977 and became acute in 1978 (Tainter et al. 1984). Similarly, an extensive decline and mortality of willow, laurel, water and southern red oaks (*Q. phellos, Q. laurifolia, Q. nigra,* and *Q. falcata,* respectively) along the South Carolina coast in late 1980 and 1981 was triggered by water shortage (Tainter et al. 1983). While the severe drought of 1978 resulted in scattered cases of decline and death, a second one in 1980 eventually resulted in rapid decline and death in 1981 of thousands of these shallow rooted trees. Tainter and others (1983) found that *Hypoxylon atropunctatum* was common on declining and dead trees. This naturally ubiquitous, normally saprophytic fungus apparently can rapidly invade trees stressed by a number of factors (Tainter & Gubler 1974), and by its colonization of cambium and sapwood it can hasten mortality (Tainter & Gubler 1974, Tainter et al. 1983).

A general decline of many oak species, first noticed in the early 1980s over wide areas of the south, prompted a survey in 1985 (Starkey & Brown 1986). Overall, of 2800 codominant and dominant trees examined, 17% were dead, 23% were in moderate to severe decline, while the rest showed only slight decline symptoms or were healthy. Analysis of annual growth indicated that healthy and declined trees did not differ significantly until about 1964. These data suggest that trees that first showed decline symptoms 6 to 7 years ago may actually have been affected 2 decades before. Although the cause of this decline is not yet determined, it is likely that water shortages are involved since mortality tended to be highest on the shallowest soils, and on ridge tops and south and west exposures.

Oak declines demonstrate the great diversity of stress factor/secondary organism interactions, and the close association in time and place of the occurrence of the stress events and the occurrence of the diseases.

It is apparent that drought has been an important factor in the etiology of a number of oak decline episodes. Drought also has been associated with declines of several other species. The 1950 decade, which encompassed one of the most intense and prolonged drought periods on record, saw the initiation or intensification of a rather large and diverse group of decline diseases including 1) sweetgum (*Liquidambar styraciflua*) blight in the southeast and south, 2) littleleaf disease of shortleaf pine (*Pinus echinata*) in the southeast, 3) ohia (*Metrosideros polymorpha*) decline in Hawaii, 4) dieback of white and green ash (*Fraxinus americana, F. pennsylvanica*) in the northeast, 5) pole blight of western white pine (*Pinus monticola*), and 6) sugar maple declines in New England and New York. A discussion of the first four follows.

Sweetgum blight. Sweetgum blight made a remarkably brief appearance associated with this drought. First noticed in 1948 in Maryland (Miller & O'Brien 1951), the disease intensified until decline and mortality occurred throughout its range (Young 1956). A southwide survey in 1954 revealed that 36% of the trees sampled were affected and the disease was twice as prevalent on upland as on bottomland sites (Hepting 1955). The evidence points to water shortage as a primary stress responsible for the problem. Most damage in the Mississippi flood plain occurred in soils with high imbibitional water capacity and high K and Na contents (Toole & Broadfoot 1959). Diseased trees possessed abnormally high fine-root mortality though the larger roots appeared healthy (Toole & Broadfoot 1959). No organism was found consistently associated with root dying (Berry 1955, Toole 1959). Sweetgum blight decreased in significance once water tables returned to normal. This disease occurred when prolonged drought periods markedly affected water tables. Other soil water relationships also may be important as predisposing factors, as is demonstrated by the significant and complex disease known as littleleaf disease.

Littleleaf disease. This problem of shortleaf (*Pinus echinata*) and to some degree of loblolly (*P. taeda*) pines is the most serious disease of this species (Campbell & Copeland 1954, Anderson & Mistretta 1982, Mistretta 1984). Annual losses range from 3 to 5% in severely affected areas in the Piedmont of Georgia, North Carolina, South Carolina, and Virginia, where extensive planting of shortleaf pine has occurred. Much research over many years has shown this disease to be a remarkable complex that still is not fully understood. Trees rarely develop symptoms before 20 years old and usually not until they reach 30-50 years of age. This is the time when, on the soils of the Piedmont, tree demands begin to exceed available water and nutrient supplies. Typical

littleleaf disease soils are highly erodable and possess poor internal drainage—usually with very firm and plastic clay subsoils that stay wet for long periods. Nutrients, especially nitrogen, are in low supply (Roth et al. 1948). Such wet soils favor root pathogens, especially *Phytophthora cinnamomi, Pythium* spp., and pathogenic nematodes. In addition, anaerobic conditions seem to favor root infection and disease development.

Ohia decline. A similar set of factors seems responsible for a major decline of ohia (*Metrosideros polymorpha*) on the island of Hawaii. An excellent summary of the history and current status of this disease and of the research to disclose its cause has been published recently (Hodges et al. 1986). In brief, although it was recognized as early as 1875, with severe episodes known early in this century, the most widespread damage occurred from 1954 to 1972. Today, about 50 000 ha are seriously affected. According to Hodges and others (1986), mortality associated with ohia decline results when the stress of poor soil drainage predisposes trees to eventually lethal attacks by secondary organisms, including the ohia borer (*Phagithmysus bilineatus*), *Phytopthora cinnamomi,* and *Armillaria.* Low soil nutrient levels, aluminum toxicity, and senescence may also play a part in the overall decline syndrome.

The rapid decline and death of large numbers of ohia trees in even-aged or even-structured stands (cohorts) have led to an interesting hypothesis that senescence itself is a predisposing factor in this decline (Meuller-Dombois 1983). Although there are many reasons to reject this hypothesis per se (Hodges et al. 1986), it is possible to conceive of situations where relative differences in susceptibility or resistance to stress factors or to secondary insects and pathogens may reflect differences among age or genetic cohorts.

Ash dieback. This decline, primarily of white ash (*Fraxinus americana*), was first reported from Canada in 1925 (Pomerleau 1953) and from the United States in 1930 (Ross 1966). The decline has been considered to be a drought-triggered canker disease. Episodes of ash dieback coincided with the severe drought periods of the 1930s and 1950s, and research has shown that at least two fungi common to naturally senescing lower branches of healthy ash can produce cankers that contribute to dieback of upper crown branches and stems of drought-stressed trees (Ross 1966, Silverborg & Brandt 1957). A survey in the mid-1960s found that 27% of the ash in northeastern USA were dead or dying (Tegethoff & Brandt 1964).

A dieback and sometimes mortality of white ash also occurs when outbreaks of ash rust (*Puccinia sparganioides*) are severe enough to cause defoliation-refoliation. Ash rust is most severe along the Atlantic coast in proximity to the alternate host, the salt marsh grasses (*Spartina* spp.). Damage has been especially severe from southern Maine to Connecticut in recent years. The fact that even those trees with severe dieback are not cankered and often recover after outbreaks cease suggests that defoliation stress does not alter bark in the same way that drought does.

In recent years, both viruses and mycoplasma-like organisms (MLO's) have been found associated with some declining ash (Hibben 1966, Hibben & Bozarth 1972, Lana & Agrios 1974, Hibben & Wolanski 1971, Hibben & Reese 1983). Matteoni and Sinclair (1985) have shown that MLO's are strongly associated with declining and dying ash in N.Y., and affected trees recently have been found in New England and in the midwest (Personal communication, Sinclair). How, or if, ash yellows decline caused by MLO, and/or infection by viruses are related to "historical" ash dieback presumably triggered by drought is yet to be shown. A possible linkage is suggested by the fact that MLO infection interferes with stomatal closure (Matteoni & Sinclair 1983).

Ash diebacks demonstrate that 1) even for a single host species, a diverse array of stress factors can each initiate a dieback syndrome, 2) in the absence of organisms able to capitalize on the particular stress- induced alterations, trees often can recover when stress abates, and 3) pathogens, previously unrecognized or understood, may be contributing either directly or indirectly to the stress etiology. These same relationships seem to be demonstrated as research progresses on declines purportedly resulting from atmospheric pollution.

The role of AAP in forest tree declines

In recent years much attention has focused on the possibility that atmospheric deposition is responsible for widescale forest decline. This concern was triggered by observations of foresters in 1979 and 1980 that Norway spruce (Picea abies) in southern Germany was in serious trouble. From 1980 to 1984, spruce forests in many other areas of western Europe showed similar problems. Surveys in 1982, 1983, and 1984 revealed not only that the problem was intensifying, sometimes rapidly, but also that many other conifers including white fir (*Abies alba*), Scots pine (*Pinus sylvestris*), larch (*Larix decidua*), and several hardwoods, notably beech (*Fagus sylvatica*), were also exhibiting symptoms of decline. Some of these symptoms were purported to be unique or new.

The chronology of the developing awareness of this decline situation, which has become interna-

tionally known as Waldsterben, the description of symptoms, the magnitude of losses, the geographic occurrences of affected forests, the various hypotheses proposed to explain its origin and development, the research needed or underway to examine the validity of the hypotheses, and the current state of our understanding have been rather exhaustively presented in recent reviews. Representative of these are the papers by Ulrich (1980), Ulrich et al. (1980), Binns and Redfern (1982), Schutt and Cowling (1985), McLaughlin (1985), and the papers by Fuher (1985), Landolt and Keller (1985), McLaughlin and Braker (1985), and Schütz (1985) that were published as a series.

At first, the onset of decline in Norway spruce in Germany was viewed with alarm, not because it was linked to atmospheric deposition, but because it followed closely on a similar decline of white fir known as Tannensterben. This long-recognized and still unresolved disease has recurred periodically for well over two centuries (Neger 1908). An exceptionally severe and widespread episode beginning about 1970, prompted a research program to find the cause (Schutt & Cowling 1985). Various hypotheses have been forwarded throughout the years to account for Tannensterben, including drought stress, root pathogens, aphids, and bacteria (Blaschke 1981, 1982). Bacterial wetwood is a major symptom in white fir (Bauch et al. 1975). Interestingly, even though outbreaks of this disease occurred long before current periods of heavy pollutant loading, Tannensterben is now included under the Waldsterben umbrella. Indeed, white fir is considered the species most seriously affected by Waldsterben in central Europe (Schutt & Cowling 1985).

The widespread geographic distribution of the damage in Europe and the seemingly synchronous development of a heterogenous array of symptoms on large numbers of diverse gymnosperm and angiosperm hosts appear at odds with previous experience with declines initiated by climatic extremes or biotic stress agents. Airborne pollution seemed the most plausible explanation. Various hypotheses have emerged to explain the role of air pollutants. The five generally considered most likely to account for all or part of the Waldsterben syndromes in Europe and for red spruce mortality and growth losses in North America are: 1) the acidification-aluminum toxicity hypothesis of Ulrich and colleagues (Ulrich et al. 1980); 2) the gaseous pollutants, especially ozone, hypothesis of Prinz and colleagues (Krause et al. 1983); 3) the foliar magnesium-deficiency hypothesis of Rehfuess (Rehfuess 1981); 4) the general stress hypothesis formulated by researchers at the University of Munich (Schutt & Cowling 1985) and 5) the excess nutrient (nitrogen) hypothesis that evolved during an exchange program of scientists in Germany and America (Schutt & Cowling 1985).

Hypothesis 1 proposes that soil acidification, resulting from deposition of anthropogenic sulfuric and nitric substance and through natural processes, mobilizes aluminum (and heavy metals), which reaches levels toxic to fine roots. Death of fine roots then leads to development of symptoms associated with reduced uptake of water and nutrients.

Hypothesis 2 proposes that gaseous pollutants, primarily ozone, were responsible for the sudden appearance over large regions, in 1981 of a needle chlorosis symptom. Ozone is also considered a primary cause of reduced growth in many species.

Hypothesis 3 proposes that low soil availability and/or increased leaching of magnesium from foliage by acid deposition enhanced perhaps by ozone, is responsible for the often observed foliar magnesium deficiency symptoms.

Hypothesis 4 proposes that air pollutants absorbed by foliage impair photosynthetic capacity and create a stress that leads to subsequent symptom development. Effects include production of growth-regulating hormones, reduced translocation of carbohydrates to roots, and impaired root development and function.

Hypothesis 5 proposes that deposition of excess nitrogen contributes to symptoms of Waldsterben directly through the toxic effects of high levels of atmospheric nitrogen, and indirectly through imbalances in activity between roots and shoots induced when foliage absorbs deposited nitrogen, and through lowered resistance to frost and to root disease fungi induced by increased nitrogen supply to foliage.

As research to examine these hypotheses progresses in Europe, it is becoming clear that none of them alone can account satisfactorily for the overall syndrome of Waldsterben. Indeed, evidence is mounting to suggest that if air pollution is a triggering factor it will likely be a result of complex, interactive or synergistic relationships among several agents. Some of the apparently contradictory relationships that need clarification are that damage is occurring to plants growing in soils that range from acidic to basic; that trees with widely differing nutrient requirements, and tolerances to pollutants (e.g. to ozone) are affected; that the occurrence of damage frequently is not well correlated spatially with depositional patterns; and that much of the syndrome, originally considered unique to Waldsterben, has actually been associated previously with abiotic or biotic factors (Kandler 1985).

Clarification of how pollutants interact with natural stress factors to affect physiological function is also of great importance. For example, although drought has been discounted as a factor triggering Waldsterben (Schutt & Cowling 1985), some of the most significant droughts in recorded history occurred in western Europe in 1975-1976 and again in 1983. The temporal correlation of these drought periods with the appearance of symptoms, given the well-documented delayed response to drought stress (Kozlowski 1979), warrants a thorough examination of the potential role of severe water shortage as a primary or exacerbating factor in Waldsterben. This potential has not gone unnoticed. Rehfuess (1981) proposed that drought was a possible cause of the decline of European conifers. He believed that a series of dry summers could interfere with root regeneration and water uptake and could lead to the onset of decline. Similar scenarios have been proposed for several of the previously mentioned North American declines including birch dieback and sweetgum blight. It is possible that the effects of drought could exacerbate or be exacerbated by one or more of the suspected pollutants. For example, interactions between drought and acid deposition, and their influence on the mobilization, concentration, uptake, and toxicity of Al have been postulated (Ulrich et al. 1980). These interactions need to be examined critically for soils of different origins or composition (e.g., organic vs. mineral soils) before the roles of airborne pollutants in declines can be established, not only in Europe but also in North America.

The alarm raised in Europe spread quickly to North America where researchers were becoming aware of a developing decline of red spruce (*Picea rubens*) in some high elevation forests in the northeastern USA (Siccama et al. 1982). Subsequent surveys have shown that aside from the red spruce problem and possibly a reported decline of sugar maple in Quebec, there exists relatively little obvious damage yet unascribable to previously documented nonanthropogenic stresses.

This is in marked contrast to the situation in Europe where, for example in 1984, over 50% of the forested areas in Germany were reported as exhibiting damage (Anonymous 1984). There are, however, reports of unexplained, synchronous, and sharp growth reductions in many tree species throughout eastern North American forests (Johnson & Siccama 1983, Raynal et al. 1980, Johnson et al. 1981, McLaughlin et al. 1983a, Tansey 1983, Sheffield & Knight 1983). Most of these reductions began in the 1960s and seemed to be temporally correlated with monitored or calculated increases in air-borne pollutant deposition or production (Likens & Butler 1981). There are, of course, other factors that result in growth reduction.

Johnson and Siccama (1983) pointed out the possibility that drought may be involved in both the high-elevation red spruce decline and in a growth decline of pitch pine (*Pinus rigida*) in New Jersey. The initiation of sharp reductions in incremental growth were correlated with drought periods of the 1960s and 1950s respectively. Schier (1985) studied pitch pine seedlings treated with "acid rain" that were grown for 1 year in intact soil cores from the New Jersey barrens and found no link between acid deposition and growth decline.

The inability thus far to implicate a primary pollution causal relationship, indeed, the present lack of any direct linkage between effects of AAP and decline in Europe or in North America, has led researchers to examine closely other factors that might be associated. Recent surveys of red spruce stands have revealed the association of declining trees with exposure to wind (Harrington 1986) and with a number of biotic agents including the root fungi *Armillaria* spp., *Scytinostroma (Corticium) galactinum, Perenneporia (Poria) subacida* (Carey et al. 1984, Rizzo & Harrington 1986), and *Phytophthora cinnamomi* and *Pythium* spp. (pers. comm. P. Wargo/ R. Bruck); insect borers, *Dendroctonus rufipennis,* (McCreery et al. 1986) and swift moths, *Pharmicis mustelinus* (pers. comm. W. Wallner); nematodes (pers. comm. P. Wargo/ R. Bruck); cytospora canker, *Valsa kunzei,* (Mielke et al. 1986); and dwarf mistletoe, *Arceuthobium pusillum* (McCreery et al. 1986). Research is currently underway to clarify the roles of these organisms in current tree decline. Because many of these organisms are known to follow stress, a considerable proportion of the current research effort is focused on the relationship of these and other organisms with 1) atmospheric deposition, 2) natural stresses such as water shortage, wind-induced root disturbance, root freezing during cold open winters, unusual freeze-thaw periods, and 3) changes in tree and soil characteristics associated with natural stand evolution.

Viewed from this perspective, red spruce decline seems to fit well into established conceptual frameworks of diebacks and declines (Marion 1981, Houston 1982). The role of air pollution stress in predisposing trees to decline was demonstrated in a series of multidisciplinary studies on the effects of photochemical oxidents in California (Miller 1983). High concentrations of ozone trapped by the San Bernardino and San

Gabriel Mountains east of Los Angeles injured the Ponderosa pine causing foliar damage, early leaf drop, decreased photosynthetic capacity, and reduced radial growth. Affected trees were rendered susceptible to fatal attacks by bark beetles and root pathogens.

Because many current decline situations are occurring in areas where atmospheric deposition is high and where natural stress events have also recently occurred, determinations of etiology are extremely difficult. The possibility that tree mortality or growth reductions are the result of anthropogenic pollutants must be weighed against the equal possibility that such responses are being triggered by natural stress events. Even more difficult to discern and to confirm is the very real third possibility that both anthropogenic and natural stress factors are together operating to trigger today's declines. Our understanding of tree declines will no doubt be expanded and modified as the results of current research efforts focused on these possibilities become available. Finally, we should not overlook the possibility that one or more of the currently unexplained declines may be the result of new or previously unrecognized pathogens. Kandler (1985) points out clearly that for many of the tree species affected by Waldsterben, the nature of associated symptoms, and the patterns of disease spread and development are more characteristic of diseases caused by biotic pathogens than those caused by abiotic factors including air pollutants. The recent association of MLO's with declining ash in North America points out the very real possibility of involvement by these and other difficult to isolate or culture pathogenic entities including viruses and viroids.

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SECTION ASSIGNMENT

SELF-TESTING/REVIEW QUESTIONS

Test your understanding of the material in the third 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 what happens to dormant and frost hardy bark tissues as their temperature is slowly decreased from the freezing point to –40°C.
   How does this differ from what happens if such tissues freeze while the tree is in active growth?
   How does it differ from the case in which unfrozen dormant bark is suddenly cooled to –40°C?
2. Describe the typical symptoms of early and late frosts on young conifers.
3. Why are recent clearcuts particularly susceptible to frost damage?
4. What is a frost ring?
5. What are the typical symptoms of a decline, and how do declines differ from other types of diseases?
6. What are the possible mechanisms that might explain red belt?
7. Why is frost usually the most damaging at early stages of stand development?
8. What are the likely causes of sunscald?

LESSON 2 Decay

LESSON OVERVIEW

CONTENT

This lesson deals with decay, both in living and dead trees, and, to a minor extent, in wood that is in the form of lumber (i.e., "in service").

The study of decay is also a good way of starting a course dealing with tree pathogens, because decay fungi are largely restricted to dead wood tissues (either dead trees or the heartwood of living trees), and represent one of the simplest disease situations that you will encounter.

Decay fungi play an important role in ecosystems. They recycle the carbon and mineral nutrients tied up in wood, and they create special habitats that are required for a number of organisms, from birds to nitrogen-fixing bacteria. Decay also causes great losses in value in standing timber, and it greatly limits the time that dead standing timber can be salvaged.

The content of this lesson is developed through discussion of the following six topics:

- Enzymatic degradation of wood
- Types of decay
- Ecological considerations
- Infection
- Rate of decay
- Decay and forest management

OBJECTIVES

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

1. to summarize the requirements for growth and reproduction of decay fungi, and to describe the manner in which various groups of decay fungi digest wood;
2. to outline the infection process, and to list the special infection pathways required by decay fungi;
3. to predict how fast decay can be expected to develop once a tree has been infected, and to identify which phenomena control the rate of decay;
4. to summarize the role of decay in natural ecosystems;
5. to predict, based on information about decay in natural ecosystems, where decay is likely to be a problem;
6. to apply the principles that underlie decay estimation techniques; and
7. to appraise how various silvicultural operations and management decisions can either promote or retard decay.

LESSON STUDY INSTRUCTIONS AND ASSIGNMENT

Start this lesson by reading Chapter 14 in Manion (1991). Then study the commentary below and the papers in the reading section at the end of this lesson, which include Etheridge and Craig (1976), Shain (1979), Shortle (1979) and Merrill and Shigo (1979). The commentary includes

notes to direct you to certain readings at appropriate times. In addition, study Pest Leaflets Numbers 55 and 62 (supplied with the course manual package).

At the end of the lesson, do the self-testing/review questions. These questions will help you test your understanding of the material covered in this lesson, and will also provide a good review when you are studying for the final exam.

After you have answered the self-testing/review questions, complete Assignment #1 (in Appendix A) and submit it to your tutor for marking. Remember to include a comment sheet for your tutor's use. Check your course schedule for the due date.

COMMENTARY

ENZYMATIC DEGRADATION OF WOOD

Wood consists of cellulose, hemicellulose, lignin and various extractives. Cellulose consists of long chains of sugar units strung end to end, and these chains give wood its strength. Lignin is a more complex material of indefinite structure. The conifer tracheid cell wall may be compared to reinforced concrete, with cellulose bundles representing the steel and lignin the concrete. The formation of celluloses is mediated by enzymes, and the first steps of degradation are almost the reverse of the last step of formation. This is not true for lignin, since the last step of lignin formation, the condensation of the various phenyl propane units into the complex, three-dimensional lignin polymer, involving many different kinds of chemical bonds (see Manion Figure 14-3), is apparently not mediated by specific enzymes, and cannot be reversed by enzymatic action. Hence lignin is much more resistant than cellulose to enzymatic breakdown.

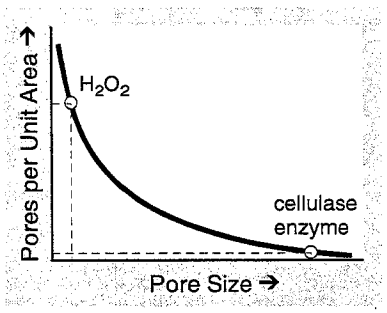

FIGURE 2.1
Distribution of pore size in lignin matrix compared to a cellulase enzyme molecule and a molecule of hydrogen peroxide.

The cellulose is at least partly protected from enzymatic degradation by the lignin in which it is embedded. The pores in the lignin matrix are too small for large, protein enzyme molecules (see Fig. 2.1). There are many fungal species that can survive and grow with pure cellulose as their only carbon source, but only a small subset of these have the peculiar ability to utilize cellulose in wood efficiently, and these are the decay fungi. Decay fungi deal with lignin in two ways. The white rots, which can digest both lignin and cellulose, slowly degrade cell walls from the inside, beginning their attack in the S3 layer of the tracheid cell wall. As the lignin is degraded and dissolved, the cellulose is exposed to enzymatic degradation. Brown rots on the other hand, do not produce enzymes that can degrade lignin. Instead, they produce hydrogen peroxide (H_2O_2), and Fe^{+++}. These small molecules can enter the pores in the lignin, and together they react with the cellulose to break the long cellulose chains into shorter segments. Once this has happened, the lignin matrix relaxes, and the pores become large enough to admit enzymes.

In white rots, the breakdown of cellulose proceeds as two related, enzyme-mediated processes. Enzymes can be named according to the substrate on which they act. Thus **cellulases** are cellulose-digesting enzymes. **Exocellulases** are enzymes that attack the ends of the cellulose chains and release soluble sugar units by hydrolysis. There are two exocellulases, since the two ends of a cellulose chain are chemically different. Since the chains are very long, there are few sites available that exocellulases can act on, and by themselves they would lead to very slow digestion of cellulose. However, white rots produce another enzyme, **endocellulase**, which breaks the long cellulose chains into smaller fragments, thus creating sites at which the exocellulases can operate (see Fig. 2.2). In brown rots the function of endocellulase is achieved by the action of hydrogen peroxide and iron.

All these enzymatic processes occur outside the living fungal cell. The enzymes involved are therefore all **extra-cellular** enzymes, produced by the fungus and released into their immediate aquatic environment. If

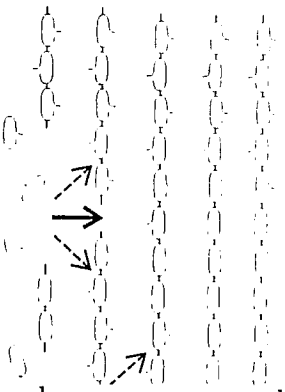

FIGURE 2.2
Long cellulose chains are broken into smaller fragments by endo-cellulases (solid arrows), creating sites where exo-cellulases can operate (broken arrows).

there is no liquid water on the cell wall surface, decay cannot proceed since there is no medium in which the enzymes can act.

The half-life of enzymes, particularly extra-cellular enzymes such as the cellulases, is quite brief. The fungus therefore controls the rate of the process through controlling the rate at which enzymes are formed and released. High sugar concentrations inhibit the production and release of endocellulase, resulting in a decrease in the number of sites at which exocellulases can operate.

TYPES OF DECAY

Decay fungi can be divided into three major groups, namely the soft-rots, the white-rots and the brown-rots, according to the type of decay they produce.

Soft-Rot

Soft-rots are Ascomycetes whose hyphae grow within the cell wall or sometimes on the inner surface of cell walls. Deterioration of the wall occurs only in the immediate vicinity of the hyphae; the enzymes apparently remain attached to the hyphal wall. Those that grow within the cell wall produce small, regularly shaped cavities within the wall. Those that grow on wall surfaces produce characteristic grooves. Soft-rot is almost wholly restricted to wood in service and is common in wet wood. There is little or no soft-rot in living trees. As the name indicates, soft-rots produce soft, spongy decayed wood.

White- and Brown-Rots

The white- and brown-rots, on the other hand, produce free extra-cellular enzymes that attack the wood material some small distance from the hyphae. The two groups are distinguished by the types of enzymes they produce, although the distinction isnít absolute; there is some gradation from one group to the other. Both groups digest cellulose and use the resulting sugar as a major energy source. In addition, the white-rot fungi produce enzymes that digest the lignin at least partially, while the brown-rots don't. Thus when white-rots decay a piece of wood, there is almost nothing left when the decay is complete; in contrast, when the brown-rots are finished, the lignin remains as a brown, crumbly material.

There are a number of ways in which the behaviour of white- and brown-rots differ, and these are mostly related to the basic difference in

the enzymes they produce and the manner in which they deal with lignin. White-rot hyphae travel from cell to cell by dissolving bore holes in tracheid walls; brown-rot hyphae can pass from cell to cell only through pits. Hardwoods are most commonly decayed by white-rots; both types of rot occur in conifers.

The initial attack of white-rots in conifers is unevenly distributed in wood. Typically advanced decay develops as small elliptical pockets (about 1 mm by 2 mm in size). This leads to a stringy or laminar decay, but, at an early stage of decay, the wood retains considerable strength. Kraft pulp yield (measured as weight of pulp produced per unit weight of wood) is also not greatly affected. Severely decayed wood is lost as fines in the chipping and screening process. Parts of the decaying wood that are at an early stage of decay, and still strong enough to withstand the chipping process without breaking into small pieces, give a nearly normal yield of pulp.

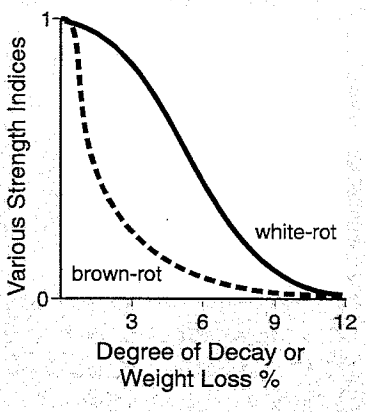

FIGURE 2.3
Comparison of loss of strength in wood infected with white-rot or with brown-rot.

Brown-rots, on the other hand, develop evenly throughout the infected wood. At a very early stage the long cellulose chains are broken into short fragments. As soon as that has happened, the strength is gone. The decaying wood is usually dark in colour and the first cracks develop across the grain, leading, in the typical case, to a cubical rot. Strength loss at early stages of decay is very rapid and pulp yield of partially decayed wood is very low (refer to Fig. 2.3).

ECOLOGICAL CONSIDERATIONS

Carbon Cycling

Decay of wood is virtually the only way in which wood is destroyed in natural ecosystems. Fire and insects kill trees but do not destroy much wood. After the tree is dead, decay fungi move in and eventually reduce the wood to carbon dioxide and water. It follows that over large natural areas (e.g., the province of B.C. a century ago) decay equals growth. The two processes are balanced, and the average amount of carbon per hectare that is tied up in wood and other organic materials is constant.

The total carbon tied up as wood in a single stand rises with stand age to some maximum and then declines to an equilibrium level in the climax forest. At the same time, the total carbon tied up as organic material in the forest floor may continue to rise with stand age, although it too may reach an equilibrium level in the climax forest. Switching from natural forests to managed forests involves a decrease in the average amount of carbon tied up as biomass in the ecosystem, particularly in those cases where the natural forests tend to be quite old at stand renewal, and under cool climatic conditions which are more prevalent on the coast than in the interior (refer to Fig. 2.4).

The rate of carbon dioxide assimilation by immature stands can be quite fast. Such stands can assimilate an amount of carbon dioxide equal to that present in the column of air above them to the limit of the atmosphere in about one year.

Currently, we cut and use wood before it decays naturally. Wood and wood products in service have a fairly short half-life, and so the return

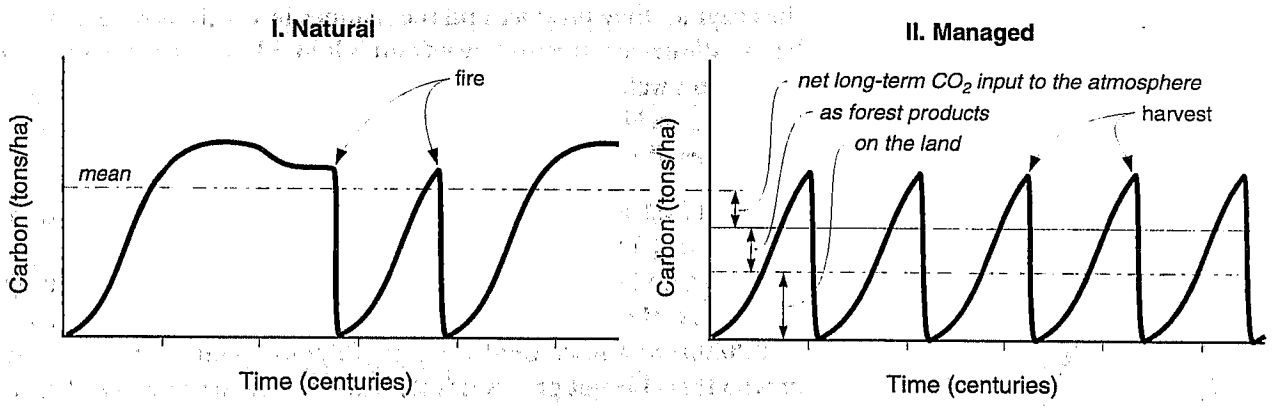

FIGURE 2.4
Comparison of amount of carbon tied up as wood in natural and in managed forests.

to the atmosphere of carbon tied up in wood that is harvested and used (for lumber, pulp, and other products) is somewhat faster than if the trees had been left in the forest to die and decay naturally. In addition, a great deal of biomass other than wood is rapidly returned to the atmosphere following logging. The overall effect of harvest therefore is to increase atmospheric carbon dioxide. Eventually a new stable equilibrium will be reached. In a fully regulated forest, carbon uptake by photosynthesis and total carbon release (largely by decay in the forest, deterioration of wood and other forest products in service, and release during manufacturing) will be in balance for all practical purposes (Fig. 2.4 illustrates these concepts). The graph labeled "I. Natural" depicts a natural forest over many centuries in which stand renewal events occur at irregular intervals; the graph labeled "II. Managed" illustrates events in a managed forest in which the rotation age is a little over one hundred years. The dotted horizontal lines represent the average amount of carbon tied up as wood in these two cases. In the case of the managed forest, one must also consider that some carbon is stored in the form of wood or wood products in service, shown by the solid horizontal line. The difference between the solid line in the managed forest graph and the dotted line represents the amount of carbon that is permanently released when such natural forests are brought under management. The magnitude of that difference varies a great deal, depending on the ecological zone and ecosystem being considered, and may even be negative (i.e., managed forests tie up more wood than natural forests) for some zones.

Habitat Creation

Partially decayed wood provides a special habitat that is required by many organisms. Cavity-nesting birds need snags or living trees with decay in which they can excavate their nesting cavities. Some small mammals also use such cavities. Decaying stumps and logs (Coarse Woody Debris) can act as nurse logs, and sometimes these are the only places where regeneration can become established. Such wood also provides essential habitat for various amphibians. Finally, decaying wood

commonly harbours nitrogen-fixing bacteria. Their action can represent a significant nitrogen input into the local ecosystem.

There may well be other significant roles of decaying wood. The difficulty is that most studies of decay have been wholly focused on the damage and loss of wood to the forest industry. Little thought has been given to possibly beneficial roles. Yet if we don't understand and appreciate these roles, our prescriptions for management may well miss the mark!

INFECTION

Spore Production

Most decay fungi belong to the Basidiomycetes, but a few are Ascomycetes. Many Basidiomycetes produce large numbers of sexual basidiospores and sometimes asexual conidia. Figure 2.5 shows how a fruiting body (conk) is constructed. The typical fruiting body has a dark, weathered upper surface, while the lower surface consists of layers of pores. Each year, or sometimes twice a year if there are distinct warm, moist seasons, the fungus grows another layer of pores and abandons the old one. The pores are lined with a layer of basidia (see Lesson 1), the layer being called the **hymenium**. When the basidiospores mature, they are shot off with a speed just enough to carry them to the center of the

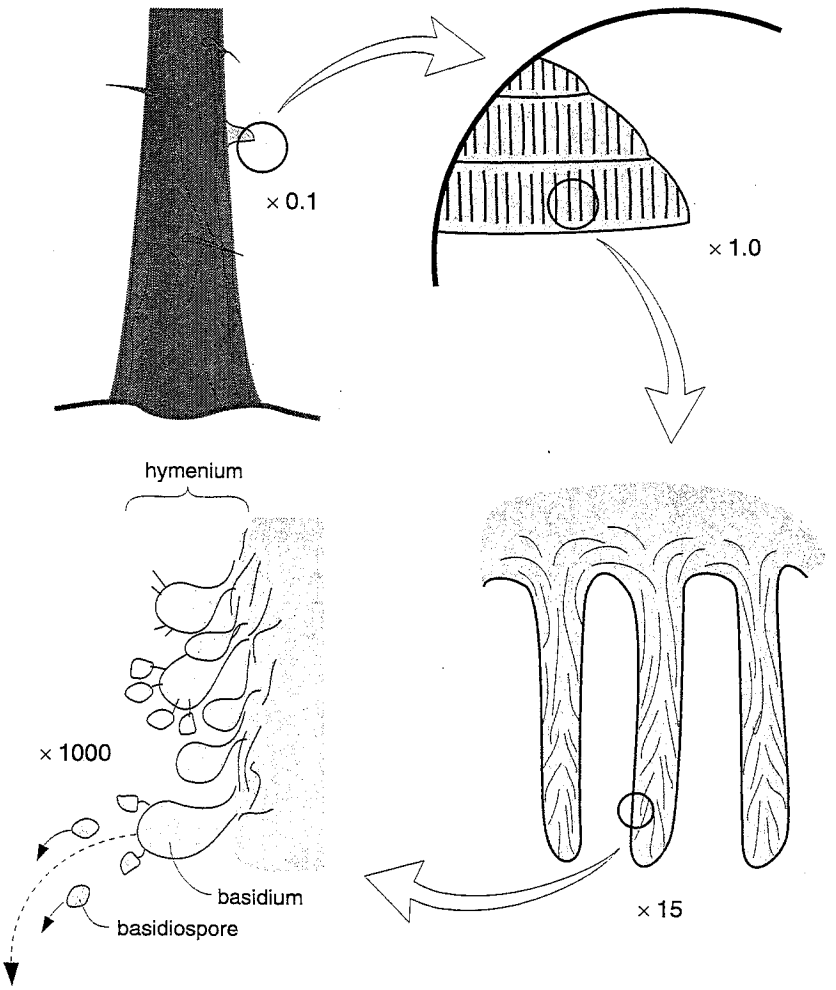

FIGURE 2.5
Progressively greater magnified views of a conk.

pore, and then they drop out of the pore under the force of gravity, and are carried away by wind. A single large fruiting body can produce tens of millions of spores per hour! So, it is not surprising that spores of decay fungi are in the air virtually year round, except during very cold or dry weather. All exposed plant surfaces receive a constant rain of decay spores. Every time you go for a walk in the forest on a warm day, you inhale several live decay spores with each breath!

Penetration of Decay Fungi

Living plant tissues (phloem and sapwood) are immune to decay (with some exceptions in the genus *Phellinus*). Thus, in living trees, only the heartwood decays. How then do decay fungi get in? Branch stubs (knots) do not function as entry courts, or, if they do, the process is very slow. Knot wood is very resistant to decay, but pruning of large-diameter, live branches can create an entry pathway, especially if some stem bark is damaged. Decay fungi can be divided roughly into three groups depending on their mode of entry: the true heartrots, wound entry heartrots, and saprot fungi.

the true heartrots

The few species in the group of true heartrots can apparently enter unwounded trees. The pathway is not always known. Good examples in B.C. are *Echinodontium tinctorium* on hemlock, and true firs; *Phellinus pini* on Douglas-fir, pine, and spruce; and *Phellinus tremulae* on aspen. Because all individuals of a host species are susceptible, decay is found in many trees in affected stands. Usually there is a critical age at which the true heartrots first appear. That age, which varies from place to place and species to species, becomes a critical age with respect to several forest management considerations.

Echinodontium Tinctorium

The case of *E. tinctorium* on hemlock is perhaps best understood. When small, tertiary branchlets close to the main stem die, they become infected with *E. tinctorium*. The fungus forms a small colony, no more than a few cubic millimeters in size, and then remains dormant. Eventually the tree bole grows around branches containing such colonies, and once the fungus finds itself well within the bole, it begins to colonize the heartwood. It is evident that all trees are susceptible, and that decay will begin to appear at a certain age or size.

reading

At this point, you should read the paper by Etheridge and Craig included at the end of this lesson on pages 55–74. This rather long paper has become a classic in work on decay infection pathways. It is included in this course material not only for its own sake, but also because it gives you a taste of the painstaking work required to establish what, in retrospect, appear to be fairly straightforward events. Moreover, you should notice that the evidence for the last stage of development of *Echinodontium* decay in hemlock, the activation of small latent branch infections, is rather scanty and circumstantial. That is typical of descriptions of many processes in forest pathology, because in most cases definitive research that establishes explanations beyond doubt has not yet been done.

Etheridge uses the term "medullary tissues" to refer to what is commonly known as the pith. You will also come across references to the fungus

Ascocoryne sarcoides, a non-decay fungus that can inhibit decay (see p. 46 under the heading "Antagonism" for a description).

wound entry heartrots

Wound entry heartrots include many species. All require wounds of some sort (scars, cracks, broken tops, or patches of dead bark killed by other parasites or sunscald, etc.) in order to gain entry to the heartwood. This group can be divided into two subgroups — primary and secondary wound heartrots. The primary wound heartrots require fresh wounds that expose virtually sterile wood in order to germinate and penetrate. *Heterobasidion annosum* is a good example. Decay fungi belonging to this group usually develop quickly once they gain entry. Wounds remain suitable for entry by this group for only a few days or weeks. Once the wound surface is colonized by micro-organisms other than decay fungi, the primary wound-invading heartrots are excluded. Secondary wound-invading heartrots continue to invade wounds for a much longer time, often following other staining fungi and bacteria. These situations are best understood as a form of succession. Various micro-organisms invade in turn, each changing the nature of the substrate (the wood) to make it suitable for the next one. The final stage will always be decay, but the length of time between wounding and the establishment of decay varies from months to decades.

Even large wounds heal over eventually, and such healing may slow the rate of decay development, possibly due to low oxygen levels. A host reaction in the sapwood may restrict decay in the case of small wounds (see below).

A special case of a wound-invading decay fungus is that of a *Amylostereum chailletii,* the spores of which are injected into wood of trees by a *Sirex* woodwasp together with its eggs. The larvae feed on the decaying wood, apparently relying on the fungal enzymes to digest the wood for them. Balsam fir and radiata pine are commonly attacked in this way. The woodwasp is initially attracted to the tree host by small wounds.

saprot fungi

The final group of decay fungi is restricted to dead trees, which include logs and slash. Once a tree dies, the sapwood becomes very susceptible to decay. Some very specific associations develop. Thus the sapwood of dead standing hemlock in south coastal forests is almost always invaded by *Trichaptum abietinum,* while the heartwood of such trees is destroyed by *Fomitopsis pinicola.* T. *abietinum* produces large numbers of small, purple to white fruiting bodies on the bark within two or three years of tree death, while *F. pinicola* produces its larger, perennial fruiting bodies some years later. *Cryptoporus volvatus,* closely associated with bark beetle galleries in various conifers, decays the recently killed sapwood. Other species are commonly found on slash.

RATE OF DECAY

Even if decay fungi manage to gain entry into the heartwood of living trees, it does not necessarily mean a great deal of decay. The rate at which decay fungi develop in heartwood columns varies widely. Maximum rates of invasion, measured longitudinally, can exceed one meter

per year, but may be as little as one centimeter per year. Radial and tangential movement is always slower than longitudinal movement, but these vary just as much. The rate becomes an important determinant of the eventual loss.

Physical and chemical factors have some effect on the rate of decay. Thus the rate is controlled in part by temperature, moisture content, mineral nutrient content of the wood (wood is particularly low in nitrogen), pH, and the presence of natural toxins. The effect of such factors has been well studied in wood test blocks *in vitro* (literally "in glass" - meaning in the lab away from living trees), but predictions based on such studies do not match well with observed rates of decay in living trees. For instance, western redcedar heartwood contains several potent toxins such as the thujaplicins and a complex set of water soluble phenolics, and redcedar wood is widely promoted as having considerable decay resistance. Nevertheless, the total volume of accumulated decay in living trees is greater for redcedar than for any other conifer in B.C. This is only partially attributable to the greater average age of redcedar. Anomalies such as these abound.

Other mechanisms play an important role. They include the factors discussed below, although more may be discovered as the biology of decay is studied in greater detail.

Antagonism

Old undecayed heartwood is seldom sterile. One can usually find various species of non-decay fungi, bacteria, and yeasts. These organisms live on some of the extractives and perhaps some hemicellulose. Attempts to inoculate living trees with decay fungi through holes drilled in the trunk usually result in the stimulation of these non-decay fungi, and only seldom lead to decay. Sometimes the organisms living in wood are antagonistic to decay fungi, meaning that they inhibit or prevent the invasion by decay fungi. A good example is certain strains of *Ascocoryne sarcoides.* This fungus is occasionally found in the heartwood of *Abies, Tsuga* and *Picea.* Some strains provide very good protection against decay, even after the tree dies, as long as the heartwood remains moist. Little is known about such antagonistic fungi. It is not sure how they get into trees, or why some trees in a stand are protected from decay by their presence, while they are absent in others.

There is a potential use of fungi such as these to provide decay resistance to wood in service (e.g., in wooden utility poles). The current chemical treatments provide protection, but among the chemicals commonly used to treat wood, some (such as chromium and arsenic) are environmentally dangerous. Some of these toxic chemicals always leach out of treated wood, and when the wood eventually decays, the remainder is released. The problem so far for using these fungi has been to get a consistent response. To be useful, a treatment must be nearly 100 percent reliable. That has not been achieved so far by treatment with biologicals. A similar situation obtains in the development of anti-stain treatments in lumber.

Destruction of Toxins

The heartwood of many tree species contains toxins that inhibit or stop the development of decay. Western redcedar heartwood is well known for its high decay resistance, attributable to thujaplicins and water soluble phenolics. In the case of this species, the heartwood of living trees is commonly invaded from the base and center of the trunk outwards by a succession of fungi (see Fig. 2.6). The first one to invade, a species of *Sporothrix*, converts the thujaplicins into a non-toxic dimer (a molecule derived from two thujaplicin molecules joined at their active end). In the process it turns the normal straw-coloured heartwood a pink or red to purple colour. Once this has happened, a second fungus, *Kirschteiniella thujina,* follows, and possibly together with *Sporothrix* detoxifies the water soluble phenolics. Then yet more species appear, and the wood turns various shades of brown. Finally the decay fungus shows up. It is found in a narrow zone (about 1 cm wide) along the edge of a central decay column. Within the decay column, the decay fungus can no longer be found. In turn it is replaced by a complex microbial community resembling that found in forest floors. The whole process can be regarded as a form of succession in which each succeeding sere changes the environment (heartwood chemistry) to prepare it for invasion by the next. The process is so effective that it largely negates the supposed durability of redcedar heartwood.

Anaerobic Conditions

All decay fungi require oxygen. In some tree species, notably western black cottonwood, normal heartwood is completely replaced by wetwood. Wetwood is nearly saturated wood occupied by a complex community of bacteria which may include nitrogen-fixing species. It has a high pH.

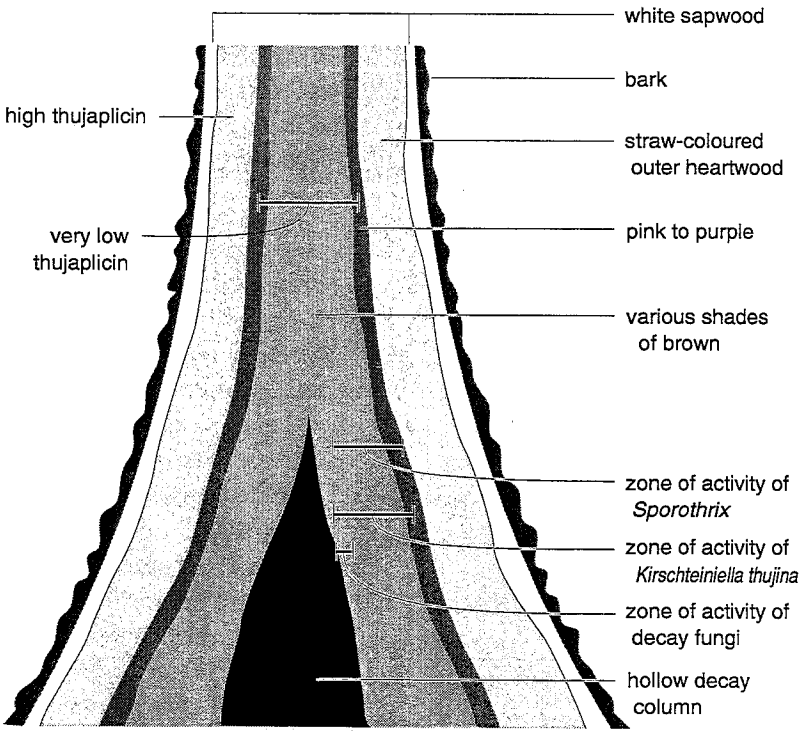

FIGURE 2.6
A vertical section through a large old western redcedar.

Sometimes it is under pressure and it may contain considerable amounts of methane. It is also essentially anaerobic (i.e., without oxygen), and it is this property that stops decay. As long as the tree trunk is free of large scars, the bacterial community uses the little oxygen that diffuses in from the sapwood, and the wood remains free of decay. Once a large, deep scar or crack is formed, too much oxygen is admitted, and the anaerobic condition is lost. Then the tree becomes very susceptible to decay. Wetwood is found in several species of hardwoods, such as aspen and elms and willows, and also occurs in hemlock and true firs, although seldom in large columns that occupy the heartwood completely. In all such cases, some decay resistance is imparted. Wetwooleads to problems in lumber drying during the manufacturing process, and for that reason is considered to be detrimental for some purposes.

Nitrogen Fixation

Nitrogen concentration of heartwood is very low and nitrogen is often a limiting factor for the rate of decay. Decay fungi require nitrogen not only for regular protein formation, but also to build their chitin cell walls. Decay fungi have special adaptations for growth in a low-nitrogen environment. Among other things, they can digest their own old hyphae and recycle the nitrogen that would otherwise be tied up in them. Enriching wood with nitrogen usually increases the rate of decay. Some decay fungi produce within their columns of partially decayed wood a set of conditions in which nitrogen-fixing bacteria can flourish. The nitrogen so produced is thought to be partly absorbed and translocated by the decay fungi to the sites of decay activity, thus presumably increasing the rate of decay. Eventually, of course, that nitrogen enters the nutrient cycle and is used by all the living organisms in the ecosystem.

Host Reactions

There is no direct host reaction to decay of heartwood, apart from some evidence for increased radial growth (appearing as butt swell) to compensate for loss of strength of the bole. However, trees react to wounding, and that reaction can have a major influence on the development of decay caused by agents entering through wounds. Two major types of barrier protection occur:

sapwood wounds

If sapwood is wounded, the tree reacts by forming a barrier between the wounded sapwood and the remaining functional sapwood. That barrier forms only during the growing season. When fully formed, usually within a few weeks of wounding, it is impervious to gases and liquids, and also serves to block the advance of decay and stain. The precise chemical and physical structure of that barrier has not yet been determined. In small, shallow wounds, the barrier may surround the wound completely and block the entry of decay fungi into the heartwood. In subsequent years, the wound heals by formation of a callus. That callus is initiated by the cambium at the point at which the barrier meets the cambium. In such cases little or no decay results. In larger or deeper wounds, the barrier still forms. but now it forms a ring around the wound extending from the cambium to the heartwood-sapwood boundary, and decay can enter the heartwood. Thus, small, shallow wounds do not

usually lead to decay, whereas large (large in this context is greater than 5–10 cm tangentially, while longitudinal extent matters little) or deep ones often do. This mechanism of barrier formation occurs in both conifers and hardwoods.

CODIT

In hardwoods, a second phenomenon known as Compartmentilization Of Decay In Trees (CODIT) occurs. In the case of large wounds, the tree forms an unusual annual ring that extends throughout the whole tree (roots, bole, and branches) in the year of wounding. The unusual ring consists of a dense layer of wood impregnated with tannins and other materials. As the tree grows, the ring becomes embedded in the sapwood and eventually heartwood. Decay fungi are unable to penetrate the ring. Thus any fungi entering through the wound are restricted to wood that was laid down before the time of wounding; they cannot penetrate the ring and invade wood formed later. After many years, the tree in question may develop a column of decay that has the exact shape of the tree at the time of wounding, with a very sharp boundary between decay and normal wood. A similar ring is formed in conifers, but it does not extend more than a couple of decimeters from the wound edge, and may not even continue around the tree.

These various phenomena, and possibly many more, determine to a great extend how rapidly decay is going to develop. Whether or not they influence the process depends on the tree species, the age, and the ecological position of the tree, as well as on such random events as scarring and frost cracks. It is not surprising, then, that the amount of decay in mature and overmature stands varies a great deal.

reading

At this point, you should read the three papers included at the end of this lesson, beginning on page 75. These papers are taken from a symposium on decay published in the journal *Phytopathology* (a U.S.A. journal devoted to the science of plant pathology). These papers were chosen because they give a good indication of the state of affairs in the study of decay. It isn't difficult to show that all sorts of things are going on in living trees invaded by decay fungi; there are physical and chemical changes in the wood, as well as changes in the microbial populations active in wood. The precise nature of these changes, however, often depends on the host species and the identity of the decay fungus involved. Also, contradictory results are not uncommon. A major research goal is to arrange the very large body of data (observations of all kinds from many places around the world, and under a variety of circumstances and conditions) into a coherent system in which cause and effect can be shown.

In their paper, Merrill and Shigo point out, quite correctly, that simple models that are concerned only with dead wood and a single decay fungus (typical of the classical model of study) are inappropriate. I have made a similar argument above, showing that *in vitro* studies do not provide the required predictive ability. Both the Shortle and the Shain papers, by proposing broad, generalizable concepts, attempt to help us identify the significant underlying processes. Such an approach is essential: it cannot be said that we have an understanding of the processes unless we can arrange all the observations into reasonable patterns that make sense biologically and that identify chains of causes and effects. In fact, the essence of science is just such an activity; mere

observation and recording of what's going on in particular instances is only the starting point!

You will notice, however, that both Shain and Shortle leave a lot of loose ends — many parts of their proposals need further refinement. Don't let that disturb you, because it's the usual state of affairs. Scientists attempt to explain events in the simplest terms possible, but must continuously "fine-tune" these generalities to account for all observations.

Next time you have a chance to observe a set of decayed logs, take some time to examine the various patterns of stained and decayed wood and the apparent barriers that seem to limit decay. Notice how commonly there are islands of stained but sound wood within decay columns. Ask yourself what has gone on. It's a necessary but humbling experience.

Decay and Forest Management

Estimating the Volume of Decay

The only effective way of measuring the amount of decay in standing trees is by destructive examination. Every year new instruments come on the market that are supposed to be able to measure decay in living trees without boring into them or cutting them down. Some are based on differences in sound transmission through normal and decayed wood; some on electrical resistance; and some on deflection under mechanical forces. You may be told that the latest version of the instrument will do the job faultlessly, but be cynical — perhaps some day such an instrument will be developed, but those currently available are at best reliable only in very limited situations. Normal variation in moisture content, wood density, ring width, defects such as knots and cracks, and islands of stain often confuse the situation and lead to faulty readings. Also, all such instruments tell you only about the state of the tree at or below DBH (unless you climb the tree with the instrument).

Estimates of decay volume are made by using **cull factors.** Cull factors are derived by making the following observations:

- All trees in representative small plots are examined for signs (fruiting bodies), or indicators (large scars, broken tops, cracks, etc.) of decay.
- They are then divided into two groups: "suspect" (bearing such signs or indicators) and "residual" (without visible signs of decay). The former group may be subdivided depending on the kind of indicators that are present and their height.
- All trees are then felled and bucked into small sections, and the volume of decay determined.
- From this information, cull factors are calculated for each group.

Each tree in a timber cruise is examined for signs and symptoms of decay, and an appropriate cull factor is applied. Cull factors vary widely depending on species, age and geographic location.

Cull factors give an estimate of the actual volume of decay, but the losses may be much greater because the lumber recovery from logs with a central decay column is greatly reduced. In fact, logs in which more than half of the volume is decayed are usually regarded as cull and left in the woods even though there is some sound wood, because it is uneconomic to produce lumber from these logs.

Age of Tree and Decay

Significant decay usually does not develop until a particular age that is characteristic for the species and site. The decay fungus involved is usually a true heartrot. That age determines the length of time that timber volumes can be "saved on the stump" without significant deterioration; however, insects must also be considered. Some rough critical ages are: 150–175 years for interior spruce; 100 years or less for interior subalpine fir; about 150 years for lodgepole pine; 200 or more years for coastal hemlock but less than 100 years for interior hemlock and cedar; 300 or more years for coastal Douglas-fir; less than 50 years for aspen in much of the interior. These are only rough estimates and they vary somewhat from place to place.

When an overall timber supply area harvesting plan is developed, this kind of information can be very useful because it tells the planner which stands are likely to deteriorate rapidly, and which will retain their value for a considerable time. Thus the likelihood of decay can be an important determinant of the sequence in which various forest types and age classes should be harvested.

Following are two examples that illustrate the importance of decay in management decisions. In the early 1960s several pulp mills and sawmills were built near Prince George, and the rate of harvesting increased rapidly. East of Prince George were large areas of old, valuable spruce; to the west were mostly smaller and younger pine. The spruce was about 150 years old and could not be expected to get much older without significant decay developing. For that reason, the decision was made to concentrate logging in the spruce forests. Later, outbreaks of spruce bark beetle speeded the logging in that area even more. The alternative, namely, to harvest both the spruce and the pine at sustainable rates was (quite rightly) rejected because it would have led to significant losses in value of spruce before it could be harvested.

The second example concerns interior hemlock, which is known to develop significant decay at an early age. Until very recently, it was not acceptable as regeneration in much of the interior cedar hemlock ecological zone. Recent studies, however, suggest that decay is minimal before about age 90. It thus appears that hemlock can produce an acceptable crop after all, leading to a major change in silviculture in the zone. For now, the use of advanced regeneration remains uncertain, and, without further studies, it should probably not be accepted. Interior western redcedar appears to be a similar situation, although we do not have enough data for a thorough assessment.

Decay in Immature Stands

Decay will become less common as our forests come under management. Nevertheless some decay will continue. Scarring caused by various forestry operations is an important factor. Much of the damage can be prevented by reasonable care and appropriate timing (e.g., by avoiding operations in early summer when the cambium is very succulent). A great deal also depends on the tree species. Douglas-fir and lodgepole pine can withstand considerable scarring when young and develop little decay. Other species such as hemlock and true firs are more easily

infected. Again this also varies from region to region, and local experience is the best guide.

Decay of Dead Standing Timber

Stands killed by fire or insects can be salvaged. The rate of deterioration depends on temperature, moisture, species, and the wood-utilizing insect population. On the coast, small trees (i.e., DBH less than 25 cm) can deteriorate within a few years, but for larger trees a rough rule of thumb is a loss of 2–4 cm radially each year. In the interior and at high elevations, decay of dead standing timber can be much slower.

Decay in Parks and Recreation Areas

Old decayed trees add to the variety of habitats available in ecosystems, and thus are often desirable in parks. However, public safety needs to be considered and high use areas such as parking lots, picnic areas and campsites should be checked for dangerous trees. On the coast a fungus of particular concern is *Phellinus tsugina* on hemlock and true fir. This species is able to invade and decay sections of sapwood in living trees. Where that happens, typical fruiting bodies are often produced. Such trees are very weak and commonly break at the decay. Wherever *P. tsugina* is spotted in high use areas, the affected tree should be removed.

Decay in Wooden Structures

The single effective rule is: "Keep it dry" (i.e., keep moisture content below the fiber saturation point), and this is achieved by appropriate design of wooden structures. If exposure to moisture cannot be prevented, treated wood should be used. Another approach is to exclude oxygen, which happens when using wooden pilings in wet soils. Notice that prevention of decay usually involves creating a set of conditions in the wood that do not allow decay fungi to grow (i.e., conditions are too dry, too toxic, no oxygen, etc.). It is not feasible to prevent spores of decay fungi from landing on wood because such spores are present everywhere in the air. Various types of paints and coatings seldom serve a useful purpose. Sooner or later they crack or peel, and decay enters.

All of the processes discussed above can be illustrated with the example of a telephone pole. The base of the pole, buried at least two meters below ground, is always water saturated. It remains sound because the rate of diffusion of oxygen in water-soaked wood is very slow, so that the wood at the base of the pole is essentially anaerobic. The top of the pole is generally too dry for decay fungi. There are of course periods when the whole pole is wet, but during periods of extreme drying, decay fungi cannot survive. Between these two regions, at ground level, conditions are just great for decay. Enough water wicks up from the soil to keep the pole moist year round. If a telephone pole decays, it always happens at ground level.

Since all this is well known, poles are always pressure treated with a wood preservative. Various preservatives have been used over time. The earliest was creosote. This was replaced in the 1950s and 60s with pentachlorophenol. More recently the preservative of choice is copper-chrome-arsenic (CCA), which gives the pole a green colour. These preservatives can be forced into the wood to a depth of about 3–6 cm

depending on species and the amount of sapwood left on the outside of the pole. The preservative-treated zone remains sound for many decades. However, as the pole dries, radial cracks appear, and these extend into the untreated interior of the pole. Such cracks form entry pathways for the spores of decay fungi, and decay begins in the interior of the pole. Thus, 10–20 years after installation, poles typically will look sound on the outside, but a good proportion of them will have some internal decay, some of them extensively.

SECTION READINGS

READING 1 *[pp. 55–74]*
Etheridge, D.E. & Craig, H.M. 1976. Factors influencing infection and initiation of decay by the Indian paint fungus (*Echinodontium tinctorium*) in western hemlock. *Can. J. For. Res., 6:* 299–318.
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READING 2 *[pp. 75–34]*
Shain, L. 1979. Dynamic responses of differentiated sapwood to injury and infection. *Phytopathology, 69:* 1143–1147.
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READING 3 *[pp. 23–34]*
Shortle, W.C. 1979. Mechanisms of compartmentalizations of decay in living trees. *Phytopathology, 69:* 1147–1151.
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READING 4 *[pp. 23–34]*
Merrill, W. & Shigo, A.I. 1979. An expanded concept of tree decay. *Phytopathology, 69:* 1158–1160.
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Factors influencing infection and initiation of decay by the Indian paint fungus (*Echinodontium tinctorium*) in western hemlock

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Sporulation by the Indian paint fungus was maximal during cool, wet periods in the fall. Basidiospores were viable throughout the year, but maximum germination occurred only after temperatures had fallen below 0 °C. It is postulated that infection of western hemlock (*Tsuga heterophylla*) occurs in spring when a single basidiospore comes in contact with the stub which remains after shade-killed branchlets, about 1 mm in diameter, are broken off at their base. Anatomical studies of branch stub formation showed that this stage is reached around age 40 and that residual stubs must be exposed almost 2 years before they can serve as infection courts. After stub closure, the fungus becomes dormant and can survive in the medullary tissues for up to 50 years or more without causing decay. The possibility is discussed that conditions associated with large branch stubs and other deep-seated injuries, such as logging scars, broken tops, or frost cracks, are responsible for reactivating dormant infections and initiating the decay process. Clarification of the infection mechanism explains observed variations in severity of decay caused by *E. tinctorium* in different forest associations and provides a simple method, based on host age and stem-ring patterns, for estimating the decay threat in individual trees and stands, without extensive, destructive sampling.

ETHERIDGE, D. E., and H. M. CRAIG. 1976. Factors influencing infection and initiation of decay by the Indian paint fungus (*Echinodontium tinctorium*) in western hemlock. Can. J. For. Res. 6: 299–318.

La sporulation du champignon responsable de la carie brune filandreuse (*Echinodontium tinctorium* E. & E.) était maximale durant les temps froids et humides de l'automne. Les basidiospores étaient viables toute l'année, mais la germination maximale se produisait seulement à des températures inférieures à 0 °C. Les auteurs postulent que l'infection de la pruche occidentale (*Tsuga heterophylla*) a lieu au printemps, lorsque qu'une seule basidiospore se dépose sur les chicots d'environ 1 mm de diamètre qui subsistent après le bris des rameaux morts à cause de l'ombre. Selon des études anatomiques de la formation des chicots, cette basidiospore se dépose sur des arbres âgés d'environ 40 ans, et les chicots résiduels doivent être exposés à peu près 2 ans avant qu'ils puissent servir de sites d'infection. Après la fermeture des chicots, le champignon devient dormant et il peut survivre dans les tissus médullaires 50 ans ou plus sans causer de carie. Les auteurs discutent la possibilité que les conditions qui prévalent dans les gros chicots et les blessures profondes telles que les cicatrices de blessures causées par les opérations forestières, les cimes cassées ou les gélivures entraînent le déclanchement du mécanisme d'infection et l'amorce de la carie. La connaissance du mécanisme d'infection permet d'expliquer les écarts de sévérité de carie causée par *E. tinctorium* dans différents types de peuplements forestiers, et elle fournit une méthode simple, fondée sur l'âge de l'hôte et les caractéristiques des cernes annuels de la tige, en vue d'estimer le danger de carie dans les arbres et les peuplements, évitant ainsi les échantillonnages destructifs élaborés.

[Traduit par le journal]

Introduction

Although many studies have been made on the biology and ecology of the Indian paint fungus, none have satisfactorily explained the observed differences in host susceptibility which characterize different forest associations. Thomas (1958) considered low host vigor to be the principal factor distinguishing associations where disease levels were high from associations where disease levels were low on the

[1]Revised manuscript received April 9, 1976.

assumption that branch stubs served as infection courts and these were retained longer on non-vigorous trees. This explanation was questioned by Maloy (1967), who found disagreement in the literature concerning the mode of entry of this fungus, claiming that most papers citing specific entry points were based on unverified observations.

Studies of infection courts for *E. tinctorium* have been complicated by lack of information on the early stages of decay. Most studies implicating branch stubs and trunk wounds were carried out in old trees where decay had been established for many years, precluding tracing infections to their point of entry. The question arises as to whether infections are established in trees before injuries occur and then extend to the proximity of scars or branch stubs without the fungus having used it as a means of entry. That wounds do not provide points of entry for this fungus receives some support from recent studies (Smith and Craig 1970; Maloy and Gross 1963; Maloy and Robinson 1968; Hudson 1972) which show that while other decay fungi, notably *Haematostereum sanguinolentum* (Fr.) Pouzar, are frequently recovered from open wounds on young host trees, *E. tinctorium* is rarely recovered until trees are much older and the wounds and decay have callused over. The possibility that wounds stimulate the development of established *E. tinctorium* decay is supported by the early studies of Meinecke (1916), who found that frost cracks greatly increased the vertical spread of the fungus, which he attributed to improved aeration.

These questions became of considerable interest after the discovery that basidiospores of *E. tinctorium* could be successfully germinated on the freshly exposed wood of living, but not of dead, branches of western hemlock (Etheridge *et al.* 1970). Branches, after death, were rapidly colonized by saprophytic microorganisms. making them unsuitable for this fungus. The low moisture content also rendered dead branch stubs unsuitable as avenues of entry. In 1972, Etheridge *et al.* showed that *E. tinctorium* was capable of infecting western hemlock through branchlet stubs less than 1 mm in diameter. These stubs were frequently formed on living shade-suppressed branches of the host after about 40 years of age, providing numerous potential entry points for the fungus. After closure of the branchlet stubs, the fungus became dormant and apparently could exist in this state for 50 or more years without causing visible decay or discoloration.

Despite these advances, we lack knowledge of events leading to the formation of infection courts, their role in relation to host susceptibility, and factors responsible for reactivation of the fungus and the initiation of the decay. Objectives of the present study were to elucidate factors affecting these processes and to assess their significance in terms of host and stand susceptibility. Stand susceptibility was interpreted in the broad sense of the forest association or habitat, as defined by Thomas (1958). Complementary studies were undertaken on basidiospore dispersal and germination to determine their significance in relation to the infective period.

Materials and General Methods

Study Areas

The principal study areas were located in predominantly hemlock forest associations near Hidden Lake, in the southeast interior region of British Columbia.[2] and at Nitinat on Vancouver Island (V.I).[3] Study material was also obtained in the Upper Columbia Region near Revelstoke (susceptible) and at Lake Cowichan, V.I. (non-susceptible). The Hidden Lake stand consisted of overmature, severely infected, fire residuals that bore fruiting bodies of the Indian paint fungus and younger codominants (130 years old) with no visible signs of decay. The disease-free area at Nitinat was located in a 100- to 130-year-old hemlock stand which presumably had originated as advance regeneration after logging.

Spore Discharge and Germination

Maximum and minimum daily temperatures. relative humidity (weekly chart recordings). and precipitation (accumulated weekly collections) were obtained during spore discharge studies in the Hidden Lake area according to standard meteorological procedures. from April 22 to October 3. 1969. from April 27 to May 31. and from September 13 to December 6. 1970. Weather data were obtained during complementary spore dis-

[2]Representative of the following 'susceptible' habitat where *E. tinctorium* is known to occur in relative abundance (Thomas 1956): *Tsuga heterophylla* (*Thuja plicata* — *Pinus monticola*) — *Pachystima myrsinites* — *Vaccinium membranaceum* — *Calliergonella schreberi.*

[3]Representative of the following 'non-susceptible' habitat where *E. tinctorium* is rare or absent (Thomas 1958): *Tsuga heterophylla* (*Pseudotsuga menziesii*) — *Gaultheria shallon* — *Polystichum munitum.*

TABLE 1. Comparison of events in branchlet stub (<2 mm in diam) formation and closure in understory and codominant sterns of western hemlock from the study area at Hidden Lake in relation to susceptibility to infection by *E. tinctorium*

Stem dominance class	Estimated susceptibility [a] of stub	No. stubs	Mean stub diam, mm	Mean age at death	Mean age [b] at onset of closure	Years required [c] for completion of closure
Understory	(+)	10	0.808	12.2a	43.9a	1.8a
Understory	(–)	11	0.62b	13.8a	40.3a	1.1b
Codominant	(–)	16	0.83a	22.3b	32.4a	0.6b

NOTE: Means followed by a letter in common are not significantly different (Newman-Kuels multiple comparison test: $P = 0.05$).

[a]Based on isolation results, (+) = colonized by *E. tinctorium* and (–) = *E. tinctorium* absent.

[b]Age at death plus number of years until stubs become flush with main stem.

[c]Number of years to cover 'flush' stubs = $Ds/2Rw$, where Ds = diam of stub (mm) and Rw = mean width of annual rings at distal end of stub.

charge studies in the Vernon area from October 29, 1969, to April 26, 1970.

Spores were collected on microscope slides covered with a thin film of Vaseline located about 1 cm below two or three naturally occurring sporophores of *E. tinctorium*; slides were collected weekly with the weather data. Spore deposits were examined with a stereo microscope using low-power magnification on transits across the top, middle, and bottom of the slides. Density was recorded as trace (T) (< 500 spores/mm^2), light (L) (> 500 < 1500), medium (M) (> 1500 < 3000), and heavy (H) (> 3000).

Aqueous spore suspensions for germination tests were prepared by immersing the tips of two or three spines from the selected sporophore in a small quantity of sterile distilled water or by adding water to a spore cast on glass. They were then dispensed on films of water agar either directly on microscope slides or on samples of host heartwood and incubated in moist chambers. Germination of free-cast spores was also made on agar films which had been installed beneath the actively discharging sporophore for a suitable period. Counts were based on three replicates, each of 100 spores. Spores were considered to have germinated if a distinct germ tube was present after 48 h.

Infection Court

To study factors affecting the formation of infection courts, 30-cm sections of stems and branches of western hemlock from appropriate study areas, after having the bark removed. were dissected with a band saw, either longitudinally at 1–2 cm and 3–4 cm from the centre so as to expose the pith of all internodal branchlets or transversely to produce discs 1- to 2-cm thick so as to expose the entire encased portion of the branchlets. Branchlets were tallied according to diameter (smaller or greater than 2 mm), exposed or overgrown, and living or dead. Ring counts were made to determine the age of the section; the diameter (inside bark) was measured at the same point to obtain an estimate of radial growth rate. Overgrown branchlet stubs in the diameter range of 0.6 to 1.5 mm were characterized according to diameter, age at death, and number of years until closure. The rate of closure was calculated according to the formula in Table 1.

Microbiological Sampling

Isolations were attempted from stem and branch sections on 1.25% malt agar slants by aseptically taking small chips of wood from freshly exposed longitudinal surfaces. All chips included a portion of the pith, except when otherwise noted. Branchlet stubs were sampled aseptically by attempting isolations from cross sections exposed by two longitudinal cuts (1–2 cm and 3–4 cm from the centre of the branch or stem), usually at two points along their axes. Slants were examined after 2–3 weeks incubation at 21 °C; sterile slants were kept a further 3–4 weeks since the recovery of *E. tinctorium* from chips was sometimes achieved only after 5 or more weeks of incubation.

Results

Factors Affecting Dispersal and Germination of Basidiospores of Echinodontium tinctorium

Discharge of spores of *E. tinctorium* at Hidden Lake and Vernon occurred when average daily temperatures ranged from 40 to 60 °F (4.5 to 16 °C) (Fig. 1). Maximum discharge occurred during and immediately after periods of rain from late summer when weekly temperature minimums first dipped below 40 °F (4.5 °C) to late fall when mean daily temperatures fell to 32 °F (0 °C). Spore discharge resumed sporadically, but in trace amounts, when mean daily temperatures were above freezing, from early February to early May. A substantial increase in spore discharge occurred during the 2nd week of May in 1969 and 1970 without notable change in temperature or rainfall from the previous week but fell off abruptly and ceased entirely during June, July, and August, when temperature lows were about 40 °F (4.5 °C).

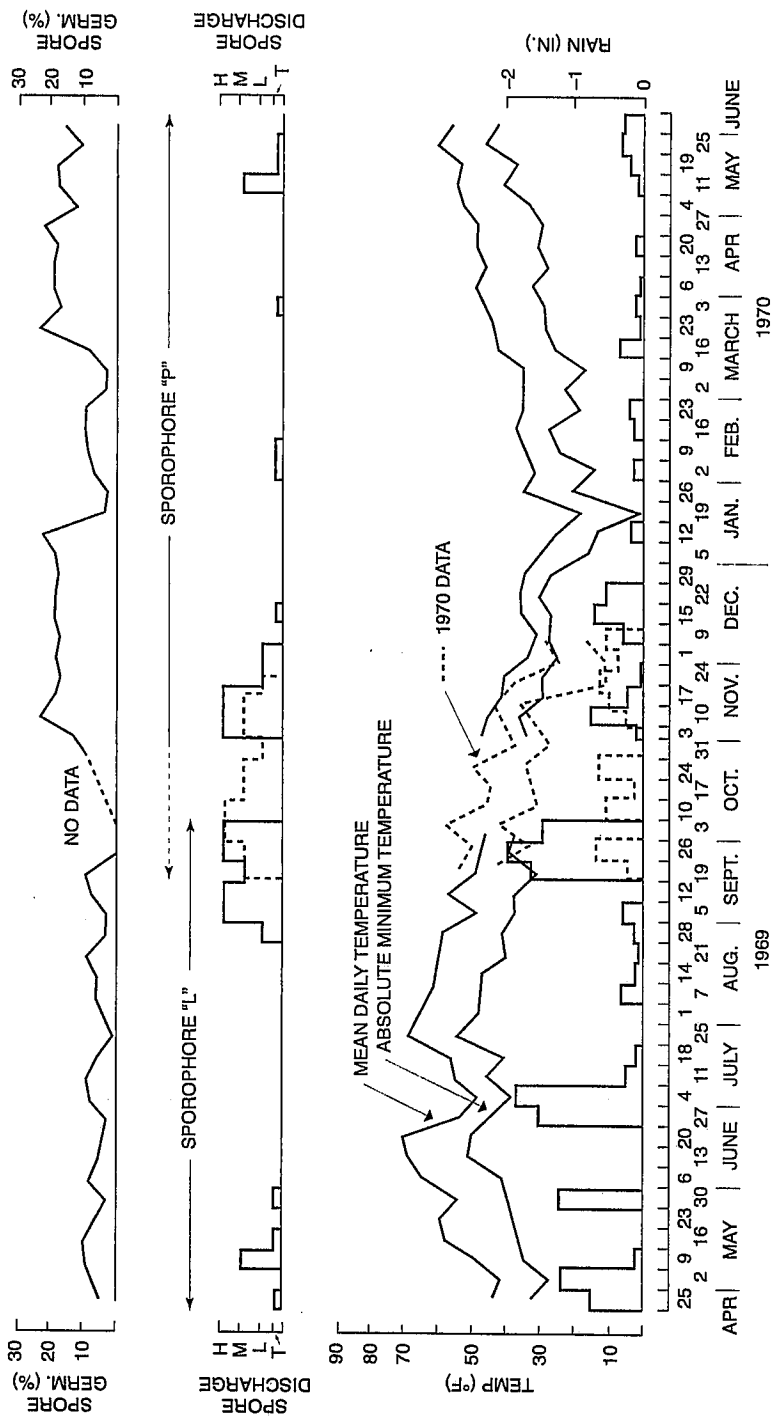


FIG. 1. Discharge and viability of basidiospores of *E. tinctorium* in relation to mean daily and absolute minimum temperature and to precipitation at Hidden Lake, B.C. (April 25 to Dec. 9, 1969, including 1970 data (broken line) and April 27 to May 31, 1970) and at Vernon, B.C. (Oct. 29, 1969, to April 26, 1970).

In laboratory tests, heavy spore discharge was obtained at relative humidities as low as 85% at temperatures ranging from 9 to 20 °C. However, this heavy discharge lasted only a few days.

Germination tests (on fresh host heartwood) were performed with spores from spines collected weekly during the spore-trapping surveys (Fig. 1) to determine seasonal variations. Maximum percentage germination (15 to 25%) occurred during late fall (November and December 1969) and early spring (March and April 1970). A marked reduction in germination (5 to 10%) occurred in spore samples collected in midwinter (January and February 1970) and from late spring to early fall (May to October 1970). However, spore viability was demonstrated during every month of the year.

Tests, using free-cast spores on agar films on glass, collected July 1972 and stored for a minimum of 8 months at temperatures of –10 °C, gave germination values of 80 to 100%. Germination tests were not made with free-cast spores from sporophores that had not received the cold treatment. However, numerous previous tests with spores from spines not exposed to low temperatures rarely gave germination values greater than 10%.

The Formation and Distribution of Infection Courts

Etheridge *et al.* (1972) demonstrated that the exposed ends of small (less than 1-mm diameter), embedded branchlet stubs serve as specific avenues of entry of *E. tinctorium* in western hemlock. Infection occurs just before stubs are overgrown, after which the fungus enters a state of dormancy. Based on the number of annual rings occurring in the overlying mantle of wood, dormancy may last up to 50 years or more before decay is initiated.

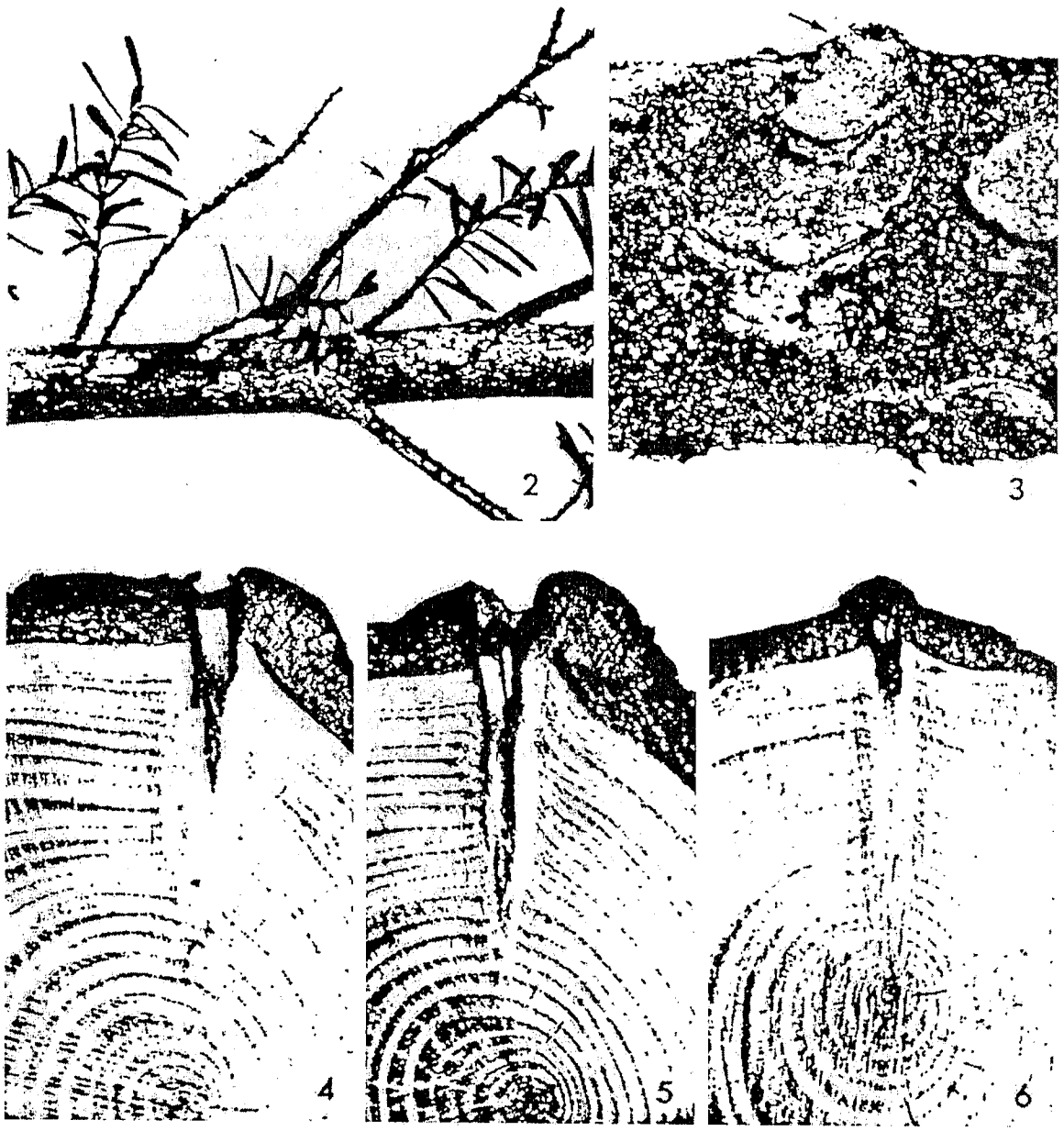
To elucidate events leading to the formation of infection courts, 10 embedded branchlet stubs (from branches sampled at Hidden Lake) which had yielded *E. tinctorium* were characterized according to diameter, age at death, and age at the onset of closure (Table 1). The mean diameter of the infected stubs was 0.80 mm; the mean age at death of the stubs was 12.2 years, while the age at the onset of closure, presumably the start of the infective period, was 43.9 years. On the average, 1.8 years were required to complete the closure process. Examples of these events are shown in Figs. 2 to 6. It can be seen that the actual points of entry for this fungus are the exposed stubs of shade-killed branchlets which have broken off within the cup-like depression formed by callus tissue at their base.

Etheridge *et al.* (1972) demonstrated that the frequency of dormant infections of *E. tinctorium* increased progressively with increasing age after 40 years; branches in younger age classes were consistently free of this fungus. To provide more detailed information on the relationships between branch ages, the formation of infection courts, and infection by *E. tinctorium,* 13 branches, ranging from 22 to 93 years, were dissected tangentially just above the pith to expose cross sections of all secondary branchlets and were systematically sampled for the presence of microorganisms. Another longitudinal cut was made to sample the medullary tissues. An average of 80 isolations were attempted from each branch. Age was determined from ring counts at appropriate points along the primary branch axis. Figure 7 is a schematic representation of an infected branch constructed from these data.

Exposed branchlet stubs occurred along the entire axis of branches but showed a progressive decrease in numbers with increasing age, from tip to base. Stub closure began when branches were between 40 and 50 years of age, and by 90 years of age, over 80% of the stubs were overgrown.

A number of miscellaneous fungi were isolated from the exposed stubs, but *E. tinctorium* was obtained only occasionally and always in association with outward extensions of previously established medullary infections. Overgrown stubs, for the most part, were sterile or yielded only *E. tinctorium*. The youngest stubs infected by *E. tinctorium* were between 37 and 50 years of age, about the age when closure began. Invasion of the medullary tissues of the primary branch by *E. tinctorium* occurred between 60 and 70 years of age, about 20 years after the first infections were detected. By 90 years, the medullary region was continuously colonized by *E. tinctorium* to within a few centimetres of the trunk. Isolates of *E. tinctorium* obtained from the distal end of

branches were predominantly haploid, indicating that they had originated from a single basidiospore. Diploids were isolated with increasing frequency after 80 years of age, presumably the result of fusion of haploid infections in the medullary region.



FIGS. 2 and 3. Potential infection courts of *E. tinctorium* on living branches of western hemlock. Fig. 2. Two recently dead. internodal branchlets (< 2 mm in diameter) on a 10-year-old branch. Fig. 3. Cup-like structure of callus around the base of a dead branchlet on a 50-year-old branch. This structure contains the residual branchlet stab which provides the entry point for the fungus. FIGS. 4–6. Cross section of living branches of western hemlock showing progressive stages of closure of branchlet stubs. The susceptible stage is from the onset of closure (Fig. 4) until stubs are overgrown (Fig. 6), embracing a minimum period of almost 2 years.

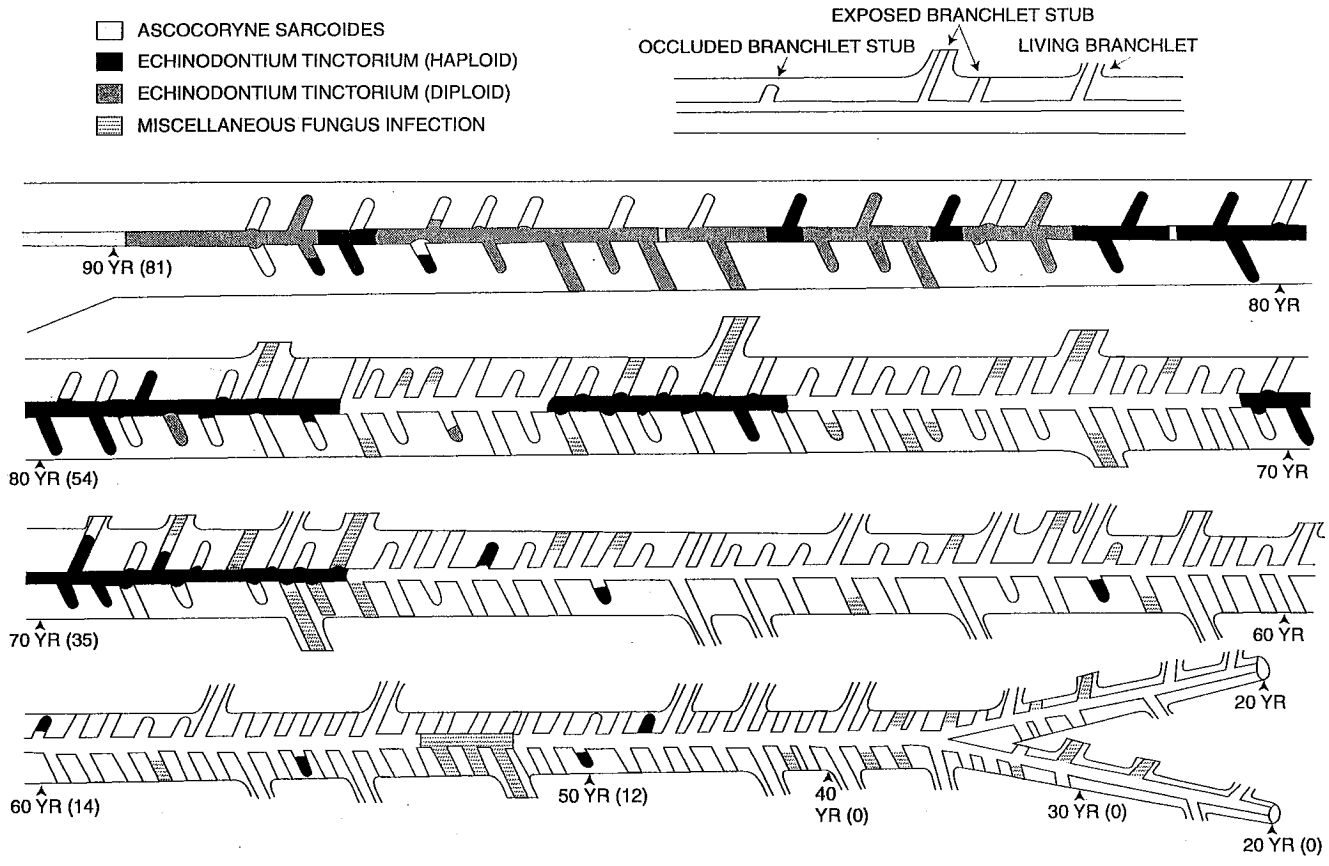


FIG. 7. Schematic representation of a 90-year-old living branch of western hemlock showing distribution of occluded and exposed branchlet stubs in relation to the occurrence of dormant haploid and diploid infections of *E. tinctorium* and infections by other fungi. Note the progressive closure of branchlet stubs (percentage in brackets) and the progressive development of *E. tinctorium* with increasing age after 40 years.

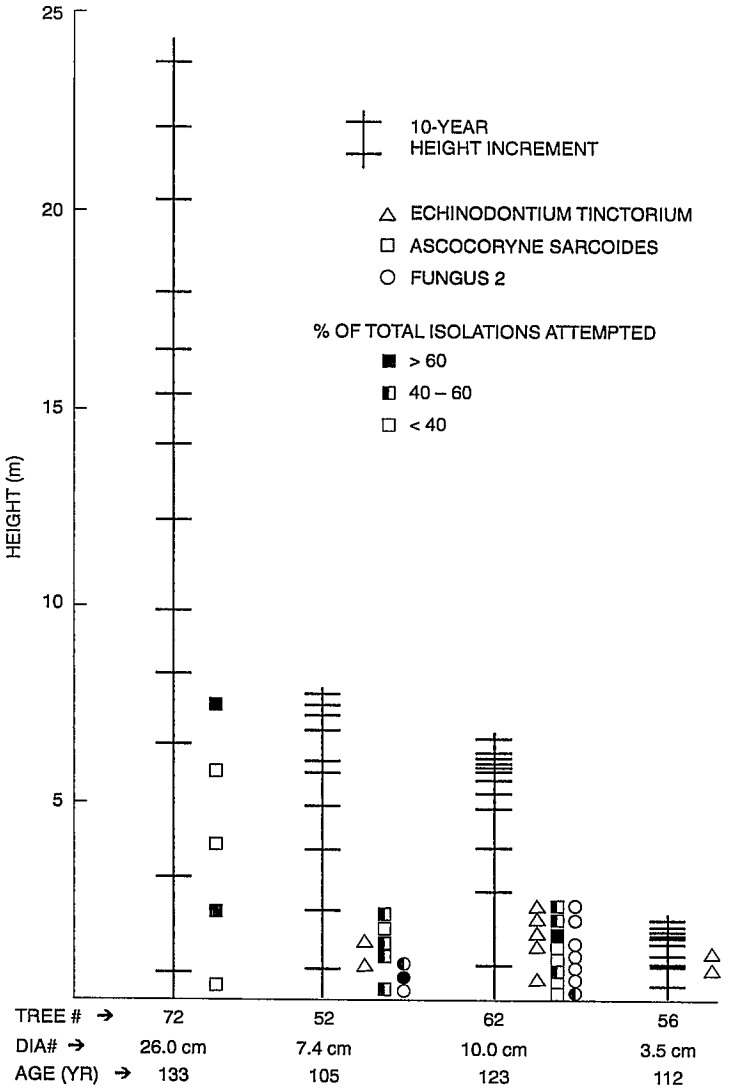


FIG. 8. Schematic representation of four living western hemlock stems showing distribution of dormant infections of *E. tinctorium* and two major fungal competitors in relation to dominance class and height-growth increments at different ages in the tree's development. Note *E. tinctorium.* in contrast with *A. sarcoides,* is confined to suppressed stems.

Only three of the branches (all 90+ years old) contained basal medullary infections of *E. tinctorium,* but none had extended more than 3 cm inside the trunk. The basal medullary region of four of the branches was colonized by outward extensions of trunk infections by *Ascocoryne sarcoides.* Only one of these, like the branch shown in Fig. 7, had infections of both *A. sarcoides* and *E. tinctorium* at the base.

Distribution of dormant infections of *E. tinctorium* in stems was investigated in one codominant and three suppressed trees, 100 to 130 years old. from the Hidden Lake area. Five or six 30-cm sections of stem were taken from the basal third of each tree and longitudinally dissected to expose all overgrown branchlet stubs, using the method employed for sampling branches. Seven hundred and eighty three isolations were attempted from the medullary tissues and embedded branchlet stubs up to 2 mm in diameter. Figure 8 shows the distribution of the three most common fungi isolated from the four

trees according to their frequency of occurrence in wood of different ages, at different heights from the ground. The medullary tissues of the codominant (no. 72) and the two larger of the suppressed stems (nos. 52, 62) were colonized exclusively by *A. sarcoides* to the upper limit of sampling. *Echinodontium tinctorium* was isolated exclusively from overgrown branchlet stubs in the three suppressed stems but not from overgrown stubs in the codominant stem, which remained uncolonized except for several examples of extensions of medullary infections of *A. sarcoides*. *Echinodontium tinctorium* was not isolated from stem sections less than 50 years of age. An unknown hyphomycete, 'fungus 2,' colonized overgrown branchlet stubs in addition to *E. tinctorium* in trees 52 and 62.

Distribution of dormant branch infections of *E. tinctorium* in relation to height from ground (age) and occurrence of other fungi was investigated in 11 codominant trees from the Hidden Lake stand. The trees were about 130 years old, 20 to 25 m in height, and had not undergone prolonged periods of suppression. A total of 110 living and 3 dead branches, with the associated stem sections, were removed from the trees and systematically sampled. Two isolations were attempted from the medullary tissues at 10-cm intervals along the main axis of each branch to a minimum diameter of about 1 cm. In addition, the heartwood of the associated stem sections and the embedded bases of 17 of the branches were sampled to determine if penetration of the trunk by *E. tinctorium* had occurred. The medullary region and embedded primary branch stubs of the entire 'branch-free' section of the bole of one of the trees was also systematically sampled. The results of all the isolations have been consolidated and are schematically presented in Fig. 9 to show the distribution of *E. tinctorium* and associated fungi in the stem and branches of a representative tree.

Dormant infections of *E. tinctorium* were detected in living branches as young as 40 years of age, but they occurred predominantly in branches 60 years of age and older. No visible decay or discoloration was associated with the infections. Incidence of infected branches and frequency of infections per branch decreased with increasing height, from almost 100% for branches at the base of the crown, which contained five or more infections, to the complete absence of infections above the midpoint of the crown. Isolates of *E. tinctorium* were predominantly haploid. Diploids were isolated with increasing frequency in successively older branches but rarely exceeded 25% of the isolates, even in the oldest branches sampled. *Echinodontium tinctorium* was obtained from the embedded bases of five branches from 1 to 3 cm inside the stem but not from the adjacent stem tissues. The medullary region of the stem was colonized exclusively by *Ascocoryne sarcoides* to about the height of the lowest living branch, which averaged 5.2 m from the ground. With the exception of one branch that had died very recently, as evidenced by the persistence of fine twigs, none of the dead branches, including partially and completely overgrown ones in the branch-free region of the bole, yielded *E. tinctorium*. A species of *Dermea*, frequently isolated from exposed ends of broken branches, and two unknown hyphomycetes, 'fungus 32' and 'fungus 2,' isolated mainly from partially covered stubs, dominated the large number of miscellaneous isolates obtained from the dead material.

Effect of Growth Rate on Susceptibility of Branchlet Stubs

Presence of *E. tinctorium* in suppressed stems and branches and its absence in non-suppressed stems suggested possible differences in growth characteristics, especially closure rates, between branchlet stubs from fast- and slow-grown wood which might affect their suitability as infection courts. To investigate this possibility, representative examples of overgrown branch stubs (<2 mm in diameter) from a non-suppressed codominant and two suppressed understory trees were characterized according to diameter, age at death, and rate of closure. Presence or absence of infections by *E. tinctorium* in the stubs was determined by cultural diagnosis before characterization. The rate of closure was calculated by the formula $E = Ds / 2Rw$, where E = closure rate, or number of years exposed to infection, Ds = diameter of stub (millimetres), and Rw = mean width of annual rings at distal end of

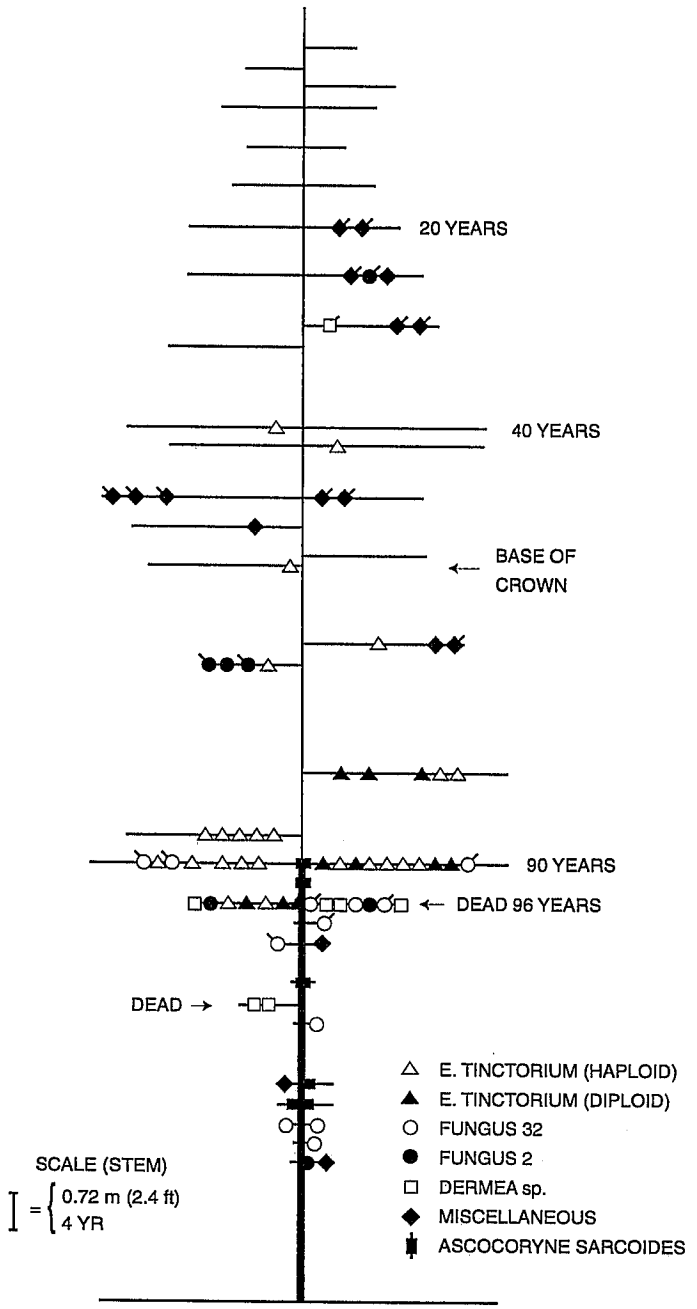


FIG. 9. Schematic representation of a 130-year-old living western hemlock showing distribution of dormant haploid and diploid infections of *E. tinctorium* and its major fungal competitors in relation to age of branches and height from the ground. Note *E. tinctorium*, in contrast with its competitors, is confined to living and relatively still-intact dead branches older than 40 years of age.

stub (millimetres). The formula was based on the assumption that a ring of 0.5 mm in width would cover a 'flush' stub, 1 mm in diameter, in 1 year. Examples of applying the formula to branchlet stubs in suppressed (A) and non-suppressed (B) wood are shown in Fig. 10. The characteristics of the stubs in relation to their susceptibility to *E. tinctorium* and to the

TABLE 2. Comparison of events of branchlet stub (<2 mm in diam) formation and closure in shade-suppressed branches of dominant and codominant western hemlock from susceptible and non-susceptible habitats[a] of *E. tinctorium* in relation to suitability as infection courts

Habitat [a]	Estimated susceptibility [b] of stubs	No. stubs	Mean stub diam, mm	Mean age at death	Mean age [c] at onset of closure	Years required [d] for completion of closure
Susceptible (Hidden Lake)	(+)	10	0.75a	20.6a	41.7a	3.34a
Non-susceptible (Nitinat, V.I.)	(–)	10	0.84a	9.3b	15.6b	1.00b

NOTE: Means followed by a letter in common are not significantly different (Newman-Kuels multiple comparison test: $P = 0.05$).

[a] According to the classification of Thomas (1958).

[b] Based on isolation results, (+) = *E. tinctorium* frequently isolated from neighboring branchlet stubs and from other branches of same tree. (–) = *E. tinctorium* absent from branches sampled.

[c] Age at death plus number of years until stubs become flush with main branch.

[d] Number of years to cover 'flush' stubs = $Ds / 2Rw$, where Ds = diam of stub and Rw = mean width of annual rings at distal end of stub.

dominance class of the trees from which they originated are given in Table 1. Duration of the closure process was the only characteristic that was significantly different between infected stubs and both samples of non-infected stubs, indicating that the length of time that stubs were exposed to infection was a critical factor affecting their susceptibility. Infected stubs from the slow-grown understory stems had been exposed for nearly three-quarters of a year longer than non-infected stubs from the same stems and over a year longer than the non-infected stubs from the faster-grown codominant. Similar conclusions can be drawn from a comparison of growth characteristics of overgrown stubs from branches of the susceptible habitat at Hidden Lake and those of non-susceptible habitats on Vancouver Island (Table 2). Although it was not possible to verify infection of stubs by cultural diagnosis, the rate of closure of stubs was over 3 years slower in branches from the susceptible habitat than in those from the non-susceptible habitat.

A slow growth rate, by its effect on interstub shoot development, could conceivably affect susceptibility by increasing the frequency of occurrence of internodal branchlet stubs. To demonstrate this relationship, counts were made of dead secondary branchlets less than 2 mm in diameter on selected primary branches from susceptible and non-susceptible habitats at Hidden Lake and Nitinat, V.I., respectively. Branches from the susceptible habitat, in the two age classes sampled, had a slower rate of growth (0.37 and 0.25 mm/year) than those from the non-susceptible habitat (0.51 and 0.46 mm/year), and a significantly greater number of dead branchlets were produced on the former (Table 3). The frequency of dead branchlets from the two habitats in the two branch-age classes were 52 and 65, and 24 and 28, respectively, indicating that more than twice the number of potential infection courts were produced by branches in susceptible habitats than in non-susceptible habitats.

Factors Affecting Reactivation of Dormant Branch Infections and Initiation of Decay

Despite the high incidence of dormant infections, there was no evidence of advance or incipient decay caused by *E. tinctorium* in any of the stems sampled. However, pockets of incipient decay, presumably originating from dormant medullary infections of *E. tinctorium,* were observed at the base of large secondary branch stubs (Figs. 11A and 11B) in two living branches. Since it was possible that the decay had been initiated as a result of conditions associated with the broken stubs, an attempt was made to reactivate *E. tinctorium* in branches by producing stubs artificially. A total of 27 primary branches, ranging in age from 70 to 113 years and, therefore, likely to contain dormant infections of *E. tinctorium,* were removed from two trees in the Hidden Lake study area so as to leave stubs which varied from 6 to 16 cm in length. A number of branches were expected to contain dormant

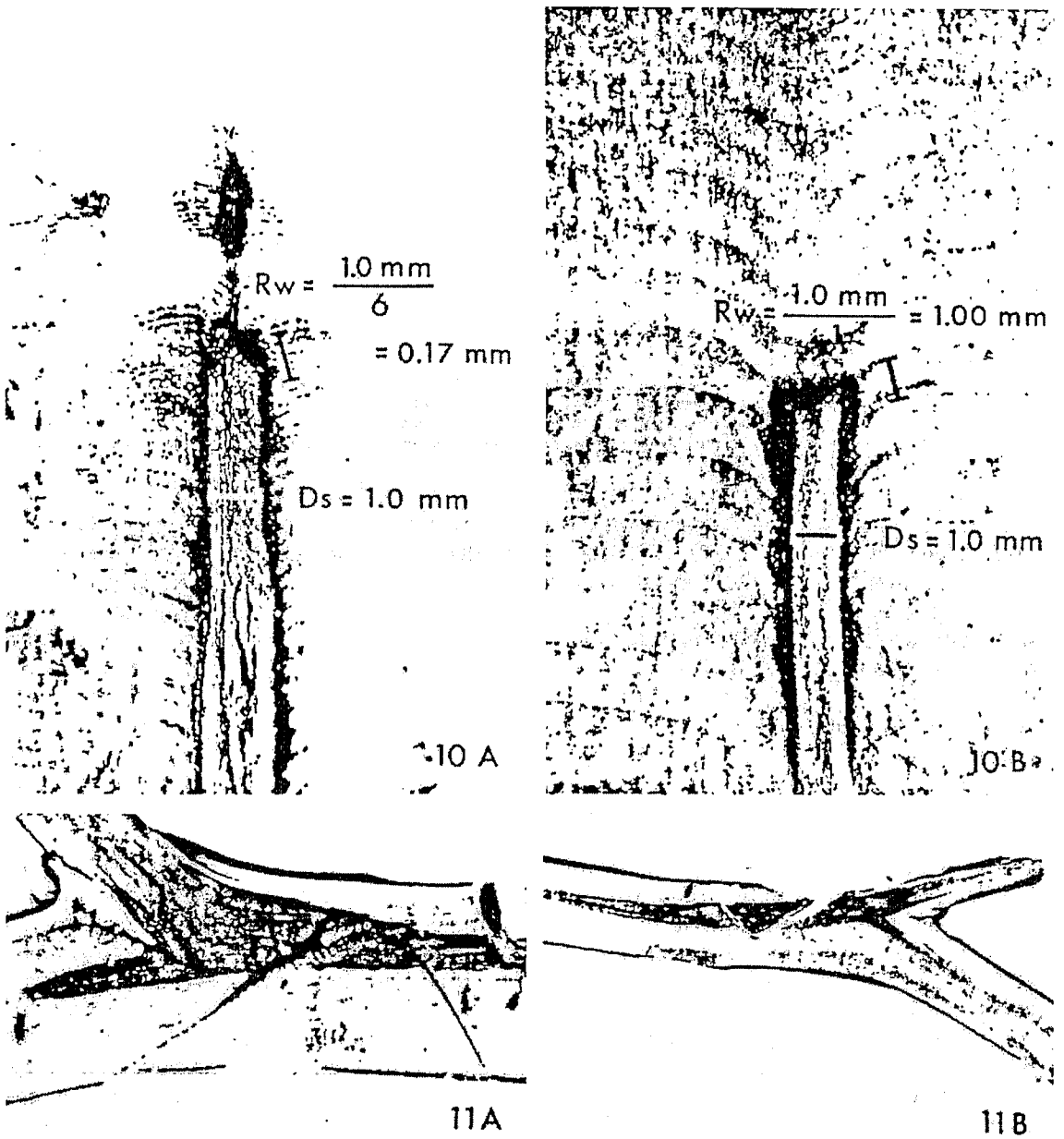


FIG. 10. Examples of overgrown branchlet stubs, 1 mm in diameter. in slow-grown (A) and fast-grown (B) wood of western hemlock. Based on the formula (see text) $E = Ds / 2Rw$, the duration of exposure assigned to 'A' was 2.9 years and to 'B' was 0.5 years. Secretion of resin has delayed closure in 'A' but effectively sealed the stub within the theoretical time limit. FIG. 11. Two examples (A. B) of incipient decay in living branches of western hemlock believed to have originated from dormant infections of *E. tinctorium* previously established in the medullary tissues. Note proximity of large secondary stubs which, presumably, were responsible for reactivating the infections.

medullary infections of the fungus at the point of severence, thus providing both detached branches and residual stubs for studying the possible reactivating effect of exposure under laboratory and forest conditions. Isolations were attempted from the pith and 0.5 cm above

TABLE 3. Frequency of dead secondary branchlets (<2 mm diam) on primary branches of dominant and codominant western hemlock from a susceptible and non-susceptible habitat of *E. tinctorium* in relation to branch growth rate

Habitat	Primary branches			Dead secondary branchlets/basal 90 cm of primary branch	
	Age	Diam, cm	Growth rate,[a] mm/yr	No.	% Occluded
Branches < 50 years old					
Susceptible	43a	3.2a	0.37a	52a	44.0a
Non-susceptible	46a	4.8b	0.51a	24b	
Branches > 50 years old					
Susceptible	64b	3.3a	0.25b	65a	77.4b
Non-susceptible	66b	5.6a	0.46a	28b	

NOTE: Means followed by a letter in common are not significantly different by *t* test ($P = 0.05$). Means are averages of three branches.

[a]Growth rate = $Db / 2A$, where Db = mean diameter (mm) outside bark at base of primary branch and A = age of stem at base of branch.

and below the pith in the surrounding heartwood. *Echinodontium tinctorium* was recovered from the pith in 7 of the 27 detached branches, but the heartwood was sterile. Sections, about 4 cm in length, cut from the end of each branch which had yielded *E. tinctorium,* were then placed in sterile containers and incubated at 21 °C in a saturated atmosphere. After 3 months, a new cut was made at the ends of the sections and isolations were attempted from the same three positions in the pith and heartwood. Similarly, isolations were attempted from new cuts made at the end, midpoint, and base of the stubs after 1 year of exposure in the forest. The results of the isolations, before and after exposure to forest and laboratory conditions, are depicted schematically in Fig. 12. Of the seven stubs that had yielded *E. tinctorium* from the pith position at the time of pruning, three were sterile and four yielded miscellaneous fungi exclusively after the year exposure in the forest. 'Fungus 2' dominated the fungi invading the stubs. Results of the isolations at the midpoint and basal cuts indicated that *E. tinctorium* was absent or had been replaced by miscellaneous fungi in the interior of the stubs. A single exception, stub 4709, 10 cm in length, had *E. tinctorium* at its base, but since the fungus was isolated only from the medullary position, the infection had presumably not been reactivated by exposure. Apparently, environmental conditions, possibly wood moisture content, were more favorable for fungal survival nearer the trunk, since two of the stubs (nos. 4708, 4719) yielded miscellaneous fungi at the base and midpoints but were sterile at the distal end where the infection originated. Although moisture content of the stubs at the time of sampling was not measured, their general appearance, based on previous studies on recently dead hemlock branches (Etheridge *et al.* 1970), suggested that the moisture content rarely exceeded that of fibre saturation (27% ovendry weight) at the base of the stubs and was considerably drier at the distal ends.

In contrast with the stubs, *E. tinctorium* had survived at the severed ends of five of the seven detached branches that had been incubated under controlled conditions for 3 months. In each case, the infections had not only remained viable but had spread into the heartwood surrounding the pith, indicating that humidity and temperature during the 3-month incubation had been suitable for reactivating this fungus. There was no incipient decay associated with any of the infections; however, previous tests with blocks inoculated with this fungus under similar conditions showed that discoloration and incipient decay were associated with active infections only after 8 to 12 months.

Effect of Host Vigor on Location and Extent of Rot Columns

The demonstrated unsuitability of large primary branch stubs as avenues of entry for this fungus suggested that most heartrot infections originated directly from dormant infections already established in the stem. While this was

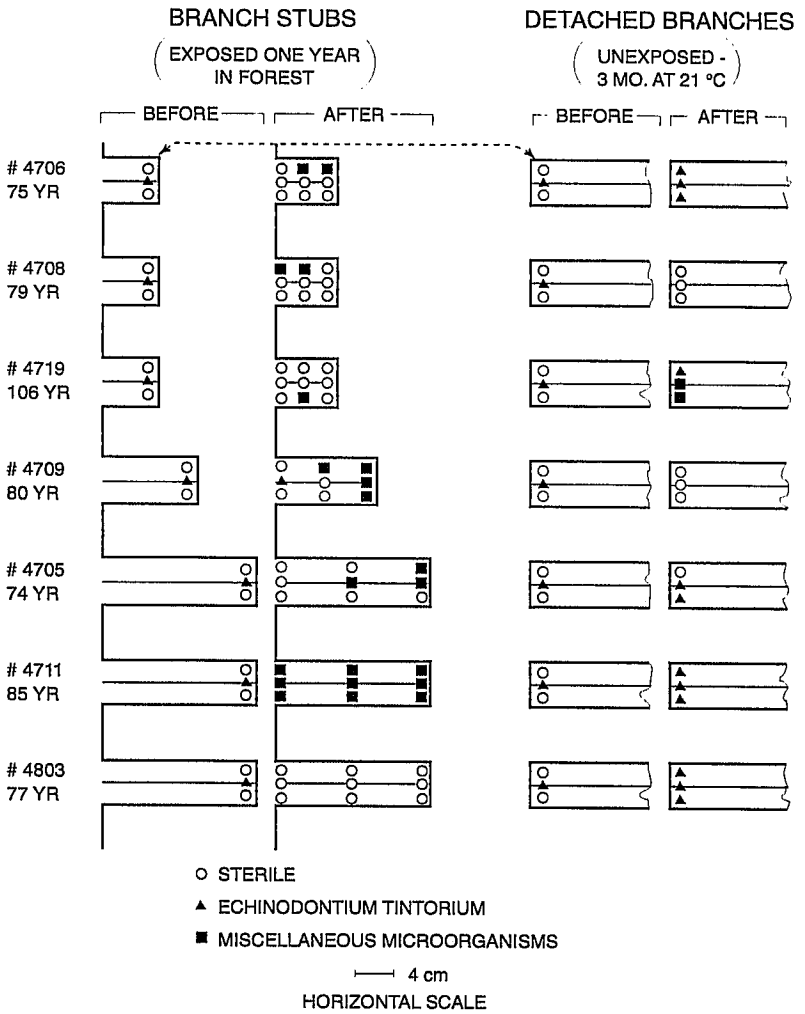


FIG. 12. Schematic representation of seven selected branch stubs of western hemlock and corresponding detached branches showing, respectively, results of isolations (points indicated), before and after exposure to forest conditions, and before and after incubation at 21 °C.

true for understory stems where low vigor ensured a continuous supply of potential infection courts, it was possible that periods of susceptibility might occur intermittently as a result of infection court formation during temporary periods of suppression in the life of dominant and codominant trees as well. This possibility was investigated by reanalyzing decay and growth increment data obtained from a study of western hemlock carried out in the Revelstoke area in 1951 (Foster *et al.* 1954). Data obtained from four trees (two dominants and two codominants) have been selected to show the relationships observed between the location and extent of trunk heartrot infections by *E. tinctorium* and 10-year growth increment patterns (Fig. 13). For this analysis, the length of each 10-year height increment was considered to be proportional to the width of the corresponding 10-year radial increment, the only data available on the periodic growth rate of the trees (see Fig. 14 for method). The association of the rot columns with periods of suppressed growth is clearly seen in Fig. 13, providing further evidence of a link between host vigor and the occurrence of infection courts. It is noteworthy that the time and duration of the supressed periods of growth appear not only to determine the dominance class of the four trees and their susceptibility to *E.*

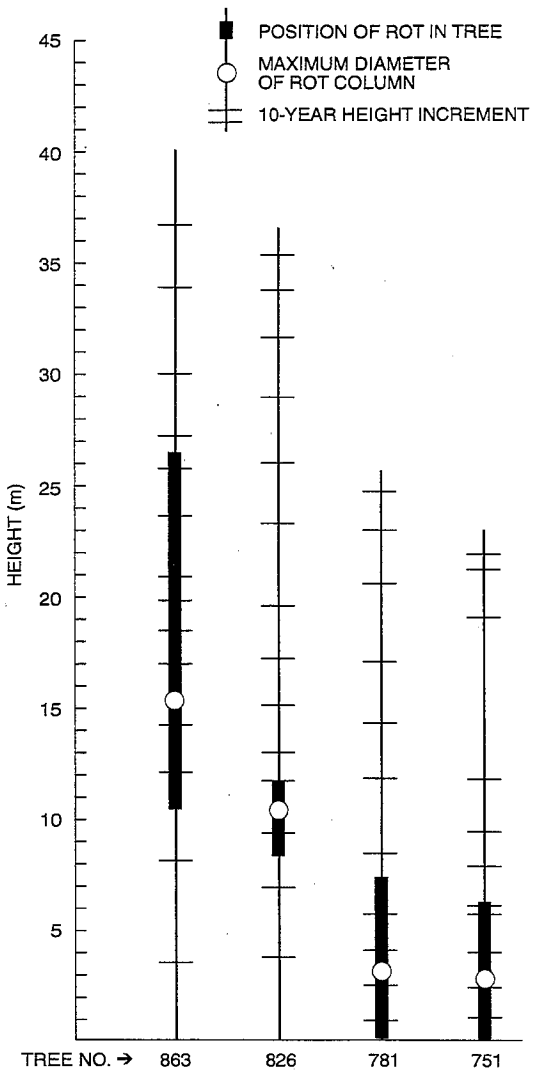


FIG. 13. Schematic representation of two dominant and two codominant western hemlock stems sampled near Revelstoke. B.C., in 1951 showing the relationship between the position and extent of trunk heartrot columns caused by *E. tinctorium* and 10-year growth increment patterns.

tinctorium but also the location and extent of the heartrot columns.

Discussion

Although the Indian paint fungus produced viable basidiospores throughout the year, suitable conditions for sporulation occurred only during spring and fall when average daily temperatures ranged from 4.5 to 16 °C (Fig. 1). It is noteworthy, however, that spring sporulation in 1970, and the more intensive fall sporulation in 1969, began, although little or no rain had fallen, emphasizing that low temperature rather than moisture is the major activating factor. Possibly temperatures below 4.5 °C stimulate production of basidiospores and only moderately high levels of air humidity are required for sporulation at higher temperatures. This explanation is supported by in vitro tests which showed that while heavy spore discharge occurred initially at relative humidities as low as 85% and at temperatures ranging from 9 to 20 °C, activity decreased rapidly after a few days, apparently when the supply of mature spores was exhausted. The fact that absolute minimums were above 4.5 °C could account for the absence of sporulation during the summer of 1969 (Fig. 1) when average temperatures were above 16 °C, despite the occurrence of a prolonged wet period, which might otherwise have stimulated activity. Maloy (1963) observed a similar cessation of sporulation by this fungus on grand fir during periods of summer rainfall when temperatures were above 18 °C.

Exposure to low temperatures also enhanced basidiospore germination; maximum germination of weekly spore collections occurred only after temperatures had fallen below 0 °C (Fig. 1), while record-high germination values of up to 100% were obtained with free-cast basidiospores which had been stored for 8 months or more at −10 °C.

This stimulating effect of low temperature could, therefore, be an important mechanism regulating the infective period of *E. tinctorium.* Such a requirement would prevent premature germination of the large numbers of basidiospores resulting from the heavy fall sporulation, ensuring that adequate supplies of viable spores were available in the spring. A similar mechanism has been postulated for other decay fungi. Denver (1960) showed that preexposure to temperatures of −7 °C for 11 weeks was essential for germination of basidiospores of *Flammula conissans* and *F. alnicola,* a requirement he pointed out that would restrict infection by these fungi to the spring season when temperatures were favorable for germination. It thus appears that sporulation is out of phase with germination in these species which require spores to be dispersed and in place on the host

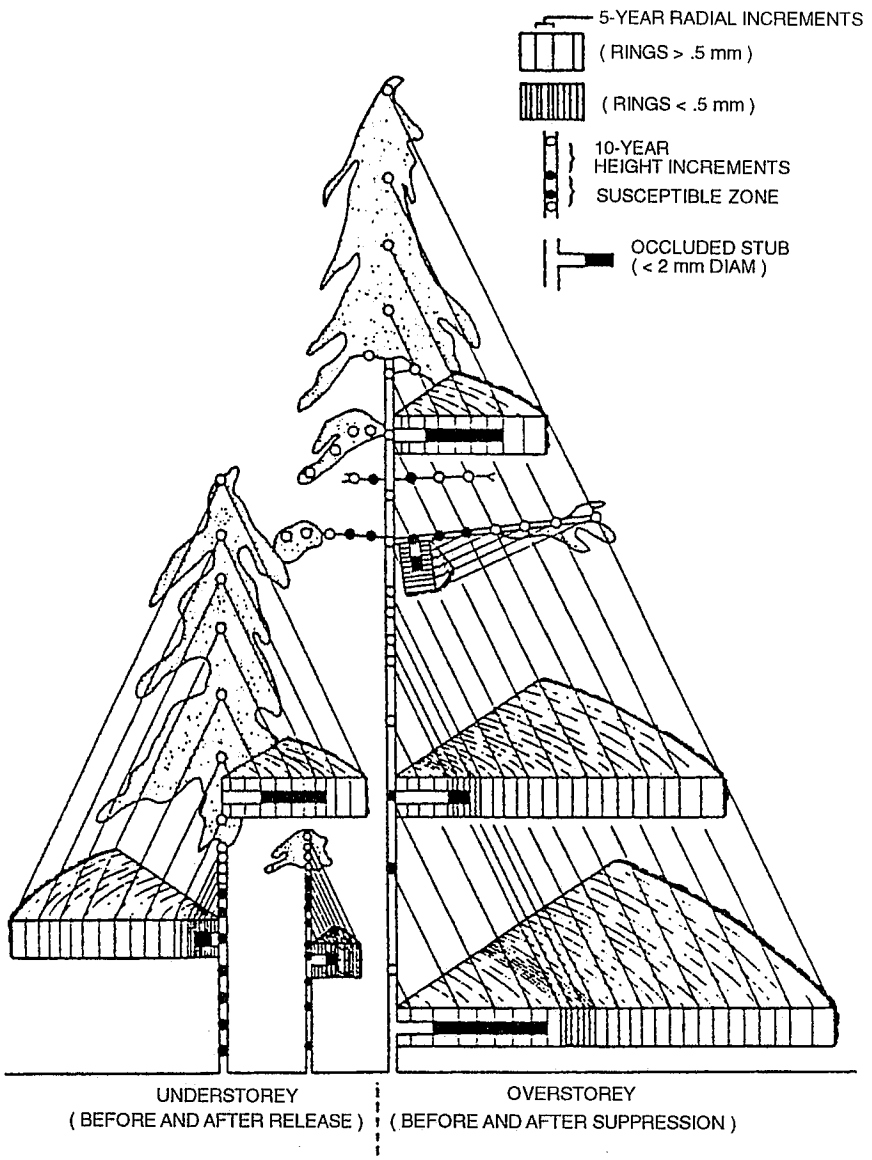


FIG. 14. Schematic representation of an overstory and two understory western hemlock showing the relationship between tree dominance, time and duration of periods of suppression (shaded areas), and the formation of susceptible infection courts. Note susceptible periods in a tree's development can be predicted by stem ring patterns which determine stub closure rates.

for a cold period before germination and infection occurs.

Haddow (1938), investigating *Fomes pini* on white pine in Ontario, demonstrated for the first time that infection by a heart-rotting fungus could take place through exceedingly small branchlet stubs at a relatively early age. He found that infections in the basal region of the trees had entered through branch stubs 1 to 6 mm in diameter. No infections were traced to large branch stubs, although they were not ruled out as potential infection courts higher up the trunk. Haddow did not consider rate of healing as a factor affecting entry of the fungus because infected stubs were kept open by the ability of *F. pini* to parasitize the callus tissues and prevent occlusion.

Echinodontium tinctorium, in contrast with *F. pini,* cannot prevent stub closure; therefore, conditions are favorable for establishment of

this fungus only so long as embedded bases of branchlet stubs are not overgrown. Thus, a major determinant of susceptibility in western hemlock is a slow rate of healing. This study has shown that minimal conditions for susceptibility in western hemlock occur when stubs of 1 mm in diameter are exposed for an average of 1.8 years, which can be expressed by the formula $E = Ds / 2Rw$ or $1.8 = {}^1/_2\ Rw$. In terms of ring width (Rw), an exposure period of 1.8 years corresponds to a minimum annual radial growth of 0.28 mm, or a frequency of almost four rings per millimetre. In western hemlock, this low rate of radial growth is characteristic of shade-suppressed lower branches and understory trees in susceptible habitats of the Indian paint fungus (Table 3).

After closure of the branchlet stub, infections by *E. tinctorium* become dormant or semi-dormant and can survive in this state for many years without initiating decay. Some infections which are confined to the medullary tissues in the region of branch stubs rarely extend for more than 2 to 3 cm from their point of origin, despite the fact that stub closure had occurred more than 50 years previously (Figs. 7 and 10A). This is a unique mode of infection, especially from the standpoint of survival ability and, according to the literature, does not appear to have an exact counterpart in any other heart-rotting fungus. Recently, Aho and Hutchins (1974) isolated what appears to be dormant infections of *Fomitopsis annosa* (Fr.) Karst., *F. pinicola* (Sw. ex Fr.) Karst., and *Ganoderma applanatum* (Pers. ex Wallr.) from small branch stubs in suppressed grand fir in Oregon, but the infection mechanism and survival would appear to differ from that of *E. tinctorium*. The trees studied by Aho (personal communication) were less than 70 years of age and many of the stubs were still exposed, suggesting the infections were relatively recent. Admittedly, evidence of survival ability is lacking specifically for the above fungi, but it has been demonstrated for several other wound parasites that infections, at least in the incipient stages, die soon after closure of the wound by which they enter (Childs 1956). When considering the remarkable longevity of *E. tinctorium* compared with wound parasites, one can only speculate whether the reported ability of this fungus to produce chlamydospores in host tissues (Etheridge *et al.* 1972) is involved in the long-term survival mechanism. Although the importance of chlamydospores as survival propagules is well documented (Garrett 1970), apparently very little is known about their role in poorly aerated environments.

The highly selective nature of the exposed ends of embedded stubs as entry points for *E. tinctorium* may be explained by its failure to become established in protruding stub spurs which, in this study, were consistently larger than 2 mm in diameter. Etheridge *et al.* (1970) found dead branches and exposed primary branch stubs of western hemlock unsuitable as a substrate for germination of basidiospores of *E. tinctorium,* mainly because of competition by non-hymenomycetous fungi which rapidly colonized the wood after death. In the present study, non-hymenomycetous fungi and bacteria were the only organisms found in both exposed and protruding stubs larger than 2 mm in diameter, whereas stubs of smaller diameters were mainly sterile or colonized exclusively by *E. tinctorium.* The exclusion of non-hymenomycetous fungi from small-diametered stubs may be due to the tendency for dead branchlets less than 2 mm in diameter to break off close to their base compared with branches of larger diameters, which characteristically break off to leave spurs. Since breakage of branchlets at their base results in the removal of previously exposed and colonized material, only the embedded and presumably sterile portion of the branch is then exposed to infection. Although it may be expected that the freshly exposed branch base would be a suitable substrate for colonization by a variety of organisms, including *E. tinctorium,* the tendency would be for infections by the latter fungus only to survive prolonged stub closure.

Various estimates, ranging from 45 to 75 years, have been given in the literature as the age of earliest infection by *E. tinctorium* (Maloy 1967). The 'age of infection' given in these reports, however, means the age at which decay becomes conspicuous and cannot be interpreted as the age when trees become susceptible to this fungus. Etheridge *et al.* (1972) demonstrated that starting at age 40 years, a very close relationship existed between age and the percentage of living branches containing dormant infections of *E. tinctorium,* indicating

that western hemlock younger than this age was not susceptible to infection. A fundamental explanation for this relationship is afforded by estimates of the number of years required for branchlet stubs to reach the occlusion stage, which in the present study, ranged from 42 to 44 years, marking the onset of the infective period. After trees have reached this age, we can envisage a zone of susceptible infection courts forming at the base of suppressed stems and branches and passing slowly upward and distally with increasing age as the radial growth covers the stubs (Figs. 7 and 8). By the same token, we can envisage, following closely behind this zone in suppressed stems of western hemlock, a zone of dormant infections and potential heartrot progressively increasing in length after age 40 years.

Despite the large number of infections by *E. tinctorium* observed in living branches of western hemlock (Figs. 7 and 9), few, if any, would lead to the establishment of trunk heartrot because of (1) the localization of infections in the medullary tissues and the virtual cessation of active mycelial development after closure of entry points; (2) the relatively rare occurrence, except in a few of the oldest branches sampled, of medullary infections in the basal portion of the branch and their still rarer occurrence in the trunk-encased portions; (3) the increasingly frequent occurrence with increasing branch age of outward extension of medullary trunk infections of *Ascocoryne sarcoides* and the competitive effect that this fungus would have on infections of *E. tinctorium* occupying the same tissues; and (4) the failure of dormant infections of *E. tinctorium* to survive in branches after death and breakage (Fig. 12). A possible exception to the above circumstances is the rare occasion when an infection is initiated in a relatively young branch near the point of juncture with the trunk and subsequently becomes encased by the radial growth of the tree. Presumably, in this situation, death and breakage of the branch would have little or no effect on the moisture content of the trunk-encased portion, while the improved aeration could be responsible for reactivating any dormant infections in the tissues and thus lead to the development of heartrot.

These observations strongly suggest that in most trees heartrot caused by *E. tinctorium* originates from dormant infections established directly in the stem and only rarely from primary branch infections. This is supported by the frequent occurrence of decay columns by this fungus in the lower portion of the bole, where natural pruning has resulted in the removal of branches many years before infection courts could have formed on them. In such cases, the reactivating agent could be any deep-seated mechanical injury such as a logging scar, frost crack, or the formation of a large branch stub which allows air to enter the heartwood where the dormant infections are located. This hypothesis receives some support from the frequently observed association between mechanical wounding and decay by *E. tinctorium*, which many early investigators believed was evidence of a causal relationship (Maloy 1967). Both Meincke (1916) and Weir (1920), while fostering this belief, attributed the increased decay development observed in the proximity of wounds to improved aeration that stimulated the growth of the fungus. Support for the improved-aeration hypothesis is also provided by the two examples of incipient decay infections by *E. tinctorium* which are shown in Figs. 11A and 11B to be closely associated with large secondary branch stubs.

It is now possible to provide a fundamental explanation for Thomas's (1958) observation that low host vigor is the principal factor distinguishing a susceptible forest association from a non-susceptible one. Although Thomas correctly believed that the low vigor of host trees influenced susceptibility by delayed healing of branch stubs, his concept was based on the prevailing belief of the time that most infections originated in large primary branch stubs. We now know that large branch stubs are unsuitable infection courts for this fungus. Moreover, the persistence of large branch stubs, even on thrifty trees in non-susceptible habitats, would rule out healing rate as a determinant of susceptibility. As indicated by the concept of infection court formation shown in Fig. 14, susceptibility to infection is determined by low host vigor only because entry points are exceedingly small. Thus, while a minimum period of about 40 years of suppression appears to be necessary for their formation, infection courts

need to be exposed only 1 to 2 years for the fungus to be established. Hence. low host vigor becomes critical only during stub closure when the annual radial growth is less than 0.30 mm. In western hemlock, this rate of growth is characteristic of shade-suppressed lower branches and understory trees that are notably susceptible to *E. tinctorium.*

Effect of host vigor on the occurrence of potential entry points for the Indian paint fungus can be illustrated diagrammatically by postulating three hypothetical causal situations in the life of a tree. Figure 14 shows the nature of relationships between tree dominance class, the time and duration of periods of suppression (shaded areas), and the formation of susceptible infection courts. The magnified radial sectors show how host susceptibility is regulated by the healing rate of branchlet stubs, as determined by the width of the annual rings. Thus, the entire branch-free stem of the small understory tree (the portion of stem older than 40 years of age) is shown to be susceptible to the Indian paint fungus because stub closure and the infective period occur over a prolonged period of suppression. Susceptible infection courts will continue to form in this tree as long as it remains suppressed. Heartrot, if it develops, may then be expected to occur throughout the bole. The enhanced growth rate after release displayed by the other understory tree has prevented the formation of new infection courts through increased rate of healing. Provided that there is no further prolonged suppression in this tree, heartrot, if it occurs, will be confined to the lower bole. Overstory trees, on the other hand, are not suppressed during the early stages of growth; consequently, infection courts can form only if such trees are suppressed later, as in the example shown. A further example (not illustrated) is the occurrence of infection courts resulting from natural growth retardation of dominant trees coincident with old age. In both examples, if heartrot develops, it will characteristically occur at more advanced ages and nearer the living crown. The diagram shows that infection courts form by the same process in suppressed living branches as in stems, provided that they have attained a minimal age of about 40 years.

Evidence of both past and current periods of susceptibility to the Indian paint fungus, hence estimates of the potential decay hazard, can be obtained from characteristic ring patterns appearing on stem cross sections at or near ground level. Consequently, stem ring patterns may provide a useful basis for evaluating the decay risk in advance regeneration of western hemlock after logging. Ring patterns may also provide a basis for characterizing stands of western hemlock according to whether heartrot by *E. tinctorium* is likely to occur in the butt, midsection, or top position of the trees. This information would seem to have application to specific stands of this species for estimating net volumes on an individual log basis.

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Symposium on Wood Decay

Dynamic Responses of Differentiated Sapwood to Injury and Infection

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Heartwood of many tree species contains phenolic compounds which inhibit wood decay fungi and are largely responsible for the durability of heartwood in service. Sapwood of most species lacks appreciable amounts of inhibitory compounds and is considerably less durable in service than is heartwood when exposed to conditions favoring decay (43).

The relative susceptibility of heartwood and sapwood to decay in living trees, however, is reversed. Decay fungi are largely confined to the hostile environment of the heartwood to the extent that heartrot is the most destructive tree disease (22). Sapwood, on the other hand, may remain relatively free of infection for many years even when neighboring heartwood is extensively decayed. Our understanding of this phenomenon is, at best, fragmentary.

Two other papers of this symposium (20,57) critically evaluate the state of knowledge of certain aspects of the trees' defenses against injury and decay. I will limit my comments to an induced mechanism of resistance by differentiated sapwood. The role of preformed inhibitors (24) and the formation of barrier zones (58), therefore, will not be included in this paper.

Observed responses of differentiated sapwood to injury and subsequent infection, or to the encroachment of decay fungi from a central core of heartrot, include the production of two types of tissue: the transition zone and the reaction zone. For the purpose of orientation, these contiguous tissues separate infected wood from moist, functional sapwood. The pale-colored transition zone is contiguous with functional sapwood and the phenol-enriched reaction zone is contiguous with infected wood. This description takes into account the histological and cultural evidence that the transition zone and the reaction zone are produced in advance of infection by decay fungi (28,45,46,65).

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TRANSITION ZONES

Moisture content. Transition zones are drier than surrounding sapwood, hence their pale color. The moisture contents of transition zone and sound sapwood of Norway spruce (*Picea abies*) attacked by *Fomes annosus,* for example, were about 40 and 120% (dry wt), respectively (1). Some authors (10,16) have chosen the name "dry zone" to describe this tissue.

A plausible explanation has been provided for the rapid formation of dry zones adjacent to wounds in sapwood containing water columns under hydrostatic tension. In conifers, gas emboli are restricted to injured tracheids, particularly in earlywood, due to the valve-like action of tori causing aspiration of bordered pit pairs. This occurs because the pressure required to deflect tori into a closed position is sufficiently less than that required to move air-sap menisci through pit pairs (17). Drying in latewood, under similar conditions, is not particularly noticeable probably because it is nonconducting due to a low degree of water saturation (17,19). Embolism also is restricted to the severed vascular components in angiosperms due to insufficient pressure to draw menisci through pits that connect vessel segments (44).

The mechanism for replacement of water with gas in internal tissues not in direct contact with the atmosphere (ie, transition zones surrounding heartwood or reaction zones encircling central columns of decay), however, is more difficult to explain. Harris (18) found that over 80% of the bordered pits were aspirated in the dry-wood zone encircling heartwood of Pinus radiata. This compared to less than 50% pit aspiration in sapwood and about 50% at the

sapwood-dry zone boundary. He reasoned that a tissue containing tracheids with more than 50% of their pits aspirated would be impermeable to water movement. Direct measurement subsequently showed that dry zones surrounding cankers caused by *Peridermium pini* on Scots pine (*Pinus sylvestris*) were quite impermeable to water (16). Harris (18) further speculated that utilization by metabolizing parenchyma of water made available by low hydrostatic tensions in inner sapwood and subsequent pit aspiration were the causes of drying.

Coutts (11) presented evidence for the involvement of living parenchyma in the formation of dry zones in the sapwood of Corsican pine (*Pinus nigra* var. *maritima*) and Scots pine. Logs injected with dilute poison and subjected to autoclaving, an anaerobic atmosphere, or cold temperature (2–5 C) produced dry zones considerably smaller than their injected counterparts that were incubated under conditions favorable for metabolism. Sizable dry zones, however, were produced when autoclaved logs were injected with an aqueous extract from a log inoculated with *Fomes annosus*. The explanation given was that lysis of tori by the fungal extract provided an avenue for withdrawal of water and entry of gas. This explanation, however, seem inappropriate for living systems in which tori in transition zones are not lysed and the zone itself is quite impermeable to the movement of water (10,16). Coutts (11), contrary to Harris (18), concluded that dry zones were produced under conditions of high hydrostatic tension. Fresh logs with hydrostatic tension presumably relaxed by placing their ends in water prior to injection produced smaller dry zones than similar ones whose ends were not placed in water. From these results it was suggested that cavitation in individual tracheids resulting from the entry of gas from adjoining parenchyma under conditions of high hydrostatic tension could explain the formation of dry zones.

To add to this conjecture, I suggest that gradients in water potential from transition zone to surrounding sapwood could account for the movement of water out of this zone, particularly after the hydrostatic tension in its tracheids was released. Water would be expected to move from such tracheids into transition-zone parenchyma whose water potential may be expected to be higher than that of sapwood parenchyma because: water in sapwood tracheids would be under greater hydrostatic tension and, therefore, less available to its parenchyma; soluble metabolites in the transition-zone parenchyma are being converted to less soluble extractives; and the possible increase in membrane permeability of transition-zone parenchyma. This early response to pathogenesis (63) could be induced by ethylene (36), which was shown to accumulate in transition zones (48,50).

It seems less likely that the metabolic processes of transition-zone parenchyma could account for the dramatic decrease in water in this tissue. For example, a sample of sapwood containing 10 g of wood substance and 10 g of water (moisture content = 100% dry wt) with a starch content of 5% would not lose more than 0.05 g of water if all of the starch was degraded by hydrolysis.

Additional experimental evidence is necessary to formulate a comprehensive explanation for the drying of this tissue.

Metabolic activity. The wide range of biochemical events observed in transition zones lends convincing evidence that this tissue contains parenchyma that is living and quite active metabolically. The obvious effects of this metabolism in the systems studied are the disappearance of starch, the accumulation of phenols, and, probably, the death of transition-zone parenchyma.

Ethylene, a gas with hormone-like properties, may play a fundamental role in this process. This gas is produced by a wide variety of plants during flowering, fruit ripening, senescence, and in response to mechanical injury and infection (2,5). It has been implicated in increased respiration and the synthesis of enzymes required in the synthesis of phenols (41,52,60). A possible involvement of ethylene in transition-zone drying by increasing permeability of cell membranes also was cited earlier.

Enhanced production of ethylene was obtained from lesions caused by the wood wasp *Sirex noctillio* and its associated decay fungus *Amylosterium areolatum* in sapwood of *P. radiata*. Because some fungi also produce ethylene (25), it was necessary to determine if this ethylene was the product of host or pathogen. Lesions with transition zones removed and cultures of *A. areolatum*, with or without *P. radiata* sapwood produced no more than negligible amounts of ethylene (48). This demonstrated that the ethylene was of host origin and that the seat of enhanced ethylene production was the transition zone.

Lesions 1–4 wk old, caused by *S. noctillio* in two multi-stemmed trees, produced about 17 times more ethylene than control tissue obtained from adjacent, sound sapwood. Comparable lesions in a suppressed tree produced only about twice as much ethylene as did controls and one-tenth that produced by lesions in the dominant trees. Three weeks after inoculation, phenols were present in considerable quantities in lesions in the dominant trees but still were not detectable in lesions in the suppressed tree (48). The major phenolic compound detected in *S. noctillio* lesions was pinosylvin (23) which was quite inhibitory (ED_{50}<25 ppm) to *A. aerolatum* in an agar medium (9). In subsequent studies, it was found that increases in ethylene production were detectable 1 day after inoculation (more than a 10-fold increase over controls) and that pinosylvin was detectable by gas-liquid chromatography (49) within two days of inoculation (50). Internal concentrations of ethylene, furthermore, were substantially higher than in controls 3 days after inoculation and a lesion 4 wk old had an internal concentration as high as 5 ppm (L. Shain, *unpublished*). Ethylene also has been implicated directly in the synthesis of pinosylvin (49) and other phenols (7,42).

Phenols have been implicated in the resistance of *P. radiata* to attack by *S. noctillio* (12). Greater resistance, therefore would be expected in individuals that produce larger quantities of ethylene, and pinosylvin, at a faster rate. It would be very desirable, from the tree-breeders' standpoint, if this capacity to respond were highly heritable. In the three-tree experiment mentioned above, however, it was not possible to distinguish whether the observed differences in response were due to the trees' crown class or genotype.

A modest attempt was made to determine whether genotype or growing conditions most affected the hosts' capacity to respond. Two clones of *P. radiata* each growing on a good and a poor site (measured by significant differences in apical growth) were subjected to a controlled *S. noctillio* attack by insects with clipped wings. Ethylene production by *S. noctillio* lesions measured 32 days after attack did not significantly differ, indicating that genotype may be playing a greater role than environment in the hosts' response. These results certainly need to be substantiated.

We are now measuring the amount of ethylene produced in response to standardized wounds in cottonwood (*Populus deltoides*). If this can be correlated with observed differences in resistance to decay and discoloration (56) it could be a rapid and convenient means for identifying resistant individuals.

Ethylene also was produced in response to wounds in all other species we have tested; ie, several species of *Ecalyptus*, black locust (*Robinia pseudoacacia*), and white pine (*Pinus strobus*) (L. Shain, *unpublished*). There is little doubt that ethylene production is a common, if not universal, early response of trees to wounding and infection.

Slight increases (about 1.4 times) were reported in oxygen uptake by *S. noctillio* lesions with their transition zones 1–6 days after attack as compared to adjacent sapwood (50). Approximately five-fold differences, however, were obtained between lesions several months old and their controls. Manometric determination of oxygen uptake by *S. noctillio* lesions with and without transition zones in the presence or absence of a phenolic substrate (1-napthol at 3.5×10^{-4} M) indicated that transition zones contribute more to the apparent respiratory rise than does the fungus in the necrotic portion of the lesion. Phenol oxidase activity, while present in transition zones, was 2–3 times greater in the necrotic tissue. With this technique, phenol oxidase was not detectable in adjacent sapwood (L. Shain, *unpublished*). By enzyme histochemistry, it was possible to demonstrate increased activity of both malic and glucose-6-phosphate dehydrogenases in the parenchyma of transition zones surrounding heartwood of *P. radiata* (51) and transition zones surrounding lesions caused by *S. noctillio*.

These modest increases in oxygen uptake by transition zones, however, are overshadowed by the rapid and greater increases that

sometimes were obtained in ethylene production.

The histochemical demonstration of starch in sapwood and its paucity or absence in adjacent transition zones (11) is good evidence that starch degradation occurs in this zone. Isolation of starch-degrading enzymes from transition zones apparently has not been reported.

Much remains to be learned about the physiology and metabolism of transition zones. Such information could greatly increase our understanding of reaction-zone formation.

REACTION ZONES

Reaction zones are necrotic tissues that are enriched with inhibitory extractives and are produced in advance of infection. Their formation has been related to a dynamic mechanism of host resistance (45,46). The term reaction zone probably is analogous to several other terms which have been used; eg, pathological heartwood (6), protection wood (28), discolored wood (8,53), wound-initiated discoloration (54), and walls 1, 2, and 3 of the CODIT model proposed by Shigo and Marx (55).

Stimulus for reaction zone formation. As host parenchyma in the transition zone die, reaction zones are formed. The occurrence of some reaction zones apparently devoid of microbial colonization (46,65) and the intraspecific, qualitative consistency of its chemical components (23,45,47) provide strong circumstantial evidence that this tissue is the end product of transition zone, but not microbial, metabolism.

It was suggested that the stimulus for necrosis of reaction zones may be provided by toxins produced by invading pathogens (11,45). The nonspecificity of this response and the production of reaction zones around presumably sterile wounds (45) argue against the necessity for such toxins. It seems more likely that the physiological and metabolic processes of the transition zone are programmed to terminate in reaction-zone formation and cell death in what could be considered a hypersensitive response. Heartwood formation, which in some ways may be similar to reaction-zone formation (45,62) also seems to occur in the absence of microbial stimuli.

The initial stimulus for the formation of transition zones and then reaction zones appears to be injury of nearby cells caused by wounding or infection. The response is measured; ie, it keeps pace with the margin of wounded or infected tissue rather than perpetuating itself throughout the entire sapwood. Investigation of the biochemical mechanisms for triggering and controlling this response should be a challenging, and perhaps rewarding, area of research.

Antifungal compounds. The accumulation of antifungal compounds in a reaction zone has provided evidence that this tissue constitutes a defense mechanism. Some of the major points of evidence are presented in the examples below. A more comprehensive coverage of this topic, including some of the anomalous results obtained by different bioassay techniques is available elsewhere (29) and in Hart and Shrimpton (20), the preceding symposium contribution.

The term reaction zone first was applied to a necrotic tissue enriched with oleoresin and phenols produced in advance of *F. annosus* infection in sapwood of loblolly pine (*Pinus taeda*) (45). The formation of similar tissues were described in other pine species in response to *F. annosus* or other injurious stimuli (12,28,38).

Oleoresin (about 30–40% dry wt) probably flowed into this zone passively upon the death of the thin-walled epithelial parenchyma which maintain oleoresin under pressure in a comprehensive resin duct system. Oleoresin is largely composed of resin acids dissolved in volatile terpenes (35). Phenols (up to 2%, dry wt), largely pinosylvin in early infections (45), probably were produced during the necrobiotic metabolism of the transition zone described above.

Evidence for reaction zone formation. The following points serve as evidence that reaction-zone formation is a function of the hosts' capacity to respond and that the accumulated compounds in this tissue are responsible for a dynamic mechanism of host resistance:

In vivo observations. One year after inoculation, *F. annosus* was isolated an average of 38.6 cm from inoculum dowels in two trees that died 4–6 mo prior to harvest and an average of 4.5 cm in comparable trees that remained alive. Reaction zones were present around all inoculations in living trees but absent in the dead trees except in control positions inoculated with sterile dowels (45). This last point, as well as others (30–32,39,45,59) were taken as evidence that decay fungi can slowly degrade reaction-zone constituents, but that living trees are capable of a continued response.

A significant negative correlation was obtained between the extent of infection of Corsican pines inoculated with *F. annosus* and their pinosylvin content (38).

The characteristic shape of the reaction zone; ie, greater penetration of decay fungi with increasing distance from the cambium (45,64), indicates that the rate of physiological activity is related directly to resistance. This was further substantiated by greater infection and a longer lag period in reaction-zone formation during the dormant season than during the growing season (33,45). Resin flow also was greatest in trees of increasing dominance and during the growing season (14,15). Finally, roots of dominant trees were invaded by *F. annosus* at a slower rate than roots of suppressed trees, but only when these roots remained attached to the tree (14,34).

Decay tests. Acetone-extracted reaction zone and incipiently decayed wood (which was resin soaked, but contained substantially less pinosylvin than did the reaction zone) were decayed significantly more by *F. annosus* than were their nonextracted counterparts (45). Wood samples naturally or artificially impregnated with resin acids were decayed significantly less by two decay fungi, particularly by the white rot fungus, *Coriolus versicolor*, than were their nonimpregnated counterparts (21).

In vitro bioassays. Inhibition of *F. annosus* and other decay fungi by pinosylvin and oleoresin or some of their components was generally obtained by a variety of bioassay techniques. Even though the effective dosages of these compounds varied considerably in some of these tests (29), the bulk of evidence supports the view that pinosylvin and some constituents of oleoresin are inhibitory to decay fungi at concentrations that occur in vivo.

A reaction zone also was described in Norway spruce as a nonspecific response to several decay fungi and to mechanical injury. This reaction zone contained a disproportionate amount of phenols, particularly the lignan, hydroxmatairesinol (up to 6% dry wt as compared to less than 0.5% in uninfected heartwood). The reaction zone in interior sapwood of spruce, unlike that in pine, was not resin soaked. This could be due to the less extensive resin duct system and thicker-walled epithelial cells in spruce than in pine (46,47).

The most convincing evidence for the presence of inhibitors in this reaction zone was the fungistatic effect of its filter-sterilized, expressed sap on *F. annosus*. Fungal growth, furthermore, was progressively greater on similar extracts from decayed wood, sound heartwood, and finally sound sapwood which supported luxuriant growth (46). The identity of compound(s) responsible for this inhibition is unclear. Results of an in vitro bioassay indicated that hydroxymatairesinol was the most inhibitory of three reaction-zone lignans that were tested (inhibition was 25, 30, and 40% of controls for concentration of 0.1, 0.2, and 0.4%, respectively) (47). A highly significant correlation also was found between the lignan content of wood samples and their inhibitory effect on *F. annosus* (1). In another bioassay, however, inhibition by hydroxmatairesinol was not detected in concentrations up to 2%, but the lignan, liovil, and another phenol found in the reaction zone, 4-methylcatechol, completely inhibited *F. annosus* at concentrations of ~0.1% and ~0.005%, respectively. It was suggested that synergism may occur in reaction zones among the numerous phenolic compounds that separately are relatively weak inhibitors (37). Inhibition of several extracellular enzymes of *F. annosus* by reaction-zone extracts was demonstrated in vitro (26).

The reaction zone in spruce was more alkaline than were neighboring tissues; eg, about pH 8.0 as compared to pH 5.5 for expressed sap from sound sapwood and incipiently decayed wood (46). This probably was due to the accumulation of inorganic

carbonates (27,46). The significance of this elevated pH is that *F. annosus* is inhibited substantially at levels above pH 7.0 (40).

Mineral content. Analyses of spruce xylem demonstrated that the mineral content of the reaction zone and decayed tissues was substantially higher than that of sound sapwood. Particular increases were noted in potassium, calcium, magnesium, and manganese (1,27,46). Elevated mineral contents which were related to higher pH values also were reported in discolored and decayed tissues of several other tree species (61).

In an earlier report (13), mineral accumulation was observed in decayed wood and it was suggested that fungi may selectively accumulate certain elements. The more recent studies cited above, however, show that mineral accumulation can occur well in advance of fungal penetration; ie, in the reaction zone and even in the transition zone (1). Furthermore, initial increases in potassium concentration in wounded tissue were not related to uptake of that element from the soil (4). Mineral accumulation in uninfected tissue of some tree species, therefore, could be considered as part of the hosts' general response to injury and infection

CONCLUDING REMARKS

A nonspecific response to injury and infection by differentiated sapwood in several tree species that were studied seems to follow the sequence: sapwood → transition zone → reaction zone → infected wood. Parenchyma in the reaction zone dies in advance of fungal penetration, probably as a result of the altered metabolism in the transition zone. During necrobiosis, compounds are produced, or accumulate, which impede but may not necessarily stop invading pathogens. Reaction-zone formation, therefore, could be considered within the hosts' arsenal of defense as a dynamic mechanism of host resistance to a wide variety of injurious agents, including insects (3). The inhibitory compounds that accumulate in the reaction zone, accordingly, could be considered to be phytoalexins.

The extractives-enriched reaction zone has been studied more intensively than the less conspicuous transition zone. If, as proposed, reaction zones are the product of transition zone metabolism, then additional studies of the latter will be required to further develop understanding of reaction-zone formation. The transition zones of many lesions are large, achlorophyllous, and woody—particularly well suited for such studies.

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Symposium on Wood Decay

Mechanisms of Compartmentalization of Decay in Living Trees

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When trees die, the decomposition and decay which releases CO_2 to the atmosphere and minerals to the soil is part of the normal cycle in undisturbed or unmanaged forests. Decay that develops in living trees, particularly in urban plantings and in forests being managed for timber production, becomes a serious problem. It is the single most destructive disease of trees and for many years has been described according to the heartrot concept (1,5).

THE HEARTROT CONCEPT

The basic assumption of the heartrot concept is that living sapwood at the outer margin of the trunk gradually matures and becomes nonliving heartwood at the core of mature trees. Exposure of the heartwood by wounding (by fire, storm damage, lumbering scars, or boring insects) leaves it open to attack by saprobic fungi which fruit, produce spores, and thus spread to other dead heartwood exposed by wounding. Simply stated, the heartrot is the decomposition of dead heartwood inside a living tree by saprobic organisms that gain entrance via wounds. Many plant pathologists have not considered heartrot to be a disease because the decay-causing agent is not interacting with living host tissue.

This simplistic concept has been challenged by later observations. In 1935, Hepting (6) reported that wounded young sweetgum trees without heartwood decayed as readily as wounded young oaks that had heartwood. Further, he observed that the decay was limited to wood formed before the wounding occurred. This led to the postulation that the susceptibility to decay of sapwood formed after wounding differed from that formed before wounding. When it was noted that a dark-colored tissue resembling heartwood was formed in response to wounding, the term "pathological heartwood" was coined to designate localized dark-colored tissue resembling heartwood that formed after wounding rather than by the normal aging process.

This was true in species such as sweetgum, not because localized discoloration was like heartwood, but because the cores of dark-colored wood also were wound-initiated discolorations (27). In sweetgum, larger wounds, such as broken tops or the simultaneous loss of several branches, gave rise to a uniform core of discolored wood. but small trunk wounds caused irregular regions of discolored wood. In both cases, the same wounds that initiated discoloration were the source of decay.

The patterns of decay and discoloration in the northern hardwoods—beech (*Fagus*), birch (*Betula*), and maple (*Acer*) —were established by Shigo (15–17, 22,24). Mature trees of those species do not form a heartwood. Wounded oaks (*Quercus*), which form a heartwood, discolor and decay in the same manner as trees without a heartwood. The results of tree-wounding experiments confirmed the following principles:

i. Wounds initiate discoloration and the process that leads to decay in living trees.
ii. Discoloration precedes decay in wood exposed by wounding.
iii. Discoloration and decay are limited to wood extant at the time of wounding.
iv. The amount of discolored and decayed wood is proportional to the number and severity of wounds.
v. Both sapwood and heartwood discolor as a result of processes initiated by wounding.
vi. The processes initiated by wounding involve both a response of the tree and of microorganisms.
vii. If both sapwood and heartwood are present at time of wounding, decay begins in discolored sapwood and spreads into discolored heartwood.
viii. If discolored wood is wounded, it does not discolor like heartwood.
ix. The electrical resistance of discolored wood is less than that of sapwood, and the electrical resistance of heartwood is greater than that of sapwood.
x. If decayed wood is wounded, discoloration and decay of freshly exposed wood is enhanced.

Thus, it has been clearly established that the presence of heartwood in the stem of a living tree is not essential for the decay process, but the discoloration process is.

Microorganisms other than decay fungi are active in wood during its discoloration. Bacteria and imperfect fungi have been considered pioneers in a succession of microorganisms (18,20,21) in which decay fungi follow. Decay fungi are in turn followed by many organisms that include not only bacteria and fungi. but also protozoa, nematodes, insects, birds, and mammals.

Of interest to the pathologist are the pioneers that interact with living tissue exposed by wounding and the fungi that decompose that tissue. It appears that both pioneer and decay fungi can interact with living sapwood, but only decay-causing fungi can induce discoloration (28). Other pioneers and decay fungi are dependent on the discoloration process that provides a nonliving substrate. Some of these dependent microorganisms invade through the open wound (17,27), while others may already be present as sparse populations in live sapwood, but cannot develop until the sapwood dies.

The growth of decay fungi is retarded during the discoloration process (28). This phenomenon is well known in the Bavendamm reaction on gallic acid medium used as an aid to identify decay fungi (2). Many fungi that cause decay in live trees cannot grow on this medium, but they darkly discolor it by secreting phenol oxidases. If such fungi are placed on live sapwood, the wood may also become darkly discolored. Hyphal growth is greatly retarded during this period. However, if the fungus is able to decolorize the discolored wood—that is, remove dark pigments—then abundant hyphal growth is achieved (28).

This pattern of color change is repeated in the live tree. Wood that is exposed by wounding becomes darkly discolored, and isolates of the pioneer bacteria and fungi are the predominant isolates. Then the wood becomes lighter in color, and the isolation frequencies for decay fungi begin to increase. Both the formation of dark wood and decolorization—the first steps of the decay process—are associated with the loss of phenols as components of the soluble dry matter (31). Phenols act as growth regulators of decay fungi, but not of pioneers. It was postulated that the initial phenol content is sufficient to inhibit decay fungi which must polymerize these substances by oxidation to remove the source of inhibition. Phenol-tolerant pioneers (28,29) flourish until the phenol content has been reduced to a level that permits decay fungi to use cell wall substances as food, at which time the pioneers are replaced. This phenomenon of selection on nondecay organisms by wood preservatives is well founded (3).

THE CODIT CONCEPT

Decay in living trees can now be explained by a model system called compartmentalization of decay in trees (acronym, CODIT) (23), and a concept of succession in which both pioneers and decayers can act as either pathogens or saprobes (28). The CODIT system explains the simple patterns of discoloration and decay observed in trees that lack heartwood and also the more complex patterns observed in trees that form heartwood and that have multiple wounds. The model is based on four "walls" that limit the spread of decay fungi and their pioneers. Wall 1 is a plugging component that limits the vertical spread. Wall 2 is an anatomical component that limits the spread parallel to rays. Wall 3 is a vital component that limits spread perpendicular to rays. Wall 4 is a differentiation component that limits spread into wood formed after wounding. Walls 1, 2, and 3 act in wood extant at the time of wounding; wall 4 forms only after wounding.

Three major factors appear to affect the decay fungi and their pioneers (28): a vitality factor, a preservative factor, and a solubility factor. The vitality factor of live sapwood prevents the growth of many pioneers and decay fungi. It varies with the species and individual tree and with the species and strain of inoculum, but is lost when live cells die. The preservative factor becomes operative during the conversion of live wood to discolored wood and it involves the formation of natural wood preservatives that reduce the high decay rates observed on rapidly killed sapwood. The preservative(s) favor tolerant pioneers and inhibits sensitive wood decay fungi. In the final stages, the importance of the preservative factor declines and that of the solubility factor increases as the dissolution of cell walls (the major source of nutrients in wood) proceeds and becomes limiting. Decay fungi, which have a superior capacity to dissolve cell wall substances, replace the pioneers that formerly were favored by the preservative factor.

Development of the CODIT system with its conceptual four walls and succession of microorganisms governed by major limiting factors has required postulation of the following molecular mechanisms to account for compartmentalization in the simplest case—a single wound in sapwood. A general response of plant tissue to injury and infection is a shift in oxidative metabolism from glycolysis and TCA cycle pathways to the acetate and pentose shunt shikimic acid pathways (8). Accumulating products of these alternate pathways may then act as growth inhibitors (a preservative factor) or help seal off the tissue (a solubility factor). Other consequences of a shift in oxidative metabolism are: loss of available energy as food reserves are converted to products of the acetate and shikimic acid pathways, stabilization of readily ionizable phenols owing to reduced polarity of their environment, increased randomness of chemical bonding in food sources generated by resonance forms of conversion products, loss of available nitrogen by precipitation of tanned proteins, and lowering of available oxygen by polyphenols.

Although large, dead tracheal cells make up the bulk of mass and volume, the metabolic shift occurs in the live parenchyma cells, which are the most abundant cells of sapwood (Table 1). The network of small, live cells react to protect the large, dead cells from decomposition after an injury. Years of study have established that products converted from food reserves stored in sapwood protect the cell wall substances of heartwood, and provide wood with a natural resistance to decay (11). In fact. the same shift in metabolism must accompany the death and lignification of the

large cells as they differentiate from cambial derivatives.

A biosynthetic change in constituents of the small live cells should be maximized in the rays. where such cells are concentrated CODIT system, wall 3 and in the last latewood, where conducting elements are smallest and least abundant (CODIT system, wall 3) (Fig. 1). Failure to generate enough plugging materials or tyloses in the remaining tissue (CODIT system, wall 1) most often leads to the failure of compartmentalization. Levels of preservative factors that are sufficient to prevent the spread of microbes in dense tissues may fail in more open tissues, and large columns of discolored and decayed wood may form.

Where walls 1, 2, and 3 fail in extant wood, the biosynthetic machinery of cambial derivatives usually succeeds. Cambial derivatives form a sheet of living tissue in which shifts in metabolic activity can produce profound changes in both anatomical structure and chemical composition. These changes maximize the reactivity of wall 3 and the density of wall 2, thus negating the inherent weakness of wall 1 (Fig. 2).

Evidence is accumulating that metabolic activity shifts during compartmentalization. In 1967, Shain (12) characterized a zone in pine that surrounds wood infected by *Fomes annosus.* This "reaction zone" was resin-soaked and had an increased phenol content. Thus, normal soluble dry matter of pine sapwood was converted to products of the acetate and shikimic acid pathways as predicted by the general scheme of plant response to injury and infection. A similar zone in spruce was later described, where phenols accumulated in the reaction zone and resinous components were less prominent (13). The reaction zone described by Shain (12,13) corresponds in general to CODIT walls 1, 2, and 3.

For a decade, several collaborators and I have studied the response of sapwood to wounding and inoculation. We used maple for the basic experiments because of its simple structure and its wide range. Later we used other species to extend principles that were developed in the studies of maple.

The results of analyses following simple homogenization of wood exposed by wounding indicated that products of a shift in oxidative metabolism accumulated in sapwood before it became discolored (Table 2). Subsequent discoloration then resulted in a reduction of phenol content, which reached a minimum in decaying wood (31). Phenol content reached a maximum in the bright-colored tissue found at the margin of discolored wood and sapwood (26). Soluble dry matter of this marginal zone was double that of sapwood. The ratio of phenol to carbohydrate (wt:wt) in dry matter readily soluble in water increased from 1:10 in sapwood to 2:1 in marginal tissue, then decreased to 1:3 in discolored wood. Dry matter from both marginal and discolored wood retarded the growth of pioneer and decay fungi alike, although growth of the latter was retarded more. In culture, a ratio of gallic acid (the common water-soluble phenol of sapwood) to glucose of 1:10 allowed more growth of pioneer fungi than of decay fungi, but at 1:100, the decay fungi grew better than did the pioneers (28).

Phenols of the bright-colored marginal zone in maple were not the same as those of the sapwood. Sapwood contains phenols primarily of the tannin-type—both the hydrolyzable tannins built

TABLE 1 Cellular composition of sweetgum sapwood

Cell type	Weight (%)	Volume (%)	Number (%)
Parenchymal	5	20	95
Tracheal	95	80	5

TABLE 2. Formation of phenols in response to injury of sugar maple sapwood (milligrams phenol per gram of moisture-free wood)[a]

Condition of wood	Acetone	Hot water	Total
Noninjured	0.7	1.8	2.5
Injured	1.2	2.3	3.5

[a]Mean of paired samples from three trees. Shavings of wood were taken from the blue-fluorescent zone (UV, 365 nm) associated with a wound and from equivalent nonfluorescent wood of the same tree (before formation of discolored wood). homogenized in acetone, boiled in water, and the phenol content was determined with Folin-Ciocolteau reagent.

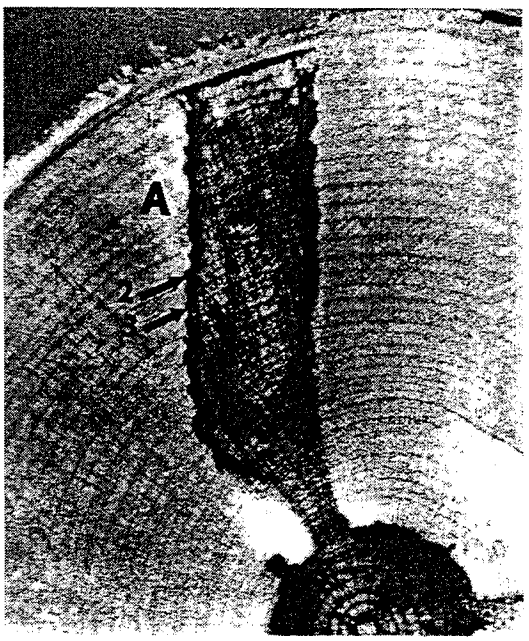

Fig. 1. Uneven edge of column of discolored wood (A); gross effect of greater effectiveness of a reaction zone formed in ray tissue (CODIT wall 3) and in latewood (CODIT wall 2).

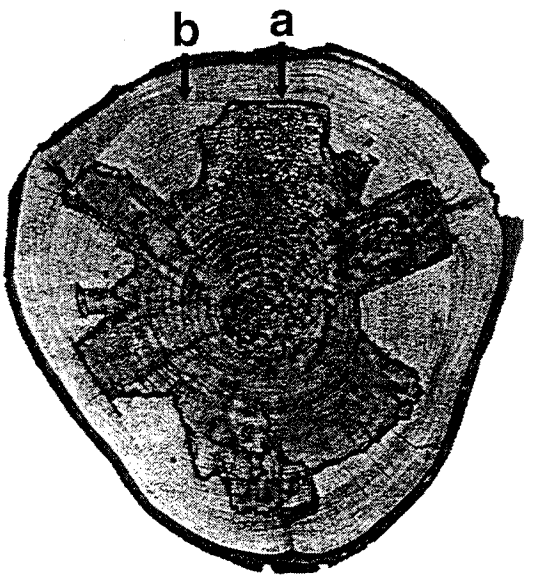

Fig. 2. Column of discolored and decayed wood 5 yr after multiple wounds were inflicted with a large screwdriver; a = barrier zone in contact with discolored wood, b = barrier zone not in contact.

on gallic acid residues and the condensed tannins built on catechin residues (30). Phenols of the marginal tissues were less polar, which suggests a relationship with the fatty products of the acetate pathway (26). The ultraviolet (UV) spectra and bathochromic shifts of these phenols when ionized suggested the presence of hydroxycinnamic acid derivatives. The phenols of the margin differed from tannins in chromatographic behavior and in susceptibility to aerial oxidation. However, further progress in their characterization requires a solution to the problem of sampling this narrow marginal zone (< 2 mm in width).

Given the working hypothesis that a reaction zone forms at the margin of columns of discolored wood and operates in a fixed structure as walls 1, 2, and 3 of the CODIT system, what regulates the effectiveness of compartmentalization? At present it appears that the genes of the tree are an important factor (25). The genes of the inoculum also appear to be important, as inferred from in vitro studies (28), and soon to be confirmed in vivo (2-yr studies nearing completion). Also, the normal response can be altered by chemical treatment of wounds (32) and by manipulating the inoculum in wounds (10). Detailed studies of mechanisms that regulate the compartmentalization process remain to be done.

The most effective barrier to the spread of discoloration and decay does not form in the wood extant at the time of wounding, but in the wood that grows afterward. This tissue, called a barrier zone, is not well understood. Sharon (14) described a "distinct tissue" in maple that corresponds to the "barrier wall" of Shigo (21). The gross features of a "barrier zone" were described by Shortle in sweetgum and yellow-poplar (27). The anatomical features of the barrier zone of sweetgum were then studied by Moore (9), who greatly expanded the pioneering work of Gerry (4). The storax secreted by resin canals in sweetgum wood formed after injury again supports the hypothesis for a shift in oxidative metabolism that results in products of the acetate pathway (β storesin) and the shikimic acid pathway (cinnamic acid). Both storax and cinnamic acid completely inhibited the growth of a decay fungus (28,29).

Studies in progress have shown that the wood formed adjacent to the wound at varying distances has fewer, shorter vessels and increased opacity to transmitted light. This zone has a higher than normal phenol content, and where it comes in contact with discolored wood, a very dark edge is formed (Fig. 2)—although the wood of the zone is not discolored.

Isoelectric focusing of isoenzymes of indole acetic acid oxidase (peroxidase) from differentiating cambial derivatives shows a loss of many isoenzymes within minutes after wounding. Compared to nonwounded controls, this loss is followed by changes in enzyme activity and levels of phenolic effectors of that activity during the next few weeks. Studies of the changes in the kinds and activities of enzymes that regulate levels of growth promotors and growth arrestors (7) as the barrier zone forms, may lead to further understanding of this process.

In general, where the barrier zone meets the tissue responsible for wound closure, it looks most like callus. Then, with, increasing distance from the callus, the appearance of the zone grades back into that of normal wood—more quickly circumferentially than longitudinally. Thus, each wound has an elliptical barrier zone around it. It is not known how the size of the ellipse or the thickness of the zone varies with the size and severity of any given wound. It is known that ring shake develops between the barrier zone and the wood which was extant at the time of wounding (19).

SUMMARY

The heartrot concept has failed to explain the patterns of discoloration and decay observed in living trees. The CODIT system and a concept of succession, in which both pioneer and decay fungi may act as pathogen or saprobe, explain much more. Working hypotheses for the study of molecular mechanisms of compartmentalization can be deduced from these general schemes by reference to three well-established principles of plant and wood pathology: a shift in oxidative metabolism is associated with injury and infection, food reserves in sapwood converted to products of the acetate (resinous materials) and the shikimic acid pathways (phenols and other aromatic compounds) cause wood to become more decay resistant, and bacteria and nondecay fungi grow selectively in wood preserved against decay fungi. There is evidence to support these hypotheses. and more is being generated at this time. I can only hope that I have outlined new patterns of thinking about decay in trees that will lead to a better understanding of this serious tree disease problem.

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Symposium on Wood Decay

An Expanded Concept of Tree Decay

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CLASSICAL CONCEPT OF TREE DECAY

More than a century ago the concept of spontaneous generation of life, and with it the autogenetic theory of plant disease, was abandoned by most scientists. Anton DeBary established the germ theory of disease. Shortly thereafter, Robert Hartig ushered in the field of forest pathology by developing a concept of tree decay centered about three major points: fresh wounds, the infection of the freshly exposed heartwood by Hymenomycetes, and the subsequent decay of the infected wood. This concept of tree decay has persisted in textbooks virtually unchanged for nearly a century. Emphasis is upon the physical and chemical characteristics of the decayed wood and on the taxonomy of the associated Hymenomycetes. Until the 1950's, all attention was focused on the Hymenomycetes, in spite of the publication of scattered research papers indicating that not only were other fungi associated with the Hymenomycetes, but also that some of those alone could cause decay in (or at least alter) wood.

In living trees, the decayed wood has been considered to be primarily heartwood, and heartwood has been considered to be the dead, nonresponsive central core of the living tree. Heartwood has been considered to be any wood darker in color than the sapwood. Many variations of heartwood have been described: wound heartwood, false heartwood, precocious heartwood, and so forth. The decay of heartwood in living trees has been termed "heart-rot," and completing the circle, "heart-rot" in turn has been defined as the decay of the heartwood, the dead, nonresponsive central core of the tree.

Many pathologists have not considered tree decay to be a disease. Because such decay was considered to be confined to the dead, nonresponsive central core, they considered the fungi involved to be saprophytes rather than parasites. They had overlooked the essential point that parasitism is defined on the basis of interactions at the organism level and, further, that there are living cells in "heartwood." Tree decay is a disease and the associated fungi are both pathogenic and parasitic.

Cavities within trees were described as the final result of complete digestion of the heartwood. The progression of decay in a longitudinal or vertical direction was considered to be similar to processes occurring in a horizontal or cross-sectional direction. Initial infection was described as germination of spores of Hymenomycetes on freshly exposed heartwood even though spores of many such fungi never have been observed to germinate on any substrate. Details on many of these points are in Chapter 16 of the textbook, Forest Pathology, by Boyce (1).

DEFICIENCIES OF THE CONCEPT

The results of studies published during the past two decades have revealed several obvious deficiencies in the classical concept of tree decay. The classical concept does not consider: the response of the living tree to wounding and to invasion by microorganisms, the bacteria and nonhymenomycetous fungi that infect tree wounds, that many tree species lack a true heartwood, and that even in true heartwood injured and infected tissue often is walled off. Thus, the classical concept must be expanded and modified.

First, it is necessary to consider how a tree is constructed and how it is pre-set chemically and anatomically to survive or react after injury to the xylem. Trees do not heal wounds that extend into the xylem: the word *heal* denotes to repair, to replace. or to restore injured and infected tissues to a previous healthy state. Trees cannot do this. Instead, a tree is a highly compartmented organism. These compartments are delimited by the last cells which form in the annual rings and the rays. After injury, the tree's defense system reacts anatomically and chemically to isolate, wall off, or compartmentalize the infecting microorganisms and damaged tissue so that the fewest compartments are affected. Compartmentalization has great survival value for a long-lived tree.

Many types of microorganisms are involved in the processes that lead to discolored and decayed wood. In some cases, the Hymenomycetes are the first organisms to interact with the tree. But in most cases the freshly exposed injured tissues are rapidly colonized by bacteria, yeasts, and many types of nonhymenomycetous fungi. The first microorganisms to penetrate into the tree alter both the sapwood and heartwood.

After a tree is wounded it reacts by forming inhibitory compounds in the tissues surrounding the wound. These inhibitory compounds limit the infection caused by most microorganisms. Nevertheless, if the microorganisms are aggressive enough, or if environmental conditions favor the microorganisms, infection will occur. There is intense competition among microorganisms for the available simple nutrients in the tree. Some pioneer microorganisms alter the wood in such a way that they stimulate spore germination of and infection by the Hymenomycetes. Other microorganisms alter the wood in such a way that they slow the rate of infection by Hymenomycetes. Regardless of the nature of the processes, succession does occur.

Many conditions affect the nature of succession: type of wound, position of wound, time of year the wound occurred, and the size and depth of the wound. Successions are complex events, and it will take time and patience to unravel their intricacies.

CURRENT RESEARCH

The papers in this symposium focused on four major points: the unique internal environment of the wood in the living tree, the dynamic response of a tree to infection, the inhibitory materials that affect resistance to infection by the Hymenomycetes, and the metabolic shunts that occur after wounding and infection.

Hart and Shrimpton (4) touched upon three critical points. First, that in studies published to date seldom have the rules of proof been followed that are necessary to establish a cause and effect relationship between a particular chemical extractive and the decay resistance of wood. Second, that we must know more about where wood extractives are localized in the cell walls and lumina, and how these extractives are bound to other wood constituents. Evidence suggests that these chemicals are not bound to cellulose but may be bound to lignin. Are they bound to hemicelluloses or other extractives? Third, that decay resistance probably is multifunctional. Most studies to date have concentrated on a single compound or class of compounds, and have ignored the multitude of other extractives present in the wood. Interactions among

extractives are highly probable and this will be a difficult subject to unravel.

In the sequence of events leading to discoloration and decay of living trees, the initial reaction is an autonomous response of the tree. An understanding of these plant responses is prerequisite to understanding the rest of the discoloration-decay sequence. These initial responses may condition the rest of the entire sequence. Mullick (7) recently postulated that blockage or cessation of xylem transport is one of the first reactions of wounded xylem tissues. Perhaps it is this response which leads to the formation of transition and reaction zones and the changes in moisture levels as discussed by Shain (8). Mullick (7) stressed the lack of conclusive proof at the present time. This is a subject worthy of critical study.

There are seven other points which we wish to raise. First, most of the research to date, as summarized in three of the four previous papers of this symposium, has been done in the laboratory. There is grave danger in extrapolating the results of a limited petri dish study to the complex sequences of events involved in the processes of discoloration and decay of wood in nature. Such in vitro studies must be done; nevertheless, it must always be foremost in our minds that the results of such studies may never relate to nature and that they may be only laboratory artifacts!

Second, decay fungi *never* occur alone, either in living trees, in slash, or in wood in use. Wood decay is the final result of a sequence of events. Studying those organisms or processes at the *end* of this sequence will not lead to an understanding of the total process.

In living trees, the microorganisms associated with the decay fungi affect pH; the zones in which they occur may be quite alkaline. They may be associated with an accumulation of minerals; some of these minerals may affect the metabolism of the organisms appearing later in the succession. The metabolic activities of the associated microorganisms undoubtedly affect CO_2–O_2 relations in the living tree; this may not occur or may occur to a lesser extent in dead trees or cut lumber. The associated microorganisms may secrete antibiotics or stimulants, they may detoxify or consume extractives, or may modify the wood constituents.

Third, successions are a complex series of events. We may have to do thousands of studies and examine thousands of trees to detect the *patterns* that exist. An understanding of the general nature of these patterns may be as close as we ever come to unraveling the intricacies involved. It may never be possible to duplicate these patterns in vitro, or even to create them at will in vivo.

Fourth, in most studies to date, including those of Hart and Shrimpton (4) and Highley and Kirk (5), the decay organisms used have been the slash-rotting fungi—those which decay litter and woody debris. Although such studies may relate to wood in use, they probably do not relate at all to decay in living trees. The true heartrotting fungi have been little used in such studies, probably because they cause very small amounts of decay in vitro. A further point of concern is the extrapolation from information about a fungus which is primarily a root-rotting fungus to those fungi which rarely if ever occur in the roots. Such extrapolations would be more acceptable if it could be demonstrated that *Fomes pini* elicits the same responses as *F. annosus* under similar conditions.

Fifth, most existing data on tree decay must be examined with great skepticism. The problem lies in the characterization of the tissues being studied. Prior to the relatively recent studies by Shigo and his co-workers, all dark-colored wood was considered to be heartwood. Much of this wood actually was discolored due to the autonomous responses of the tree to wounding and the invasion and subsequent activities of various pioneer microorganisms. We now know that heartwood and discolored wood are not the same, and do not react the same. Thus, there is no way to interpret the results of earlier studies.

Sixth, it has long been stated that the true heartrotting fungi invade through dead branch stubs, and this seems to be true in hardwoods. In conifers, however, stubs of branches which have died naturally often are the most decay resistant portions of the wood. Further, Etheridge and Craig (3) showed that *Echinodontium tinctorium,* a true heartrotting fungus, invaded only through small living branches. Other heartrotting fungi in conifers may behave similarly, and it also may be true for certain fungi occurring in hardwoods. For example, Etheridge (2) was unable to find *Fomes igniarius* growing inwards through dead branches of aspen. If the fungus occurred in a dead branch, it always occurred as an outward growth from an internal column of decayed wood.

Even if some fungi do enter living trees through branch stubs, at the present time it seems unwarranted to assume that all of the cells in the branch stub are dead and that the tissues are incapable of reacting to this invasion. For example, Shigo and Shortle (9) recently showed that even so-called dead, unresponsive true heartwood of oak can, and does, react to invasion by the fungi involved in the discoloration-decay syndrome.

Seventh, many techniques (particularly inoculations) used in in vivo studies have been inadequate. In retrospect, some of these are ludicrous.

The most commonly used technique has been to insert a large wooden dowel overgrown by a test fungus into a deep hole in a tree. Such an inoculum load is probably a billion-fold greater than any occurring in nature, even if one ignores the fact that the natural inoculum probably consists of basidiospores! Further, the mycelium was placed in direct contact with freshly injured xylem tissues. To add insult to injury, in many studies oat seeds or similar nutrients were added to the wound to give the inoculum an even greater boost. The only information obtainable from such studies is a description of what happens when trees are inoculated in such a manner. All such data are artifacts, which have no relationship to the events that occur in nature. Further. when isolations were made from such trees, all nonhymenomycetous organisms were considered to be merely contaminants. Over a century after Robert Hartig showed that Hymenomycetes were involved in tree decay, still virtually nothing is known about the germination of hymenomycetous spores in various infection courts, the initial penetration into the suscept plant, or the subsequent colonization of the host (6).

A LOOK TO THE FUTURE

Clarification of the decay processes to include compartmentalization and succession provides new opportunities to regulate tree decay. A tree is constructed in an orderly manner, and when it is injured and infected, it responds in an orderly way (compartmentalization). When microorganisms infect trees, they do so in an orderly manner (succession). The more completely the nature of compartmentalization and succession are understood, the better are our chances for regulating the processes of tree decay.

We can now select individual trees within a species that compartmentalize discolored and decayed wood to very small volumes. Viewed in this way it is not so important whether the tree can be infected, but that the infection has been walled-off or compartmentalized to a very small volume within the tree. In this sense, it is now possible to select decay-resistant trees.

This compartmentalization effect also appears to function in tree roots. This may explain why some trees live for many years following multiple root infections, whereas some trees die very quickly after becoming infected.

The anatomy of trees in some species may favor a higher degree of compartmentalization than that of trees of other species. This may help to explain why some trees, such as species of *Populus* and *Betula,* have very long columnar defects, whereas species of *Acer,* especially *A. saccharum,* have very short columnar defects. With detailed anatomical information it may become possible to develop schemes that will outline the limits for defect potential in different tree species. It also would aid geneticists selecting tree breeding stock for reduced defect potential.

By understanding the successional patterns of colonizing microorganisms, it may be possible to utilize biological control agents that could stall or stimulate the decay process.

In summary, we must now consider the living tree, and how it compartmentalizes injuries and infections. We also must consider the impact of many microorganisms operating in successional patterns in living trees. Better regulation of the decay process depends upon a clearer understanding of this expanded concept of tree decay.

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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.
After you answer the questions in this part, proceed to Appendix A and complete Assignment #1 for submission to your tutor for marking.

1. What special feature distinguishes decay fungi from all other fungi that digest cellulose?
2. What enzymatic features differ between white and brown-rots?
3. List two major roles that decay fungi play in natural ecosystems.
4. What are the minimum requirements of decay fungi for growth and reproduction?
5. How do living trees become infected with decay?
6. List and describe at least five ways in which the spread of decay in the heartwood of living trees may be either retarded or promoted.
7. How do trees react to decay of their heartwood?
8. How do trees react to wounding of the bark in a way that influences decay development?
9. Examine and describe at least two decay fungus fruiting bodies found on living or dead conifers in your area. Specify colour, size, shape. location, features of the lower surface, and type of decay produced.
10. Which species of conifer is thought to have the greatest amount of decay in your area?
11. What are cull factors, and how are they derived?
12. What are the basic rules for prevention of decay in wooden structures?

LESSON 3 Root Diseases

LESSON OVERVIEW

CONTENT

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

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

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

OBJECTIVES

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

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

LESSON STUDY INSTRUCTIONS AND ASSIGNMENT

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

COMMENTARY

Types of Root Diseases

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

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

The Soil Environment

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

Antagonism

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

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

Mycostatis

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

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

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

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

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

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

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

Root Structure

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

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

The Main Basidiomycetous Root Pathogens

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

Spore Infection

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

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

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

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

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

Phellinus weirii

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

infection and spread

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

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

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

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

identification

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

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

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

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

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

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

assessment

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

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

management

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

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

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

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

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

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

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

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

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

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

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

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

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

special considerations

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

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

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

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

Armillaria ostoyae

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

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

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

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

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

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

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

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

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

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

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

Heterobasidion annosum

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

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

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

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

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

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

Inonotus tomentosus

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

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

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

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

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

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

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

Phaeolus Schweinitzii

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

Root Diseases Caused by Non-Decay Fungi

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

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

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

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. Construct a table that lists, for each of the major root diseases, tree species that are susceptible, moderately resistant, and immune.
2. Why are root diseases sometimes called diseases of the site?
3. List the various ways in which root pathogens can advance proximally along living roots.
4. Relate tree age at infection and the length of time it will take to kill the tree (if ever) for all the major root diseases.
5. In which way does mycostasis restrict the spread of root diseases?
6. Describe the role of root pathogens in natural ecosystems.
7. Why is stump treatment with fungicide sometimes recommended for *Heterobasidion annosum,* but not for the other root diseases?
8. What are the advantages and problems associated with the two main management approaches (stump removal and replacement with resistant species) for *Phellinus weirii*? For *Armillaria ostoyae*?

LESSON 4 Wilt, Foliage, Bark and Seedling Diseases

LESSON OVERVIEW

CONTENT

This lesson is divided into four self-contained sections, each dealing with a particular group of diseases, as indicated by the title of the lesson. Each section has its own reading assignment, and is followed by a set of self-testing/review questions.

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

- Wilts: Diseases of the vascular system
- Foliage diseases
- Cankers
- Seed and seedling diseases

OBJECTIVES

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

1. to recognize typical signs and symptoms of each of the four groups of diseases;
2. to describe their life cycles and infection pathways;
3. to identify some of the common diseases in each of the groups;
4. to assess the impact of these diseases on management goals and how to take preventive or remedial action to limit damage.

READING

Specific reading assignments are suggested in each section.

LESSON STUDY INSTRUCTIONS AND ASSIGNMENT

Study each section in turn, answering the self-testing/review questions before proceeding to the next section.

After you have studied all the sections, complete Assignment #2 (in Appendix A) and submit it for marking. This assignment covers content in Lessons 3 and 4.

COMMENTARY

SECTION 1

WILTS: DISEASES OF THE VASCULAR SYSTEM

reading

Start by reading Chapter 13 of Manion (1991), then study the material that follows in this manual. To test your understanding of the material in this section, answer the self-testing/review questions before proceeding to the next section.

Wilt diseases are diseases of the vascular system of hardwoods. The pathogen lives in the most recently formed xylem and interferes with normal water translocation. In most cases, the causal agents belong to the Ascomycotina, but the perfect or sexual stage is often rare or unimportant in the life cycle. Infection usually requires insect (e.g., bark beetle) vectors but it may also occur via root grafting to adjacent infected trees, or occasionally via wounds (e.g., through the use of unsterilized pruning tools). The pathogen commonly produces small spores which are carried passively from the point of penetration up and down the xylem. The typical symptom is sudden wilting early in the season, at a time when the transpiration stream runs mostly through the large spring wood vessels. These large, diseased vessels are often stained.

CAUSES OF WILT

Possible causes of wilting are:

1. formation of tyloses that block vessels, in an attempt by the host to limit the spread of the pathogen;
2. blockage of vessels by fungal hyphae and spores;
3. breakdown and solubilization of cell wall material resulting in increased viscosity and hence slower flow rates;
4. introduction of air bubbles into the vessels through disruption of the cell wall;
5. production of toxins by the pathogen.

Of these, the last is probably the major mechanism. The toxin(s) may disrupt normal stomatal function, leading to excessive transpiration. While the other mechanisms may contribute, they cannot by themselves explain the sudden wilting of all the foliage on major branches or the whole crown.

DUTCH ELM DISEASE

In North America, the best known example of wilt is Dutch elm disease (DED) caused by *Ophiostoma ulmi* (formerly known as *Ceratocystis ulmi*). Symptoms include sudden wilting of large sections of crowns in mature elms and a ring of brown stain in the outer vessels of branches in the affected crown parts. This pathogen was introduced into Quebec from Europe during the second world war, probably on elm lumber cut from unbarked logs which carried live bark beetles. There may have been an earlier introduction in U.S.A. It has since spread west to the Rockies

and will no doubt arrive in Vancouver and Seattle in the next decade or so. Transmission is by insects, mainly the European elm bark beetle (*Scolytus multistriatus*) and, to a lesser extent, the American elm bark beetle (*Hylurgopines rufipes*). When these bark beetles emerge from infected trees, they carry spores of the pathogen on their bodies and in their mycangia. They feed in branch crotches and leaf axils of nearby trees, occasionally transmitting the disease, and then mate and bore a gallery, infecting the brood tree. In the major springwood vessels the fungus produces small asexual conidia which are carried passively through the vascular system. The fungus also fruits in the beetle galleries, producing abundant coremia (another asexual spore stage) and occasionally perithecia.

MANAGEMENT TECHNIQUES

The American elm is extremely susceptible. It is also an important shade and landscape tree, and hence there has been much concern about this disease. Initial control was by the use of DDT to kill the bark beetles. This was fairly effective but required large and repeated applications.

Massive DDT applications to control DED on the Michigan University campus triggered the publication of a book by Rachel Carson, *Silent Spring*, in the early 1960s. This book is considered germinal to the current popularity of the North American environmental awareness movement.

Current management techniques are aimed at protecting large, valuable elms in urban settings from the disease. Some are directed at the vector, and for these a good understanding of the local vector life cycle is required. Others are aimed at the pathogen. The main methods include:

1. Sanitation — if all trees showing symptoms are immediately removed and burned, infected beetles will not emerge. Sanitation does not work if there is a large population of untreated elms in the vicinity (e.g., a forest). Some towns in New England which have good sanitation programs have retained more than 70% of their elms, compared to 100% mortality elsewhere. Speed, and access to all elms on public and private property are essential for a successful sanitation program. Costs are not really a factor, since elm trees must be removed after they die anyway.

2. In elms planted closely together in rows, the pathogen can spread from tree to tree via root contact. In such cases taking out alternate trees can usually stop further spread.

3. Disease development in infected trees can sometimes be retarded or stopped by injection with systemic fungicides such as Benomyl. This is an expensive treatment that must be repeated every time the tree is reinfected, and hence is suitable only as a stop-gap measure for very valuable trees.

4. Insect trapping using pheromones can reduce insect populations and infection.

5. Breeding of resistant trees is in progress, and current releases show some promise, although there is concern about the stability of such resistance. Apparently, the pathogen exists in several forms of variable aggressiveness; circumstantial evidence suggests that new aggressive strains have arisen from time to time.

Other Wilt Diseases

Other major wilt diseases include oak wilt caused by *Ceratocystis fagacearum* in certain parts of the eastern hardwood forests, and *Verticillium* wilt of eastern maples and elms caused by *Verticillium dahliae* or *V. albo-atrum.* In the case of *Verticillium* wilt, the pathogen survives in soil as dormant sclerotia, and infection takes place through small root wounds. Also, the development of symptoms is much slower. Black stain root disease, described in the lesson dealing with root diseases, also has many characteristics of a wilt disease. However, since it occurs in conifers, it is not normally included with wilt diseases.

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. What are the mechanisms that account for the symptoms of wilt diseases?
2. Which control measures against Dutch elm disease are aimed at the vector insect? Which deal with the pathogen directly?
3. If you were concerned about the expected arrival of Dutch elm disease in Vancouver, what measures would you take now to prepare for the arrival of the disease?

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 micro-organisms 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?

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?

SECTION 4

SEED AND SEEDLING DISEASES

reading Read the commentary, then answer the self-testing/review questions.

Up to this point, this course has dealt with diseases of particular tissues and organs. We have discussed root diseases, decay of wood, and diseases of bark, foliage, and the vascular system. That covers nearly all tree parts (nothing about flowers and fruits so far), and you may well wonder what else there is to discuss. From here on, this course deals with diseases caused by special groups of pathogens, namely the rusts and the dwarf mistletoes, and, in this section, diseases of seedlings. Seedling diseases deserve a special category because seedlings in nurseries are exposed to special and artificial conditions that lead to a set of diseases that are rare or absent in the field, and because young seedlings consist largely of succulent tissues not protected by periderms, and they don't have the energy reserves of larger trees to respond to diseases and to repair damage.

Seedlings occur in two kinds of places: as natural regeneration in the forest, and in nurseries (both bare-root and container). Virtually nothing is known about seedling diseases in the forest, while the nursery situation has been intensively studied. Stands of mature trees produce large quantities of seed. Seed fall often exceeds a million live seeds per hectare. Some of this seed is eaten by rodents, some is killed during the winter months while it lies dormant on the seedbed, and some if it germinates in the spring, and then dies within a short period of time. Causes of death after germination can include drought, grazing by rodents, insects and slugs, and diseases. Sometimes the forest floor microbial community includes potent pathogens of seed and seedlings. This is the case, for instance, for Engelmann spruce and subalpine fir seed deposited on undisturbed forest floors in the ESSF. Interestingly, seed deposited on nurse logs or on exposed mineral soil in these forests does not suffer nearly as much from these diseases., and that may be why these are the only successful seed beds in this zone. Much needs to be learned in this regard, and as silvicultural alternatives to clearcutting and planting are being explored, more will be learned about the pathology of natural seedlings in various seed beds.

Conifer seed can be destroyed by pathogens before it begins to germinate. The seed fungus, *Calocypha fulgens,* is commonly the cause. *C. fulgens* occurs naturally in the forest floor. Cones that are in contact with the forest floor can become infected, and the seed in such cones is destroyed. The typical symptom is that the endosperm and embryo remain firm but somewhat shrunken and mummified. In contrast, pre-emergence damping-off is a condition in which the seed contents become soft, rotten, and water-logged. *C. fulgens* can spread from seed to seed while seeds are in storage, and particularly during stratification.

Thus a few infected seeds in a seed lot can result in major losses. The remedy is to avoid prolonged contact of cones with the forest floor during cone collection and storage. Infected seed lots require the use of fungicides during stratification to avoid further spread.

Damping-off is a disease in which succulent stem tissues are invaded shortly after germination, and the seedling is killed. This can be caused by a large number of pathogens, all producing similar symptoms. In **pre-emergence** damping-off, infection occurs shortly after the radical begins to emerge from the seed, and the seedling never emerges above the soil surface. In **post-emergence** damping-off the attack usually occurs at the ground line. Initially the cortex is invaded and it becomes brown and shrunken. The seedling then topples over, but the cotyledons remain green and turgid for a while. The common pathogens causing damping off are various species of *Fusarium, Pythium, Phytophthora, Rhizoctonia,* and *Cylindrocladium.* In all cases the inoculum is present in the soil as dormant spores, and these germinate in the presence of young roots, presumably following stimulation by root exudates. Continued use of the same nursery beds will serve to increase the inoculum and the severity of the problem from year to year. However, environmental conditions are also critical. Most damping-off fungi are favoured by a soil pH higher than about 5.7, and therefore also somewhat higher than the optimum for seedling growth. Water logging also promotes damping-off. Thus good soil management techniques, combined with crop rotation, can minimize losses. When the epidermis on seedling stems and roots is replaced with the first periderm, four to eight weeks after emergence, damping off ceases because (1) exudates are greatly reduced and (2) the pathogens cannot penetrate the periderm readily.

Older bare-root seedlings are also affected by a number of pathogens. Various species of *Fusarium* also attack older seedlings. Sometimes the stem is invaded and the upper part of the seedling killed; sometimes invasion is through the roots, and tops look stunted. The other pathogens that cause damping-off may also continue to infect larger older seedlings, causing lesions on stems and roots. In all these cases, the growing conditions are critical. Apparently several of these pathogens may be present on, and even inside, seedlings without causing any symptoms, until times of unusual stress such as extreme heat, flooding or drought. Such periods of stress cause a change in behaviour: instead of acting as endophytes, roots and stems are rapidly invaded and killed. Sometimes these pathogens are carried on or in asymptomatic seedlings, and are expressed after the seedlings are planted in the field.

Sirococcus blight caused by *Sirococcus strobilinus* can be serious in container nurseries. In most cases, the pathogen is introduced on the seed. The typical symptoms are necrosis of the base of the cotyledons, while their tips remain green for a while. The fungus produces abundant asexual spores on the dead tissue, resulting in rapid spread within the nursery, and, if uncontrolled, in large losses. Container nurseries are also subject to grey mold caused by *Botrytis cineria. B. cineria* is a weak pathogen and a common saprophyte on dead organic matter. Spores are

present everywhere. The fungus becomes established on dying needles low within the dense stands of seedlings. In that moist, shaded environment, spore production is rapid, and all the lower needles become infected. From such needles the pathogen spreads into the stem, causing small lesions, and sometimes girdling seedlings. The simplest cure, increased aeration around the base of seedlings, lowers the relative humidity within the seedling canopy, and reduces both spore production and infection to tolerable levels.

A final problem is that of storage molds. Many regeneration systems require that seedlings be stored under cold and dark conditions for months before planting. Temperature is critical during storage. At one or a few degrees below zero, mold pathogens do not develop. However it is a common practice to allow a thawing period of a week or more, and this plus even longer periods at just above zero during transportation and storage in the field, is the critical time. Such conditions are ideal for various molds. Seedlings that were green when placed in storage may be totally molded and essentially dead when they are finally taken out of the box. At one time the fungicide captan was widely used to prevent storage molds. However prolonged exposure to that fungicide experienced by planters has had negative health impacts, and the practice has pretty well been discontinued. It now appears that care during transportation and storage, so that the temperature is not allowed to rise above zero until the last possible moment, will effectively remove the problem. Even a few days at an elevated temperature can result in significant molding.

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 lesson 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.
After you answer the questions in this part, proceed to Appendix A and complete Assignment #2 for submission to your tutor for marking.

1. Why is it useful to consider seedling diseases as a special category of diseases?
2. What are the symptoms of "damping off"?
3. Seedling diseases of bare-root nurseries often involve pathogens that differ from seedling diseases that are common in container nurseries. Why is this?
4. hat conditions are required for the development of gray mold?
5. How can damage by "storage molds" be prevented?

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

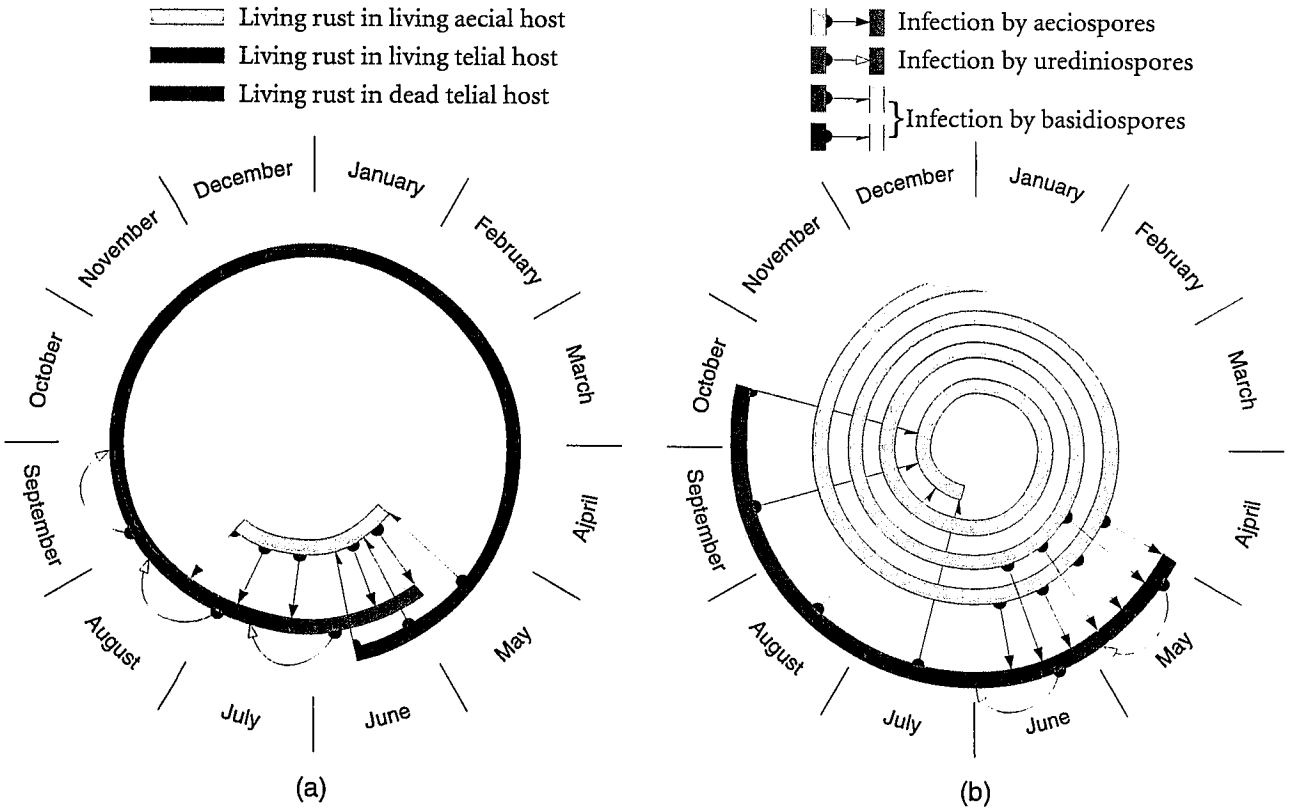

FIGURE 5.1 Life cycles of (a) *Melampsora* and (b) *Cronartium*. [Adapted from W.G. Ziller, 1974, *The Tree Rusts of Western Canada*.]

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.

LESSON 6 The Dwarf Mistletoes

LESSON OVERVIEW

CONTENT

Dwarf mistletoes, common parasites on several important conifers, are vascular plants that belong to the genus *Arceuthobium* in the family Loranthaceae. Their characteristics are such that there are many opportunities to reduce losses by careful silvicultural prescriptions. Chief among these characteristics are the obligate parasitic nature of dwarf mistletoes, the low rates of spread, the host specificity, and the ease of detection.

This lesson starts with a description of the life cycle of dwarf mistletoes and the manner in which they affect trees. Following that, there is a description of some of the common mistletoe species. Finally, there is a detailed discussion of two of these (*Arceuthobium tsugense* and *A. americanum*), looking at mistletoe and stand dynamics and the way that these can be manipulated to reduce damage.

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

- The life cycle of dwarf mistletoe
- Rate of spread and intensification of mistletoe
- Effect of mistletoe on its host
- Species of dwarf mistletoes, hosts and geographic range
- Silviculture in stands infected by mistletoe
- Lodgepole pine dwarf mistletoe
- Hemlock dwarf mistletoe

OBJECTIVES

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

1. to recognize dwarf mistletoe symptoms and identify the various species;
2. to assess the impact of dwarf mistletoes on trees and stands;
3. to describe how these organisms spread and reproduce, and under which conditions they are likely to become serious threats;
4. to prescribe treatments that will reduce the impact of these parasites on various management goals.

LESSON STUDY INSTRUCTIONS AND ASSIGNMENT

Start this lesson by reviewing Chapter 17 in Manion (1991). Then study the commentary in this lesson and Pest Leaflet No. 44.

After covering the material in this lesson, complete the self-testing/review questions at the end.

The next assignment to be submitted for marking is due at the end of Lesson 8.

COMMENTARY

The Life Cycle of Dwarf Mistletoe

A good way of visualizing the development of dwarf mistletoe in a stand is to think of it as an epidemic that continues over the life of the stand. The major parameter that describes an epidemic is the infection rate *r*, which is defined as the number of new infections that arise from each established infection per unit time and unit target area. If *r* is constant over time, (and only a small part of the available host tissue is infected), there will be a geometric increase in the amount of infection in the stand. We will find, however, that *r* depends critically on a number of factors, and that it varies greatly over time. The main factors are the effect of shading and weather on seed production, the spatial distribution of the target area, and the amount of non-host target area in the stand. As you study the life cycle, therefore, pay particular attention to the aspects that influence *r*. You will then appreciate how mistletoe epidemics will develop over time under different conditions.

Dwarf mistletoes are small vascular plants whose "roots" are embedded in the living xylem and phloem of branches and stems of their conifer hosts. All are obligate parasites and can survive only on living host tissues. The aerial parts consist of leafless, segmented shoots (2–20 cm in length, depending on species) that bear flowers and fruits. The fruit is a small berry containing a single small (about 3 mm long) seed. When the fruit matures, it breaks away from its stalk and then the seed is shot out through the abscission layer. Hence there is virtually no directional control of the flight of the seed. Seeds are expelled explosively at initial velocities of about 100 km/hr, and travel up to 15 meters (rarely as far as 20). The seeds are covered with a sticky material called viscin. At high speed they may bounce off solid objects, but when they slow down, they stick. Most seeds that land on a proper host stick to its needles. The next time it rains, the viscin swells into a gelatinous mass, and the seed slides along the needle and lodges at the needle base (or slides off the needle and drops lower in the crown or to the ground). Then the viscin dries and glues the seed in place.

The probability that a seed will land on a host is determined largely by the available target area. You can think of that probability as the proportion of all possible flight lines that intercept a host. In young stands, before crown closure, many seeds end up on the ground or on the herb and shrub layer. In stands with a closed canopy, the location of the infection is also important. Seed from infections in the upper crown has a higher probability of landing on the host than seed produced at the base of the canopy. Non-host trees act as sinks for mistletoe seed. The crowns of such trees intercept seed in proportion to their relative target area, but that seed does not participate in the ongoing mistletoe epidemic.

Seed dispersal occurs in fall for most species. The seed overwinters on the twig. Some of it is parasitized by various fungal pathogens and dies. In spring surviving seeds produce a radicle. (Unlike most vascular plant seed, mistletoe seed does not have a fully developed embryo with

an apical meristem.) The radicle extends for a short distance until it meets an obstruction, then forms a mass of tissue called the **holdfast**, and penetrates the host by a combination of mechanical force and enzymatic digestion of the periderm tissues. Inside the cortex and phloem of the living host, the mistletoe produces a number of strands of tissue that contain poorly organized phloem and xylem. This "endophytic system" then proliferates in the living bark for several years. When it reaches the cambium, the cambium lays down xylem around it, and thus it becomes embedded in the xylem rays. The strands of the endophytic system in the xylem are known as **sinkers**. Sinkers do not grow actively into the xylem. Hence, the oldest annual ring to have sinkers in it also defines the year of infection. Xylem laid down beneath infected bark is abnormal. The rings are often wide, and the wood has many characteristics of reaction wood.

The endophytic system extends both proximally and distally along branches at about 4 cm per year, but slower on low-vigour or old infections. After two to five years it grows to the surface and produces aerial shoots. These segmented aerial shoots bear flowers in their axils starting one to three years after the shoots first appear. Mistletoe is dioecious, that is, individual infections originating from a single seed are either male or female. Pollination is largely by insects. Flowering occurs at various times depending on the species. For all species, the fruit matures late the following year. (Incidentally, the following reference has some good illustrations of flowers and fruits: Hiratsuka, Y. 1984. *Forest tree diseases of the prairie provinces.* Canadian forestry Service, Northern Forestry Centre. Information Report NOR-X-286.) In summary, the length of time from a seed landing on a host to the production of the first new seed by the new infection is a minimum of about five years (two years before production of shoots, another two to produce flowers, and one for the seed to mature), but more commonly it takes seven years. Under ideal conditions in a greenhouse the cycle can be as short as three years.

Next we consider the continued development of dwarf mistletoe on the host. Because the endophytic system is perennial, it will continue to extend as long as the host tissue on which it is located lives, and it may continue to produce new aerial shoots each year. Aerial shoots live for no more than three to five years. Most are broken off mechanically, particularly by snow and ice. In windy regions, such as Alaska, many aerial shoots are broken off before the first crop of seed on them matures. Here the infection rate is small, and the level of infection remains much lower than farther south. Infections on low-vigour or shaded branches produce few (or no) shoots and seeds. Such infections remain alive however, and can resume seed production if the branch on which they occur is exposed to light, as, for instance, following thinning or partial cutting. On some hosts, leafless branches below the live crown that carry infections close to the bole, remain alive (the mistletoe must somehow send a hormonal signal to the tree to prevent abscission), and such branches can also produce new foliage if exposed to light.

Infections on major branch axes in good light induce the host to produce epicormic shoots which develop into **brooms**. The brooms are systemically infected by some but not all mistletoe species (in systemic infections, some of the endophytic mistletoe tissue is carried in the terminal meristem of the epicormic shoots). The presence of brooms (of a shape characteristic of the host species) in the living crown is the main way in which mistletoe infection in large trees can be diagnosed from the ground.

There are some fungal parasites of dwarf mistletoe flowers and aerial shoots. These fungi sometimes reduce seed production, but mainly in places where infection levels are high. In addition mistletoe-infected bark is susceptible to certain canker fungi, and mistletoe infections are sometimes killed by them.

Surprisingly, there is little variation in resistance. All the individuals of a host species are susceptible, and one doesn't see defensive host reactions in some but not all the individuals within such species. Crown form and needle arrangement have some effect on the proportion of the mistletoe seed (whose flight path is through the crown) that ends up "glued" to twigs in a place where it can infect. For example, a rare drooping needle form of ponderosa pine is quite resistant because most of the seed slides off the needles and falls to the ground during the first rains after seed dispersal.

Rate of Spread and Intensification of Mistletoe

Seeds are expelled in random directions. Mistletoe may advance as much as 15 meters in a single jump, but after that it takes about seven years before the next jump can take place. The resulting spread rates are as follows:

- Single infected residual trees or infected stand edges will shed mistletoe seed over about a 15 meter radius and eventually infect all hosts within that range. Thus 15 evenly distributed infected residuals per hectare will result in 100% infection of the regeneration.
- Lateral spread within an even-aged canopy is about 0.5 meter per year.
- Long distance spread occurs mainly via birds. Many birds carry seed on their feathers at times of seed dispersal. Normal preening removes the seed, and some of it lands on susceptible hosts. Mammals also carry seed, but it is not known how much ends up on host trees.
- Upwards spread in the canopy is somewhat less than horizontal spread, and is about 0.4 meter per year at maximum but much less in dense canopies and in cases where only the lower crown is infected.

Intensification is the process by which the number of infections per tree (as well as their size, the number of brooms, and the energy drain represented by them) increases over time. A caution is required — expressing mistletoe severity as number of infections per tree requires that tree size also be considered. For example, 20 infections on a 10-year-old hemlock would be considered severe infection, whereas the same number of infections on a large, old tree would be very light. In young stands, before canopy closure, the number of infections per tree

increases exponentially over time with a doubling time of two to five years. Two factors are important: first, the target area (live crown surface per hectare) increases each year, so that each year a larger proportion of the total amount of mistletoe seed produced lands on a host; and second, all infections produce seed, and the number of infections increases each year, leading to a rapid increase in total seed production per hectare. During this time of mistletoe development, about half the infections do not yet have aerial shoots and are therefore essentially invisible. Hence sanitation as part of a juvenile spacing operation will miss many infections, and within a few years, mistletoe levels will return to those before the spacing.

After the canopy closes, intensification slows. Most infections will be located in the lower (older) parts of the crown, and these remain alive but may no longer produce seed, depending on the degree of shade, while branch suppression results in the death of many infections as the crowns lift. Juvenile spacing leads to rejuvenation of the lower crown infections and rapid intensification until the crowns close again. Large brooms do not form until the rapid lifting of crowns slows. The branches on young trees (less than 15 years old) in well stocked stands do not live long enough to develop large brooms.

Effect of Mistletoe on its Host

Dwarf mistletoe infections are energy sinks. Branches distal to infections continue to grow normally, and use their share of nutrients and water, but most of the photosynthate produced by such branches is intercepted and used by the infection (both to support elevated levels of metabolism and to produce the mistletoe tissues), and is not available for growth of the host. The overall drain on the host energy supply is small and the effect on height growth and increment virtually undetectable until large brooms form. This doesn't usually happen until the host is at least forty years old. From that time on, the brooms use a disproportionate part of the water and mineral nutrient supply available to the tree (broom foliage is usually lush) without contributing to the energy needs of the tree. Eventually, diameter and height growth begin to decrease, and then the parts of the crown that are not infected or are lightly infected start to decline. About forty to sixty years after broom initiation, the tops die back, and eventually the tree dies, but usually not until age 120 or more. (All these ages are approximate.) Much depends on site quality, stocking and between-tree competition, sources of the initial inoculum, and the mistletoe species involved. An important result of the manner in which mistletoe develops on its host is that young infected stands will show normal height and diameter growth and crown development for several decades. After that, when brooms begin the form, volume increment declines sharply, so that at harvest the merchantable volume may be less than half that of healthy stands.

Several mistletoe infection rating systems have been developed. The most widely used is Hawksworth's seven point scale (ratings range from *0* to *6*). The living crown is divided into three equal parts, and each part is rated as *0* (uninfected), *1* (lightly infected without brooms), or *2*

(heavily infected, usually with brooms). The sum of the three values is the rating for the tree. In pole-sized trees, the total rating increases by about one class every decade (with considerable variation). In mixed stands containing non-host species, the rate of intensification is much slower and serious losses may not occur. This arises because a good part of the mistletoe seed that is produced lands on non-host trees and doesn't participate in the epidemic.

Increment losses can be very roughly related to the average mistletoe rating of dominant and co-dominant trees in the stand as shown in Table 6.1. There is considerable variation depending on mistletoe species, tree species composition, site, stand density, and climate.

TABLE 6.1
Increment loss related to the Hawksworth rating.

Hawksworth Rating	Increment Loss (%)
0 – 2	0 – 5
3	5 – 15
4	10 – 35
5	30 – 70
6	65 – 95

A second type of loss arises from stem infections. The wood laid down in the immediate area of such infections is reaction wood, and lumber cut from such stems exhibits severe twisting and bending when it is dried. Sections of the bark on older stem infections may die, creating openings for decay. In some areas, stem infections by dwarf mistletoe are considered "suspect" characteristics for the purpose of decay estimation.

Species of Dwarf Mistletoes, Hosts and Geographic Range

Dwarf mistletoes are most common in California and Mexico. Many of the species are parasites of pines. Farther north and east, the number of species drops rapidly.

There are five species of dwarf mistletoe in Canada. One of these occurs on black spruce from southern Manitoba and Saskatchewan to Newfoundland. Three species are restricted to B.C., and one ranges from B.C. to Manitoba. There may be a sixth species of mistletoe on shore pine along the coast in B.C. See Table 6.2 for more information about the species found in B.C. Each species has a major host and one or more secondary and occasional or rare hosts. Neither the secondary nor the occasional or rare hosts are severely damaged by the parasite. Mistletoe can survive in pure stands of "secondary" hosts, but not on occasional or rare hosts. Such host species become infected only if they grow in close proximity to the main host.

To allow dwarf mistletoe to survive, (remember the low rate and short distance of spread) the host must occupy a forest site continuously over time; hence, mistletoe is restricted largely to situations where its host is a climax species. Thus Douglas-fir mistletoe is found only in the ponderosa pine zone and in the driest parts of the interior Douglas-fir

TABLE 6.2
The dwarf mistletoe (*Arceuthobium*) species of British Columbia.

Species	Range	Native Hosts		
		Principal	Secondary	Occasional/Rare
A. americanum (lodgepole pine dwarf mistletoe)	Interior	lodgepole pine	ponderosa pine	white and Engelmann spruce
A. tsugense (hemlock dwarf mistletoe)	Coast	western and mountain hemlock	shore pine* Pacific silver fir	spruces, grand and subalpine fir
A. laricis (larch dwarf mistletoe)	Southeast Interior	western larch	lodgepole pine and alpine larch	ponderosa and white pine, subalpine and grand fir, Engelmann spruce.
A. douglasii (Douglas-fir dwarf mistletoe)	Extreme southern Interior	Douglas-fir	grand fir, Engelmann spruce	ponderosa and lodgepole pine, western larch

*The mistletoe on shore pine may be a separate species or a special variety of hemlock dwarf mistletoe.

zone. Lodgepole pine mistletoe relies on regular fires to renew pine stands. In areas where fire frequency is so low that the succession occasionally proceeds from pine to spruce and subalpine fir, lodgepole pine mistletoe is rare or absent. Larch mistletoe also depends on fires and other disturbances to regenerate its host. Larch is a fire resistant, long-lived pioneer species within its range in B.C. Finally, hemlock dwarf mistletoe is found throughout the coastal western hemlock zone, where its main host forms an important component of the climax forest.

In order of their impact on wood production, *A. americanum* rates first, *A. tsugense* second, and *A. laricis* a distant third. Larch mistletoe is very serious and has the greatest relative impact of all mistletoes in stands where it does occur, but province-wide it is a minor disease because of its limited geographic range and the minor position of larch among conifer species within that range. Compared to the other dwarf mistletoes, Douglas-fir dwarf mistletoe is a mere biological curiosity.

SILVICULTURE IN STANDS INFECTED BY MISTLETOE

Several characteristics of dwarf mistletoe make it amenable to management by appropriate silviculture. These characteristics are the obligate parasitic habit, the considerable host specificity, the slow rates of spread, and the obvious symptoms that make detection easy. In older stands, mistletoe is usually identified by the presence of brooms. Some care is required here. When branches in the lower and mid crown are exposed to light, as along roads, clumps of dense foliage commonly develop. Such clumps have at times been mistaken for mistletoe brooms.

A principal aim of silviculture in stands infected by dwarf mistletoe is to reduce the amount of infection to levels that are not damaging. Generally this means mistletoe ratings of less than 3 on crop trees at the time of harvest. In some ecological zones this can be achieved by judicious manipulation of stand density and removal of tall infected residual trees at the time of harvest. In other zones mistletoe spreads so fast that damage can be prevented only by eradication of mistletoe at the time of harvest, and the creation of mistletoe-safe boundaries. As cutblock size

decreases, movement of mistletoe from infected edges into cutblocks becomes more and more important. In almost all situations, even-aged stand management using the clearcutting system is required. The other silvicultural systems (shelterwood and selection systems) all lead to heavy infection of potential crop trees at an early age. Thus dwarf mistletoe infection is a major consideration in "new forestry" schemes that are currently being promoted. Similarly, strip cutting in infected stands leads to rapid infection of regeneration in the strips from the infected edges on either side.

Sometimes mistletoe problems can be avoided by switching to non-host species, or by establishing stands with a significant component of non-host species. In the latter case, the non-host crowns will intercept a significant part of the mistletoe seed without contributing to seed production, leading to much lower rates of spread and intensification.

Lodgepole Pine Dwarf Mistletoe

Lodgepole pine dwarf mistletoe occurs in Canada on lodgepole, ponderosa, and jack pine, and ranges from the B.C. coast mountains to the Ontario-Manitoba border. In B.C. it extends north to Lake Williston. Infection is heaviest on the interior plateaus east of the coast mountains and in the extensive pine stands of the east Kootenays. Scattered infection occurs throughout the interior cedar hemlock (ICH) zone and the Engelmann spruce subalpine fir (ESSF) zone, but the disease is seldom serious in these locations.

Natural forest fires have always played a major role in the distribution of lodgepole pine dwarf mistletoe. Hot fires in infected stands kill all trees, and the resulting stands are even-aged, often dense, and mistletoe-free. On the other hand, cool fires leave patches of unburned infected trees, which infect the regeneration. The resulting stands are patchy, partly two-storied, and heavily infected. Mountain pine beetles also leave living infected trees, which lead to heavy infection of the subsequent stand. While it is sometimes argued that our efforts at fire control have led to an increase in dwarf mistletoe infection, that is not necessarily so. Fire control is much more successful with cool than with hot fires. Thus the types of fires that lead to severe infection in subsequent stands are being controlled, while the really hot fires continue to occur, though perhaps at lower frequency.

Lodgepole pine stands have a relatively low leaf area index (needle surface area per unit ground area). This results in less obstruction of seed flight than with other species such as hemlock. Also, height growth is slow and rarely exceeds 0.4 meter per year. These two factors together mean that the potential vertical rate of spread of mistletoe is greater than height growth of the tree. It follows that survival of dwarf mistletoe on small advanced regeneration after clearcutting will lead to infection of virtually the whole canopy of subsequent stands, and dwarf mistletoe ratings will commonly reach 5 or even 6 at harvest. Such stands suffer severe increment loss. In the case of lodgepole pine, therefore, the aim must be eradication of dwarf mistletoe at harvest.

It is important, however, to distinguish between experience and prediction. The previous paragraph draws consequences from the best current understanding of dwarf mistletoe behaviour. Whether this will actually happen remains to be seen. The difficulty is that there are no older infected stands that have arisen from clearcut logging that left only small infected residuals. It isn't possible to go to a set of such stands that are at least about forty years of age to measure the result of various degrees of eradication at harvest. Such stands do not exist, and stands much younger than forty years cannot provide the necessary answers. The long life cycle of dwarf mistletoe, combined with the necessity of determining what happens to intensification and the vertical rate of spread after crown closure mean that about forty years must elapse before the long-term development of mistletoe can be observed. Some evidence from the oldest clearcuts in the Chilcotin suggests that early predictions were overly pessimistic, and that mistletoe may not develop as quickly as was feared. Infection in older, naturally regenerated stands does not provide a good guide because most of these have originated in situations in which dwarf mistletoe survived on tall residual trees. Clearcut harvesting has created a new situation.

Eradication is difficult and the options are often limited by economics because one is often dealing with sites of low potential productivity. All advanced regeneration must be killed, since small infected trees in the understory are usually both common and asymptomatic. Slashburning is the most efficient way but with whole-tree logging there often isn't enough slash to carry a broadcast burn and loss of nutrient is a concern. Also in many places serotinous cones in the slash provide the seed for regeneration, and these cones are destroyed. There is a very narrow burning window in which a cool quick fire leaves enough cones with viable seed for regeneration while all advanced regeneration is killed, but most years the right conditions do not occur.

Other methods of eradication include: hand clipping; treatment with a mechanical chopper (good only when the small trees are frozen so that they are brittle, and there is no snow — otherwise too many survive); herbicides to kill all advanced pine regeneration (not practiced but a distinct possibility that should be tried); and possibly a seed tree method in which enough trees are left standing to provide seed for regeneration. A hot slash fire will then kill these trees, burn the slash and all advanced regeneration, open the serotinous cones on the seed trees, and prepare a seedbed. The seed trees can be removed after the seed is shed. This latter method needs to be tried under a variety of circumstances, but so far it hasn't been tried in B.C. However, slash burning affects nutrient supply and may have other detrimental effects; thus it cannot be used everywhere.

Dwarf mistletoe can move from infected edges into a young pine stand, infecting a strip about 40–50 meters wide after a rotation. Thus eradication of dwarf mistletoe from cutblocks must be combined with the establishment of mistletoe-free cutblock boundaries. The smaller the block, the more important this becomes. Such mistletoe-free boundaries might consist of healthy stands, non-host stands, lakes, rivers and

swamps, and major roads or other right-of-way. For lodgepole pine it is often difficult to find such boundaries. At one time it was suggested that strips of non-host species such as Douglas-fir be planted along infected edges, but this doesn't work well. Survival of planted Douglas-fir is often poor, while lodgepole pine regenerates naturally in such strips and outgrows the Douglas-fir that is established, necessitating several cleanings. Also, if successful, one ends up with narrow strips of Douglas-fir within a pine stand, and different treatments and rotation ages are required for the two. Planting spruce leads to similar problems. Instead, the current practice is to regenerate pine throughout the cutblock, and to destroy the infected pine regeneration (a 30-meter strip) when the adjacent stand is harvested.

While complete eradication of mistletoe from cutblocks may be the best way of ensuring a healthy new stand, the cost is considerable. In practice, a compromise is applied. Cutting permits in mistletoe-infected areas have a clause that requires the removal of all trees greater than a specified height (usually 0.3 to 0.5 meter). Some mistletoe will survive on the smaller trees. It may well be that in the end this approach will prove to be insufficient to prevent significant losses. On the other hand, removal of tall infected residuals is certainly an improvement over doing nothing.

In immature stands there isn't much that can be done. Infected overhead residuals should always be removed since they are sources of much mistletoe seed. Also, that seed is likely to be deposited high in the crowns of the main canopy, in a location where infections can easily develop into brooms.

The incremental loss resulting from mistletoe (percentage of final volume) is less in open than in dense stands in spite of the somewhat greater rate of intensification in open stands. Open-grown trees may have more infections per tree, but they also have much more foliage. The number of infections per unit of foliage (a measure of the relative energy drain) may in fact be less in open than in dense stands. Hence infected stands may be spaced. The return isn't as good as for healthy stands, but infected stands that are treated will yield some volume. During the spacing operation, the more heavily infected trees, and those that bear small brooms or stem infections, can be removed, resulting in a slight improvement in the mistletoe rating of the stand.

Hemlock Dwarf Mistletoe

Hemlock dwarf mistletoe is found in the coastal forests of Washington, British Columbia, and Alaska. It does not occur in the natural range of western hemlock east of the coast mountains and the Cascades. The disease extends into the mountain hemlock zone in British Columbia, but it is seldom serious there. Wildfires play a role in determining the local distribution of infection over part of the range. In the north and in the cooler, wetter areas, wildfires are rare, and mistletoe is widely distributed; in the drier parts of the range, wildfires have served to eliminate mistletoe from large areas. However, in the broken, mountainous terrain of the coastal forests, scattered patches of trees in swamps

and moist draws commonly escape even large, hot fires, and in these locations mistletoe often survives.

Often, climatic events limit seed production. Fall frosts before seed dispersal destroy the seed; wind storms, snow, and ice lead to loss of aerial shoots and hence also reduce seed production. As the frequency of such events increases (with elevation and latitude) the rate of intensification decreases, so that in parts of the wet subzone of the coastal western hemlock (CWHb) and the higher elevation zones, hemlock dwarf mistletoe is not very damaging. Cool, wet weather may also interfere, perhaps by promoting fungal parasites of mistletoe seed. All this helps explain why measured rates of intensification in southern Alaska are much lower than those in the Cowichan valley in southern Vancouver Island.

Hemlock forms a dense, high leaf area index (LAI) canopy, so that the effect of shading on seed production is pronounced. Also, on the better sites, tree height growth exceeds the vertical rate of spread of mistletoe. In immature, even-aged, dense stands on medium to good sites, the canopy outgrows dwarf mistletoe. The upper crown is uninfected, and shades the infected lower crown, thus reducing mistletoe seed production and hence the rate of vertical spread. Figures 6.1 and 6.2 demonstrate mistletoe effects in good and poor sites. In stands on a good site, dwarf mistletoe ratings at rotation age seldom exceed 2 to 3, and damage is minimal. Of course, there will be some stem infections that lower the value of the trees carrying them. If such stands are left till well after rotation age, they begin to break up, and mistletoe will again spread upwards and eventually infect the whole crown.

On good sites, therefore, mistletoe-safe boundaries are still important, and removal of tall residuals (over two meters) left after

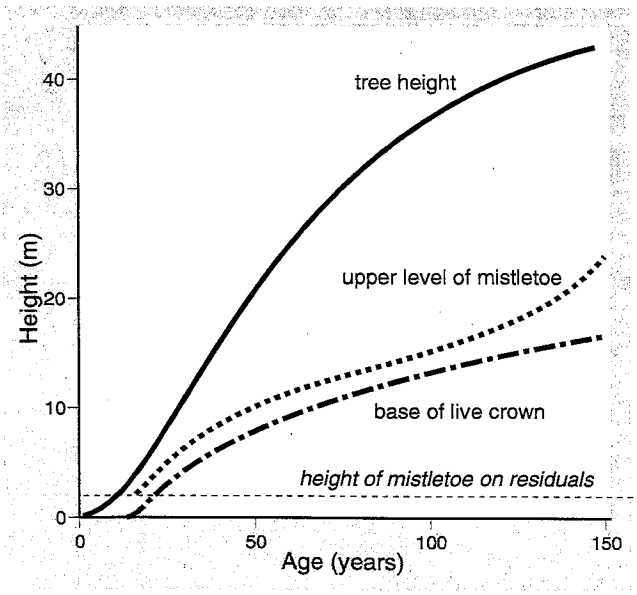

FIGURE 6.1
Mistletoe infection does not reduce height or volume growth under conditions in a good site.

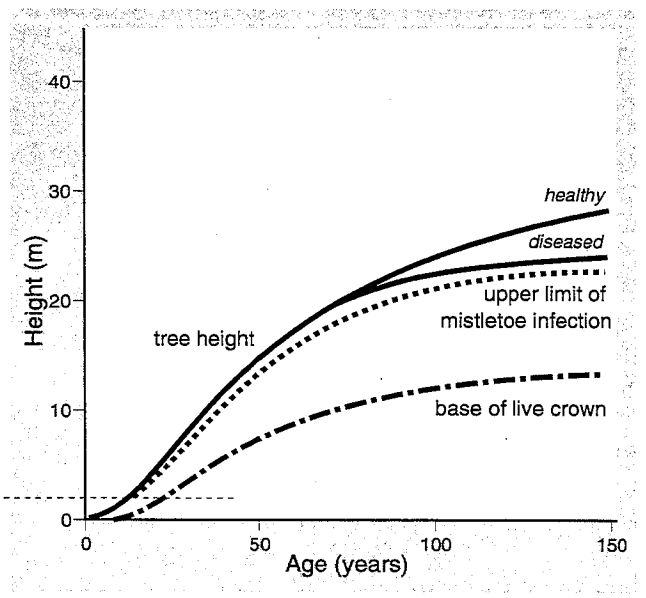

FIGURE 6.2
On low and poor sites, conditions favorable to mistletoes result in substantial timber losses.

clearcutting is still essential, but complete eradication of all advanced (and probably infected) regeneration may not be necessary. Two important qualifications need to be considered: Steep slopes result in a much greater rate of vertical spread because mistletoe seed produced in the lower crown infects the tops of down-hill trees, and, hence, total eradication is required on steep (40%+) slopes. Also, extreme forms of juvenile spacing that leave the canopy relatively open through much of the rotation will result in maximum rates of vertical spread. A much larger part of crowns will then be infected, and mistletoe ratings at maturity may range from 3 to 5, leading to considerable loss of volume.

On low and poor sites, the story is very different. The canopy is less dense, and height growth is often less than the vertical rate of spread; hence heavy infection and substantial losses result. Figure 6.2 shows the outcome. Such low sites are difficult to treat. Slashburning is usually the only feasible method of eradication, but it is also very damaging to most low-productivity hemlock sites. A species change to Douglas-fir, western red cedar, or *Abies* can solve the problem.

While most of the current recommendations for dealing with dwarf mistletoe are based on our understanding of the biology of these organisms, few have been applied over a long enough period to allow assessment. There is a great need for well-established trials (including proper controls) of various mistletoe management techniques under a variety of ecological conditions. We must also establish the relationship between mistletoe rating and yield with a great deal more precision, and in a species- and site-specific way. Such studies and trials will point the way to further improvements in mistletoe management.

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. List the five species of dwarf mistletoe that occur in Canada, and describe how you would distinguish each on the basis of morphology, host, and geographic location.
2. Draw a life cycle diagram for lodgepole pine dwarf mistletoe.
3. Why are dwarf mistletoes usually parasites of climax species?
4. How do mistletoes damage their hosts?
   Why does it take so long before damage becomes obvious?
5. How are the rate of spread vertically, host canopy density, and host height growth, related to the degree of sanitation required at harvest?
6. Why is lodgepole pine dwarf mistletoe sanitation during juvenile spacing of limited use?
7. What is the relationship between slope and vertical rate of spread of dwarf mistletoes?
8. Lodgepole pine dwarf mistletoe occurs throughout the interior plateaus east of the coast mountains. What determines the natural distribution of infected and healthy stands in these regions?
9. Lodgepole pine occurs in most of the interior cedar hemlock zone. Why is *A. americanum* relatively rare in this zone?

LESSON 7 Inheritance of Resistance

LESSON OVERVIEW

CONTENT

The use of resistant stock is an attractive way of dealing with many tree diseases. The question is whether such resistance will last. Experience with many agricultural crops has shown that new, resistant varieties lose that resistance because new, virulent forms of the pathogen arise, often within ten years. Since tree crops need to be on the ground much longer than that, we need much more stable forms of resistance, or else such crops may well be lost before maturity. In this lesson we will discuss various types of resistance mechanisms and the manner in which they are inherited, and the issue of stability of resistance.

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

- Mechanisms of resistance
- Variation in resistance
- Host-parasite interactions
- Qualitative resistance
- Quantitative resistance
- Strategies for breeding resistance trees

OBJECTIVES

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

1. to list the types of diseases which may be dealt with by using resistant stock;
2. to outline the processes of selection and testing that will provide useful resistance; and
3. to describe how to achieve the desired stability of resistance in managed pathosystems.

READING

You should start by reading Chapter 20 in Manion (1991), then study the material in the commentary below.

LESSON STUDY INSTRUCTIONS AND ASSIGNMENT

At the end of the lesson, answer the self-testing/review questions before proceeding to the last lesson.

COMMENTARY

Mechanisms of Resistance

Mechanisms of resistance of plants to disease can be divided roughly into two types, namely passive and active. **Passive resistance** is due to plant characteristics that are present before infection, while **active resistance** is attributable to mechanisms that are triggered in response to invasion by the parasite.

Passive Resistance

Passive resistance mechanisms are attributable to such things as the structure and chemical composition of the cuticle and epidermis, structure and position of stomata, the nature of any periderms, toxins present in some plant tissues, and possibly, lack of essential physical or chemical stimuli. All these are plant characteristics that can be observed and measured even if the pathogen is not present.

Active Resistance

Active resistance mechanisms, on the other hand, come into play after a pathogen propagule has landed on a host and has started to penetrate. One major active mechanism is the formation of periderms in response to the killing of host cells by the invading parasite. Notice that pre-existing periderms may serve to exclude pathogens or slow their penetration, and such periderms are considered a passive mechanism. On the other hand, formation of new, possibly different, periderms in response to invasion is an active mechanism.

A second common active resistance mechanism is the formation of phytoalexins. Phytoalexins are toxic compounds produced by plant cells in response to invasion by pathogens. These compounds may be produced by dying host cells or by the living cells immediately surrounding the necrotic area. In general, the speed of formation of phytoalexins and the concentrations reached depend on the host organs being invaded and the vigor of the host, as well as on the identity of the pathogen. Each host species produces a particular phytoalexin (or set of phytoalexins) in response to invasion by all sorts of pathogens (and also following wounding). Different species of pathogens exhibit different tolerances to a particular phytoalexin. If a particular pathogen does not induce fast phytoalexin formation on a particular host, and if that pathogen is relatively tolerant to the phytoalexin being produced, then disease results. In the other cases, phytoalexin production provides resistance. In the case of obligate parasites, such as rusts and dwarf mistletoes, successful parasites do not trigger phytoalexin production at all. If they did, they would be eliminated.

Sometimes, phytoalexins produced in response to invasion by one organism may provide protection against a second organism, that, if it were by itself, would not induce phytoalexins. The phenomenon is called **cross protection.**

Variation in Resistance

Most tree pathogens exhibit marked host- and tissue-specificity. For instance, *Rhabdocline pseudotsugae,* the cause of Douglas-fir needle cast,

does not attack aspen bark, while *Hypoxylon mammatum,* the cause of a common canker on aspen, does not attack Douglas-fir needles. It is not wholly clear why that should be so. *H. mammatum* will grow quite well on dead, sterilized Douglas-fir needles, and similarly, *R. pseudotsugae* can be grown on dead, sterile aspen bark! At the species level, resistance is the rule, and disease the exception. Plant pathogens can attack and invade only a small subset of all plants, sometimes only a single species or variety. Our concern is with those combinations of hosts and parasites that do lead to disease.

Sometimes, all individuals of a host species are about equally susceptible to the pathogen (some of the root diseases, such as those caused by *Phellinus* and *Armillaria* are good examples). In other instances, there is considerable variation in resistance within a given host species. In the former case, the pathogen also is not differentiated into special forms, each pathogenic on a subset of the total host population; in the latter case, the pathogen may consist of a set of races that are adapted to certain host species or even varieties.

Any program of selection and breeding for resistance requires that there is some variation in resistance in the host. If all host individuals are equally susceptible, there is nothing to select. Furthermore, that variation in resistance must be genetically determined. If the resistance were induced by certain environments, the offspring of a resistant host would not necessarily be resistant, particularly if it were placed in a different environment. In general, variation in resistance and the heritability of that resistance is greatest to diseases caused by obligate parasites and facultative parasites that spend most of their life cycle in a parasitic mode. Thus hosts of rusts, mildews, foliage diseases, and cankers often show much variation in resistance, while decays and root diseases don't. Strangely, intraspecific variation in host resistance to dwarf mistletoes is relatively minor, even though these organisms are obligate parasites.

At the very outset, then, it is obvious that selection and use of resistant trees is a possible option for some but not all diseases. In those cases where natural variation in resistance is minimal, it is theoretically possible to create such variation either by mutagenic agents or by importing genes from closely related plant species, but the process is slow and uncertain.

Host-Parasite Interactions

Now we turn to the ways in which resistance is inherited. But our concern is not only with resistance, but also with pathogenicity, that is, the way in which the ability to cause disease is inherited in the parasite. As we have discussed, for diseases in which there is a good deal of intraspecific variation in host resistance, there is usually also a good deal of variation in virulence. That variation in virulence can take two forms. Sometimes, some isolates of the parasite are more pathogenic than others, regardless of the host variety on which they are tested. (In such cases, the less pathogenic isolates may survive because, under environmental

conditions other than those used in the experiment, they may be the most successful.) At other times, different isolates of the pathogen may be pathogenic on different subsets of the host population. These two ways in which variation in pathogenicity occurs are illustrated in an idealized and extreme manner in Table 7.1.

TABLE 7.1
Disease severity (on a nine-point scale: 0 = no disease, 9 = very severe disease) when different host varieties of the same species are inoculated with different isolates (races) of a particular pathogen.

A. Qualitative resistance:

	Host Variety		
Pathogen Race	A	B	C
1	9	9	0
2	9	0	9
3	0	9	9

B. Quantitative resistance:

	Host Variety		
Pathogen Race	A	B	C
1	3	4	5
2	4	5	6
3	5	6	7

Several points are illustrated in Table 7.1. First, in part A, the outcome of inoculating particular host varieties with particular pathogen races is either severe disease or no disease. There are no in-between cases. Hence this form of the interaction is called **qualitative**, because the outcome can be expressed as two non-overlapping classes, namely "resistant" or "susceptible." On the other hand, in part B, disease severity lies along a continuum from light (3) to heavy (7). This type of interaction is called **quantitative**, because disease severity must be measured or quantified in order to describe the outcome.

A second point illustrated by the table is that in the case of qualitative resistance, the amount of disease on resistant interactions is very low or zero, while on the susceptible reactions it is very high. On the other hand, in the quantitative case, most of the outcomes are at an intermediate disease level. Resistance based on the mechanisms that operate in part A will often be complete, while resistance of the form described in part B is usually only partial.

A third point relates to the question, "Which is the most resistant host, and which the most pathogenic race of the parasite?" In the quantitative case (Table 7.1B), host variety *A* is the most resistant to all pathogen isolates, while pathogen race *3* is the most pathogenic on all. (Remember, however, that under different experimental conditions, the order of both host varieties and pathogen races may change.) In the qualitative case, there is no straightforward answer. In statistical terms. Table 7.1A shows insignificant main effects of either host or pathogen, but a significant interaction between host and pathogen. In Table 7.1B the reverse is the case.

Different terms are used to describe the ability of pathogens to cause disease. In a situation such as described in Table 7.1A, one speaks of **virulence**. Thus, race *1* is virulent on varieties *A* and *B*, but **avirulent** on

variety *C*. On the other hand, in a situation such as described in Table 7.1B, the proper term is **aggressiveness**. Thus, race *3* is the most aggressive of the pathogen races.

QUALITATIVE RESISTANCE

A situation such as described in Table 7.1A arises when both resistance of the host and virulence of the pathogen are conditioned by single, powerful genes. The **gene for gene hypothesis** applies. That hypothesis states that for every locus on the host genome that may bear a gene for resistance, there is a corresponding locus in the pathogen genome that may have genes for virulence able to overcome the resistance genes in the host. Generally, major genes for resistance are dominant, while major genes for virulence are recessive. In a single pathosystem, there are usually several pairs of loci bearing such genes. The simplest case is shown in Table 7.2, with the outcome rated as resistant (*R*) or susceptible (*S*). For the host, the gene for resistance (*R*) is dominant, and the gene for susceptibility (*r*) is recessive. For the pathogen, the dominant gene for avirulence is *V*, and the recessive gene for virulence is *v*.

A little more complex is the case where genes at two loci interact. This is shown in Table 7.3. Since the effect of *RR* and *Rr* is the same (*R* is dominant), these two possibilities are both represented as R^-, and similarly, *VV* and *Vv* are shown as V^-. The subscripts *1* and *2* refer to the two loci.

Notice that in Table 7.3, if there is any locus on the host having an *R* gene that is not matched by a homozygous recessive pair of virulence genes (*vv*) on the corresponding locus in the pathogen, the outcome is resistant (*R*). All other cases lead to susceptibility (*S*)

TABLE 7.2
Resistance (*R*) or susceptibility (*S*) of the host to a pathogen in a simple "gene for gene" case.

Pathogen Genes	Host Genes		
	RR	*Rr*	*rr*
VV	*R*	*R*	*S*
Vv	*R*	*R*	*S*
vv	*S*	*S*	*S*

TABLE 7.3
Resistance (*R*) or susceptibility (*S*) of the host to a pathogen in a more complex case where genes at two loci interact.

Pathogen Genes	Host Genes			
	$R_1^-R_2^-$	$R_1^-rr_2$	$rr_1R_2^-$	rr_1rr_2
$V_1^-V_2^-$	*R*	*R*	*R*	*S*
$V_1^-vv_2$	*R*	*R*	*S*	*S*
$vv_1V_2^-$	*R*	*S*	*R*	*S*
vv_1vv_2	*S*	*S*	*S*	*S*

exercise As an exercise, try to construct host and parasite genomes that would give the outcome described in Table 7.1A. You will find that you need three loci to get the desired result.

In this discussion, we have referred to *R* and *V* genes as conditioning resistance and virulence. That is perhaps a little misleading. These genes apparently code for proteins or enzymes that are required for the normal functioning of the parasite or the host. The two forms of each gene (*R* and *r*, or *V* and *v*) represent small changes in amino acid sequence that may affect the efficiency of the processes that the relevant gene products are involved in, but allow these processes to continue.

While many *R* and *v* genes have been identified in several pathosystems, the manner in which they function at a biochemical level remains unknown. Perhaps it is best to think of a matching process. If the host and parasite genes are properly matched, the two organisms can live together for a while, and disease results. If they are not matched, a resistance mechanism such as phytoalexin production is triggered. In the above schemes, *r* genes match with both *V* and *v* genes, while *R* genes match with *v* genes only, and that is commonly the case.

You should now appreciate at a deeper level what is involved when new resistance genes are introduced in a host, and when that resistance is eventually lost. One starts with the situation at which a particular locus on the host carries the homozygous recessive form of the gene (*rr*) and the corresponding locus on the parasite has *VV*. All the other relevant loci also have "matched" genes, and the host will be susceptible. If a new resistance gene that changes the host locus to R^- is now introduced, all plants bearing that combination will be resistant. Such plants will have a decided reproductive advantage, and before long the R^- gene will be common in the host population. Now there is strong selection pressure on the parasite. Sooner or later a new *v* gene appears, either by mutation, or by immigration from a geographically distant part of the parasite population, or perhaps, by a change in frequency of the *v* gene in the parasite population. Remember that the virulence gene *v* is recessive. If the frequency of the *v* gene in the original parasite population is 0.001 (and there is truly random mating), then the frequency of parasites having *vv* is 0.0012 or one in a million. However, if only those few propagules of the parasite that have that particular genetic composition can cause disease and reproduce, then the frequency of *v* genes will increase rapidly, once the corresponding *R* gene has been introduced in the host population.

One trick that breeders can employ is to introduce two different *R* genes (at two different loci) at once. Now the parasite must change at both loci before disease can occur, and the probability of that happening is very much smaller than a change at one locus only. Hence the resistance will last longer. Another approach is the use of **multilines**. In this approach, the crop consists of several host varieties, each bearing a

different set of *R* genes. A particular parasite race may be virulent on one of these, but avirulent on the others. In such a case, there will be some disease, but the rate of buildup will be slow, because most of the spores produced on a diseased plant will land on plants with different sets of *R* genes, and hence will not develop. It is of course possible for the pathogen to develop a supervirulent variety in which all loci carry *vv,* and that variety will be able to cause disease on all the host lines, but, as we shall see below, such a variety is not likely to be very vigorous, and it too may not cause serious levels of disease.

It is generally assumed that the gene products of the resistance and virulence loci function in the normal growth of the host and parasite. Generally, the *r* and the *V* forms of the genes are slightly more efficient in that function. Thus *R* genes that are not required to maintain resistance (for instance because the genetic composition of the parasite at the matching locus is *vv* or because the parasite is not present at some locality), will tend to disappear in favour of *r* genes. Similarly, if *vv* is not required in order for the parasite to cause disease (because the matching host locus has *rr*), the *V* genes will be favoured. Thus if one were to inoculate a crop that has *rr* genes with a half-and-half mixture of *vv* and V^- parasites, then after several cycles, the V^- form would dominate. In this model it is clear that resistance is not "free" — the host pays a small price in efficiency in order to gain a large advantage (resistance to a disease).

In conclusion, resistance based on major *R* genes is inherently unstable. It is certainly suitable for annual crops, but its use for forest trees is questionable. Perhaps there are cases in which the probability that matching virulence genes will appear is so low that it is safe to plant the tree crop. However, that probability cannot be estimated ahead of time, and so uncertainty remains.

QUANTITATIVE RESISTANCE

In reference to Table 7.1B, we find a very different situation. First, there is no matching of resistance or virulence genes. If any two host parents are crossed, the offspring will have a range of resistance. Some will be more resistant than the most resistant parent, some more susceptible than the most susceptible parents, while most will be intermediate. If both parents are very resistant (or very susceptible), the resistance of most of their offspring will lie somewhere between that of the parents and the population mean, depending on the heritability of the characteristic. This is a very different outcome than in the qualitative case (Table 7.1A), where the offspring of a cross will be either very resistant or very susceptible, and where the proportion of these will follow standard Mendelian ratios.

Quantitative resistance is probably due largely to passive resistance mechanisms. None of these provide complete protection, nor is a particular mechanism either present or absent. Rather, it is a matter of degree. Consider, for instance, a case where resistance is related to cuticle thickness, such that the thicker the cuticle, the more resistant the plant.

In a population of plants there will be a range of cuticle thickness, and hence a continuous range of resistance levels. At the same time, the ability of the pathogen to penetrate cuticles will also vary. Some races will do so readily, and occasionally penetrate even very thick cuticles, while other races will find it much more difficult. Again it is a matter of degree. There will be constant selection pressure for characteristics that lead to greater resistance and greater aggressiveness, but, in natural populations, these two processes are apparently balanced, and the overall outcome in terms of the amount of disease does not change.

An important characteristic of quantitative resistance is that it is stable over time. On the other hand, the degree of resistance imparted is not great, and disease will still occur, even in resistant host varieties, but at a lower level than in susceptible varieties.

Before leaving this comparison of qualitative and quantitative resistance, one further comment. Table 7.1 was simplified to make a point; one would not expect to find such situations in the real world. Rather, one would expect to find the effect of major genes (part A) superimposed on that of minor genes (part B). If major resistant genes were present and unmatched, the resistance level would be very high. If they were absent or matched, the resistance level would be determined by the quantitative resistance of the host. Thus a more realistic situation is obtained by superimposing parts A and B as shown in Table 7.4.

Strategies for Breeding Resistant Trees

In the few natural pathosystems that have been investigated in detail, quantitative resistance is the rule. Sometimes major *R* genes occur, but these are generally uncommon. Many more studies have looked at agricultural crop pathosystems. In many of these, resistance is based on a few major *R* genes. Almost all such systems have proved to be unstable, and new *R* genes must be introduced constantly. Some *R* genes are "better" than others, meaning that they tend to last longer, but in almost all cases, it is just a matter of time before they are overcome.

The obvious strategy, then, is to imitate natural pathosystems, and base a resistance breeding program on passive resistance mechanisms that are conditioned by many "minor" genes. However, there are two main difficulties with this approach. The first is that disease-free trees in the field, in areas where most trees are infected, do not bear a label reading, "My resistance is quantitative." Sometimes, disease-free trees have escaped infection simply by chance and remain susceptible. Mostly, however, disease-free trees are truly resistant, but it is usually not clear whether such resistance is based on qualitatively or quantitatively

Table 7.4
Typical distribution of levels of resistance when both qualitative and quantitative mechanisms play a role.

Pathogen Race	Host Variety		
	A	B	C
1	3	4	0
2	4	0	6
3	0	6	7

inherited resistance mechanisms. (Sometimes the stage at which disease development is arrested may indicate the type of resistance.) However, one thing is sure: the more stringent we make the selection criteria, the more likely it is that selected trees will be resistant because of major *R* genes. One can, of course, determine the nature of the resistance carried by a tree by judicious crossing with other trees and exposing the offspring to a variety of pathogen isolates. It should be clear from the above however, that often the *vv* race of the parasite may not yet be around, and that makes the test much more difficult to interpret.

The second difficulty is that resistance based on quantitatively inherited, passive resistance mechanisms is only partial and often not very great. The gains made during each cycle of selection and breeding are small, and the resistance may not be strong enough to make the "resistant" selection attractive from a silvicultural point of view.

In natural pathosystems, natural selection for resistance and for virulence (or aggressiveness) has been going on for a long time. The outcome of that selection process is a pathosystem in which the pathogen survives and causes some damage to the host, but in which the survival of the host is not threatened. The *Cronartium* rusts on lodgepole pine are a good example. In that pathosystem, there is a good deal of variation in infection in the short run caused by variation in weather patterns. In the long run, however, disease levels are stable. Also, in that system, there is considerable variation in resistance in the host population. (Whether there is parallel variation in virulence/aggressiveness on the part of the parasite population is not known.) Furthermore, the bulk of the resistance appears to be quantitative. Major genes for resistance do occur, but their frequency is not high. Is it worthwhile to attempt to improve resistance by further selection in such natural pathosystems? Many would say not. The risk of destabilizing the system is greater than the benefits. In the case of introduced diseases, the situation can be very different. The host and parasite have not co-evolved, and disease incidence can be very high. Here an appropriate goal is to produce a host and rust pathosystem that resembles a natural system.

The natural balancing point between natural selection for resistance on the part of the host and for virulence/aggressiveness on the part of the parasite is usually a situation in which disease occurs but in which the majority of the host plants escape serious damage. The case of foliage diseases provide an instructive example which will also serve to make a further point. Local pathosystems of trees and foliage disease parasites appear to reach an equilibrium in which some disease occurs, but most of the host trees survive without a major impact on growth. (At the same time, however, there occur large short-term fluctuations and major epidemics as discussed in Chapter 4. The host-parasite equilibrium that is the subject of this section is always a long-term one, and recognizes that short-term fluctuations will occur.) These local pathosystems occur in a wide variety of environments, some of which are favourable for the pathogen (e.g., frequent moist warm periods during early summer) and some are not. Each of these local populations

reaches about the same balancing point: populations in locations that are very favourable for the pathogen have nearly the same amount of disease as populations growing under conditions that are unfavourable for the pathogen. Host populations from areas where the environment favours the pathogen show a great deal more resistance than host populations from areas that don't. When such populations are moved from low-risk to high-risk areas, they can sustain a great deal of disease and suffer severely. So, it seems that resistance develops by natural selection wherever it is needed, but only to the point that survival of the host is no longer threatened, and no more.

Finally, a point on genetic diversity. Natural populations of trees (particularly conifers) are, genetically speaking, among the most diverse of all living organisms. Now it is well known that great gains in short-term productivity can be achieved by reducing that diversity, the extreme case being the use of clones. Intensive poplar culture is an example. This practice has had an interesting effect on pathogens. One of the more widely studied pathosystems is that of *Melampsora* rust on poplars. In natural cottonwood-*Melampsora* pathosystems, resistance is mostly quantitative, and major genes for resistance and virulence, if they occur, are certainly not important. However, parasite populations change when they develop on uniform cottonwood plantations. Only those races that are particularly well adapted to the clone(s) being used survive. So, after a short while, the parasite population is also much more uniform than the original natural population. In places such as Australia, where both the host and the parasite are introduced, and there are no nearby natural populations of the rust to confuse the issue, resistance is mostly qualitative and unstable. There are several examples of clones that were at one time resistant, but that have lost their resistance (due to a change/ mutation in the parasite), and are now so susceptible that they are useless for wood production. In fact, the disease severity seen on such clones far exceeds any that is ever seen on natural populations. These phenomena are widely recognized, and much has been written about the number of clones required to retain a "natural" pathosystem. The theoretical answer has usually been something like twenty to sixty clones used in intimate mixture. Time will tell whether these theoretical calculations have in fact considered all the relevant factors.

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 lesson 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. Define and give examples of active and passive resistance mechanisms.
2. Which classes of diseases are good candidates for selection and breeding for resistance and why?
3. Contrast quantitative and qualitative inheritance of resistance.
4. What is meant by the "gene for gene" hypothesis?
5. Why is the disease resistance of many agricultural crops inherently unstable?
6. What are the two main difficulties in breeding disease-resistant trees for use as forest trees?
7. How can tree breeding and intensive culture using clones upset the natural equilibrim between host and parasite?

LESSON 8 Forest Pathology in the Context of Forest Management

LESSON OVERVIEW

CONTENT

In this lesson we will extend some of the concepts that have been covered in this course by considering them in the context of silviculture and forest management. The relevance of forest tree diseases to various kinds of forestry issues will be emphasized. A large section of this lesson will be devoted to the assessment of disease losses.

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

- Disease losses and the purpose of management
- Estimation of disease-caused losses
- Stands vs. forests
- Concluding remarks

OBJECTIVES

At the conclusion of this lesson, you should be better able to relate what you have learned earlier in this course about trees and diseases to broader issues in forestry. You will also develop a greater appreciation for the complexity of assessing forestry losses due to diseases.

READING

Study the material in the commentary that follows.

LESSON STUDY INSTRUCTIONS AND ASSIGNMENT

As you read the material, try to relate it to your knowledge of forest biology and management. Unless your understanding of diseases is well integrated with all the other things you know about forests, you will find it difficult to translate your knowledge into appropriate practice.

Answer the self-testing/review questions, then complete Assignment #3 (in Appendix A), which deals with the content of Lessons 5, 6, 7 and 8, and submit it to your tutor for marking. Check the course schedule for the deadline.

COMMENTARY

Disease Losses and the Purpose of Managment

Most of the diseases that you have studied in this course are native diseases that have always been part of natural forest ecosystems, and that sometimes play critical roles in such ecosystems. For instance, life on Earth as we know it would soon come to a halt without the recycling of carbon tied up in wood back to the atmosphere by decay fungi. Or, to take another example, the diversity of stand types resulting from the action of various root diseases may well be critical in maintaining essential habitat for various organisms that in turn play a major role in the stability of forest ecosystems.

Why then are diseases usually considered to be damaging? The answer, I think, is that "damage" has meaning only in the context of some human goal or purpose. If the purpose of management of a particular tract of forest is to produce wood, or to provide forage, or to provide water, or some combination of such purposes, then diseases will sometimes frustrate that purpose. Diseases don't destroy the forest, but they may lead to a type of forest that isn't very suitable for the particular purpose we have in mind.

Another, and important way in which diseases can be damaging, occurs when we alter the natural forest in order to achieve some purpose, usually by silvicultural means such as particular harvesting systems, planting, spacing, fertilization, use of species that wouldn't occur naturally, and so on. Such actions may upset the natural balances and result in disease development to levels that would not occur in natural forests. A good example would be rapid regeneration by the planting of conifers following harvesting, a practice which favours the survival and spread of several root diseases. Natural regeneration patterns with long regeneration periods and mixtures of species allow a large part of the root disease inoculum in stumps to die off before a new crop is infected. I'm not arguing that planting should be abolished, but I am saying that at times and in certain places, it can have detrimental effects.

Estimation of Disease-Caused Losses

Incidence, Severity and Intensity

A central and difficult issue is the measurement of damage. It is usually fairly easy to obtain a measure of the amount of disease. Two terms are commonly used in this regard: **incidence** is the frequency of occurrence of a disease, measured as the proportion or percentage of plants or plant parts infected; and **severity** is a measure of the amount of disease on these infected plants. Thus the incidence of white pine blister rust in a white pine plantation might be 70%, meaning that 70% of the trees are infected, and the severity could be 3.2 infections per tree, meaning that infected trees have, on average, 3.2 infections each. (Sometimes it is more appropriate to express severity as the average number of infections per tree for all trees rather than just the infected trees.) Obviously, in most situations, incidence and severity are related. As the proportion of infected plants increases, the number of infections per diseased individual also increases. That need not always be the case, however. If,

for instance, part of a host population carries a major gene for resistance that imparts virtual immunity to a disease, then, once the susceptible plants have been infected, the incidence will not increase any further, but the severity may continue to increase. The product of incidence and severity (both as proportions), is sometimes called **disease intensity**, and is usually related most closely to the amount of damage incurred.

Spatial Distribution

The spatial distribution of infection may be important, especially if one is speaking of young stands. If, for instance, 20% of the trees at age 10 are infected by a lethal disease, but such trees are randomly distributed throughout the stand, then (assuming no further spread beyond that age) the disease can sometimes be regarded as a natural thinning agent, and the effect on volume production may be positive. For example, western gall rust often behaves in this fashion. Lethal stem infections occur mainly during the first fifteen to twenty years, and such infections are usually randomly distributed in lodgepole pine stands (and such stands are commonly rather dense). If, on the other hand, the infected trees are clumped, as is the case with root diseases, then close to 20% of the area is rendered non-productive, (and if the original stand had too high a stocking level, that stocking level is retained in the uninfected portions of the stand). In such a case, the loss in volume at maturity may be close to the incidence of the disease at an early age. Thus in addition to incidence and severity, one would also want to know about the spatial distribution of a disease.

Disease Intensity and Volume Losses

Another consideration relates to the relationship between disease intensity and volume loss (accepting for the moment that the main purpose of management for the stand under consideration is wood volume production). Take, for instance, the case of a foliage disease such as Douglas-fir needle cast caused by *Rhabdocline pseudotsugae.* Needles are infected immediately after they emerge from the bud, but remain green and symptomless for about 11 months. Then sections of the needle turn red-brown, the fungus reproduces on the needle, and infected needles are shed. What is the impact (the loss in volume increment) if 50% of the needles are infected each year? Clearly, there are several aspects to this question. Infection probably results in increased rates of metabolism in the infected (but symptomless) needles. That increase may or may not be offset by compensatory increases in photosynthesis in either diseased or healthy needles, or, perhaps, the presence of the pathogen may lead to decreased rates of photosynthesis. Furthermore, how serious is the loss of older needles? Certainly, the current year's needles are the most efficient at photosynthesis, but the degree is not known precisely. A disease incidence of 50% of needles infected does not necessarily result in a 50% reduction in increment. The losses might be considerably greater, or considerably less. Studies that have related disease intensity in forests to productivity, usually as current annual increment (CAI), are few, and many of them are based on questionable assumptions.

Closely related to the above is the issue of tolerance. We have seen that losses due to a foliage disease are probably the result of the overall net decrease in photosynthate production. The extent of that reduction depends on the interplay of many processes at the physiological level. All individuals in a population do not exhibit the same reduction in photosynthate in response to the same disease intensity. Tolerant individuals exhibit a smaller loss per unit of disease intensity than intolerant ones. Disease tolerance is a valuable, genetically-determined trait that can be selected for, parallel to disease resistance. In fact, variation in tolerance may be as important as variation in resistance.

There is a fourth aspect to the estimation of losses from measures of incidence and severity, and that is the time dimension. Losses occur because, *at the time of harvest,* the expected volume (and/or quality) isn't there. Losses in stand volume at an early age are of concern only if they result in a reduced final volume. Hence we need to know how a disease intensity that fluctuates over time will affect final volume. This introduces a further set of uncertainties that are best demonstrated by considering how one might measure losses.

The basic experimental approach to measure losses would be to compare the growth of diseased trees with that of healthy controls. Obviously, the controls should be exposed to the same environment as the diseased trees. One way to achieve this is to compare the volume (or increment) of healthy and diseased trees growing side by side in the same stand. Alternatively, one might set out to describe the relationship between disease severity and volume increment, because trees growing together in the same stand, and thus presumably exposed to the same environment, usually show variable amounts of disease, rather than being either healthy or diseased to a particular severity. The relationship between disease severity and volume production for a particular individual or clone is, of course, a measure of the tolerance of that clone. When one tries to determine that relationship for a population of trees, variation in tolerance will be a source of error that we must accept.

The central difficulty in an experiment of the sort described here is that trees in a stand are in competition with each other. Severely diseased trees will be poor competitors. Compared to healthy trees, their root systems won't have as much energy supply and their ability to absorb their share of nutrients and water will be reduced. That share will be available to the healthy trees. Furthermore, as time passes, diseased trees increasingly will occupy inferior crown positions. The result is that the healthy trees in the stand use a disproportionately large part of the available nutrients, water supply and light. The healthy trees grow faster than they would if all trees in the stand were healthy and competing on even terms for space, nutrients and water. The difference in volume (or increment) between healthy and diseased trees (or the slope of the relationship between disease severity and volume increment) is the result of both the direct effect of the disease and the effect of increment transfer from diseased to healthy trees. Another way of stating this is that the decrease in volume production per hectare (usually as CAI) is less

than the average difference in increment between healthy and diseased trees multiplied by the number of diseased trees.

The effects of disease on competition are shown in Figure 8.1. The height of trees in (A) represents average tree volume on the site if all trees were healthy. In (B), some of the trees are diseased, others are healthy. (Remember, tree height represents average tree volume.) The healthy trees in (B) are larger than those in (A) because they experience less competition from their diseased neighbours than the healthy trees in (A) from their healthy neighbours. In (C) all trees are diseased. Here the effect of unequal competition is removed, and as you might expect, the diseased trees in (C) are larger than the diseased trees in (B). Figure 8.1 demonstrates that if we estimate the losses due to disease as the difference between healthy and diseased trees as shown in (B), then we will be overestimating the effect of disease considerably.

The above analysis serves to remind us that the parameter of interest is volume per hectare, rather than tree volume. The effect of disease should be estimated by comparing the productivity of healthy and diseased stands rather than comparing healthy and diseased trees within stands. That approach, however, raises another difficulty. The stands to be compared should be the same in all respects except for the presence of the disease. Two aspects are important: the stands should have the same history of establishment and pattern of stocking over time; and they should be on sites of the same productivity. The former is often difficult to achieve, and the latter difficult to measure.

Let us consider stand productivity first. One of the best measures of productivity is site index that is estimated from height-over-age curves. If the disease affects height growth, then the site index of diseased stands will be underestimated. In fact, we need to know the very thing we are trying to determine, namely the decrease in productivity caused by disease, before we can select appropriate stands to make that determination! An alternative method is to use ecological approaches to estimate site productivity. Such methods are less precise, and may also be confounded by the presence of a disease. For instance, if disease results in a less dense canopy, or in reduced water or nutrient uptake, understory vegetation in the diseased stand may be much richer than in healthy stands.

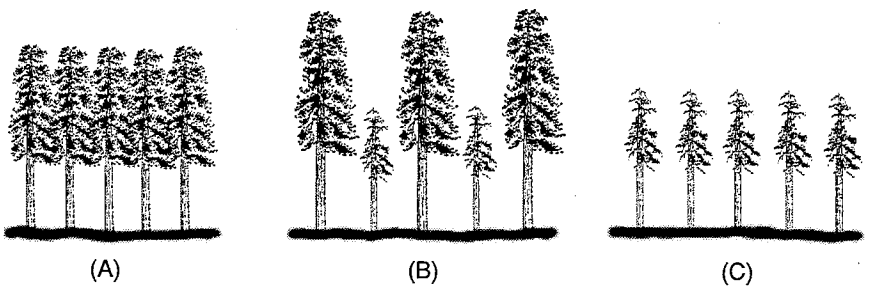

FIGURE 8.1
Simplified illustration of the relative size of trees in stands that are (A) all healthy, (B) mixed healthy and diseased, and (C) all diseased.

The requirement for a similar stand history also introduces uncertainties. Take, for instance, the case of lodgepole pine dwarf mistletoes. Infected stands have usually originated after a disturbance (e.g., cool fires, or a mountain pine beetle epidemic) that has removed most but not all trees in the previous stand, and that has left the forest floor largely undisturbed. Neighbouring healthy stands will be healthy because the same disturbance has been more severe (e.g., a hot fire) removing all trees and a large part of the forest floor. The effect of stand history on volume production is often hopelessly confounded with the effect of the disease.

In conclusion, estimation of losses caused by diseases is a very tricky process. The best approach would be to start with young, healthy stands that can be shown to be identical in site productivity and stand history, and to introduce the disease artificially into some of these. Usually a considerable amount of time must elapse before a valid assessment of the effect of disease can be made. Studies of this sort are very rare for forest diseases. In the meantime, you should be aware that most published figures on disease loss are based on questionable methods, and in fact are mostly best guesses rather than factually based estimates. Furthermore, such estimates are usually made by the very people whose status and jobs depend on diseases being serious. I'm afraid that this has on occasion resulted in rather generous estimates of losses.

Now that you have been properly forewarned, it is time to look at Table 8.1 (adapted from Woods and Van Sickle, 1994, B.C. Ministry of Forestry Publication BC-X-354) which lists estimates of annual losses to various insects and broad groups of diseases. Notice first that the single largest loss, amounting to about half of all pest-caused losses, is attributed to butt and heart rots. Most of this loss accrues in old declining stands, and little can be done about it other than appropriate scheduling of harvests. As such old stands are harvested, the loss associated with decay will decline. At the same time, however, losses to root diseases will increase unless strong remedial action is taken. Much of the decay listed under butt rots is in fact caused by root disease pathogens acting as butt-rots in these very old stands. Losses due to

TABLE 8.1
Annual mortality and growth loss from forest pests in British Columbia, 1988–1992.

Pest	Mortality	Growth Loss	Total	
			volume	percent
Mountain pine beetle	2,055	0	2,055	7.7
Other bark beetles	1,031	0	1,031	3.8
Conifer defoliators	1,671	969	2,640	9.8
Hardwood defoliators	0	162	162	0.6
Subtotal Insects:	4,757	1,131	5,888	21.9
Butt and heart rots	0	13,680	13,680	51.0
Dwarf mistletoes	0	1,797	1,797	6.7
Root rots	4,128	1,350	5,478	20.4
Subtotal Pathogens:	4,128	16,827	20,955	78.1
Total:	**8,885**	**17,958**	**26,843**	**100.0**

dwarf mistletoe could in theory be eliminated in one rotation. However, the large clearcuts required to do so are no longer acceptable, and various constraints on practice for good biological and social reasons mean that dwarf mistletoe losses will be with us for a long time.

STANDS VS. FORESTS

You are probably familiar with the expression, "You can't see the forest for the trees." A similar expression can sometimes apply to foresters: "You can't see the whole forest for the stands." Silviculture, including disease management, is typically focused on stands. In the case of forests managed primarily for wood harvest, the aim is to increase stand productivity to a maximum. However, we need to remember that the management unit is a *forest* consisting of *many stands,* and that the aim should be to optimize the wood flow of wood and other resources from the *forest.* Optimizing the productivity of an individual stand does not necessarily lead to that goal.

A common problem in many management units (e.g., a timber supply area, or TSA) is one of age-class distribution. In many TSAs there is an area of operable mature timber that will support the allowable cut for perhaps one or two decades, while the remainder of the area is occupied by young, well stocked, vigorous stands that won't reach maturity for another forty or more years. Older immature stands are often rare. This age-class gap presents a major management problem. In order to sustain communities one needs a steady supply of wood. Yet in many TSAs, that will be difficult to achieve. In such TSAs one can identify a period early in the next century during which it will be difficult to maintain the harvest at rates close to the long run sustainable yield. It is clear that in such management units a prime purpose of silviculture is to bring some of the younger immature stands to a harvestable size as quickly as possible. Juvenile spacing and fertilization are the common techniques.

If you now think back to the various ways in which we can deal with tree diseases, you will realize that almost always they involve something that must be done at the time of harvest and stand renewal. Take for instance the two most damaging groups of diseases, the dwarf mistletoes and the root rots. For dwarf mistletoe, the approach is eradication at harvest or a switch to resistant species. Little can be done to affect the course of the disease in immature, infected stands. Similarly, for the root rots, control is achieved at harvest by stump removal or a switch to resistant species. Again, treatment of immature stands is not economically feasible. Thus the opportunities to increase stand productivity through disease management do not usually come at a stage of stand development that will allow the manager to use them to solve the looming age-class problem.

Another example of the way in which the structure of the whole forest affects decisions about individual stands is provided by young, mistletoe-infected stands. Often it can be shown that the overall yield of a piece of land will be greater if infected stands are destroyed and

replaced with healthy stands than if such stands are maintained until they reach a harvestable size. If the decision is based on the goal of maximizing *stand* productivity, the answer is clear — destroy the stand and start again. An implication of that decision, however, is a change in the pattern in which wood becomes available from the forest over time. That change can sometimes be very detrimental.

The conclusions from this section are clear. Decisions about individual stands can be made only in the context of the whole forest and the purpose of management of that forest. This approach, however, seems to be very difficult to apply. Very often, the people who are required to make decisions about stands do not know the status of the whole forest, resulting inevitably in poor decisions.

It has been argued throughout this course that forest pathology is a subset of silviculture; that techniques and approaches to limit the impact of diseases should be seen as silvicultural techniques, competing with other silvicultural opportunities for a limited budget. In this section that argument has been extended by examining the context in which all silvicultural decisions must be made. It is not until that context is understood and appreciated that the correct decisions for the treatment of forest tree diseases can be made.

Concluding Remarks

Now that you have nearly completed this first course in forest pathology, it is worthwhile to consider again the objectives that were set out at the beginning. The emphasis throughout has been to give you enough information to allow you to consider forest tree diseases in decisions concerning the forest. Throughout there has been a practical slant. There is a large body of scientific knowledge in plant pathology that has been skimmed over very lightly, and there is a great deal more to learn. Almost nothing has been said, for instance, about the use and mode of action of fungicides, mostly because the use of fungicides can seldom be justified in forests in the light of ecological, economic, and social considerations. Nothing has been said about the physiology of diseased tissues, and the large body of information about the delicate biochemical interactions of pathogens and hosts. The reason is that foresters must necessarily base their decisions on an understanding at the ecological level, and need not, and indeed cannot, know all the physiological and biochemical processes that give rise to ecological phenomena.

You will all be aware that the Forest Practices Code contains requirements and guidelines for disease management. Many of you may wonder why there have been no references to that code in this course. There are two main reasons. First, administrative devices such as codes and guidelines come and go over very short time intervals; they are also specific to particular administrative regions (in this case B.C. or certain Forest Regions within B.C.). Wherever you find employment you will have to become familiar with the local administrative procedures, and such procedures are constantly revised. So learning about a particular

code at a particular time doesn't prepare you for a lifetime of work — at best, it prepares you for your first job.

The second and more important reason for omitting any reference to the Forest Practices Code is that, in the view of the course author, the Code is a step backwards in forest practice. The Code, with its regulations and guidelines, attempts to define acceptable practice. But it will inevitably fail to do so, because it cannot possibly take into account all the factors and considerations that must enter into a prescription for a specific situation. Each ecosystem and each stand is unique — there are no two stands anywhere in the world that are identical in all respects and that therefore can be adequately dealt with by a common set of prescriptions. In every particular situation there will be a number of critical factors which must be taken into account in a prescription, but the set of these will differ from site to site. Competent professionals with local experience are much better at identifying such factors than any general checklist could possibly do. So, simply meeting Code requirements (and no more) usually means a sub-optimal prescription. Most professionals will agree with this, and many try to go beyond the code, or try to ignore parts of the code that are irrelevant to the local situation. However, the administrative burden of the code, and particularly the extra work involved in obtaining relief from inappropriate provisions, greatly reduces the ability of professionals to do their work efficiently. For these reasons, reasons which would require a book to set out in full, I believe that the Forest Practices Code as it is currently formulated, is a step backwards that will result in a level of practice much lower than would be possible with current staffing and economic resources. Training of professionals should be directed at two goals, namely to achieve an understanding of (1) the function and response of ecosystems, and (2) the way society functions, so that the forester's special technical knowledge can be put to appropriate use. Most of this course is aimed at the first of these, in the expectation that better understanding will lead to better practice.

The first objective of the course was that you should be able to recognize and identify the common and damaging diseases of Pacific coast forests. Apart from the large number of decay fungi, about a dozen or so species of pathogens are responsible for most of the losses, and these have all been discussed and described in detail.

The second objective was that you should be able to interpret disease signs and symptoms of forest and shade trees around the world. You should now know enough about tree diseases that you can recognize the presence of a disease in trees or stands, and that you can make a pretty good guess about the type of disease you are dealing with, particularly in coniferous stands. The details will of course vary from place to place, and if you are going to work in places other than the Pacific Northwest, you will need further study to become familiar with the locally important diseases. That is even true within the Pacific Northwest; some diseases are of great importance in certain specific localities, but minor when considering the whole area, and not all such diseases have been

covered. This course is only the start of a lifetime of learning about the forest.

The final objective was that you should know enough about the common diseases of the North American Pacific coast forests to deal with them effectively by appropriate silvicultural prescriptions. You will now recognize that this final objective has been the main guide in the selection of material studied.

Good luck!

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 lesson 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.
After you answer the questions in this part, proceed to Appendix A and complete Assignment #3 for submission to your tutor for marking.

1. For a particular forest type in your area, list three possible purposes of management, and describe and explain which of the common tree diseases in that area might be of concern for each of these, and which are neutral or beneficial.
2. What is meant by *disease incidence, severity, intensity,* and *tolerance*?
3. Why is the spatial distribution of a disease an important consideration in addition to disease incidence, severity and intensity?
4. What is the main confounding effect when tree disease losses are estimated from a comparison of the growth of infected and healthy trees growing together within a stand?
5. What is the main difficulty in experimental design when tree disease losses are estimated from a comparison between healthy and diseased stands?
6. Explain, giving local examples, why decisions about disease management in individual stands must be placed in the context of the purpose of management of the whole forest.

APPENDIX A

Assignments to be Submitted for Marking

ASSIGNMENT INSTRUCTIONS

There are four assignments for you to submit to your tutor for marking (the last one after completion of the course laboratory). Remember to include a comment sheet (supplied to you on pink paper) with each assignment for your tutor's comments about your work. Check your course schedule for the dates that the assignments should reach your tutor.
To make steady progress in the course, you will find it helpful to maintain a regular study place and time if possible.

Answer the questions in each of the assignments in your own words — avoid copying sections from the course manual, text, or other references. Your tutor will find it much easier to assess what you understand well, and where you need further help, if you use your own words to describe the situation as you see it.

Complete the self-testing/review questions at the end of each of the lessons covered by an assignment before completing the assignment and sending it in for marking.

reminder

There is also a disease specimen collection that you must submit at the beginning of the laboratory session. See page ix for a description of this assignment.

ASSIGNMENT 1

on Lessons 1 and 2

This assignment consists of 15 questions. Answer each in one or two paragraphs.

Do this assignment after you have completed Lesson 2. Check your course schedule for the deadline date by which your tutor should receive the assignment.

1. Facultative parasites often go through a parasitic and a saprophytic stage in the course of their life cycle. Explain the survival strategy of such organisms as exemplified by their behaviour during the parasitic and saprophytic stage.

2. What is the difference between disease signs and symptoms?

3. Not all diseases are caused by pathogens. Describe and distinguish between the two groups of diseases that are not caused solely by the invasion of parasites.

4. How must Koch's postulates be amended in order to apply them to obligate parasites such as rusts or virusses?

5. Draw and describe a generalized fungal life cycle. Show when the processes of somatogamy, karyogamy, and meiosis occur, and identify which stages are the haplophase, the dikaryophase, and the diplophase.

6. Distinguish between taxonomy and nomenclature, explain why a particular fungal species may have more than one valid name, and describe why it is important to be aware of all the Latin names that can be used to describe a particular species.

7. Distinguish between dormancy and frost-hardiness.

8. If you were presented with a disease condition in which crowns of trees were dying back, how would you determine whether you are dealing with a decline or with a disease of which a pathogen is the primary cause?

9. Describe the sequence of enzymatic actions that results in decay of wood, distinguishing between white and brown rots.

10. At which stage in the decay process is the strength of the cell wall lost?
    Why does wood decayed by brown rots lose strength faster than that decayed by white rots?

11. What is the difference between true heartrots and wound-invading decay fungi?
    Which are likely to be most common in (a) young stands and (b) old stands?

12. Give three examples of the ways in which forest management decisions and silvicultural prescriptions and standards can be affected by decay development.

13. Describe how various microorganisms living in sound heartwood of trees can (a) promote or (b) retard decay development.

14. How are cull factors derived and used?

15. Discuss and give examples of how the approaches to disease management depend on the purpose and goals of management

ASSIGNMENT 2

on Lessons 3 and 4

This assignment consists of 11 questions. Answer each in one or two paragraphs.

Do this assignment after you have completed Lesson 4. Check your course schedule for the deadline date by which your tutor should receive the assignment.

1. Describe and explain the possible causes of mycostasis and explain how that phenomenon restricts the spread of root diseases to certain specific pathways.

2. How can one decide whether or not spore infection plays an important role in the spread of a root disease, and how does that knowledge help in devising control procedures?

3. Describe the common, stand-level symptoms of root disease in even-aged conifer stands during: (a) the regeneration phase; (b) the pole stage; and (c) the near-climax stage?

4. What are the specific symptoms by which the following pathogens can be recognized: *Phellinus weirii, Armillaria ostoyae, Heterobasidion annosum,* and *Inonotus tomentosus*?

5. The two main approaches to the control of root disease are the eradication of the pathogen or the use of resistant tree species. Discuss the advantages and disadvantages of each. Are there occasions when a "do nothing" approach would be appropriate?

6. What are the characteristic infection pathways and mode of spread within trees of wilt diseases?

7. Describe a typical foliage disease epidemic.
   What set of circumstances triggers the start of such epidemics, and which phenomena usually result in their collapse?

8. What (if anything) can be done to control foliage diseases?

9. Sketch and describe a mature necrophylactic periderm. What are the steps involved in the formation of such a periderm, and where on a tree would you expect to find necrophylactic periderms?

10. Describe the relationship between tree vigour, periderm formation, and the development of annual, perennial, and diffuse cankers.

11. List and give the symptoms of the major bare-root and container-nursery diseases of conifers.

ASSIGNMENT 3

on Lessons 5, 6, 7 and 8

This assignment consists of 18 questions. Answer each in one or two paragraphs.

Do this assignment after you have completed Lesson 8. Check your course schedule for the deadline date by which your tutor should receive the assignment.

1. Draw and describe a typical life cycle of rust that shows the progression of spore stages and the nuclear condition of hyphae in the various hosts and spore forms.
2. Under what conditions is it effective to prune young white pine to reduce blister rust infection?
3. Relate white pine blister rust disease hazard to the use of various methods to reduce or control the disease.
4. Why is it that the first 40 or so years in the life of a white pine stand are critical for blister rust development and damage?
5. What is the relationship between wave years, stand age, and damage by western gall rust?
6. Draw and describe a typical dwarf mistletoe life cycle.
7. How and why does the rate of infection of dwarf mistletoes change as canopies close?
8. Why does the presence of non-host species slow the rate of intensification of dwarf mistletoe?
9. Why is selective removal at juvenile spacing of trees with mistletoe symptoms unlikely to be very effective in lodgepole pine?
10. Relate site productivity and mistletoe spread and intensification in western hemlock.
11. Why do young stands infected by dwarf mistletoe grow at near-normal rates, while older stands are often severely affected?
12. Relate active and passive resistance mechanisms to quantitatively and qualitatively inherited mechanisms.
13. How is the resistance imparted by major resistance genes often overcome?
14. What are the main problems with breeding trees for quantitatively inherited resistance?
15. Compare breeding for resistance for introduced and native pathogens, and the arguments for and against such programs.

16. Why, and under which circumstances is it important to know the spatial distribution of disease within a stand when estimating disease impacts.

17. Describe the confounding effect of competition when attempts are made to estimate losses due to pathogens by comparing trees growing together within a stand

18. The difficulties referred to in question 17 can be avoided by comparing diseased and healthy stands. What new problems are introduced by such comparisons?

ASSIGNMENT 4

after lab session

This assignment is to be submitted after the laboratory session has been completed, and is due three weeks before the date the final exam is written.

During the lab field day you will see at least seven common diseases. Some of these will be encountered in several stands and under different conditions. All of them will have been discussed in the readings and course manual.

Select the three diseases most relevant to your work situation (or, if that doesn't apply, the three you find most interesting). For each of these:

1. describe the various symptoms;
2. relate impacts (on timber volume, quality, and non-timber values) to stand history and conditions as well as management goals; and
3. prescribe appropriate remedial actions under at least one explicitly stated goal of management.

Your answer for each disease should take about one-thousand words.

APPENDIX B Sample Final Examination

THE UNIVERSITY OF BRITISH COLUMBIA

FINAL EXAMINATION

FORESTRY 309

Forest Pathology

INSTRUCTOR: Dr. B.J. van der Kamp TIME: 2 hours

SPECIAL INSTRUCTIONS: Answer all questions in booklet provided.

Marks

5 1. Define: aecium
phytoalexin
ectotrophic
heteroaecious
soft rot

4 2. a) You are examining a 100 year old mixed conifer stand in the Columbia Valley near Golden, B.C. You notice that there has been heavy recent mortality in white pine such that 75% of the white pine has died in the last two years. What might be the cause of death and how would you verify your diagnosis.

b) In an even-aged, pure, 80 year old lodgepole pine stand you notice long, narrow, resinous lesions on the boles of many trees. What might they be, and how would you verify your diagnosis.

9 3. Answer three of the following four questions.

a) What is meant by "cross protection"?

b) Why is Douglas-fir dwarf mistletoe restricted to a very limited range in the driest southern interior forests?

c) Define and compare qualitative and quantitative resistance to diseases.

d) Partial cutting of Douglas-fir in interior forests is often followed by a flare-up of armillaria root disease. How does this come about?

7 4. Describe the state of the art in the management and control of either Phellinus weirii in coastal forests or Arceuthobium americanum in the central interior.

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