
Tree Wounding and Partial-cut Harvesting

A Literature Review for British Columbia



PEST MANAGEMENT REPORT NUMBER 14



BRITISH
COLUMBIA

Ministry of Forests — Forest Health — Vancouver Forest Region

Tree Wounding and Partial-cut Harvesting

A Literature Review for British Columbia

PEST MANAGEMENT REPORT NUMBER 14



Ministry of Forests — Forest Health — Vancouver Forest Region

Tree Wounding and Partial-cut Harvesting

A Literature Review for British Columbia

Stefan Zeglen

PEST MANAGEMENT REPORT NUMBER 14



Ministry of Forests — Forest Health — Vancouver Forest Region

Canadian Cataloguing in Publication Data

Zeglen, Stefan.

Tree wounding and partial-cut harvesting : a
literature review for British Columbia

(Pest management report ; no. 14)

"Intended to complement the Forest Practices Code
Tree wounding and decay guidebook." — Preface.

Includes bibliographical references: p.
ISBN 0-7726-3206-5

1. Trees — Wounds and injuries — British Columbia.
 2. Trees — Diseases and pests — British Columbia.
 3. Wood-decaying fungi — British Columbia.
- I. Vancouver Forest Region (B.C.). II. Title.
III. Series.

SB764.C3Z43 1997 634.9'6 C97-960084-7

Prepared by

Stefan Zeglen
B.C. Ministry of Forests
2100 Labieux Road
Nanaimo, BC V9T 6E9

© 1997 Province of British Columbia

Copies of this report may be obtained, subject to supply, from:
Forestry Division Services Branch
Production Resources
595 Pandora Avenue
Victoria, BC
V8W 3E7

Preface

This review was created to provide a consolidated reference to the diverse literature available on tree damage, partial-cut harvesting, and decay fungi. It is meant to be as specific as possible to British Columbia but extracts findings from literature world-wide.

This document is intended to complement the Forest Practices Code *Tree Wounding and Decay Guidebook*. It provides supporting and explanatory material for many of the concepts presented in the guidebook and discusses several subjects in greater detail.

This review is based on work originally conducted under contract to the Ministry of Forests, Prince Rupert Forest Region, by Dr. Ralph Nevill. Particular thanks are due to Dr. Brenda Callan, Pacific Forestry Centre, Canadian Forest Service, for assistance on fungal taxonomy and Paul Nystedt, Production Resources, Ministry of Forests, for co-ordinating publication design, editing, and printing.

If you have any comments on this document or require further information on this subject matter, please contact:

Stefan Zeglen
Forest Pathologist
Vancouver Forest Region
British Columbia Ministry of Forests
2100 Labieux Road
Nanaimo, B.C. V9T 6E9
Telephone: (250) 751-7001
Fax: (250) 751-7190
E-mail: SZEGLN@MFor01.for.gov.bc.ca

Executive Summary

Many factors influence wounding and subsequent decay in trees. In partial-cut harvesting operations, the amount of wounding increases with the size of the equipment used and with the number of passes through the stand. Wounded trees compartmentalize the wound and subsequent decay by forming barriers that limit the decay column. In this way, the wound will not exceed the diameter of the tree at the time the wounding took place. No further expansion into the sapwood occurs unless additional wounding takes place.

Some species are more susceptible to wounding than others. Thin-barked species and species with non-resinous wood such as true firs and hemlocks, are more frequently wounded than thick-barked species with resinous wood such as Douglas-fir.

The amount of decay increases proportionally with wound size and age. Most wounds larger than 9 dm² (1 ft²) become infected, regardless of tree species. After wound size, width and depth of injury are the most important characteristics to determine the severity of decay. Wound location is another important factor because the incidence and extent of decay is greater with increased proximity to the soil. The amount of wounding and size of injury are greater in spring and early summer when the bark is loose.

Decayed trees can be detected using physical decay indicators. These include signs of decay such as conks or punk knots and indicators of infection courts such as wound scars, frost cracks, crooks, forks, mistletoe brooms, dead or broken tops, and dead vertical branches. After harvest takes place, damage caused by sunscald, wind, and mistletoe, as well as root decay infections may cause further losses.

Damage to the residual stand may be reduced by harvest strategies that minimize the movement of equipment within the stand and by gaining co-operation of harvest personnel.

Contents

Preface	iii
Executive Summary	v
1 Introduction	1
2 Physical Response of Trees to Injury	1
2.1 Background	1
2.2 Changes in Sapwood at Wounding	3
2.3 Compartmentalization	4
2.4 Wound Closure	5
2.5 Microbial Invasion and Host Response	5
2.6 Wood Decay Processes	6
3 Wood Decay Organisms	6
3.1 Representative Wound Parasites	10
3.2 Representative True Heartrots	12
4 Injury and Decay Related to Stand Management	14
4.1 Injury During Harvest	14
4.2 Indirect Injury Resulting from Stand Management	18
4.3 Biotic Factors Affecting Stand Management	19
5 Factors Affecting Decay Development and Recognition	20
5.1 Tree Species	20
5.2 Location of Injury	20
5.3 Size of Injury	21
5.4 Age of Injury	21
5.5 Season of Injury	22
5.6 Decay and Tree-related Factors	22
5.7 Forest Site Type	23
5.8 Growth Changes Produced by Injuries	23
5.9 Wound Dressings	23
5.10 External Indicators of Decay	24
6 Stand Management Strategies to Prevent Wounding	26
6.1 Guidelines for Reducing Wounding	26
7 Summary	27
Appendix 1 Answers to Commonly Asked Questions	29
Literature Cited	33

Tables

1. Scientific and common names of wound parasites and “true heartrot” decay fungi, decay type, and recorded hosts in British Columbia	7
2. Common wound parasites of forest trees, their common means of entry, and most frequently reported hosts in British Columbia	9

Figures

1. White pocket rot caused by *Phellinus pini*. The pattern of decay is the formation of small pockets that often enlarge and coalesce over time. 6
 2. Brown cubical rot caused by *Fomitopsis pinicola*. Note the dry, crumbly appearance of the decayed wood. 6
 3. Fruiting body of *Heterobasidion annosum*. This structure is usually found at the base of a dead tree. 10
 4. The fruiting bodies of *Fomitopsis pinicola* are common in almost all forest types of the province. 11
 5. Fruiting body of *Echinodontium tinctorium* is usually found associated with the branch stub that served as its entry point into the tree. This fungus produces a brown stringy rot of conifers. 13
 6. Two *Phellinus pini* fruiting bodies. This fungus commonly enters through branch stubs of conifers. 13
 7. Commercial thinning using mechanized harvesters is becoming more common. 15
 8. A horse logger prepares to skid some logs from this shelterwood. 16
 9. Bark removal caused by skidding felled timber is a common example of tree damage in partial-cut stands. 20
 10. Note the gouging of the sapwood in the lower part of this large wound. 21
-

1 Introduction

This literature review provides information on wound response, decay fungi, and logging injury, as well as the types of damage and subsequent decay that may occur from partial-cut harvesting activities. For subject areas, sufficient material is available from which to draw conclusions and provide examples. For example, many reports document the response of trees to wounding and these reports have been frequently reviewed (Shigo 1966, 1967, 1984; Shigo and Hillis 1973; Shigo and Marx 1977; Shortle 1979, 1984; Boddy and Rayner 1983; Rayner 1986; Blanchette 1992; Boddy 1992). Adequate information is also available on decay fungi and the factors that affect decay development. However, information about damage to the residual stand caused by harvesting equipment is lacking in the literature. When it does exist, damage to the residual stand is usually of secondary interest and few of these reports are current.

Fortunately, many of the studies of decay fungi have taken place in British Columbia (Bier et al. 1946; Bier and Foster 1946; Bier et al. 1948; Buckland et al. 1949; Foster and Foster 1951; Foster et al. 1954; Thomas and Thomas 1954; Foster et al. 1958; Thomas 1958; Parker and Johnson 1960; Smith and Craig 1968, 1970; Etheridge and Craig 1976; Morrison and Johnson 1970; Wallis and Reynolds 1970; Wallis et al. 1971; Wallis and Morrison 1975; Morrison et al. 1986). In addition to these reports, studies have also been conducted on similar tree species and decay fungi occurring in the Pacific Northwest region of the United States (Wright and Isaac 1956; Shea 1960; Hunt and Krueger 1962; Aho 1974, 1977; Aho and Simonski 1975; Aho and Roth 1978; Aho and Filip 1982; Aho, Fiddler, and Filip 1983; Aho, Fiddler, and Srago 1983; Aho et al. 1987, 1989). These reports are rounded out by the findings of workers in other regions.

The information provided by these and other studies show that with adequate precautions, thinning and partial-cut harvest operations can take place with acceptable levels of damage to the residual stand.

2 Physical Response of Trees to Injury

Wounds are common events in the life of trees. A wound occurs every time a branch is lost or the bark to the xylem is broken. Tree wounds are the starting points that may lead to discoloration and decay caused by invading micro-organisms. Wounds may vary in size, but it is the area of xylem exposed during wounding and the amount of xylem disrupted beneath the bark that determines wound severity. Some wounds may only occur in the bark, where a series of complex reactions restore bark function. However, these types of wounds will not be dealt with here. Excellent reviews on bark wound responses have been written by Mullick (1977), Biggs et al. (1984), and Biggs (1992).

2.1 Background

2.1.1 *The heartrot concept*

Tree decay was first investigated over a century ago by Robert Hartig (Merrill et al. 1975), who developed the concept of heartrot. This concept assumes that as living sapwood matures it becomes the non-living heartwood at the core of mature trees. When a mature tree is wounded, the dead heartwood is decomposed by saprophy-

tic organisms that gained entrance through the wounds. Once all the heartwood is digested, a hollow results (Boyce 1961; Merrill and Shigo 1979; Shortle 1979). As the decay-causing agent does not interact with living host tissue, many plant pathologists have argued that heartrot is not a disease (Shortle 1979).

The heartrot concept has been repeatedly challenged. Hepting (1936) noted that wounded sweetgum trees, which have no heartwood, decay as readily as wounded oaks that have heartwood. Hepting also observed that the wound need not extend into the heartwood for decay to take place. Decay was limited to the sapwood and heartwood that formed before the wound occurred. Dark-coloured tissues that formed in sapwood adjacent to the wounded area were termed "pathological heartwood." This discoloration was not a result of the normal ageing process.

2.1.2 *New concepts of wound response*

Over the last thirty years, a renewed interest in the discoloration and decay associated with wounds has led to numerous, frequently reviewed studies (see Shigo 1966, 1967, 1984; Shigo and Hillis 1973; Shigo and Marx 1977; Shortle 1979, 1984; Boddy and Rayner 1983; Rayner 1986; Blanchette 1992; Boddy 1992). These studies confirm Hepting's findings that decay can occur other than by wounds to the heartwood. In place of the classical heartrot concept, models have been proposed from a number of different viewpoints. These models are based on active defence mechanisms and include:

- compartmentalization of decay in trees [codit] (Shigo and Hillis 1973; Shigo and Marx 1977; Shigo 1984);
- reaction zone formation (Shain 1967, 1971, 1979); and
- non-specific response brought on by changes in micro-environmental conditions after wounding (Boddy and Rayner 1983; Rayner 1986; Boddy 1992).

While it is uncertain which model most closely accounts for the mechanisms of tree defence, they all provide an effective system that limits decay in living trees. This report will discuss responses to fungal invasion using the codit model.

2.1.3 *Tree anatomy*

To better understand the mechanisms of decay, the basic structure of living trees should be considered first. Simplified, a tree stem is composed of bark, vascular cambium, sapwood, and heartwood. Bark is the commonly used, non-technical term used to describe all the tissues outside the vascular cambium. The vascular cambium is a thin, cylindrical layer that generates new cells. Cells formed in the outer cambium differentiate into the phloem, or inner bark, and transport photosynthate downward from the leaves or needles. Cells laid down by the inner cambium compose the xylem, or wood, which transports water and minerals upward. These cells may differentiate into vessels, tracheids, or axial parenchyma (Esau 1977).

Conifer wood is relatively homogeneous structurally, and consists primarily of tracheids and ray parenchyma cells. Axial and radial resin ducts are found in the wood, notably in genera *Picea*, *Pinus*, and *Larix*. Aging begins as soon as cells are formed. Water transport becomes functional in tracheids when their living cell contents die, which is usually within days or weeks after formation. Parenchyma

cells are thin-walled, contain living cytoplasm for one to many years, and store energy reserves (Esau 1977).

Sapwood contains living parenchyma cells and conducts water. Heartwood forms in the centres of the stem and provides mechanical support to the tree. It is no longer capable of conducting water because of secondary changes that include the depletion of nutrient reserves, necrosis of parenchyma cells, and formation of cell deposits that may darken the xylem (Shigo and Hillis 1973; Hillis 1987). Extractives often accumulate in the heartwood and may consist of polyphenols and other compounds. The chemical properties of heartwood differ in origin from wound-associated, discoloured wood. Heartwood formation rates will vary for different species and the dark colour is not always discernible (Hillis 1987).

2.2 Changes in Sapwood at Wounding

Injuries that extend into the tree's sapwood disrupt the water conduction system, expose the injured xylem to air and micro-organisms, and lead to drying of the tissues that surround the wound.

The first visible reaction to wounding is the discoloration of the xylem that surrounds the wound. This response is relatively rapid and most changes occur within a few weeks of injury (Shigo 1986). Parenchyma cells adjacent to the wound have elevated respiratory activity (Wardell and Hart 1970). As the wood discolours, starch is removed from parenchyma cells and the cell lumina fill with phenols, fatty acids, and resin acids (Shain 1967). The discoloured wood of hardwoods contains gums, gels, and suberin, while terpenes, resin, and polyphenolics form in conifers. The pigmented substances produced are the result of the degeneration of nuclei and cytoplasm that leads to cell death (Wardell and Hart 1970; Sharon 1974; Blanchette and Sharon 1975).

Above and below the wound, vessels or tracheids that would normally conduct sap become plugged with pigmented substances that tend to make the wood toxic to micro-organisms and more or less impervious to water (Blanchette 1992). Pits that connect the tracheids in gymnosperms close. Parenchyma cells may also form tyloses that plug vessels or tracheids. Tyloses are the hypertrophied parenchyma cells that balloon out into the lumen. Tyloses induced through injury, as well as tylosoids in resin canals, are frequently observed in the tracheids of species such as pine, which have thin-walled parenchyma (Yamada 1992). Tyloses are found in tracheids or vessels that are filled with air and may form because of loss of water pressure. After formation, tyloses can undergo secondary thickening, and if abundant may completely occlude tracheids or vessels. Although tyloses may slow the colonization process, many decay fungi are capable of growing through them (Blanchette 1992).

In addition to the physical alterations described, many other changes take place in discoloured wood. Moisture, ash, and mineral content, as well as pH are higher in discoloured wood (Shigo and Sharon 1970; Tattar et al. 1971; Shigo and Hillis 1973; Shortle 1979). Phenols increase and the discoloured wood becomes resistant to colonization by many decay fungi (Shigo and Sharon 1970; Tattar et al. 1971; Shortle and Cowling 1978; Shortle 1979). Phenol-tolerating organisms, however, are often found in discoloured tissues (Tattar et al. 1971; Shortle and Cowling 1978; Shortle 1979).

The amount of discoloured wood that develops after injury depends on the size of the wound and the depth of penetration into the xylem. Deep wounds have more

discoloration than surface wounds (Shigo and Sharon 1968; Shortle and Cowling 1978; Shortle 1979). For discoloration to occur, the xylem must interact with air. Sucoff et al. (1967) found no discoloration when xylem was injured without breaking the bark. The discoloration process apparently is not dependent on micro-organisms in the xylem (Sharon 1974), although their presence may intensify the formation of cell occlusions leading to greater discoloration (Blanchette and Sharon 1975; Shortle et al. 1978; Blanchette 1982a).

The season in which wounds occur may also influence the degree of discoloration, although some discrepancies in the literature exist. Several authors (Leben 1985; Shain and Miller 1988; Mireku and Wilkes 1989; Shigo 1991) report that the most extensive discoloration occurs with autumn or winter wounds, intermediate discoloration with summer wounds, and the least discoloration with spring wounds. Basham (1978) reported greater discoloration to *Acer saccharum* (Marsh.) from spring wounds compared with summer or autumn wounds, but noted that after two years spring wounds had less decay than those made in the autumn.

2.3 Compartmentalization

Following injury, micro-organisms generally colonize the vicinity of the wound. Codit is a conceptual model used to describe the processes by which trees respond to wounds. The model explains the response as a two-step process. First, "reaction zones," or chemical boundaries, are formed at the time of injury in living parenchyma cells of the sapwood and by enzyme activity in the heartwood. Then, the cambium responds by forming a "barrier zone" to isolate new tissues from those present at the time of wounding (Shigo and Marx 1977; Shortle 1979; Shigo 1984). The reaction zone results from processes associated with the discoloured xylem. The codit system describes these reactions as four "walls" each based on the tree's anatomical features (Shigo and Marx 1977; Shortle 1979). "Wall 1" is formed when the vessels and tracheids are plugged in the vertical direction above and below the wound. The last cells in a growth ring form "wall 2," which limits the spread inward. "Wall 3" limits the spread perpendicular to the ray cells. As soon as the tree is wounded walls 1–3 begin to form. "Wall 4," or the barrier zone, separates the xylem formed before wounding from xylem formed after wounding (Shigo and Marx 1977).

Wall 1 is the weakest barrier and thus columns of discoloured and decayed wood are often elongate and narrow. Wall 2 is moderately strong, but its effectiveness depends on the depth of the injury. The inward spread of pathogens behind shallow wounds that kill the cambium are resisted more effectively than deeper wounds. If wall 2 fails to the pith, the pathogen spreads inward. Wall 3 is the strongest of the first three. However, hollows will form as wall 3 fails over time and its boundaries will open like a fan (Shigo and Marx 1977).

The second part of the codit model describes the formation of wall 4. Wounding stimulates the vascular cambium to produce abnormal xylem with reduced numbers of tracheids or vessels and a high number of parenchyma cells. Thus, the barrier zone separates existing wood from that formed after wounding. The parenchyma cells of the barrier zone produce chemicals that result in a layer of new xylem more impervious than that produced by walls 1–3. Wall 4 is the strongest

wall and prevents microbes from invading the tissues formed by the cambium after wounding (Shortle 1979).

The effectiveness of compartmentalization is thought to be under genetic control of both the host tree and pathogen (Shigo et al. 1977), and a recent investigation suggests that trees can be screened for superior compartmentalizing genotypes (Shain and Miller 1988).

2.4 Wound Closure

Wounds close as the result of callus formation by the vascular cambium. Activity of the cambium around wounds exceeds that of non-wounded parts of the tree. This activity produces more wood than elsewhere at the same level on the stem. Callus—bark plus sapwood produced at the edge of the wound—expands faster tangentially than radially, which allows the wound to close while the tree expands in girth. After the wound closes by the fusion of callus rolls, the advance of discoloration and decay ceases. Over time, trees may sustain multiple wounds that result in overlapping regions of discoloration and decay (Shigo 1984).

2.5 Microbial Invasion and Host Response

The processes of compartmentalization create the barriers that resist invasion by decay fungi. However, these physical and morphological barriers do not make the tree immune to microbial colonization. As long as the wound remains open, its surface can be invaded by micro-organisms that occur on the bark and in the air. Compartments can fail when micro-organisms become established, and this causes new barriers to establish at greater depths and distances from the wound (Shigo and Marx 1977; Shortle 1979; Shigo 1984).

The reaction of the tree to wounding cannot be separated from its reaction to the micro-organisms because trees possess no mechanisms to prevent microbial invasions (Shigo and Marx 1977; Shortle 1979; Shigo 1984). Bacteria and yeasts are the primary colonists followed by organisms, chiefly ascomycetous fungi, that grow into the reaction zone and detoxify inhibitory chemicals or use them as nutrients. These organisms may be resisted, but not halted, and their advance causes a continual expansion of the reaction zone away from the wound. Many of these organisms cause a dark discoloration of the wood by converting chemicals in the reaction zone, but they do not cause decay (Shortle 1979).

Some decay fungi can interact with the living sapwood, but most depend on the discoloration process to provide a non-living substrate (Shortle and Cowling 1978). The discoloured wood contains a high phenolic content that retards the growth of most decay fungi. If the decay fungus can detoxify these chemicals (i.e., remove the dark pigments), abundant fungal growth will occur. However, phenol-tolerant pioneers will usually flourish until the phenol content is reduced and the pioneer organisms are replaced by decay fungi (Shortle 1979). Thus, pioneer organisms initially break down the host's defences and then invade the xylem, which is most critical in the processes leading to decay. After ascomycetous fungi have detoxified the wood, decay fungi (Basidiomycetes, Hymenomycetes), which are able to degrade cellulose and lignin, become active often months to years after wounding. This sequence of pioneer micro-organisms followed by wood decay fungi is known as microbial succession (Shortle 1979; Blanchard and Tattar 1981).

When wounds penetrate the heartwood, invading micro-organisms are also resisted by chemical means. Discoloured wood forms in the heartwood, but this is

attributed to chemicals secreted by parenchyma cells during the process of heartwood formation and not to an active response controlled by the tree (Shigo and Hillis 1973; Shigo and Marx 1977).

2.6 Wood Decay Processes

The largest group of fungi to degrade conifer wood is Basidiomycetes. Some members of the Ascomycetes can also cause decay, but their role in the decay of conifers is not known (Eriksson et al. 1990). Based on morphological characteristics, wood decayed by Basidiomycetes is separated into two categories: brown rots and white rots. Brown rotted wood is characterized by extensive degradation of cellulose and hemicellulose, but not lignin, whereas cellulose, hemicellulose, and lignin are degraded in white rotted wood (Eriksson et al. 1990). Heartrot and saprot fungi may cause either white or brown rot.

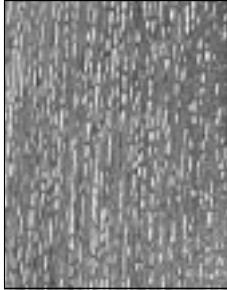


Figure 1. This white pocket rot is caused by *Phellinus pini*. The pattern of decay is the formation of small pockets that often enlarge and coalesce over time. (Photo courtesy of E. Allen)

2.6.1 White rot

White rotted wood is characterized by degradation of all organic constituents, with lignin often being degraded more rapidly than cellulose. Most white rot fungi leave no residue of coloured breakdown products, and the final coloration of decayed wood varies from whitish-yellow to whitish-tan. In an advanced stage of decay, the predominantly cellulosic residue is brittle, soft, spongy, stringy, or laminated (Eriksson et al. 1990).

With white stringy rots, the wood does not rapidly lose strength in the early stages of decay because the length of cellulose polymers is reduced relatively slowly. In more advanced stages of decay, the wood may be soft and weak, but does not fracture across the grain.

White rots that produce brittle or crumbly wood show random cleavage of cellulose molecules, which results in reduced wood strength in the early stages of decay. Decaying wood easily fractures across the grain and becomes crumbly.

2.6.2 Brown rot

Brown rot is more common in conifers than hardwoods, although some brown rots also attack hardwoods. Wood infected by brown rot fungi has a dry, cubical appearance and brown rot is often referred to as "dry rot." Although moisture is required for brown rot fungi to grow, they can survive for long periods in a dormant state.

Brown rots are characterized by random cleavage of cellulose molecules, with early loss of wood strength and brittleness similar to white rot fungi, but lignin is used slowly and incompletely. Coloured breakdown products remain, which impart a dark colour. The decaying wood shrinks and cracks into more or less cubical chunks.



Figure 2. This example of a brown cubical rot is caused by *Fomitopsis pinicola*. Note the dry, crumbly appearance of the decayed wood. (Photo courtesy of D. Morrison)

3 Wood Decay Organisms

The fungi that cause decay in living trees in British Columbia are noted in Table 1. Depending on the method of infection, decay fungi may be separated into two groups. "Wound parasites" invade living trees through wounds, while with "true heartrots" wounds are seldom the principal infection court (Etheridge 1972, 1973). Both groups of fungi are Basidiomycetes in the order Aphyllophorales and most are pore fungi.

Table 1. Scientific and common names of wound parasites and “true heartrot” decay fungi, decay type, and recorded hosts in British Columbia.

Scientific name	Common name	Type of decay	Hosts ^a
Wound parasites			
<i>Amylostereum chailletii</i> (Pers.:Fr.) Boidin		white rot	Ba, Bg, Bl, Lw, Ss, Sw, Pl, Pw, Fd, Cw, Hw
<i>Antrodea serialis</i> (Fr.:Fr.) Donk		brown pocket rot	Bl, Se, Ss, Pl, Fd, Cw
<i>Antrodea carbonica</i> (Overh.) Ryvarden & R. L. Gilbertson		brown cubical rot	Bg, Ss, Fd, Hw
<i>Bjerkandera adusta</i> (Willd.:Fr.) P. Karst.	scorched conk fungus	white mottled rot	Bl, E, Ac, At
<i>Ceriporiopsis rivulosa</i> (Berk. & Curtis) R. L. Gilbertson & Ryvarden [= <i>Poria rivulosa</i> (Berk. & Curtis)]	white butt rot or white laminated rot	white ring rot	Cw^b , Hw, Fd, Ss, Sw, Ba
<i>Chondrostereum purpureum</i> (Pers.:Fr.) Pouzar	silver leaf disease	white rot	Dm, Dr, Ep, At, M, V, W, Bl, Sw, Fd, Cw, Hw
<i>Coniophora puteana</i> (Schumach.:Fr.) P. Karst.		brown cubical trunk rot	Ba, Dr, Lw, Se, Sw, Py, Fd, Cw, Hw
<i>Cylindrobasidium laeve</i> (Pers.:Fr.) Chamuris		white rot	Ba, M, Dr, Sw, Ac, W
<i>Fomes fomentarius</i> (L.:Fr.) J. Kickx fil.	white spongy trunk rot	white rot	E , D, Ac
<i>Fomitopsis cajanderi</i> (P. Karst.) Kotlaba & Pouzar		brown cubical pocket rot	Se, Sw, Pl, At, V, Fd, Hw
<i>Fomitopsis officinalis</i> (Villars.:Fr.) Bondartsev & Singer [= <i>Fomes officinalis</i> (Villars.:Fr.) Faull.]	brown trunk rot	brown cubical trunk rot	Lw , Ba, Bg, Se, Ss, Pl, Py, Pw, Fd, Hw
<i>Fomitopsis pinicola</i> (Sw.:Fr.) P. Karst. [= <i>Fomes pinicola</i> (Sw.:Fr.) Cooke]	red belt fungus	brown crumbly rot	Ba, Bg, Bl, Se, Ss, Sw, Pl, Py, Pw, Fd, Cw, Hm, Hw, Lw, Dr, Ep, At, Ac
<i>Ganoderma applanatum</i> (Pers.) Pat.	shelf fungus (Artist’s conk)	white mottled rot	M, D, E, V, Q, W, Ba, Bg, Ss, Sw, Fd, Hm, Hw, Cw
<i>Ganoderma oregonense</i> Murrill	lacquer fungus	white spongy rot	Ba, Bg, Ss, Fd, Hw
<i>Hericium abietis</i> (Weir ex. Hubert) K. Harrison [= <i>Hydnum abietis</i> (Weir ex. Hubert)]	yellow pitted rot	long pitted rot	Ba, Bg, Bl , Hm, Hw, Ss
<i>Heterobasidion annosum</i> (Fr.:Fr.) Bref. [= <i>Fomes annosus</i> (Fr.:Fr.) Cooke]	annosum root disease	white spongy rot	Ba, Bg, Ss, Sw, Pl, Fd, Cw, Hw , Mb, D
<i>Laetiporus sulphureus</i> (Bull.:Fr.) Murrill [= <i>Polyporus sulphureus</i> (Bull.:Fr.) Fr.]	brown cubical rot	brown cubical rot	Hw , Se, Ss , Sw, Qg, B, L, Pw, Py, Fd, Cw
<i>Neolentinus kauffmanii</i> (A.H. Smith) Redhead & Ginns [= <i>Lentinus kauffmanii</i> (A.H. Smith)]	brown pocket rot of Sitka spruce	brown pocket rot	Ss
<i>Neolentinus lepideus</i> (Fr.:Fr.) Redhead & Ginns	scaly cap fungus	brown cubical rot	Pw, Cw
<i>Peniophora gigantea</i> (Fr.:Fr.) Masse		white sap rot	Ba, Sw, Pl, Pw, Py, Fd
<i>Peniophora polygonia</i> (Pers.:Fr.) Bourd. & Galzin		brown heart rot	Ac, At
<i>Peniophora pseudopini</i> Weresub & S. Gibson		red ray rot	Pl, Fd
<i>Perenniporia subacida</i> (Peck) Donk [= <i>Poria subacida</i> (Peck) Sacc.]	stringy butt rot	white spongy rot	Ba, Bg, Bl , Lt, Se, Ss, Sw , Pl, Pw, Fd, Cw, Hw , M, D, Ra, E, V, Ac, W
<i>Phaeolus schweinitzii</i> (Fr.:Fr.) Pat. [= <i>Polyporus schweinitzii</i> (Fr.:Fr.)]	velvet top fungus	brown cubical butt rot	Ba, Bl, Lt, Lw, Ss, Sw, Pl, Pw, Py , Fd , Cw, Hw , Qg

Table 1. Continued

Scientific name	Common name	Type of decay	Hosts ^a
Wound parasites (Continued)			
<i>Phellinus hartigii</i> (Allesch. & Schnabl.) Bondartsev [= <i>Fomes hartigii</i> (Allesch. & Schnabl.)]	white trunk rot of conifers	white heartrot	Hw , Ba, Bl, Fd
<i>Pholiota adiposa</i> (Fr.:Fr.) P. Kumm.	yellow cap fungus	brown mottled rot	Ba, Bg, Bl, Pw, Hw, W
<i>Pholiota populnea</i> (Pers.:Fr.) Kuyper & Tjall.-Beukers [= <i>Pholiota destruens</i> (Brond.)]	yellow laminated butt rot of poplars	white heart rot	Ac
<i>Piptoporus betulinus</i> (Bull.:Fr.) P. Karst	brown cubical rot of birch	brown crumbly rot	E
<i>Pleurotus ostreatus</i> (Jacq.:Fr.) P. Kumm.	oyster mushroom	white flaky rot	Bg, Dr, Sw, At, Ac
<i>Postia balsamea</i> (Peck) Jülich		brown cubical butt rot	Bl, Lw, Ss, Fd, Cw, Hw
<i>Postia placenta</i> (Fr.) M. Larsen & Lombard		brown cubical rot	Ss, Pw, Fd, Cw, Hw
<i>Postia sericeomollis</i> (Romell) Jülich [= <i>Polyporus sericeomollis</i> (Romell)]	pocket and butt rot of cedar	brown cubical rot	Cw , Bl, Lw, Se, Ss, Sw, Pl, Py, Fd, Cy, Hw
<i>Scytinostroma galactina</i> (Fr.) Donk	yellow stringy butt rot		Bg, Bl, Dr, Lw, Pl, Pw, Ac, Fd, Cw, Hw
<i>Spongipellis delectans</i> (Peck) Murrill [= <i>Polyporus delectans</i> Peck]	brown stringy trunk rot of hardwoods	brown stringy rot	Ac
<i>Stereum hirsutum</i> (Willd.:Fr.) S. F. Gray		white rot	Dr
<i>Stereum sanguinolentum</i> (Albertini & Schwein.:Fr.) Fr. [= <i>Haematostereum sanguinolentum</i> (Albertini & Schwein.:Fr.) Pouzar]	red heart rot	red heart rot	Pl, Pw, Py, Se, Sw, Cw, Hm, Hw, Fd, Lw, Lt, Ba, Bg, Bl
<i>Trametes versicolor</i> (L.:Fr.) Pilat	cedar pocket rot	white spongy saprot	M, Dr, Ra, E, Gp, Ac, At, V, Fd, Qg, Cw, Hw
<i>Trichaptum abietinum</i> (Dickson:Fr.) Ryvarde [= <i>Hirschioporus abietinus</i> (Dickson:Fr.) Donk]	purple conk fungus	pitted saprot	Ba, Bg, Bl, Lw, Sb, Ss, Sw, Pl, Py, Pa, Pw, Fd, Cw, Hw, Ra, V
<i>Veluticeps fimbriata</i> (Ellis & Everh.) Nakasone	brown cubical pocket rot	brown cubical pocket rot	Hw, Ba, Bg, Bl, Hm, Fd, Se, Ss
True heartrots			
<i>Echinodontium tinctorium</i> (Ellis & Everh.) Ellis & Everh. [= <i>Fomes tinctorius</i> Ellis & Everh.]	brown stringy trunk rot (Indian Paint fungus)	brown heart rot	Hm, Hw , Ba , Bg, Bl , Ss, Sw, Fd, Cw
<i>Phellinus igniarius</i> (L.:Fr.) Quél. [= <i>Fomes igniarius</i> (L.:Fr.) J. Kickx fil.]	hardwood trunk rot	white trunk rot	M, D, Ra, E, G, Ac, W
<i>Phellinus pini</i> (Thore:Fr.) Ames [= <i>Fomes pini</i> (Thore:Fr.) Fr.]	red ring rot	white pocket rot	Ba, Bg, Bl, Lw, Sb, Se, Ss, Sw, Pl, Pw, Py, Fd, Cw, Cy, Hm, Hw
<i>Phellinus tremulae</i> (Bondartsev) Bondartsev & Borisov [= <i>Fomes igniarius</i> (L.:Fr.) J. Kickx fil. var. f. <i>tremulae</i> Bondartsev]	aspen trunk rot	white trunk rot	At

^a Host abbreviations refer to genus and species symbols for trees as found in Minimum Standards for the Establishment and Remeasurement of Permanent Sample Plots in British Columbia. September 1995. Forest Productivity Councils of British Columbia. Victoria, B.C. 22 pp.

^b Bold indicates most important host(s).

Wound parasites include both heartrotting and saprotting fungi. Some are pioneer fungi or primary invaders in the microbial succession and are able to colonize living tissues (*sensu*, Shigo and Marx 1977). Most, however, are secondary invaders that attack wood already colonized by other micro-organisms. Little is known about the infection court requirements of this group of fungi (Merrill 1970; Etheridge 1973). In British Columbia, only half of the fungi reported as wound parasites can be differentiated by their mode of attack (Table 2). Many may also attack slash or continue a saprobic existence on dead trees or wood in service (Etheridge 1973). Most wound parasites are confined by the barrier zone to wood laid down before the time of injury (Shigo 1975).

Table 2. Common wound parasites of forest trees, their common means of entry, and most frequently reported hosts in British Columbia.

Fungus and mode of attack	Basal trunk wounds	Trunk wounds	Broken tops	Fire scars	Sunscald	Frost
<i>Amylostereum chailletii</i> (P-SR)*	Ba, Bg, Hw**	Ba, Bg, Hw				
<i>Bjerkandera adusta</i> (P-SR-D)		Ac, At				
<i>Coniophora puteana</i> (P-SR)	Ba, Fd			PI		
<i>Fomitopsis cajanderi</i> (P-S-HR-D)	Fd	Fd	Fd			
<i>Fomitopsis officinalis</i> (P-S-HR-SR)		Lw, Fd, Ss	Ss			
<i>Fomitopsis pinicola</i> (P-S-HR-SR-D)	Hw, Fd, Ss, Ba, Cw, Ss	Hw, Fd, Ss, Ba, Cw	Hw, Ss			Hw
<i>Ganoderma applanatum</i> (S-HR-SR-D)	Hw	Ac	Hw			
<i>Ganoderma oregonense</i> (S-HR-SR)	Hw	Bg				
<i>Heterobasidion annosum</i> (P-HR-SR)	Hw, Ba, Bg	Hw, Cw	Hw			Hw
<i>Laetiporus sulphureus</i> (S-HR-SR-D)	Hw, Fd, Ba, Ss	Hw, Cw	Hw, Ss			
<i>Neolentinus kauffmanii</i> (HR)	Ss	Ss	Ss			
<i>Peniophora polygonia</i> (P-HR)		At				
<i>Peniophora pseudopini</i> (P-HR)		Fd		PI		
<i>Phaeolus schweinitzii</i> (P?-S?-HR-SR)	Fd, Ss	Ss	Ss	Fd		
<i>Phellinus hartigii</i> (P-HR-SR-D)		Hw	Hw			
<i>Pholiota adiposa</i> (P-SR)	Hw, Ba					
<i>Scytinostroma galactina</i> (S-HR-D)	Ba	Ba, Hw	Bg			
<i>Stereum sanguinolentum</i> (P-HR-SR)		Hw, Ba, Bl, Bg, Ss, Fd	Fd, Bl	PI	Hw	Fd
<i>Veluticeps fimbriata</i> (S-HR-D)		Hw, Ba, Ss		PI		

* Symbols in brackets: P-primary invader, pioneer organism capable of attacking uncolonized heartwood and sapwood; S-secondary invader, attacks exposed colonized heartwood and sapwood, often displacing pioneer fungi; HR-causes heartrot of living trees; SR-causes saprot of living trees; D-may continue to develop in dead material or cause damage to timber in service.

** Host abbreviations refer to genus and species symbols for trees as found in Minimum Standards for the Establishment and Remeasurement of Permanent Sample Plots in British Columbia. September 1995. Forest Productivity Councils of British Columbia. Victoria, B.C. 22 pp.

True heartrots may be distinguished from wound parasites by the following characteristics:

- they rarely rely on wounds as their major infection courts;
- their characteristic decay is usually confined to heartwood;
- they consistently produce fruiting bodies on living trees; and
- they are rarely primary invaders of slash or dead material (Etheridge 1972).

Only a few decay fungi are included in this group, of which *Echinodontium tinctorium* is probably most representative.

3.1 Representative Wound Parasites

Among the most destructive wound parasites found in British Columbia are the annosum root disease fungus, *Heterobasidion annosum* (formerly *Fomes annosus*); the red belt fungus, *Fomitopsis pinicola* (formerly *Fomes pinicola*); and the red heartrot fungus, *Stereum sanguinolentum*. True heartrot fungi include *Echinodontium tinctorium* and the red ring rot fungus, *Phellinus pini*. These fungi are common to British Columbia, although they may be more prevalent in some areas than others (Wood 1986). A description of less common wound parasites may be found in Allen et al. (1996).

3.1.1 *Heterobasidion annosum*

Heterobasidion annosum is distributed throughout the northern hemisphere. This fungus is more commonly regarded as causing root rot rather than stem rot. Recent research, however, may provide an explanation for this perception. This species consists of two intersterility variants (Chase 1989), designated “S” (spruce) and “P” (pine) after the hosts on which they are most commonly found (Korhonen 1978). The P variant causes a root rot and typically infects freshly cut stumps, while the S variant is more likely to cause trunk rots. In Europe and eastern North America, the S variant is more prevalent, but in western North America both variants are common (Chase 1989). *Heterobasidion annosum* (together with *Phaeolus schweinitzii* is one of the few fungi that are primarily regarded as root rot fungi, but which may also act as wound parasites.

Stem wounds of western hemlock (*Tsuga heterophylla* [Raf.] Sarg.) are particularly susceptible to attack by *H. annosum* in coastal regions of British Columbia (Buckland et al. 1949; Foster and Foster 1951; Wallis et al. 1971), but not in the Interior (Foster et al. 1954). Douglas-fir (*Pseudotsuga menziesii* [Mirb.] Franco.) and amabilis fir (*Abies amabilis* [Dougl. ex Loud.] Forbes) suffer only light damage (Bier et al. 1948; Buckland et al. 1949; Foster et al. 1954; Thomas and Thomas 1954).

Heterobasidion annosum produces perennial fruiting structures that may be woody to leathery bracket- or crust-like structures up to 30 cm across, depending on the location. The upper surface of the basidiocarps is buff to dark brown when exposed to the light. The lower surface is cream coloured and poroid. The fruiting bodies of *H. annosum* are seldom produced on living trees (Etheridge 1973).

Basidiospore production is optimal during the periods of cool, wet weather (below 10° C), which can occur throughout much of the year in coastal areas of



Figure 3. The fruiting body of *Heterobasidion annosum*. This structure is usually found at the base of a dead tree. (Photo courtesy of D. Morrison)

British Columbia (Reynolds and Wallis 1966). The spores may infect new trunk wounds, root wounds, or freshly cut stumps. Spores that land in stem wounds may cause trunk rot, while those that land on roots may cause both root and butt rots. Stumps must be infected by *H. annosum* within a few weeks of harvest, otherwise other micro-organisms will colonize and exclude the fungus. Stumps that are successfully infected by the spores can lead to colonization of the roots. This is of particular significance in thinned or partial-cut stands. If a healthy root of an adjacent tree grows in contact with the infected stump root, the fungus will spread to the healthy tree. Further spread to adjacent residual trees occurs at root contacts (Morrison and Johnson 1978).

In decreasing order of susceptibility to infection by spores of *H. annosum* are the stumps of amabilis fir, western hemlock, Sitka spruce, Douglas-fir, and lodgepole pine. Stumps of western redcedar are rarely susceptible (Morrison and Johnson 1970; Morrison et al. 1986). Amabilis fir stumps are infected throughout most of the year, whereas infection of Douglas-fir decreases after March. Hemlock are infected more than Douglas-fir from April through August, while infection of Sitka spruce is intermediate between the two (Morrison and Johnson 1970).

Heterobasidion annosum causes a white, spongy rot. In the early stages, the decay appears as a yellow-brown to red-brown stain. As decay begins, elongate white pockets begin to form when the host tissues are dissolved. Black flecks often form in the white decay pockets. Gradually, the wood turns soft and stringy, but never brittle.

3.1.2 *Fomitopsis pinicola*

Fomitopsis pinicola is widely distributed throughout the northern hemisphere and is found in all regions of British Columbia (Etheridge 1973). It is one of the most important pathogens of old-growth stands and occurs on both coniferous and hardwood hosts (Table 1). In addition to causing decay in living trees, the fungus is also capable of invading dead trees and slash. Douglas-fir, western hemlock, spruces, and true firs are all highly susceptible to attack, with the most serious losses occurring in coastal forests (Bier et al. 1948; Buckland et al. 1949; Foster and Foster 1951).

The perennial basidiocarps of *F. pinicola* are hard, woody, and bracket- to hoof-shaped with a red band near the white- to cream-coloured edge. The oldest parts of the upper surface turn grey to dark brown or black. The undersurface or spore layer is white to cream and poroid. Although basidiocarps may grow to 60 cm in width, they are usually 5–25 cm across.

The precise requirements for *F. pinicola* infection are unclear (Etheridge 1973). *Fomitopsis pinicola* forms sporophores at wounds and spore discharge occurs throughout most of the year except during hot, dry periods of summer or below-freezing periods in winter (Mounce 1929). Decay by the fungus in true firs, western hemlock, and Douglas-fir is commonly associated with scars such as those made by falling trees (Buckland et al. 1949; Foster and Foster 1951; Thomas and Thomas 1954). Branch stubs are also common sites of decay, as are broken tops in true fir and western hemlock (Buckland et al. 1949; Foster and Foster 1951). Once established, the fungus readily develops in both sapwood and heartwood, causing a brownish discoloration, which may extend longitudinally up to 1 m beyond decayed wood (Etheridge 1973).



Figure 4. The fruiting bodies of *Fomitopsis pinicola* are common in almost all forest types of the province. (Photo courtesy of S. Zeglen)

Decay caused by *F. pinicola* is a crumbly, brown, cubical rot of both the sapwood and heartwood. Incipient decay appears as a light-brown stain. In more advanced decay, the wood breaks into small, crumbly cubes.

3.1.3 *Stereum sanguinolentum*

Stereum sanguinolentum causes decay in living conifers and slash in both the tropics and temperate regions of the world. It rarely causes decay of hardwoods. It is principally a pioneer organism that attacks freshly wounded sapwood that is uncolonized by other organisms. The fungus causes a white mottled rot and is reported to affect all coniferous species in British Columbia except yellow-cedar (*Chamaecyparis nootkatensis* [D. Don] Spach), juniper (*Juniperus* sp.), and yew (*Taxus brevifolia* Nutt.) (Table 1).

Damage by *S. sanguinolentum* is generally more prevalent in Interior rather than coastal forests of British Columbia. It is the most important cause of decay in amabilis fir, subalpine fir (*Abies lasiocarpa* [Hook.] Nutt.), and white spruce (*Picea glauca* [Moench] Voss and var. *albertiana* [S. Brown] Sarg.) in the Interior (Bier et al. 1948; Parker and Johnson 1960; Smith and Craig 1968; Smith and Craig 1970). Damage to amabilis fir is light in coastal areas (Foster et al. 1958), while damage is light to moderate in Douglas-fir, (Thomas and Thomas 1954; Craig 1970) and western hemlock (Buckland et al. 1949; Foster et al. 1954; Wallis et al. 1971).

The fungus produces thin, annual, leathery fruiting bodies that may be crust-like with a narrow, bracket-like margin when growing on a vertical substrate. Fruiting bodies often form extensive, crust-like areas when they occur on fallen material that is in contact with the ground. The upper surface is grey to olive brown. Depending on age, the undersurface or spore layer is grey to light brown turning blood red when bruised (Etheridge 1973).

Basidiospore production and infection by *S. sanguinolentum* are greatest during wet, cool (e.g., 7–24° C) periods when susceptibility of wound surfaces is optimal. The basidiospores are released after periods of rain and are dispersed by wind. In the British Columbia Interior, these conditions normally occur only in the spring and fall, while in coastal areas they may occur during most months of the year. Wounds that occur in winter are considered more susceptible to *S. sanguinolentum* than those occurring at other times of the year (Etheridge 1973). This may be because other micro-organisms are less competitive at this time or it may be related to temperature or micro-environment.

Stereum sanguinolentum causes a red heartrot. Incipient decay is firm and appears as a red-brown heartwood stain. In advanced decay, the wood becomes soft and crumbly and is light brown to red brown. Etheridge (1973) reported decay to advance at 12–13 cm/yr for most British Columbia species, but rates as high as 40 cm/yr have been reported for Norway spruce (Kallio 1976; Hansen and Hansen 1980; Atta and Hayes 1987).

3.2 Representative True Heartrots

3.2.1 *Echinodontium tinctorium*

Echinodontium tinctorium is an important cause of heartwood decay of conifers in western North America. In British Columbia, true firs are highly susceptible throughout most of their range (Bier et al. 1948; Foster et al. 1958; Thomas 1958). Depending on habitat, western hemlock is moderately to severely attacked in

Interior forests (Foster et al. 1954), at higher elevations on the coast, and on below-average sites (Foster et al. 1958; Thomas 1958). The fungus has not been found on the Queen Charlotte Islands (Foster and Foster 1951).

The basidiocarps of *E. tinctorium* are perennial, hoof-shaped, 4–30 cm in diameter, and generally form on the underside of dead branch stubs of living trees. The upper surface is rough, cracked, and black. The lower surface is grey brown to nearly black with hard, coarse spines. The interior of the basidiocarp is rust red, with the pigment extending into the decayed wood (Etheridge 1972).

Basidiospores are dispersed during cool, wet weather in autumn and spring. Large wounds are an unsuitable infection court for this fungus. In western hemlock, infection occurs when spores of the fungus enter tiny, shade-killed twigs, about 1 mm in diameter, that grow along a main branch. After spore germination and mycelial development, the fungus continues activity until the branchlet is overgrown. At this point, *E. tinctorium* produces resting spores, or chlamyospores. These can remain dormant for 50 or more years without causing decay. The dormant fungus becomes activated when mechanical injury, frost cracking, or the breakage of old, large branches allow air to enter the stem (Etheridge and Craig 1976). A similar mode of infection has been proposed and confirmed for true firs (Aho 1977; Aho and Filip 1982; Aho et al. 1987). In firs, wounds as small as 56 cm² within 30 cm of dormant *E. tinctorium* infections are capable of activating the fungus (Aho et al. 1987).

Echinodontium tinctorium may not be an important cause of decay in second-growth stands because of its method of infection and because it usually causes serious damage in trees over 150 years old. However, Aho and Filip (1982) found that 22% of 50-year-old white fir (*Abies concolor* [Gord. and Glend.] Lindl. ex Hilderbr.) had *E. tinctorium* infections. Aho et al. (1987) reported similar findings. They suggested that *E. tinctorium* may cause serious decay losses before such stands become merchantable.

In both true firs and western hemlock, *E. tinctorium* causes a brown stringy rot of the heartwood. In the early stages of decay, the heartwood appears light brown or water-soaked. In the later stages of decay the wood darkens to red brown or yellow brown with small, rust-coloured flecks. Finally, the wood is reduced to a yellow-brown, fibrous, stringy mass (Etheridge 1972).

3.2.2 *Phellinus pini*

Phellinus pini is one of the most widely distributed and destructive heartrots in North America. It is found in all regions of British Columbia and attacks all conifers except yellow-cedar and yew (Lowe 1969). Decay damage caused by *P. pini* is severe to coastal Douglas-fir (Thomas and Thomas 1954), moderate to severe to both coastal and Interior western hemlock (Buckland et al. 1949; Foster and Foster 1951; Foster et al. 1954; Foster et al. 1958), while damage to true firs is light (Buckland et al. 1949; Foster et al. 1958).

The basidiocarps of *P. pini* vary from small, nearly flat, annual incrustations to large, hard, perennial, hoof-shaped structures up to 30 cm wide. On perennial specimens, the upper surface is zoned grey brown to black with rough, concentric furrows. The margin is often velvety to the touch. The undersurface is greyish brown with round, irregular-shaped pores. The interior of the basidiocarp is cinnamon brown with a corky texture. Basidiocarps are usually produced at branch stubs on living trees. In addition to basidiocarps, the fungus also commonly produces “punk



Figure 5. The fruiting body of *Echinodontium tinctorium* is usually found associated with a branch stub. This fungus produces a brown stringy rot of conifers. (Photo courtesy of E. Allen)



Figure 6. Two *Phellinus pini* fruiting bodies. This fungus commonly enters through branch stubs of conifers. (Photo courtesy of E. Allen)

knots," which are bulging masses of tightly packed fungal hyphae protruding at branch stubs.

As with many decay fungi, details of the infection process for *P. pini* are unclear. In eastern white pine the process of infection by *P. pini* appears similar to that of *Echinodontium tinctorium* in western hemlock (i.e., entering through small branches or leader stubs). When the site of entry is overtaken by heartwood, decay begins (Haddow 1938). However, *P. pini* was seldom recovered from the branch stubs of jack pine (*Pinus banksiana* Lamb.), but entry through felling scars or broken tops could be demonstrated (Basham 1975). In British Columbia, *P. pini* infections are reported to occur in Douglas-fir and western hemlock at both branch stubs and wounds such as felling scars or broken tops, with branch-stub infections accounting for the majority (Buckland et al. 1949; Foster and Foster 1951; Thomas and Thomas 1954).

The decay caused by *P. pini* is a white pocket rot characterized by selective removal of lignin (Blanchette 1982b). In the early stages of decay, wood appears reddish in pines and purplish in Douglas-fir and spruces. Later, small, spindle-shaped pockets lined with white fibres appear and slowly coalesce. The wood in advanced stages of decay is soft, light coloured, and fibrous. Decay by *P. pini* is usually confined to the heartwood of mature trees, either in one continuous column or several discrete columns originating at branch stubs. Typically, the most extensive decay occurs in the trunk, but butt rot is common and sometimes the rot extends into the roots (Etheridge 1972).

4 Injury and Decay Related to Stand Management

All harvesting systems result in some injury to the residual stand. Injuries incurred during harvesting may include mechanical wounding to roots, root collars, and stems, as well as broken tops caused by falling trees. Indirect injuries may also occur and include sunscald to exposed trunks and windthrow of remaining trees. Regardless of cause, all types of injuries are susceptible to decay.

Although numerous studies have investigated decay resulting from logging injury, they usually provide little information on the harvesting methods that caused the injuries. Conversely, injury to the residual stand is a secondary issue in most thinning and harvesting studies. Few thinning and harvesting studies that report damage to residual trees have been conducted recently in British Columbia or other areas of the Pacific Northwest. Therefore, studies of injury caused by stand management activities conducted in eastern hardwoods are also considered in this review.

4.1 Injury During Harvest

In general terms, damage to the residual stand increases with the size of equipment used and the number of passes through the stand. However, reports on damage caused by different types of harvesting equipment and silvicultural systems are often contradictory. Generalizations such as "chainsaw felling is less damaging than machine felling" must be considered in the context of the tree species present and the extraction equipment and silvicultural system used. As well, the extent of residual stand damage very often depends on the skills and decisions of the personnel involved.

4.1.1 Injury related to machinery

Felling — When damage to the residual stand resulting from chainsaw felling and mechanical felling is compared, chainsaw felling causes less damage. However, the amount of damage will depend on the silvicultural system in use and the method of extraction. Often the reduced damage from chainsaw felling is offset by damage that occurs during skidding.

In a thinning study conducted in Washington State, chainsaw felling was compared with a rubber-tracked feller-buncher and a tracked feller-buncher with an accumulator (Kammenga 1983). Chainsaw felling was slow and often resulted in a tangle of trees that made skidding difficult. Both types of feller-bunchers lowered the trees hydraulically to the ground and bunched them, thereby increasing skidder efficiency and reducing damage.

A study in northern Ontario compared damage to the residual stand caused by chainsaw felling and tractor skidding, and mechanical felling using a Timberjack RW-30 tree-length harvester and a grapple skidder. The harvesting system used had no significant effect on the proportion of trees damaged. In each stand, 14 and 19% of residual trees were injured in chainsaw-felled and machine-harvested areas, respectively (McLaughlin and Pulkki 1992). In contrast, two studies conducted in hardwood stands produced slightly opposite conclusions. In one study, no significant difference existed between machine and chainsaw felling in stands with 20-m spacing between skid trails. On average, skidding accounted for 29% of injuries, while felling and bunching operations accounted for 62% (Kelley 1983). In the other study, machine and chainsaw felling accounted for approximately 30 and 13% of the total damage, respectively, while the remaining damage was caused by skidding (Ostrowsky et al. 1986).

Skidding — Studies that compare damage caused by ground skidding to cable yarding systems have been conducted in California (Aho, Fiddler, and Srago 1983), Oregon (Aulerich et al. 1974), and British Columbia (Maxwell and McIntosh 1976). These reports provide conflicting results as to which extraction system caused the least damage. Cable yarding was less damaging in two experimental studies (Aulerich et al. 1974; Maxwell and McIntosh 1976); however, this was not the case in surveys of randomly chosen commercially thinned stands (Aho, Fiddler, and Filip 1983). Such differences demonstrate the need for co-operation between the contractor and forest manager to ensure damage to residual trees is minimal.

In Oregon, tractor skidding and cable yarding resulted in wounds of approximately 0.5 dm² (9 in²) or larger to 25 and 30% of residual trees, respectively (Aulerich et al. 1974); however, the percentage of residual trees with scars larger than approximately 4 dm² (72 in²) dropped to 11 and 7% for tractor skidding and cable-yarding, respectively. Tractor skidding resulted in 58% of the wounds in contact with the soil, whereas 98% of the wounds caused by cable-yarding were at least 30 cm above the soil line. Wounds in contact with the soil nearly always become decayed (Wright and Isaac 1956).

In British Columbia, damage to residual Douglas-fir and western hemlock caused by ground-skidding systems using rubber-tired and soft-tracked skidders was compared with North American and European cable-yarding systems (Maxwell and McIntosh 1976). Based on a scar area of 9 dm² (144 in²) or greater, rubber-tired and soft-tracked skidders damaged 35 and 10% of residual trees, respectively. Only 3%



Figure 7. Commercial thinning using mechanized harvesters is becoming more common. (Photo courtesy of S. Zeglen)

of residual trees were damaged by the North American cable-yarding system and 8 to 10% of trees were damaged by the two European systems.

In the California study, surveys were conducted in five young white fir and Douglas-fir stands after thinning by conventional methods (tractor- and rubber-tired skidders and cable) (Aho, Fiddler, and Srago 1983). Between 22 and 50% of the residual trees were wounded in the five stands. In three of the stands, 8–15% of residual trees were damaged so badly as to be unusable. The grapple skidder produced both the highest percentage of wounding and death to residual trees, 50 and 15%, respectively. Cable yarding was almost as damaging—46% of residual trees were wounded and 8% killed. Tractor skidding was also highly damaging: 35% of residual trees were wounded and 15% killed. Skidding with rubber-tired skidders produced the least damage with between 22 and 33% of residual trees damaged.

In the same study, logging injuries were much lower in four other stands that were thinned using techniques designed to reduce logging damage. The percentage of residual trees with wounds ranged from 5% for rubber-tired skidding to between 11 and 14% for tractor skidding (Aho, Fiddler, and Srago 1983).

Skidding damage by machinery and horses — Hunt and Krueger (1962) reported that tractor skidding in partially cut stands resulted in larger wounds and a higher percentage of injury to residual trees than horse skidding. Similar findings were reported by Worthington (1961).



Figure 8. A horse logger prepares to skid some logs from this shelterwood. (Photo courtesy of D. Coates)

4.1.2 Injury related to thinning or silvicultural system

In general, unmanaged stands experience more frequent and damaging pest attacks than managed stands. Harvesting systems may influence both damage that occurs during harvest and damage that occurs after harvest by factors such as wind, sunscald, or diseases. Care must be taken with stand selection so that partial cutting is not prescribed for stands with root disease or mistletoe (Mason et al. 1989).

Single-tree selection systems often pose the greatest risk of injury and potential loss from stem decay to the residual stand (Kelley 1983; Kellogg et al. 1986; Ostrofsky et al. 1986), although this is not always the case (Boe 1974; Biltonen et al. 1976). Single-tree selection also favours shade-tolerant species such as true firs that suffer the greatest losses to stem decay (Aho 1977). Injury to residual stems can be minimized by measures such as predesignated skid trails. This and other measures to minimize damage to the residual stand are discussed in the following sections.

Shelterwood and seed-tree systems may also result in injury to overstorey trees (Boe 1974; Biltonen et al. 1976). Overstorey trees should be removed when seedlings become established. Care must be taken, however, to prevent excessive damage to developing regeneration (Mason et al. 1989).

Group-selection systems present a lower risk of injury to residual trees and thus less stem decay than selection cuttings because more space is provided for felling and skidding operations (Mason et al. 1989).

The cutting intensity also determines the type and intensity of damage to the residual stand. Boe (1974) found damage to residual trees increased with a higher cutting intensity. Cutting intensity may also influence the type of injury. A light thinning (e.g., <30% of the basal area of the stand) results in the highest number of injuries to the root collar. A high proportion of these become decayed. By contrast,

a heavy thinning (e.g., 45% of the basal area of the stand) results in injuries higher on the stem that are not as subject to decay (Wright and Isaac 1956).

The effect of three different pre-commercial thinning treatments on damage to residual trees was studied in two western hemlock–Sitka spruce stands in Oregon (Kellogg et al. 1986). Treatments included two selection cuts, narrow spacing (5.5 × 5.5 m), wide spacing (7.5 × 7.5 m), and herringbone design strip. All stands were felled by chainsaw and cable yarded. Only 12% of residual trees were damaged from yarding in the strip treatment, while 47 and 61% of residual trees were damaged in the narrow and wide treatments, respectively. Most injuries to residual trees occurred during yarding; only 3.9% of scars occurred during felling, 7.9% from loading activities, and 5.4% from line damage.

Damage to residual trees was assessed in two shelterwood cuttings and selective thinnings in mixed hardwood stands in Vermont (Kelley 1983). Trees were harvested with a Hydro-ax feller-buncher and removed with a grapple skidder, except for one selection thinning where trees were felled by chainsaw and skidded full length using a conventional cable skidder. Injuries to residual trees in the shelterwood cuttings ranged from 27 to 34%, while 42 to 47% of residual trees were injured in the thinned stands.

In another study, hardwood stands in Maine were selectively thinned by mechanical and chainsaw felling (Ostrosky et al. 1986). Treatments included mechanical felling with 20- and 40-m spacing between skid trails, chainsaw felling with 40-m spacing between skid trails, and mechanical thinning with no predesignated skid trails. A Hydro-ax feller-buncher was used primarily for cutting skid trails, while a smaller Morbell feller-buncher was used for thinning. Trees were removed using a grapple skidder. Most injuries to residual trees resulted from felling and bunching operations regardless of treatment. Chainsaw felling or mechanized thinning with 20-m spacing between skid trails resulted in the least damage with injuries to 22% of residual trees. Mechanized thinning with 40-m spacing resulted in damage to 53% of the residual stand, but the highest damage occurred in treatments with mechanized thinning and no predesignated skid trails, where 82% of residual trees were damaged.

Five thinning treatments were compared in an even-aged, 50-year-old, hardwood stand in Michigan (Biltonen et al. 1976). Treatments included strip thinning (3-m cut strips) by mechanical felling; selection thinning by chainsaw felling; selection thinning by mechanical felling; strip thinning, with selection thinning between strips (3-m cut strips), by mechanical felling; and shelterwood thinning by mechanical felling. Mechanical felling was done using a feller-buncher and trees were extracted using a grapple skidder. Chainsaw-felled trees were left in place. Strip thinning with selection thinning between strips resulted in seams approximately 3 dm² (50 in²) or larger to 60% of residual trees, followed by strip thinning 40%, shelterwood thinning 29%, selection thinning 10%, and chainsaw-felled trees 0%. Damage was high in the strip-thinning treatments because the feller-buncher could not turn in the narrow strips. This forced the operator to place cut trees to the right of the machine in the uncut stand.

Growth and mortality factors associated with three regeneration cutting methods were studied in the conversion of old-growth California redwood (*Sequoia sempervirens* [D. Don.] Endl.) into younger, managed stands (Boe 1974). Treatments included small-block clearcutting, selection cuttings (50% sawtimber volume

removal), and shelterwood cuttings (75% sawtimber volume removal). Mortality to residual trees was directly related to the volume cut: in selection cuttings 20% of sawtimber, 70% of saplings, and 80% of poletimber were killed, while in shelterwood cuttings 50% of sawtimber, 80% of saplings, and 80% of poletimber were killed. Injury to residual trees was opposite to mortality: in selection cuttings 35% of sawtimber and 5% of saplings and poletimber had logging scars, whereas in shelterwood cuttings 25% of sawtimber, 7% of saplings, and 3% of poletimber had logging scars. Wind damage after harvest caused further mortality in the shelterwood cuttings. The high mortality of sawtimber resulted in negative net basal area growth for both selection systems (Boe 1974).

Comparisons between single-tree and group-selection cuttings in ponderosa pine were combined with two sizes of skidding tractors in central Idaho (Foiles 1952). Both harvest systems and skidding methods resulted in similar amounts of damage: approximately 6% mortality to remaining sawtimber-sized trees and 12% mortality to understorey trees in each stand. No data on injury to the residual trees was provided.

Damage occurring after harvest — Lodgepole pine stands in Alberta were treated under three cutting systems: improvement cuts (two thinning cuts and a sanitation cut); harvest cuts (diameter limit, seed tree, shelterwood, group selection, and clearcut); conversion cuts (half and total overstorey), and two control stands (Walker and Johnson 1975). Eleven years after harvest, mortality to remaining trees in all treatments ranged from 10 to 70%. The conversion cut with half overstorey removed suffered the greatest mortality of lodgepole pine (70%), followed by the diameter limit cut (36%), shelterwood cut (26%), conversion cut with total overstorey removed (23%), heavy crown thinning (19%), group selection (16%), sanitation cut (15%), and heavy understorey thinning (12%). The control blocks suffered 10 and 19% mortality mostly due to windfall. No data on tree injury was provided. Mortality was caused primarily by windfall, while sunscald accounted for additional mortality. Average annual volume increase for all treatments was less than 2% per year. The authors concluded that stand mortality invalidated all potential growth gains from stand management activities.

4.2 Indirect Injury Resulting from Stand Management

Indirect injury after stand management treatments may result from sunscald and wind, and biotic agents such as mistletoes or root diseases (Wright and Isaac 1956; Alexander 1986).

4.2.1 Sunscald

Sunscald typically occurs when the trunk of thin-barked species, such as western hemlock, is suddenly exposed to full sunlight. Sunscald lesions on western hemlock occur on the southwest face of the stem and are almost always associated with decay (Wright and Isaac 1956; Wallis et al. 1971). Other species at risk include lodgepole pine (Walker and Johnson 1975) and young Douglas-fir (Stein 1955). Incidence of sunscald is significantly greater in heavy versus lightly cut stands: 5 and 16% of western hemlock had sunscald lesions in lightly and heavily thinned stands, respectively (Wright and Isaac 1956).

4.2.2 *Windfall*

Windfall, or the probability of windfall, must be considered in any partial-cut management scheme (Borough et al. 1982). In addition to the immediate loss of basal area, falling trees strike and injure those that remain and open the stand to further windfall and sunscald. In coastal areas, Wright and Isaac (1956) noted that removal of more than 20 to 25% of the board-foot volume of a stand may result in unacceptable loss due to windfall. In Interior areas, the first cut should remove no more than 30% of the basal area in single-storeyed stands with low windfall risk and 20% in moderate-risk situations. Clearcutting is recommended for high-risk situations (Alexander 1986).

4.3 **Biotic Factors Affecting Stand Management**

4.3.1 *Dwarf mistletoe*

Dwarf mistletoes, *Arceuthobium* spp., are some of the most serious diseases affecting conifers. They are parasitic plants whose fruits are forcibly ejected for distances of up to 10 m. The seeds are covered with a gelatinous material that facilitates movement from needles to branches where infection takes place (Alfaro 1983). Dwarf mistletoe infections reduce growth and seed production and increase mortality. Mistletoe infections on the outer branches may have little effect, but stem infections are more damaging. Young trees may be killed quickly, while older trees with well-developed crowns may show no appreciable effects for years. Suppressed mistletoe stem infections on older trees may become infected by decay fungi such as *Fomitopsis pinicola* (Etheridge 1973).

Partial-harvest systems, such as shelterwoods, are particularly inappropriate in mistletoe-infested stands. In well-stocked stands, most infections take place between adjacent trees, and spread within the stand is slow. After stand closure mistletoe infections generally lose vigour and shaded stem infections may die. Opening an infested stand canopy creates potential problems; it may reinvigorate old dwarf mistletoe infections, increase the rate of spread due to reduced stand density, and expose the most vulnerable, regenerating understorey trees. As the threshold levels for treatment options may vary across the province, foresters conducting partial-cut harvest in stands with dwarf mistletoe should consult the Forest Practices Code *Dwarf Mistletoe Management Guidebook* (B.C. Ministry of Forests 1995a).

4.3.2 *Root disease*

Partial-harvest systems in stands with root disease present several problems. Root disease fungi are not killed when the trees they infect are harvested. Instead they are often invigorated. As the most important root disease fungi differ among the various regions of the province, and foresters conducting partial harvest in stands with root disease should consult the Forest Practices Code *Root Disease Management Guidebook* (B.C. Ministry of Forests 1995b).

4.3.3 *Decay resulting from logging*

Injury — Numerous studies conducted in western North America provide an overview of the types of losses resulting from decay that may occur in species found in British Columbia.

Studies by Buckland et al. (1949) and Foster and Foster (1951) demonstrated that scars incurred during logging activities were the major entrance points for decay fungi. Dissections of logging injuries 17 years after harvest revealed over 50%

of Douglas-fir and 90% of western hemlock had associated decay (Shea 1961). In a similar study, Hunt and Krueger (1962) observed six-year-old logging scars on immature Douglas-fir and western hemlock of which 23 and 61% were infected. Wright and Isaac (1956) examined numerous logging scars of various ages on western hemlock, true firs, and Sitka spruce, and produced curves that assign specific decay volumes to scars of known age and size. Wallis et al. (1971) determined the usefulness of Wright and Isaac's curves for predicting decay in western hemlock in coastal British Columbia. The authors concluded that the curves accurately predicted decay volumes for scars greater than 9 dm² (1 ft²), but overestimated decay for smaller wounds.

Decay losses associated with wounding in true fir stands have been studied extensively in the U.S. Pacific Northwest. Decay in sawtimber-sized grand fir (*Abies grandis* [Dougl. ex D. Don] Lindl.) was responsible for a loss of 14% of gross merchantable volume, more than double found in Engelmann spruce, Douglas-fir, and western larch (Aho 1977). In south-central Oregon, dissections of approximately 50-year-old white fir revealed 22% had *Echinodontium tinctorium* infections (Aho and Filip 1982). Similar findings were reported in slightly older stands of white and grand fir in Washington and Oregon (Aho et al. 1987). The authors suggested that *E. tinctorium* may cause serious decay losses before such stands become merchantable. In northern California, wound area due to logging damage and age were most closely related to the extent of decay in true fir stands (Aho, Fiddler, and Srago 1983; Aho et al. 1987).

Post-harvest tree and stand damage levels for British Columbia are discussed in the *Tree Wounding and Decay Guidebook* (B.C. Ministry of Forests 1997).

5 Factors Affecting Decay Development and Recognition

The incidence and extent of decay in trees is highly variable and depends on many factors such as tree species, location, size, season of injury, and stand dynamics, as well as decay organisms.

5.1 Tree Species

Generally, thin-barked species and species with non-resinous wood, such as true firs and hemlock, are more readily infected by decay fungi than thick-barked species with resinous wood such as Douglas-fir (Shea 1960; Hunt and Krueger 1962; Aho, Fiddler, and Filip 1983). Spruce and pine are exceptions in the resinous wood category: spruce are very susceptible to infection when wounded, while pines are less so (Aho, Fiddler, and Filip 1983).

In coastal regions, incidence of decay is generally comparable between true firs, western hemlock, and spruce (Buckland et al. 1949; Wright and Isaac 1956; Foster et al. 1958), but the rate of decay is higher for spruce (Wright and Isaac 1956). In Interior regions of British Columbia, incidence and rate of decay are higher for spruce (Parker and Johnson 1960), whereas in Oregon incidence of decay is higher in true firs (Aho 1977). Incidence and rate of decay are lowest for Douglas-fir in all regions (Wright and Isaac 1956; Hunt and Krueger 1962; Aho 1977).

5.2 Location of Injury

Location of initial injury on the stem affects the frequency of infection and amount of subsequent decay. Generally, wounds below diameter breast height are more



Figure 9. Bark removal caused by skidding felled timber is a common example of tree damage in partial-cut stands. (Photo courtesy of S. Zeglen)

likely to be infected by decay fungi than those occurring higher. Root and stem wounds that contact the soil nearly always become infected, regardless of size, and have faster decay rates than wounds higher in the stem (Wright and Isaac 1956; Parker and Johnson 1960; Shea 1960; Hunt and Krueger 1962; Karkkainen 1971; Isomaki and Kallio 1974; Aho, Fiddler, and Srago 1983). This may be due to microclimatic effects that favour fungal colonization (Wright and Isaac 1956; Isomaki and Kallio 1974). Although frequency of injuries with decay declines with increasing height, as many as 80% of injuries above diameter breast height may be infected (Wallis et al. 1971; Aho, Fiddler, and Srago 1983).

5.3 Size of Injury

Wound size is one of the most important characteristics related to the amount of decay (Isomaki and Kallio 1974; Aho, Fiddler, and Srago 1983; Aho et al. 1989). Generally, 60 to 85% of wounds larger than 9 dm² (1 ft²) on western hemlock, true firs, or spruce are decayed, which are roughly double that of smaller wounds (Wright and Isaac 1956; Parker and Johnson 1960; Hunt and Krueger 1962; Wallis et al. 1971; Isomaki and Kallio 1974; Wallis and Morrison 1975; Aho, Fiddler, and Srago 1983; Aho et al. 1989). Wounds on Douglas-fir larger than 9 dm² are also more frequently decayed than smaller wounds, but the percentage of these wounds with decay is lower (Shea 1960; Hunt and Krueger 1962).

Depth and width of wounds are also important factors to determine the severity of decay. Wide scars are associated with a greater percentage of decay loss (at least double) than long, narrow scars (Isomaki and Kallio 1974; Wallis and Morrison 1975). Deep wounds or wounds with splintered wood have more decay than smooth wounds (Wallis et al. 1971; Isomaki and Kallio 1974; Wallis and Morrison 1975; Aho, Fiddler, and Srago 1983).

5.4 Age of Injury

Together with wound size, wound age is most closely correlated with extent of decay volume. For most tree species, wounds less than 10 years old are unimportant because decay has not had sufficient time to develop (Filip et al. 1984). After 10 years the percentage of wounds with decay increases with time regardless of wound size (Wright and Isaac 1956; Parker and Johnson 1960; Hunt and Krueger 1962; Wallis et al. 1971; Isomaki and Kallio 1974; Wallis and Morrison 1975; Aho, Fiddler, and Srago 1983; Aho et al. 1989).

The annual increase of decay after wounding directly affects the recoverable wood volume. For the first three to five years, the rate of decay in western hemlock, true firs, and spruce is relatively rapid, even though the volume of decay is small. After 5 to 10 years, the rate of decay continues more slowly (Wright and Isaac 1956; Parker and Johnson 1960; Aho, Fiddler, and Srago 1983; Aho et al. 1989). In western hemlock, vertical decay advances 30 cm/yr for the first three years, but slows to 5 to 10 cm/yr over the next 12 years. Fifteen years after wounding, vertical progress of decay may be less than five cm/yr (Wright and Isaac 1956). The rate of radial depth of penetration is also fastest in the first three to five years (Wright and Isaac 1956; Parker and Johnson 1960).

Wright and Isaac (1956) presented curves based on the relationship between age and area of injury, and decay volume for western hemlock, Sitka spruce, and



Figure 10. Note the gouging of the sapwood in the lower part of this large wound. (Photo courtesy of S. Zeglen)

true firs. Beginning roughly three years after injury, decay volumes in wounds larger than 9 dm² double every 10 years for western hemlock and true firs. These findings have been verified for western hemlock in British Columbia (Wallis et al. 1971). Decay apparently begins more slowly in Sitka spruce, but once established it progresses considerably faster. Eight years after wounding, decay volumes in wounds larger than 9 dm² double every three years.

5.5 Season of Injury

Reports on the effects of the season in which injuries occur and resulting decay appear to conflict. Many refer to “sap season” (i.e., spring through early summer), when sap in the trees is actively flowing and the bark is known to be loose (Wright and Isaac 1956; Maxwell and McIntosh 1976; Aho, Fiddler, and Filip 1983). Researchers working with decay fungi recommend against thinning activities at this time, especially in stands of thin-barked species, as the trees are more susceptible to injury (Aho, Fiddler, and Filip 1983; Aho, Fiddler, and Srago 1983; Aho et al. 1989).

These recommendations seem to contradict studies that compare the extent of decay associated with wounds deliberately created at different times of the year. Wounds occurring early in the growing season have less decay than those made during the rest of the year (Leben 1985; Shain and Miller 1988; Mireku and Wilkes 1989). Positive pressure from flowing sap, which results in reduced cavitation in the tracheids, may explain this finding (Leben 1985). However, the small size of injuries created for these studies bears little resemblance to the size of most logging scars. Logging injuries that occur in spring are often larger than those that occur at other times of the year (Isomaki and Kallio 1974; Aho, Fiddler, and Filip 1983). Thus, the recommendation that thinning activities be avoided during spring and early summer appears reasonable.

5.6 Decay and Tree-related Factors

5.6.1 Age relationships

The percentage of trees with decay and average volume of decay per tree increases progressively with tree age for most species (Buckland et al. 1949; Foster and Foster 1951; Foster et al. 1954, 1958; Thomas and Thomas 1954). Few western hemlock and true firs under 150 years of age are decayed, but from this age onwards the percentage of infected trees increases from less than 10 to 100% with average volumetric losses from 1 to 50% (Buckland et al. 1949; Foster and Foster 1951; Foster et al. 1954, 1958). A similar pattern occurs for Douglas-fir; however, the percentage of trees infected and volumetric losses are substantially lower (Thomas and Thomas 1954; Shea 1961; Hunt and Krueger 1962).

5.6.2 Diameter relationships

The percentage of infected trees and average volume of decay per tree also increases progressively with increasing diameter (Foster and Foster 1951; Thomas and Thomas 1954; Foster et al. 1954, 1958; Parker and Johnson 1960). Because diameter increases with age this is expected, but a high degree of variation in diameter may occur between trees of the same age (Buckland et al. 1949).

5.7 Forest Site Type

Although reports about the relationship between site and volume of decay conflict, this may be partly due to tree species or decay organisms. For coastal Douglas-fir, decay volumes increase with increasing site quality, although the increase in decay volumes on good sites is offset by increased tree growth (Thomas and Thomas 1954). However, for western hemlock growing on the Queen Charlotte Islands (Foster and Foster 1951) and in the Upper Columbia region of British Columbia, decay increases with decreasing site quality (Foster et al. 1954). Apparently, this occurs even though different fungi are responsible for the decay in each area. On the Queen Charlotte Islands, *Phellinus pini*, *Fomitopsis pinicola*, and *Heterobasidion annosum* are the principal decay organisms of western hemlock, whereas in the Upper Columbia region *E. tinctorium* causes over 60% of all decay. In this region, *E. tinctorium* occurs principally in areas with lower site indices, whereas *P. pini*, the next most commonly recovered decay organism, occurs mostly on the better sites (Foster et al. 1954).

Isomaki and Kallio (1974) found that decay volume for Norway spruce in Finland increased with increasing site quality or with fertilization. The most frequently recovered organisms were *Heterobasidion annosum* and *Stereum sanguinolentum*. They suggested that the wounds of trees growing on good or fertilized sites provided a better substrate for fungal growth than trees growing on poor or unfertilized sites.

The amount of decay may be greater on northerly versus southerly aspects (Isomaki and Kallio 1974; Filip et al. 1984); however, this effect is not always observed (Wright and Isaac 1956; Aho, Fiddler, and Srago 1983).

5.8 Growth Changes Produced by Injuries

Some reports that have examined the effect of decay fungi on tree growth show conflicting results. Buckland et al. (1949) found no difference in growth rates between healthy and decayed amabilis fir or western hemlock. No significant difference in radial growth existed between wounded and unwounded jack pine in thinned and unthinned stands (McLaughlin and Pulkki 1992). Similarly, in hardwood stands in West Virginia, no difference in five-year-diameter growth occurred between unwounded trees and trees with wounds 6 dm² or larger (Lamson and Smith 1988).

In a Finnish study, where injuries were classified by depth, width, and location on the stem, radial growth was significantly reduced (Isomaki and Kallio 1974). Radial growth decreased with both increasing depth or width of wounds. Superficial wounds reduced radial growth by approximately 10%, while deep wounds resulted in a 20% reduction. The effect of wound width was also considerable. Wounds 5 to 8 cm wide resulted in a 10% reduction of radial growth, while those 17 to 30 cm wide caused a 35% reduction of radial growth. For trunk and root collar wounds, height growth was reduced approximately by 15 and 40%, respectively. No wound classification was attempted in the former studies.

5.9 Wound Dressings

Because the consequences of wounding are often severe, it is not surprising that foresters have looked to other professionals for potential remedies. Horticulturists

and urban foresters have used wound dressings for years even if this may be, as suggested by Shigo and Shortle (1983), “for cosmetic reasons.”

Most commonly available wound dressings have either an asphalt, latex, or shellac base. Some may incorporate fungicides in their formulation, but most do not (Mercer 1983). *In vitro* tests of asphalt paints show they are not fungitoxic nor do they provide even a mechanical barrier to several wood decay fungi. Only with the addition of fungicides such as copper naphthenate will asphalt paints inhibit fungal growth and provide a mechanical barrier (Dooley 1980). In a study on living trees, several asphalt paint formulations and orange shellac failed to provide long-term protection against discoloration and infection by decay fungi (Shigo and Shortle 1983).

Asphalt-based wound dressings fail to provide long-term protection from decay fungi because with age, wound dressings tend to split and crack allowing the entry of decay organisms (Clifford et al. 1987; Biggs and Peterson 1990). Another reason for failure is that withdrawal of water and nutrients from the wood beneath the wound surface cause a negative pressure that draws fungal spores on or near the wound surface into the damaged wood (Lonsdale 1984). This might suggest that wound dressings containing systemic fungicides may provide more long-lasting results, but this is not the case. Studies of latex wound dressings and dressings containing systemic fungicides such as tridemifon show that these materials provide only 12 months’ protection at best (Mercer et al. 1983; Clifford et al. 1987).

In Europe, wound dressings are currently used to treat trees damaged during harvest (Dimitri 1983; Kallio 1983). Products registered in Germany include “Drawipas,” “Lac Balsam,” and “Silvisan” (Dimitri 1983). Lac Balsam is a latex paint; Silvisan is a latex paint containing the fungicides copper naphthenate and pentachlorophenol (Mercer 1983); Drawipas contains the fungicides thiabendazole and captofol (Schumann 1985). Both Lac Balsam and Silvisan were effective for no more than one year in studies on pruning wounds (Mercer et al. 1983; Clifford et al. 1987). In a German study, wounds were cleaned with a special scribing knife, an axe, or left uncleaned and painted with either Drawipas, Lac Balsam, or Silvisan (Schumann 1985). Discoloration, indicative of decay, was observed on 38% of scribed wounds, 69% of wounds cleaned with an axe, and 60% of uncleaned wounds. No significant difference existed between the wound dressings. These differences suggest that treatment benefits do not outweigh application costs.

5.10 External Indicators of Decay

Internal decay is often difficult to detect. A study that estimated defects in Sitka spruce on the Queen Charlotte Islands noted that of 31 species of wood decay fungi only one, *Phellinus pini*, consistently produces the conks that indicate internal decay. All of the other decay fungi produce no evidence of internal decay (Bier and Foster 1946). Similar observations were made for western hemlock (Foster and Foster 1951). However, other indicators exist that can be used to identify decayed trees (Foster et al. 1953; Aho 1974, 1982; Aho and Simonski 1975; Aho and Roth 1978).

5.10.1 Descriptions of defect and decay indicators

Trees with any of the following indicators should be removed during thinning or partial harvest and not left as crop trees. Decay indicators are of two types:

-
1. signs of decay, such as conks or punky knots; and
 2. signs of infection courts such as wounds, scars, frost cracks, crooks, forks, mistletoe brooms, dead or broken tops, and dead vertical branches.

Conks — Conks, or sporophores, are the fruiting bodies of decay fungi and are certain signs of decay. Conks are significant indicators because of the considerable decay usually associated with them, but not all trees infected by these fungi produce conks. At certain times of the year, other fungi such as *Pholiota* sp. and *Hericum abietis* may also produce sporophores on or near injuries. Although these sporophores indicate decay, the type of injury is a more reliable indicator (Aho 1974, 1982; Aho and Simonski 1975, Aho and Roth 1978).

Basal injuries — Basal injuries include open or closed wounds in contact with the ground and may be of any size. Scars may be caused by fire or mechanical injuries and are important indicators of decay (Aho 1974, 1982; Aho and Simonski 1975, Aho and Roth 1978). Recent wounds (i.e., <10 years old) will not be significantly decayed. However, recently wounded trees should not be left as crop trees if the next anticipated stand entry is in 30 to 40 years.

Trunk injuries — Trunk injuries include open and closed wounds larger than 9 dm² and more than 10 years old. Such injuries may occur anywhere along the stem above the ground to below the merchantable top diameter.

Frost cracks — Frost cracks are open cracks or seams that are caused by freezing. They are particularly common in firs and are associated with excessive moisture (wetwood). Often, trees with frost cracks have conks of *E. tinctorium* higher on the stem. Usually, decay associated with the conks will not extend into the wetwood or frost-cracked area. Trees with only frost cracks generally do not have decay associated with the cracks (Aho 1974, 1982; Aho and Roth 1978) and may be left as crop trees.

Top injuries — Top injuries are injuries that occur within the merchantable portion of the stem and include tops broken by wind, ice, or snow, and spike tops resulting from insects, rust fungi, dwarf mistletoe, animals, or unknown causes (Aho and Roth 1978). These trees should not be left as crop trees, but some may be useful as wildlife trees.

Crooks — Crooks are sudden bends in the merchantable bole. In younger trees, crooks may result from ice, snow, or animals. In older trees, they may result from breaks or leader death that occur high in the tree. Crooks in young trees are usually not associated with decay as there is no wounding, whereas crooks in older trees often become decayed (Aho and Roth 1978).

Forks — Forked trees occur when two or more lateral branches replace a dead or broken leader.

Dead vertical branches — Dead vertical branches are old leaders that may have been killed by the same factors noted for top injuries. Decay is usually present when dead vertical branches are associated with a crook (Aho and Roth 1978).

Dwarf mistletoe infection — Stem infections result in pronounced swellings and cankers. Cankers may become decayed when the bark sloughs off and exposes the wood below (Aho 1982; Aho and Roth 1978).

Using these decay indicators in the Queen Charlotte Islands, 31% of healthy amabilis fir were decayed, while 74% with wound symptoms and 100% with conks were decayed. For western hemlock the figures improved: 29% identified as healthy were decayed, while 79% with wound symptoms and 100% with conks were decayed (Foster and Foster 1951).

6 Stand Management Strategies to Prevent Wounding

The most effective way to reduce decay and the resulting volume losses in residual trees is to prevent wounding during stand activities. This requires not only good planning and logging practices, but also well-trained personnel.

Harvest planning and layout should occur simultaneously (Kellogg et al. 1986). The logging plan must include such factors as volume removal and logging system requirements. Skid trails or cable-yarding corridors should be laid out before felling and should be compatible with the terrain. Much of the damage to the residual stand depends on the efforts of logging personnel. Crews must have a working knowledge of the felling and skidding techniques that minimize stand damage.

6.1 Guidelines for Reducing Wounding

The following recommendations to reduce stand damage during harvest are summarized from several reports (Wallis and Morrison 1975; Aho, Fiddler, and Filip 1983; Aho, Fiddler, and Srago 1983; Filip et al. 1984). Although intended primarily for thinning operations, many of the principles may be applied to other silvicultural systems.

6.1.1 Before harvest

1. Manage on short rotations. Keep rotations to less than 150 years. Even in unmanaged stands incidence of decay increases after this age because of natural injuries.
2. Restrict the operating season. Do not let harvesting occur during spring and early summer when sap is flowing and the bark is not tight. Trees are easier to wound and injuries are often larger.
3. Thin stands as young as is practical. Decay following injury is confined to wood existing at the time of injury. Therefore, the smaller the tree at the time of injury, the smaller the volume of wood that will be susceptible to decay.
4. Consider local environmental conditions when evaluating stand management options. In locations with high wind risk, removing more than 25% of stand volume may cause excessive windfall losses.
5. Match the size and type of equipment to topography, tree size, soil type, and soil condition.
6. Select crop trees with at least 50% live-crown ratio, at least 20 cm of current leader growth to ensure release, the best form and growth, and no evidence of previous wounding or decay (e.g., fruiting bodies of decay fungi along the stem).

-
7. Plan skid trails before logging. Use straight-line trails wherever possible and avoid sharp turns. This not only reduces skidding distance, but eliminates the damage that may occur at the turns.
 8. Designate “rub” or “bump” trees. These trees are to be harvested last. Cull logs may also be placed along the edges of skid trails to protect residual trees.
 9. Match log length with final spacing. Close spacing requires that short logs be skidded, while wider spacing allows longer logs to be skidded with minimal damage to residual trees. Skidding whole trees increases damage to residual trees.
 10. Gain the co-operation of operators. Help operators realize that most damage to residual trees is unnecessary and avoidable. Both training and supervision may be necessary to provide desired results.

6.1.2 During harvest

1. Log skid trails first. Trees on skid trails should be felled and skidded first. Cut stumps low to the ground to avoid shunting equipment or logs into residual trees.
2. Use directional felling. Trees should be felled so as to minimize the need for skidder manoeuvring.
3. Logs should be limbed before skidding. Limbs should be cut flush with the bole because stubs may redirect skidded trees into residual trees. Stubs may also snag on residual trees causing deep injuries.
4. Wound dressings are considered unnecessary. Chemical and biological control measures have not proven effective in preventing decay. In some cases, wound dressings may even encourage decay (Shigo and Shortle 1983).
5. Stump treatments are generally unnecessary. Unless thinning or harvesting in areas with a high incidence of *Heterobasidion annosum*, treating the stump surface with chemicals is unnecessary.

7 Summary

The following generalizations apply to decay from all causes in coniferous species:

1. Wounds are the most common entry points for decay fungi.
2. Trees compartmentalize wounds and subsequent decay; that is, decay columns will not exceed the current diameter of the tree unless additional wounding takes place.
3. Wounds occur more frequently, and amount of decay is greater, in thin-barked species and species with non-resinous wood, such as true firs and hemlocks, than in thick-barked species with resinous wood such as Douglas-fir.
4. Amount of decay increases proportionally with frequency of tree wounding in the stand. Wounds both activate dormant infections by *Echinodontium tinctorium* and provide entry courts for decay fungi.

-
5. Amount of decay increases with wound size and age. Most wounds larger than 9 dm² (1 ft²) become infected, regardless of tree species. After wound size, width and depth of injury are the most important characteristics in determining severity of decay.
 6. Amount of decay increases with increasing proximity to the soil. Most injuries in contact with the soil become infected, regardless of size. Wounds below diameter breast height are more likely to be infected by decay fungi than those occurring higher on the stem.
 7. Amount of decay increases proportionally with stand age and diameter where diameter is proportional to age.
 8. Amount of wounding and size of injury is greater in spring and early summer when the bark is loose.
 9. Amount of wounding increases with increasing size of equipment and with repeated entry through the stand. Damage to the residual stand may be reduced by harvest strategies that minimize movement of equipment within the stand and by gaining the co-operation of harvest personnel.
 10. Wound dressings are unnecessary. None of the chemical or biological methods provide tree wounds with long-term protection against decay fungi.
 11. Damage resulting from thinning or partial harvest can occur to the residual stand after harvest takes place. Sunscald and wind damage, as well as mistletoe and root disease infections, are types of injury that may occur.
 12. Reasonable detection of decayed trees may be achieved using physical decay indicators. These include signs of decay such as conks or punk knots, and indicators of infection courts such as wound scars, frost cracks, crooks, forks, mistletoe brooms, dead or broken tops, and dead vertical branches.

APPENDIX 1 Answers to Commonly Asked Questions

1. Which partial-cut harvesting methods result in the least damage to residual crop trees?

In general terms, damage to the residual stand increases with increased size of equipment and repeated movement through the stand, but reports in the literature are often contradictory. Such differences suggest that the extent of damage to the residual stand may depend as much on the decision of harvesting personnel as on the type of harvesting equipment or silviculture systems.

Comparisons of damage to the residual stand resulting from chainsaw felling and mechanical felling suggest that chainsaw felling may cause less damage. However, much is dependent on the silvicultural system in use and the method of extraction. A reduction in damage from chainsaw felling may be offset by damage occurring during skidding.

Studies comparing damage caused by ground skidding (rubber-tired and tracked vehicles) to cable-yarding systems found cable yarding less damaging in two experimental studies, but not in surveys of randomly chosen, commercially thinned stands. Such differences suggest that the extent of damage to the residual stand may depend on the skills and decisions of harvesting personnel.

Single-tree selection systems often pose the greatest risk of injury and potential losses from stem decay to the residual stand, although it is not always the case. Single-tree selection also favours shade-tolerant species such as true firs, which suffer the greatest losses to stem decay.

Shelterwood and seed tree systems may also result in injury to overstorey trees. Overstorey trees may be removed once seedlings become established, but care must be taken to prevent excessive damage to developing regeneration.

Group selection systems present a lower risk of injury to residual trees than selection cuttings as there is more space for felling and skidding operations.

Intensity of cut also determines the type and amount of damage to the residual stand. Boe (1974) found damage to residual trees increased with increased intensity of the cut. Cutting intensity may also influence the type of injury. A light thinning, <30% of the basal area of the stand, resulted in the highest number of injuries to the root collar, of which a high proportion become decayed. By contrast, a heavy thinning, >45% of the basal area of the stand, resulted in injuries higher on the stem that are not as readily decayed.

2. How much damage can a tree sustain before it becomes unlikely to survive until the next stand entry?

References to how much damage a tree can tolerate and still survive are not readily available in the literature. However, it seems reasonable that a tree's ability to tolerate damage is dependent on the amount of damage to its component parts (e.g., damage to the crown, stem, or roots).

Some damage is obvious; a broken top below the living crown results in tree death. A tree may survive a broken top above the living crown, but be stunted and soon overtopped by surrounding trees. Pruning studies show increasing height and basal area growth loss with increasing pruning intensity compared with unpruned trees. Similar effects may occur for trees with broken tops. Broken tops are also sites for infection by decay fungi.

Effects of damage to the trunk and roots of trees are more difficult to predict. Kelley (1983) suggested that wounds greater than one-third the diameter of the tree were severe, but provided no data on impact of such wounds on growth. Isomaki and Kalio (1974) noted that trunk wounds reduced both radial and height growth by 15%, root collar wounds reduced height growth by 40%, and damage to subsoil roots reduced height growth by 25%. However, no descriptions of wound dimensions were provided.

These observations do not directly answer the question posed, but they may provide guidelines for felling damaged crop trees after harvest.

3. *What is the maximum size a wound on the bole can be before the tree can no longer properly heal itself? On the roots? Is wound location on the tree important?*

Trees do not heal wounds, they compartmentalize them; that is, trees do not have the ability to restore damaged tissues to their original function. Barriers are placed in tissues adjacent to the damaged area to prevent moisture loss and the entry of air and micro-organisms. Assuming the crown and roots are not damaged, and given sufficient time, trees can close wounds of almost any size. However, whether or not a tree has the ability to close a wound does not preclude the possibility of a large wound causing extensive decay.

Wound location is a critical factor in the development of decay. Regardless of wound size, almost all root injuries and stem injuries in contact with the soil become infected. The lowest percentage of infections take place above diameter breast height.

4. *Do many small wounds have the same effect as one large wound? What is the effect of strip wounds? Is there a "good" time of year to injure trees (i.e., conduct harvesting activities)? Is there a bad time?*

When considering small wounds, location and proximity to other wounds are the most important factors. Most wounds near (below diameter breast height) or in contact with the soil will become infected with decay fungi. However, if the tree crown is not damaged and the tree has no other wounds, it should be able to close these wounds relatively rapidly and compartmentalize any decay.

Wound width is a more important factor than length in determining the extent of decay. Wide wounds are associated with a greater percentage of decay loss (at least double) than long, narrow wounds.

Spring and early summer is the time of year when sap is actively flowing and bark is loose. This time of year is sometimes referred to as "sap season." Thinning and partial-cut harvesting operations should be avoided during this time, especially in stands of thin-barked trees such as hemlocks and true firs.

5. *Which decay fungi are wound invaders? What is the probability of invasion occurring given a wound of a certain size? What are other common entry points of decay fungi?*

Wound-invading decay fungi of British Columbia are shown in Table 1. Known entry points for some of these fungi are shown in Table 2. The most important wound-invading fungi in British Columbia include *Heterobasidion annosum*, *Fomitopsis*

pinicola, and *Stereum sanguinolentum*. Other important decay fungi such as *Echinodontium tinctorium* and *Phellinus pini* are true heart-rotting fungi and do not invade through wounds.

Generally, logging scars under 9 dm² are not serious. Less than half of the wounds of this size become decayed and compartmentalization usually confines the decay of these wounds to a small area. For wounds of this size, location is probably the most important factor in determining infection and subsequent decay; however, most injuries of 9 dm² and larger become infected regardless of stem location. Incidence and extent of decay increases with increasing wound size.

Width and depth of injury are also important considerations. Isomaki and Kallio (1974) showed that growth decreased with increasing width and depth of wound. Wounds wider than one-third of the stem's circumference are considered severe. Deep wounds invariably become decayed as they provide a more suitable microclimate for decay fungi, whereas wounds that do not disrupt the xylem suffer less decay.

In contrast to wound-invading fungi, heartrot fungi such as *E. tinctorium* and *P. pini* enter the stem through branch stubs. In the case of *E. tinctorium*, the fungus enters tiny shade-killed twigs, about 1 mm in diameter, growing along a main branch. Mycelial development stops once the infection is overgrown and the fungus becomes dormant. Dormancy persists until the fungus becomes activated by mechanical injuries, frost cracks, or the breakage of old, large branches that allow air to enter the stem. Wounds as small as 56 cm² within 30 cm of dormant *E. tinctorium* infections are capable of activating the fungus.

6. *Do commercial tree paints or chemical treatments provide effective, post-wound protection? For how long? Are they cost-effective for forestry operations?*

Wounds are long-term events in the lives of trees. Wounds may remain open up to fifteen years after they were made. Studies show that the most effective wound dressing, even those containing systemic fungicides, provide at best only 12 months' protection. This may be due to cracking and peeling or other factors.

German studies suggest application of wound dressing may be cost-effective, but this finding is hard to accept considering that incidence of decay is only reduced and not eliminated. In North America, researchers working with decay fungi do not recommend the use of wound dressings.

7. *How do decay fungi, which have entered through wounds caused by logging, affect stand dynamics?*

There are no reports in the literature that show that wounded and subsequently decayed trees directly affect stand dynamics. However, after partial harvesting, unharvested, decayed trees are prone to windfall due to the opening of the stand. Trees with large amounts of decay may break during storms and strike healthy trees. These newly wounded trees may also develop decay.

8. *Are there stand types that should not be partial-cut harvested for reasons of stand damage alone? Where there is a choice, should certain species be preferentially selected for removal in thinning or harvesting operations?*

Stands with a high component of thin-barked species such as western hemlock, true fir and spruce may be unsuitable for partial-cut harvesting. These species are more

easily damaged during harvesting operations than thick-barked species such as Douglas-fir. In newly opened stands the stems of these species are especially prone to sunscald and these lesions invariably become decayed.

Whenever possible, thick-barked species such as Douglas-fir or western red-cedar, which are very resistant to decay, should be preferred as crop trees.

9. Is there a reliable method to detect internal decay in trees prior to conducting thinning or partial-cut harvesting operations?

Reasonable detection of decayed trees may be achieved using physical decay indicators. These include signs of decay such as conks or punk knots, and indicators of infection courts such as wound scars, frost cracks, crook forks, mistletoe brooms, dead or broken tops, and dead vertical branches.

Using the above decay indicators, Foster and Foster (1951) found 31% of amabilis fir identified as healthy were decayed, while 74% with wound symptoms and 100% with conks were decayed. For western hemlock the figures improved to 29% identified as healthy with decay, 79% with wound symptoms and 100% with conks were decayed.

There is no known device available for reliably detecting internal decay in trees. Shortle et al. (1978) demonstrated the utility of a machine called a "Shigometer" for detecting decay in telephone poles and living trees. However, others have been unable to reproduce the results presented in this study. Another method known as "computer tomography" shows promise, but the methodology is not always reliable.

10. What other potential pest or site environmental problems are enhanced by the wounding of residual trees following thinning or partial-cut harvesting?

Mistletoe and root disease infections are enhanced in partial-cut stands. Stand opening reinvigorates suppressed hemlock mistletoe infections and allows seeds to travel further. In addition, many silvicultural systems, such as shelterwoods, allow the mistletoe overstorey to infect the regenerating stand.

In many stands, partial cutting produces fresh stumps that can serve as infection courts for *Heterobasidion annosum*. From infected stumps, the fungus may spread to healthy trees in the stand via root contacts. The fungus may either kill infected trees directly or indirectly by windfall due to decay-weakened roots.

Generally, stands in areas prone to high winds may not be suitable for partial-cut harvesting. In areas prone to high winds, no more than 20 to 25% of the basal area of the stand should be removed in coastal regions and no more than 30% in Interior regions. Sunscald of thin-barked species is another environmental factor that may lead to decay. Sunscald is more prevalent in heavily than lightly thinned stands.

Literature Cited

- Aho, P.E. 1974. Defect estimation for grand fir in the blue mountains of Washington and Oregon. U.S. Department of Agriculture Forest Service, Portland, Oreg. Research Paper PNW-175. 12 pp.
- . 1977. Decay of grand fir in the Blue Mountains of Oregon and Washington. U.S. Department of Agriculture Forest Service, Portland, Oreg. Research Paper PNW-229. 18 pp.
- . 1982. Indicators of cull in western Oregon conifers. U.S. Department of Agriculture Forest Service, Portland, Oreg. General Technical Report PNW-144. 17 pp.
- Aho, P.E. and G.E. Filip. 1982. Incidence of wounding and *Echinodontium tinctorium* infections in advanced white fir regeneration. Canadian Journal of Forest Research 12:705–08.
- Aho, P.E. and L.F. Roth. 1978. Defect estimation for white fir in the Rogue River National Forest. U.S. Department of Agriculture Forest Service, Portland, Oreg. Research Paper PNW-240. 18 pp.
- Aho, P.E. and P. Simonski. 1975. Defect estimation for white fir in the Fremont National Forest. U.S. Department of Agriculture Forest Service, Portland, Oreg. Research Paper PNW-196. 9 pp.
- Aho, P.E., G.E. Filip, and F.F. Lombard. 1987. Decay fungi and wounding in advance grand and white fir regeneration. Forest Science 33:347–55.
- Aho, P.E., G. Fiddler, and G.E. Filip. 1983. How to reduce injury to residual trees during stand management activities. U.S. Department of Agriculture Forest Service, Portland, Oreg. General Technical Report PNW-156. 17 pp.
- . 1989. Decay losses associated with wounds in commercially thinned true fir stands in Northern California. U.S. Department of Agriculture Forest Service, Portland, Oreg. Research Paper PNW-403. 8 pp.
- Aho, P.E., G. Fiddler, and M. Srago. 1983. Logging damage in thinned, young-growth true fir stands in California and recommendations for prevention. U.S. Department of Agriculture Forest Service, Portland, Oreg. Research Paper PNW-304. 8 pp.
- Alexander, R.R. 1986. Silvicultural systems and cutting methods for ponderosa pine forests in the Front Range of the central Rocky Mountains. U.S. Department of Agriculture Forest Service, Fort Collins, Colo. General Technical Report RM-128. 22 pp.
- Alfaro, R. 1983. Survey and appraisal of dwarf mistletoe in second-growth western hemlock stands. *In* Proceedings of the workshop in management of hemlock dwarf mistletoe. J. Muir (editor). B.C. Ministry of Forests, Victoria, B.C. Pest Management Report No. 4:10–21.
- Allen, E.A., D.J. Morrison, and G.W. Wallis. 1996. Common tree diseases in British Columbia. Nat. Res. Can., Can. For. Serv., Victoria, B.C. 178 pp.
- Atta, H.A. and A.J. Hayes. 1987. Decay in Norway spruce caused by *Stereum sanguinolentum* Alb. & Schwa. ex Fr. developing from extraction wounds. Forestry 60:101–11.

-
- Aulerich, D.E., K.N. Johnson, and H. Froehlich. 1974. Tractors or skylines: what's best for thinning young-growth Douglas-fir. *Forest Industries* 101(12):42–45.
- Basham, J.T. 1975. Heartrot of jack pine in Ontario. iv. Heartwood-inhabiting fungi, their entry and interactions within living trees. *Canadian Journal of Forest Research* 5:706–21.
- . 1978. Early sugar maple stem discoloration and microorganism invasion in simulated wounds of felling and fire scars. *Phytopathology* 68:1693–99.
- Bier, J.E. and R.E. Foster. 1946. The relation of research in forest pathology to the utilization of overmature timber. Significance of conk rot of Sitka spruce on Queen Charlotte Islands. *B.C. Lumber* 30:38–40.
- Bier, J.E., P.J. Salisbury, and R.A. Waldie. 1948. Studies in forest pathology. v. Decay in fir, *Abies lasiocarpa* and *A. amabilis*, in the Upper Fraser Region of British Columbia. Canadian Department of Agriculture, Publication 804, Technical Bulletin No. 66. 35 pp.
- Bier, J.E., R.E. Foster, and P.J. Salisbury. 1946. Studies in forest pathology. iv. Decay of Sitka spruce on the Queen Charlotte Islands. Canadian Department of Agriculture, Publication 783, Technical Bulletin No. 56. 35 pp.
- Biggs, A.R. 1992. Anatomical and physiological responses of bark tissues to mechanical injury. *In* Defense mechanisms of woody plants against fungi. R.A. Blanchette and A.R. Biggs (editors). Springer-Verlag, Berlin. pp. 13–40.
- Biggs, A.R. and C.A. Peterson. 1990. Effect of chemical applications to peach bark wounds on accumulation of lignin and suberin and susceptibility to *Leucostoma personii*. *Phytopathology* 80:861–65.
- Biggs, A.R., W. Merrill, and D.D. Davis. 1984. Discussion: response of bark tissues to injury and infection. *Canadian Journal of Forest Research* 14:351–56.
- Biltonen, F.E., W.A. Hillstrom, H.M. Steinhilb, and R.M. Godman. 1976. Mechanized thinning of northern hardwood pole stands: methods and economics. U.S. Department of Agriculture Forest Service, St. Paul, Minn. Research Paper NC-137. 17 pp.
- Blanchard, R.O. and T.A. Tattar. 1981. Field and laboratory guide to tree pathology. Academic Press, New York, N.Y. 172 pp.
- Blanchette, R.A. 1982a. Progressive stages of discoloration and decay associated with the canker-rot fungus, *Inonotus obliquus*, in birch. *Phytopathology* 72:1272–77.
- . 1982b. Decay and canker formation by *Phellinus pini* in white and balsam fir. *Canadian Journal of Forest Research* 12:538–44.
- . 1992. Anatomical responses of xylem to injury and invasion by fungi. *In* Defense mechanisms of woody plants against fungi. R.A. Blanchette and A.R. Biggs (editors). Springer-Verlag, Berlin. pp. 76–95.
- Blanchette, R.A. and E.M. Sharon. 1975. *Agrobacterium tumefaciens*: a promoter of wound healing in *Betula alleghaniensis*. *Canadian Journal of Forest Research* 5:722–30.
- Boe, K.N. 1974. Growth and mortality after regeneration cuttings in old-growth Redwood. U.S. Department of Agriculture Forest Service, Berkeley, Calif. Research Paper PSW-104. 13 pp.
-

-
- Boddy, L. 1992. Microenvironmental aspects of xylem defenses to wood decay. *In* Defense mechanisms of woody plants against fungi. R.A. Blanchette and A.R. Biggs (editors). Springer-Verlag, Berlin. pp. 96–132.
- Boddy, L. and A.D.M. Rayner. 1983. Origins of decay in living deciduous trees: the role of moisture content and a reappraisal of the expanded concept of tree decay. *New Phytologist* 94:623–41.
- Borough, K.W., F.H. McKinnell, and P.R. Carter. 1982. Effects of stocking and thinning on wind damage in plantations. *New Zealand Journal of Forest Science* 12:244–68.
- Boyce, J.S. 1961. *Forest pathology*. McGraw-Hill. New York, N.Y. 572 pp.
- B.C. Ministry of Forests and B.C. Environment. 1995a. *Dwarf Mistletoe Management Guidebook*. Victoria, B.C. 20 pp.
- . 1995b. *Root Disease Management Guidebook*. Victoria, B.C. 58 pp.
- . 1997. *Tree Wounding and Decay Guidebook*. Victoria, B.C. 19 pp.
- Buckland, D.C., R.E. Foster, and V.J. Nordin. 1949. Studies in forest pathology. vii. Decay on western hemlock and fir in the Franklin River area, British Columbia. *Canadian Journal of Research* 27:312–31.
- Chase, T.E. 1989. Genetics and population structure of *Heterobasidion annosum* with special reference to western North America. *In* Symposium on research and management of annosus root rot in western North America. U.S. Department of Agriculture Forest Service, Berkeley, Calif. General Technical Report PSW-116.
- Clifford, D.R., P. Gendle, and M.E. Holgate. 1987. Gel formulations for the treatment of pruning wounds. ii. Results with differing gel and fungicide components and comparison with sealant compositions. *Annals of Applied Biology* 110:501–14.
- Craig, H.M. 1970. Decay following scarring of Douglas-fir in the dry-belt region of British Columbia. Canadian Forest Service, Pacific Forestry Research Centre, Victoria, B.C. Report BC-X-43. 14 pp.
- Dimitri, L. 1983. Preliminary results of chemical application techniques to prevent wound decay. *In* Proceedings of the sixth international conference on root and butt rots of forest trees. Melbourne, Australia, August 1983. G.A. Kile (editor). IUFRO working party S2.06.01. pp. 332–40.
- Dooley, H.L. 1980. Methods for evaluating fungal inhibition and barrier action of tree wound paints. *Phytopathology* 64:465–68.
- Eriksson, K.E., R.A. Blanchette, and P. Ander. 1990. Microbial and enzymatic degradation of wood and wood components. Springer-Verlag, Berlin. 407 pp.
- Esau, K. 1977. *Anatomy of seed plants*. 2nd ed. John Wiley and Sons, New York, N.Y.
- Etheridge, D.E. 1972. True heartrots of British Columbia. Canadian Forest Service, Pacific Forest Research Centre, Victoria, B.C. Forest Pest Leaflet No. 55. 14 pp.
- . 1973. Wound parasites causing tree decay in British Columbia. Canadian Forest Service, Pacific Forest Research Centre, Victoria, B.C. Forest Pest Leaflet No. 62. 15 pp.
-

-
- 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. *Canadian Journal of Forest Research* 6:299–318.
- Filip, G.M., P.E. Aho, and M.R. Wiitala. 1984. Strategies for reduction of decay in the Interior Douglas-fir and grand fir types. *In* *Silvicultural management strategies for pests of the Interior Douglas-fir and grand fir types*. D.M. Baumgartner and R. Mitchell (editors). Cooperative Extension, Washington State University, Pullman, Wash. pp. 73–80.
- Foiles, M.W. 1952. An appraisal of logging damage to advance reproduction of Ponderosa pine. U.S. Department of Agriculture Forest Service, Ogden, Utah. Research Note No. 99. 4 pp.
- Foster, R.E. and A.T. Foster. 1951. Studies in forest pathology. viii. Decay of western hemlock in the Queen Charlotte Islands, British Columbia. *Canadian Journal of Botany* 29:479–521.
- Foster, R.E., G.P. Thomas, and J.E. Browne. 1953. A tree decadence classification for mature coniferous stands. *Forest Chronicle* 29:359–66.
- Foster, R.E., H.M. Craig, and G.W. Wallis. 1954. Studies in forest pathology. xii. Decay of western hemlock in the Upper Columbia Region, British Columbia. *Canadian Journal of Botany* 32:145–71.
- Foster, R.E., J.E. Browne, and A.T. Foster. 1958. Studies in forest pathology. xix. Decay in western hemlock and amabilis fir in the Kitimat Region of British Columbia. Canadian Department of Agriculture, Forest Biology Division. Publication No. 1011. 52 pp.
- Haddow, W.R. 1938. The disease caused by *Trametes pini* (Thore) Fries in white pine (*Pinus strobus* L.). *Transactions of the Royal Society of Canada* 29:21–80.
- Hansen, R.F. and R.H. Hansen. 1980. Microorganisms which invade *Picea abies* in seasonal stem wounds. i. General aspects. Hymenomycetes. *European Journal of Forest Pathology* 10:321–39.
- Hepting, G.H. 1936. Decay following fire in young Mississippi Delta hardwoods. U.S. Department of Agriculture. Technical Bulletin No. 494. 32 pp.
- Hillis, W.E. 1987. Heartwood and tree exudates. Springer-Verlag, Berlin. 268 pp.
- Hunt, J. and K.W. Krueger. 1962. Decay associated with thinning wounds in young-growth western hemlock and Douglas-fir. *Journal of Forestry* 60:336–40.
- Isomaki, A. and T. Kallio. 1974. Consequences of injury caused by timber harvesting machines and the growth and decay of spruce (*Picea abies* (L.) Karst.). *Acta Forestalia Fennica* 136. 25 pp.
- Kallio, T. 1976. *Peniophora gigantea* and wounded spruce. Part ii. *Acta Forestalia Fennica* 149. 27 pp.
- . 1983. Significance of wound decays in coniferous stands: the possibilities of their control. *In* *Proceedings of the sixth international conference on root and butt rots of forest trees*. Melbourne, Australia, August 1983. G.A. Kile (editor). IUFRO working party S2.06.01. pp. 314–24.
- Kammenga, J.J. 1983. Whole-tree utilization system for thinning young Douglas-fir. *Journal of Forestry* 81:220–24.
-

-
- Karkkainen, M. 1971. A study on tree injuries caused by mechanized timber transportation in thinnings. IUFRO Meeting, Royal College of Forestry, Stockholm, Sweden, Sept. 1969. pp. 136–40.
- Kelley, R.S. 1983. Stand damage from whole-tree harvesting in Vermont hardwoods. *Journal of Forestry* 81:95–96.
- Kellogg, L.D., E.D. Olsen, and M.A. Hargrave. 1986. Skyline thinning a western hemlock–Sitka spruce stand: harvesting costs and stand damage. Forest Research Laboratory, Oregon State University, Corvallis, Oreg. Research Bulletin No. 53. 21 pp.
- Korhonen, K. 1978. Intersterility groups of *Heterobasidion annosum*. *Communications Instituti Forestalis Fenniae* 94:1–25.
- Lamson, N.I. and H.C. Smith. 1988. Effect of logging wounds on diameter growth of sawlog-size Appalachian hardwood crop trees. U.S. Department of Agriculture Forest Service. Research Paper NE-616. 3 pp.
- Leben, C. 1985. Wound occlusion and discoloration columns in red maple. *New Phytologist* 99:485–90.
- Lonsdale, D. 1984. Available treatments for tree wounds: an assessment of their value. *Arboricultural Journal* 8:99–107.
- Lowe, D.P. 1969. Check list and host index of bacteria, fungi and mistletoes of British Columbia. Canadian Forest Service, Pacific Forest Research Centre, Victoria, B.C. Report BC-X-32. 392 pp.
- McLaughlin, J.A. and R.E. Pulkki. 1992. Assessment of wounding at two commercially thinned jack pine sites. *Northern Journal of Applied Forestry* 9:43–46.
- Mason, G.N., K.W. Gottschalk, and J.S. Hadfield. 1989. Effects of timber management practices on insects and diseases. *In* The scientific basis for silvicultural and management decisions in the National Forest System. U.S. Department of Agriculture Forest Service, Washington, DC. WO-55. 179 pp.
- Maxwell, H.G. and J.A. McIntosh. 1976. Yarding and skidding potential in west coast commercial thinning. *Canadian Forest Industries* 96:41–45.
- Mercer, P.C. 1983. Callus growth and the effect of wound dressings. *Annals of Applied Biology* 103:527–40.
- Mercer, P.C., S.A. Kirk, P. Gendle, and D.R. Clifford. 1983. Chemical treatments for control of decay in pruning wounds. *Annals of Applied Biology* 102:435–53.
- Merrill, W. 1970. Spore germination and host penetration by heartrotting Hymenomycetes. *Annual Review of Phytopathology* 8:281–300.
- Merrill, W., D.H. Lambert, and W. Liese. 1975. Important diseases of forest trees. Contributions to mycology and phytopathology for botanists and foresters by Robert Hartig. *Phytopathology Classics*, No. 12. American Phytopathological Society, St. Paul, Minn.
- Merrill, W. and A.L. Shigo. 1979. An expanded concept of tree decay. *Phytopathology* 69:1158–60.
- Mireku, E. and J. Wilkes. 1989. Seasonal variation in the ability of the sapwood of *Eucalyptus maculata* to compartmentalize discoloration and decay. *Forest Ecology and Management* 28:131–40.
-

-
- Morrison, D.J. and A.L.S. Johnson. 1970. Seasonal variation of stump infection by *Fomes annosus* in coastal British Columbia. *Forest Chronicle* 46:200–06.
- . 1978. Stump colonization and spread of *Fomes annosus* 5 years after thinning. *Canadian Journal of Forest Research* 8:177–80.
- Morrison, D.J., M.D. Larock, and A.J. Waters. 1986. Stump infection by *Fomes annosus* in spaced stands in the Prince Rupert Forest Region of British Columbia. Canadian Forest Service, Pacific Forest Research Centre, Victoria, B.C. Report BC-X-285. 12 pp.
- Mounce, I. 1929. Studies in forest pathology. ii. The biology of *Fomes pinicola* (Sw.) Cooke. Canadian Department of Agriculture. Bulletin No. 11. 56 pp.
- Mullick, D.B. 1977. The nonspecific nature of defence in bark and wood during wounding, insect and pathogen attack. *Recent Advances in Phytochemistry* 11:395–441.
- Ostrofsky, W.D., R.S. Seymour, and R.C. Lemin, Jr. 1986. Damage to northern hardwoods from thinning using whole-tree harvesting technology. *Canadian Journal of Forest Research* 16:1238–44.
- Parker, A.K. and A.L.S. Johnson. 1960. Decay associated with logging injury to spruce and balsam in the Prince George Region of British Columbia. *Forest Chronicle* 36:30–45.
- Rayner, A.D.M. 1986. Water and the origin of decay in trees. *In* Water, fungi and plants. P.G. Ayers and L. Boddy (editors). University Press, Cambridge, Mass. 525 pp.
- Reynolds, G. and G.W. Wallis. 1966. Seasonal variation in spore deposition of *Fomes annosus* in coastal forests of British Columbia. *Canadian Forest Service Bimonthly Research Notes* 22:6–7.
- Schumann, G. 1985. Effect of careful pretreatment of wounds on application of protective agents and on prevention of decay in Norway spruce. *Forsttech. Inform.* 37:87–94.
- Schwartz, V., A. Habermehl, and H.W. Ridder. 1989. Nondestructive identification of heart- and wound-rots in the stem of standing trees by computer tomography. *Forstarchiv* 60:239–45.
- Shain, L. 1967. Resistance of sapwood in stems of loblolly pines to infection by *Fomes annosus*. *Phytopathology* 57:1034–45.
- . 1971. The response of sapwood of Norway spruce to infection by *Fomes annosus*. *Phytopathology* 61:301–07.
- . 1979. Dynamic responses of differentiated sapwood to injury and infection. *Phytopathology* 69:1143–47.
- Shain, L. and J.B. Miller. 1988. Ethylene production by excised sapwood of clonal eastern cottonwood and the compartmentalization and closure of seasonal wounds. *Phytopathology* 78:1261–65.
- Sharon, E.M. 1974. An altered pattern of enzyme activity in tissues associated with wounds in *Acer saccharum*. *Physiology and Plant Pathology* 4:307–12.

-
- Shea, K.R. 1960. Decay in logging scars in western hemlock and Sitka spruce. Weyerhaeuser Forest Research Note No. 25. 6 pp.
- . 1961. Deterioration resulting from logging injury in Douglas-fir and western hemlock. Weyerhaeuser Forest Research Note No. 36. 5 pp.
- Shigo, A.L. 1966. Decay and discoloration following logging wounds on northern hardwoods. U.S. Department of Agriculture Forest Service. Research Paper No. 43. 23 pp.
- . 1967. The early stages of discoloration and decay in living hardwoods in northeastern United States: a consideration of wound-initiated discoloration and heartwood. IUFRO Congress Proceedings 9:117–33.
- . 1975. Compartmentalization of decay associated with *Fomes annosus* in trunks of *Pinus resinosa*. Phytopathology 65:1038–39.
- . 1984. Compartmentalization: a conceptual framework for understanding how trees defend themselves. Annual Review of Phytopathology 22:189–214.
- . 1986. A new tree biology, facts, photos, and philosophies on trees and their problems and proper care. Shigo and Trees Associates, Durham, N.H. 595 pp.
- . 1991. Modern arboriculture: a systems approach to the care of trees and their associates. Shigo and Trees Associates, Durham, N.H. 424 pp.
- Shigo, A.L. and E.M. Sharon. 1968. Discoloration and decay in hardwoods following inoculations with Hymenomycetes. Phytopathology 58:1493–98.
- . 1970. Mapping columns of discoloured and decayed tissue in sugar maple, *Acer saccharum*. Phytopathology 60:232–37.
- Shigo, A.L. and H.G. Marx. 1977. Compartmentalization of decay in trees. U.S. Department of Agriculture Forest Service. Information Bulletin No. 405. 73 pp.
- Shigo, A.L. and W.C. Shortle. 1983. Wound dressings: results of studies over 13 years. Arboricultural Journal 9:285–94.
- Shigo, A.L. and W.E. Hillis. 1973. Heartwood, discoloured wood, and microorganisms in living trees. Annual Review of Phytopathology 11:197–222.
- Shigo, A.L., W.C. Shortle, and P.W. Garrett. 1977. Genetic control suggested in compartmentalization of discoloured wood associated with tree wounds. Forest Science 23:179–82.
- Shortle, W.C. 1979. Mechanisms of compartmentalization of decay in living trees. Phytopathology. 69:1147–51.
- . 1984. Biochemical mechanisms of discoloration, decay, and compartmentalization of decay in trees. International Association of Wood Anatomists. Bulletin No. 5:100–104.
- Shortle, W.C. and E.B. Cowling. 1978. Interaction of the live sapwood and fungi commonly found in discoloured and decayed wood. Phytopathology 68: 617–23.
- Shortle, W.C., J.A. Menge, and E.B. Cowling. 1978. Interaction of bacteria, decay fungi, and live sapwood in discoloration and decay of trees. European Journal of Forest Pathology 8:293–300.
-

-
- Smith, R.B. and H.M. Craig. 1968. Decay in advanced alpine fir regeneration in the Prince George District of British Columbia. *Forest Chronicle* 44:37–44.
- . 1970. Decay in advanced alpine fir regeneration in the Kamloops District of British Columbia. *Forest Chronicle* 46:217–20.
- Stein, W.I. 1955. Pruning to different heights in young Douglas-fir. *Journal of Forestry* 53:352–55.
- Sucoff, E., H. Ratsch, and D.D. Hook. 1967. Early development of wound initiated discoloration in *Populus tremuloides* Michx. *Canadian Journal of Botany* 45:649–56.
- Tattar, T.A., W.C. Shortle, and A.E. Rich. 1971. Sequence of microorganisms and changes in constituents associated with discoloration and decay of sugar maple infected with *Fomes connatus*. *Phytopathology* 61:556–58.
- Thomas, G.P. 1958. Studies in forest pathology. xviii. The occurrence of the Indian paint fungus, *Echinodontium tinctorium* E. & E., in British Columbia. Canadian Department of Agriculture, Forest Biology Division. Publication No. 1041. 30 pp.
- Thomas, G.P. and R.W. Thomas. 1954. Studies in forest pathology. XIV. Decay in Douglas-fir in the coastal region of British Columbia. *Canadian Journal of Botany* 32:630–53.
- Walker, N.R. and H.J. Johnson. 1975. Growth and regeneration response to various stand treatments in a mature lodgepole pine stand. Canadian Forest Service, Northern Forest Research Centre, Edmonton, Alta. Information Report NOR-X-137.
- Wallis, G.W. and D. J. Morrison. 1975. Root rot and stem decay following commercial thinning and guidelines for reducing losses. *Forest Chronicle* 51:203–07.
- Wallis, G.W. and G. Reynolds. 1970. *Fomes annosus* root and butt rot: a threat to managed stands in coastal British Columbia. *Forest Chronicle* 46:221–24.
- Wallis, G.W., G. Reynolds, and H.M. Craig. 1971. Decay associated with logging scars of immature western hemlock in coastal British Columbia. Canadian Forest Service, Pacific Forest Research Centre, Victoria, B.C. Report BC-X-54. 8 pp.
- Wardell, J.F. and J.H. Hart. 1970. Early responses of sapwood *Quercus bicolor* to mechanical injury. *Canadian Journal of Botany* 48:683–86.
- Wood, C. 1986. Distribution maps of common tree diseases in British Columbia. Canadian Forest Service, Pacific Forest Research Centre, Victoria, B.C. Report BC-X-281. 68 pp.
- Worthington, N.P. 1961. Tree damage resulting from thinning in young-growth Douglas-fir and western hemlock. U.S. Department of Agriculture Forest Service. Research Note No. 202. 7 pp.
- Wright, E. and L.A. Isaac. 1956. Decay following logging injury to western hemlock, Sitka spruce, and true firs. U.S. Department of Agriculture Forest Service. Technical Bulletin No. 1148. 34 pp.
- Yamada, T. 1992. Biochemistry of gymnosperm responses to fungal invasion. *In* Defense mechanisms of woody plants against fungi. R.A. Blanchette and A.R. Biggs (editors). Springer-Verlag, Berlin. pp. 147–64.
-