CAUSES OF DIEBACK OF DOUGLAS-FIR IN THE INTERIOR OF B.C.

By

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ABSTRACT

Frost damage to sapling size plantation Douglas-fir [*Pseudotsuga menziesii* (Mirb.) Franco.] in the central interior of B.C. was identified as the major cause of dieback and canker through tree dissections. Prominent frost rings in the wood and frost damage to buds and shoots corresponded to the dates of dieback initiation and canker events throughout the history of the plantations. Frost rings and frost cankers were reproducible using an artificial freezing technique. Symptomatic frost-damaged buds and elongating shoots were described for Douglas-fir, white spruce [*Picea glauca* (Moench) Voss] and subalpine fir [*Abies lasiocarpa* (Hook.) Nutt.]. The effect of growing season frost was most noticeable on early flushing trees.

Frost and dieback damage was most severe on concave and flat landforms, which are conducive to cold air pooling on nights with strong radiative cooling.

Several pathogens isolated from recently killed stems were identified from fruiting bodies and culture. *Leucocytospora kunzei* (Sacc.) Urban was the pathogen most commonly isolated from the edge of expanding cankers and progressive dieback margins. *Sclerophoma semenospora* Funk was commonly found fruiting on dead stems and leaders killed by frost or by mechanical means.

Cinara pseudotaxifoliae Wilson feeding caused latent cankers on one year old leaders of Douglas-fir, which are thought to be activated by frost.

Boron levels of both healthy and severely affected trees were in the intermediate range, and were not considered to play an important role in frost or pathogen susceptibility for Douglas-fir in the interior.

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CHAPTER 1
INTRODUCTION

In the summer of 1985 and 1986, a number of reports of dieback and canker damage to Douglas-fir [*Pseudotsuga menziesii* (Mirb.) Franco.] appeared in the Cariboo, Prince George and Kamloops forest regions. Surveys by Mussio (1987) and Lewis and Cozens (1987) showed that the problem was most severe in valley bottoms and concave landforms. Damage consisted of various degrees of dieback. A species of *Sclerophoma* was commonly found fruiting on the dead tissues, and hence the problem became known as the *Sclerophoma* dieback of Douglas-fir.

At an early stage of the current study, it was recognized that frost appeared to play a major role in the development of typical dieback. Trees with dieback symptoms almost invariably also showed frost damage in the form of frost-killed buds and shoots, and frost rings. Whereas, nearby trees without dieback damage showed little evidence of frost damage. Frost damaged trees were then colonized by weak parasites, resulting in progressive dieback.

Growing season frosts are recorded in the wood in the form of frost rings. The position of these frost rings in the wood can be related to the time when dieback and canker was initiated. Thus, trees from several geographically, well separated areas were dissected to determine when and how frequently frost damage occurred in the history of symptomatic trees.

Although the initiation and length of the period of bud flush varies from year to year in relation to climate, the flushing date for an individual tree relative to its neighbors is a fixed genetic characteristic. The degree of

damage associated with early flushing trees was compared with that of adjacent late flushing trees in several plantations using about 100 trees in each replication to test the hypothesis that early flushing trees would be more severely damaged.

Since the disease only occurred on certain landforms, two surveys were done to map the distribution of the problem in relation to the topography. This was done to provide a more accurate description of where the problem was located, and what environmental conditions were involved.

Dieback appeared to occur in two major forms. In the less severe form, termed frost dieback, leaders and sometimes upper laterals were killed by late spring or early fall frosts. Recovery involved the formation of new leaders from surviving laterals, which sometimes resulted in forks or crooks. In the more severe form, the original frost damage was followed by progressive dieback involving weakly parasitic fungi. The pathogens isolated from the diseased bark were identified by comparison with cultures grown from spores of fruiting bodies identified from recently killed bark.

These same fungi were also found infecting the bole through frost-caused cankers and aphid-induced cankers. The role of frost in causing these cankers was investigated by simulating growing season frost through direct freezing of bark and by bud removal experiments.

Boron deficiency symptoms appear similar to those of frost dieback. Since boron deficiency is common in some places in the interior of B.C., the role of a boron micronutrient deficiency, acting as a stress factor, was studied through B fertilization, in an effort to alleviate frost and progressive dieback damage.

The hypothesis that progressive dieback occurred on certain landforms conducive to frost pooling was tested through a survey in the Kamloops forest region.

Most of the work described in this thesis was conducted in five Douglas-fir plantations in the Horsefly district, all established after clearcutting and slashburning (Figure 1). Four of these plantations were located in gently rolling terrain about 1, 3, 7 and 13 km east of Gavin Lake all in the Interior Cedar Hemlock biogeoclimatic zone (ICHe2). The first three were planted in 1971, and the last one in 1973. These locations are referred to as Gavin Lake, Edney Creek-1, Edney Creek-2, and Hazeltine Creek respectively. The fifth Douglas-fir stand was located 40 km east of Horsefly on the lower slopes of a major mountain valley in the ICHh1 and planted in 1971. It is referred to as the Horsefly plantation. Three more plantations were located at Summit Lake in the Sub-Boreal Spruce zone (SBSj1), Kenneth Creek (SBSj1), and Shesta Lake (SBSk3) in the Prince George region. They were established in 1971, 1973 and 1963 respectively. In all cases, the seed provenance was uncertain. The seed was collected somewhere in the central interior, probably in the Interior Douglas-fir zone (IDF). Since some observations indicated that natural Douglas-fir was also susceptible to dieback, the effect of provenance was not emphasized in this study.

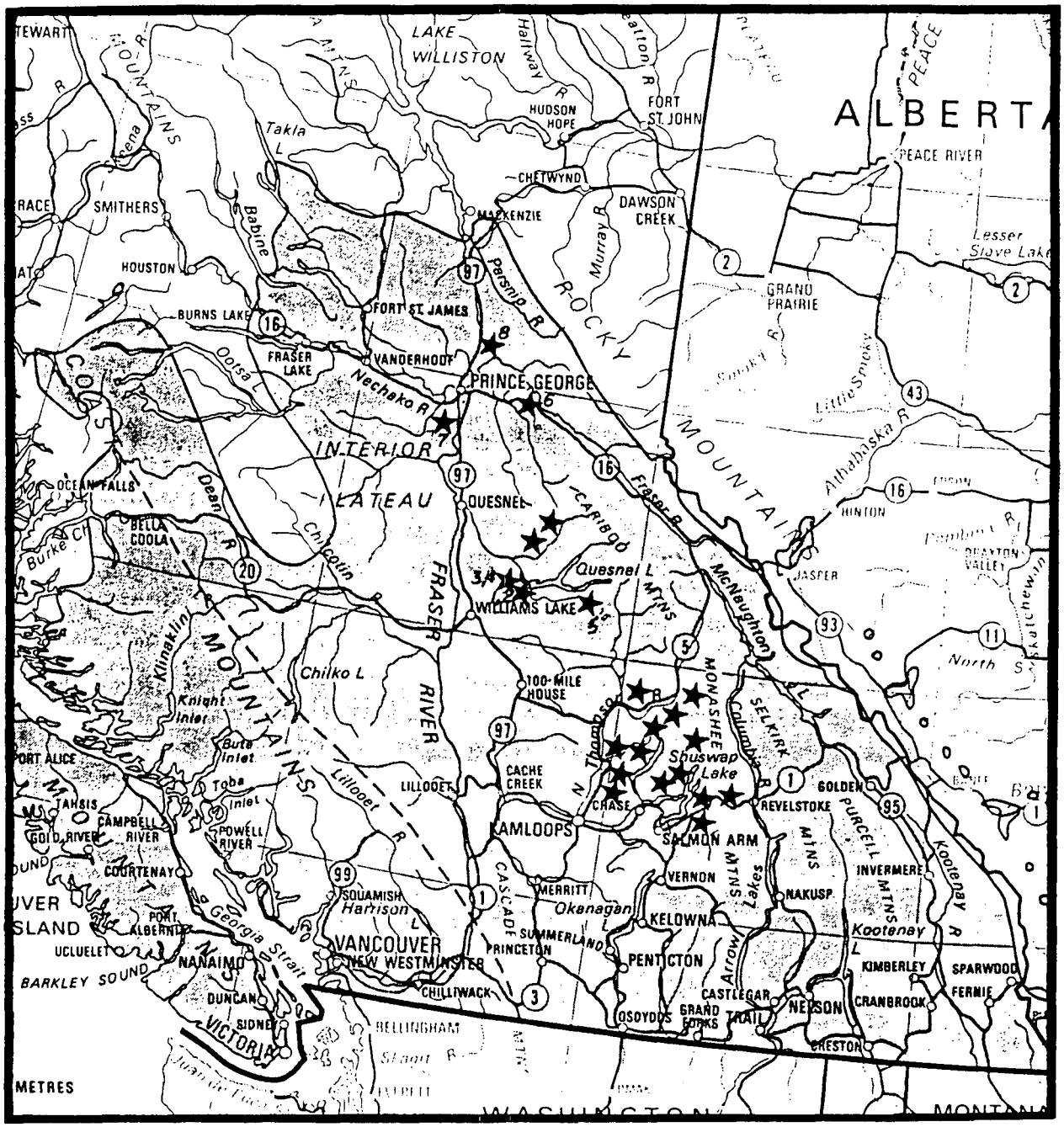


Figure 1. Map of B.C. showing geographical distribution of Douglas-fir (shaded) and some of the plantations that were used in this study (stars). 1 - Gavin Lake, 2 - Edney Creek-1, 3 - Edney Creek-2, 4 - Hazeltine Creek, 5 - Horsefly, 6 - Kenneth Creek, 7 - Shesta Lake, 8 - Summit Lake; plantations surveyed in the Kamloops region are represented by a cluster of stars in the Kamloops vicinity.

CHAPTER 2
LITERATURE REVIEW

In 1985 and 1986 an outbreak of dieback of Douglas-fir saplings in plantations was reported from the Prince George, Cariboo, and Kamloops forest regions. Surveys by Mussio in 1987 and Lewis and Cozens in 1987 demonstrated that the incidence of dieback and branch flagging was as high as 74% in the Cariboo, and 70% in the Prince George forest region respectively.

Canker and dieback of conifers in plantations have been described for well over a century in Europe and for several decades in North America. Day (1928, 31, 45) wrote extensively about dieback and canker of several conifer species, including Douglas-fir, pines, larches and spruces in Britain. His basic tenet was that frost damaged these trees periodically, giving rise to cankers and/or dieback. Trees with developmental cycles that were out of synchronization with the seasonal cycle, could be affected by growing season frost in the spring or in the fall.

One aspect of dieback and canker of conifers that has led to problems among researchers is the misdiagnosis of frost damage. Frost damage to mature and young foliage, elongating shoots, cambium and bark can all be attributed to pathogenic activity, primarily because dead and damaged tissues are rapidly colonized by fungi. Frost damage to elongating shoots is usually correctly identified. Bright red buds, usually killed all on the same night, are a good indicator of frost damage. When other tissues are damaged by frost, misdiagnosis can occur, since the death of the tissue is not always automatic. Day (1928) demonstrated how frost caused the death of cambial tissues of young Douglas-fir. He also showed how larch cankers

could also arise in the same manner. However in the case of larch, frost rings are not very conspicuous, and as a result, his argument didn't convince all skeptics.

A strong point in favor of Day's frost hypothesis was that dieback and cankers occurred at the same time that frost rings appeared in the wood at and adjacent to the injury. The first large scale freezing operation was carried out by Day and Peace in 1934. They froze a total of 3,467 two to five year old potted trees of 10 different species, 529 of which were Douglas-fir, in a large refrigerator chamber at various temperatures. They were able to simulate frost damage for many species, and cause larch to develop wounds around short shoots similar to those that developed naturally.

Manners (1957) treated *L. decidua* in the spring. He froze the bark locally with a stream of cold gas for a period of two hours at a temperature of either -5 or -10°C on either two or three to four year old stems. He was able to produce cankers similar to those described by Day and Peace (1934). He found that, if the canker healed by the end of the growing season, the healing was usually permanent.

Since it was apparent that frost affected trees primarily during the growing season, some attention was given to flushing date. Munch in 1928 refers to an early and a late flushing race of Norway spruce (*Picea abies* Karst). On frosty sites he noted that the early flushing race was usually killed out. In one particular location, as a result of a frost on June 8, 1925, 90% of the early flushing race lost its leading shoot. Only 12% of the late flushing race lost theirs. Day and Peace (1934) described a period of severe night frost in 1927 at Oxford, England lasting from April 27 to May 2, which affected early flushing provenances of Douglas-fir in a provenance

trial at a local nursery. Few other observations have since been made on the relation of flushing date to dieback.

Day and Peace (1946) extensively studied the topography of affected sites as a major factor influencing the climate with respect to frost. In the U.K. he described how the aspect, length and steepness of a slope affect the local climate. Aspect affects chiefly the amount of incoming solar radiation. Thus a southern aspect is warmer, and the trees become frost susceptible earlier here than on a north facing slope. The length and steepness of the slope determine the amount of cold air shed and the rate of drainage respectively.

Flat areas such as plains and plateaus lend themselves to shallow but severe ground frost, since the cold heavy air can not drain away. Flat valley bottoms are similar, except that cold air can drain into them from the surrounding slopes. Hummocked areas act as relatively warm islands among cold pockets. The soil and general ground characteristics such as moisture content and vegetation also affect the formation and drainage of cold air.

Larches, pines, and Douglas-fir all have a considerable history of frost damage and dieback. The connection of frost to dieback and the various pathogens affecting these trees is discussed below.

The Larch Canker, *Lachnellula willkommi* (Hartig) Dennis

Originally the larch canker pathogen *Lachnellula willkommi* [syn. *Trichcoscyphella willkommii* (Hart.) Nannf.] was believed to be a wound parasite. Hartig (1880) was the first to demonstrate this through artificial inoculations. Hiley (1919) showed that infection commonly took place from

a dead branch or short shoot, which the pathogen had previously colonized as a saprophyte. Day (1931) related the occurrence of growing season frost and frost rings to canker formation around short shoots. Langner (1936) also noted that frost rings often coincided with the occurrence of canker. Manners (1957) reported that the anatomy of naturally occurring cankers was very similar to that produced either by artificial inoculation with *Trichcoscyphella willkommii* or by local freezing. Yde-Anderson (1980) asserted that, although larch was frequently damaged by frost in the spring and fall, cankers were not the result of frost damage.

Scleroderris Canker, *Gremmeniella abietina* (Lagerb.) Morelet

Pine and spruce have been affected by *Gremmeniella abietina* (syn *Scleroderris lagerbergii* Gremmen) for over a century in Europe and close to four decades in North America. The list of articles written on this problem is significant although the reports in terms of new discoveries are not. There are only a few different interpretations of the etiology of the disease and the adherents of the two main views are numerous for either side. One group supports the pathogenicity of the fungus, whereas the other focuses more on the environmental predisposition of the host. Dorworth (1971) believed that management foresters were more likely to accept the frost pocket explanation, "frost being a phenomenon that falls more nearly within the realm of visible observation..."

Patton et al. (1984) studied the mode of infection and the early stages of colonization by *Gremmeniella abietina*. They stated that infection occurs through the buds or current season's shoots. Using serial sections and scanning electron microscopy, they described how infection through the stomata of bracts, that subtend the short shoots, in the summer and fall

lead to infection of the bud or shoot the following spring. The fungus remains dormant in the bract until the spring, when it crosses the periderm separating the base of the bract from the stem. Once the fungus is across the periderm, rapid colonization of the stem occurs, and incipient symptoms develop in late February to March.

Pomerleau and Ray (1957) and Duffy and Fraser (1963) both working in Ontario described frost damage and confirmed this through measurements of temperatures on frosty nights. Day (1928, 31, and 45), and Day and Peace (1934 and 1946) also confirmed frost damage with temperature measurements and detailed descriptions of frost pockets and evidence of frost damage in the form external and internal evidence of frost damage to actively growing tissues. Boudru (1947) and Blokhuis (1966) working on Corsican pine (*Pinus nigra* Arnold) in Europe both pointed to frost damage as the cause of dieback.

Unlike the frost pocket explanation, which is backed up through several direct lines of evidence, there is little hard evidence in favor of the other view. Several authors state that the disease symptoms identified as *Gremmeniella abietina* do not require frost pre-conditioning. Their explanation is that constant high humidity, the presence of free water, and an adequate spore source alone are sufficient for disease (Dorworth 1971). Although these conditions are stated as sufficient, no substantial evidence, other than laboratory inoculations is ever given to support this view, or to discredit the frost pocket explanation. On the contrary the marked difference in temperature inside and outside frost pockets on frosty nights is easily demonstrated.

Pomerleau (1971) strongly opposed the view that primary attack by *Gremmeniella abietina* was the cause of dieback and canker. He examined the conditions of plantations in four localities in Quebec. He found a "straight horizontal line of demarcation between living and dead branches of living trees." The height of the line on the surviving trees was greater in depressions where fewer trees survived. Stem cankers were common at the base of the trunk to 30 cm above ground. Through dissection of over 50 trees, he found that a rather extensive surface of the cambium had died suddenly in 1966, 1965 and occasionally in 1964. Frost rings were formed beyond the dead portion, indicating the date of the injury.

Pomerleau (1971) successfully demonstrated that late frosts were responsible for extensive damage and killing of pines. Pomerleau and Ray (1957) recorded minimal air temperatures as low as -8°C at 30 cm above ground during certain nights from June 15 to July 15 in 1955 and 1956. Also, the frequency and the intensity of these temperature drops were more frequent in ground depressions.

Yokota (1975), in an analysis of the climatic data associated with an outbreak of *Gremmeniella abietina* canker of todo-fir [*Abies sachalinensis* (Fr. Schm.) Mast.] in Hokkaido, Northern Japan, attributed the outbreak to the effect of two climatic conditions. Damaging early fall frosts, which would have enabled the establishment of the fungus in frost damaged tissue, followed by a heavy snowfall resulting in an outbreak in the spring of 1970.

Dieback and Canker Pathogens of Douglas-fir

The following pathogens are discussed in relation to dieback and canker of Douglas-fir since they were identified by the author as pathogens present at the expanding canker or progressive dieback margin.

Sclerophoma semenospora and *S. pithyophila* were collected by Lewis and Cozens (1987) and Mussio (1987) in the Prince George and Cariboo forest regions respectively. Although in this study they were not found progressing down the main stem, they were the predominant colonizer of frost killed leaders and branch tips.

Funk (1980) discusses the *Sclerophomas* associated with dieback of Douglas-fir in B.C. He lists three species of *Sclerophoma* found on dying tips of young Douglas-fir. *S. xenomeria* and *S. pithyophila* were implicated in dieback diseases associated with drought. *S. semenospora* was suspected as being pathogenic on stressed trees.

Funk and Shoemaker (1971) referred to *Xenomeris abietis* Barr (teleomorph of *Sclerophoma xenomeria* Funk) as being weakly parasitic, and capable of invading and killing phloem tissues in Douglas-fir and Western Hemlock [*Tsuga heterophylla* (Raf.) Sarg.], but artificial inoculations were unsuccessful in demonstrating its pathogenicity.

Pathogens other than *Sclerophoma* were typically found invading the fresh tissue adjacent to the frost-killed member. These pathogens were isolated and identified by comparison of mycelial growth with that produced from spores obtained from apothecia or pycnidia. The most common pathogen was *Leucocytospora kunzei*. It was found by the author throughout the Kamloops, Cariboo and Prince George forest regions. This organism causes an important and common disease of Colorado Blue Spruce (*Picea pungens* Engelm.), a widely planted landscape ornamental in the United States and Canada. *Cytospora* canker of spruce and other conifers is caused by *Leucostoma kunzei* (Fr.) Munk ex Kern (syn. *Valsa kunzei* Fr.),

which is most commonly seen in its imperfect form, *Leucocytospora kunzei* (Sacc.) Urban (syn. *Cytospora kunzei*) (Proffer and Hart 1988).

Waterman (1955) describes three varieties of *Valsa kunzei*, based on their stromatal variations. *V. kunzei* var *kunzei* was found on Douglas-fir, European larch (*Larix decidua* Mill.), and Eastern hemlock (*Tsuga canadensis*). The other two varieties affected spruce and pine respectively. He reported that on Douglas-fir the conspicuous branch and trunk cankers often have resin flow associated with the cankered areas. This symptom was not very common on Douglas-fir in the interior of B.C.

In North America, *Cytospora* canker was first reported killing Douglas-fir saplings on the Pike National Forest in Colorado by Roeser in 1929. Approximately 2/3 of the trees under 28 cm in diameter were attacked, and some 1.5 % of the trees died annually from 1924 to 1927. Later, Waterman (1955) identified the pathogen as *Cytospora kunzei* var. *kunzei* (Sacc.). During the 1964 outbreak in the same area, up to 30% of the trees in some areas (less than 40 acres in size) were infected, and 10 % of the seedling and sapling size trees had died. Both the 1928 and 1964 outbreaks were attributed to deficient precipitation by Hinds and Stewart (1965).

A report of canker and dieback of Douglas-fir in the Cariboo forest region by Funk et al (1965), indicated that a species of *Valsa* had been found in the dead top, which was killed by stem girdling cankers. The role of the *Valsa* sp. was not investigated since the stem cankers caused by the *Dermea* sp. were considered to be the primary cause of the dieback. Most of the affected trees were vigorous and growing on good sites, and thus the involvement of a climatic predisposing factor was proposed. Early winter

frosts of unusual severity were reported for the region at the time. The *Dermea* sp. belonged to a weakly parasitic genus, which also suggested the predisposal by climatic factors (Funk et al 1965).

Tympanis laricinia (Fckl.) Sacc. was found by the author in several locations in the Cariboo and Prince George forest regions. Groves (1952) regarded the genus *Tympanis* as being of minor importance as pathogens. Funk (1981) mentions *P. menzeisii*, *P. glauca*, *Abies lasiocarpa*, and *Larix* spp. as hosts. Smerlis (1970) also mentions *T. laricinia* as being associated with dieback and cankers of conifers.

Pragmopora pithya (Fr.) Groves causes stem cankers of Douglas-fir. In eastern North America, *P. pithya* is a virulent canker pathogen of conifers (Smerlis 1973). Funk (1975) found *P. pithya* to be the chief fungus associated with cankering around the feeding galleries of lepidopterous larvae (*Laspayresia pseudotsugae* Evans). The problem occurred in a 65 hectare Douglas-fir plantation on a flat valley bottom in the Cowichan Valley. The disease outbreak began in 1971 and then subsided. The rainfall was normal for that period. Inoculations failed to establish the pathogenicity of *P. pithya*. Funk stated that, "the general subsidence of the disease in the later years indicates that the predisposing factors had passed." These same larval feeding galleries and their associated cankered areas were also observed by the author in the Cariboo F.R., where they sometimes developed into girdling stem cankers on frosty sites.

Houston (1969) describes a perennial canker caused by *P. pithya* on Eastern white pine. This organism proved to be aggressively pathogenic. Houston stated that "even the very pathogenic *P. pithya* apparently required a wound infection court. The results of isolation trials indicated

that ant-induced lesions were suitable infection courts for many fungi including *P. pithya*."

Endothiella aggregata Funk was described by Funk in 1983 as a red coelomycete that is associated with diseased bark of various western conifers. Funk stated that, "although pathogenicity tests have not been made, the *Endothiella* is considered to be a secondary invader of killed bark. It appears to be a secondary invader of bark attacked by a primary pathogen and possibly killed before invasion by *E. aggregata*." This interpretation is valid for some of the material examined in this study, however some tops were examined that featured *E. aggregata* as the sole pathogen present at the advancing disease margin.

CHAPTER 3

SYMPTOMS OF FROST DAMAGE ON DOUGLAS-FIR AND ASSOCIATED CONIFERS

External Symptoms of Frost Damage to Buds and Shoots

Examination of frost damaged buds and shoots revealed some interesting facts. Buds and shoots of Douglas-fir and other conifers can be damaged and killed by frost either before they have become frost hardy in the fall (early fall frosts) or after they have dehardened in the spring (late spring frosts). Late spring frosts lead to various degrees of damage. In general, the damage is restricted to the current year's growth; the mature internodes formed the previous year are unaffected. Severe frost leads to death of the flushing buds or elongating shoots. The previous year's internode and its needles may survive such a frost but be unable to regenerate new buds. Unless at least one of the buds has survived the frost, the internode eventually dies.

Early fall frosts often result in the death of exposed buds and either the distal portion or the whole of the current year's internode. Leaders are more often affected than upper laterals. There are no obvious symptoms of the damage immediately following the frost in the fall, but by the following spring, most of the needles are red, and the buds fail to open. In the usual case, one of the laterals or one of the uppermost living buds takes over, and the tree recovers. Some height growth may be lost, and a slight crook may develop. The worst damage results when two or more laterals compete for dominance, resulting in a fork.

In addition to the above, two less common forms of damage occur. In the first of these, the new shoot survives, but both the needles and the shoot fail to elongate normally (Figure 2 A - D). Sometimes these short shoots die later in the summer or the following winter (Figure 2 D). At other times they survive and produce a few buds which flush that same year (Figure 2 B). This symptom is common in Douglas-fir, white spruce [*Picea glauca* (Moench) Voss], and subalpine fir [*Abies lasiocarpa* (Hook.) Nutt.] (Figure 2 A - D). In subalpine fir and white spruce, the buds did not have conspicuous lammas growth.

In the second form, only the base of the new shoot and a few needles survive, while the tip is killed (Figure 2 E, F). The base of the new shoot forms a very short swollen stem segment bearing a few large kinked needles and several lateral buds (Figure 2 F). These new buds flush the next year and produce a dense cluster of branches (Figure 2 E). This type of bud damage appeared to result from the frosts of Jan. 14, 1982; Dec. 14, 1984, and Jan. 30, 1989. On these days a rapid drop in temperature occurred within the first hour of freezing. Temperatures dropped from above 0°C to between -7 and -14°C within an hour. Although this type of bud damage is rare, it does occur with a periodicity that parallels the equally rare occurrences of the frosts mentioned above. Weiser and White (1965) observed that hardened foliage of northern white cedar (*Thuja occidentalis* L.) was killed by a rapid temperature drop, especially when the starting temperature was near the freezing point of water (cited from Alden and Hermann 1971). Several other examples are cited by Alden and Hermann (1971) showing how rapid temperature drop after sunset can cause injury.

Figure 2. Symptoms of frost damage to buds of Douglas-fir, subalpine fir and white spruce.

A. Frost damaged buds of subalpine fir flushing in the spring of 1987, (picture early June, 1987). Four of the five buds failed to elongate properly and have no live needles. One bud on the right shows normal elongation and development. This symptom appears similar to that of a bud that has not undergone proper chilling requirement (pers. comm. Dr. Worrall).

B. Frost damaged elongating shoot of Douglas-fir with dead shoot tip, and lammas growth developing from several buds near the shoot tip that have set and flushed in the same season. The tip of the shoot appears to have been well developed at the time of death. The damage apparently resulted from a spring frost that occurred while the shoot was still very small, yet elongating. Shoots that flush through the end of the bud were observed to have the tips killed by frost, yet the base and some needles survive (photo early June 1987).

C. Frost damaged terminal shoot of white spruce. Note the dead needles of the terminal shoot that developed in 1987. The buds which set in the fall of 1987 flushed normally in the spring of 1988. Frost damage to the terminal shoot appears to have resulted either from the severe spring frosts of 1987 or possibly from a 1986 fall frost (photo May 21, 1988).

D. Frost damage to elongating buds of Douglas-fir. This photo was taken two or three years after the damage occurred (photo June 1987).

E. 17 year old Douglas-fir with rossetting branch habit of the 1985 internode of the main stem and upper laterals. The rossetting condition occurred when the buds that formed in 1984, were damaged at the bud apex, and thus, did not elongate properly in 1985. A 1984 fall frost or possibly a rapid temperature drop on December 14, 1984 would have resulted in the damage. There was no visible tissue at the end of the shoot indicating that the damage occurred while the bud was in a very earrly stage of development (photo June 1987).

F. Terminal bud which developed in 1987 showing frost damage. Note the large kinked needles, and the multiple buds formed in a tight row at the tip of the short swollen shoot. This shoot would have been damaged by a 1986 fall frost or possibly by a 1987 spring frost.

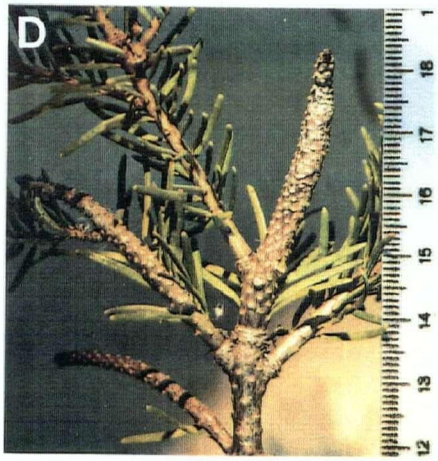
G. Terminal bud killed by 1986 spring frost after it began elongating. The ring of buds seen below the frost killed terminal bud were formed in 1986 and flushed in 1987 spring (photo June 1, 1987).

H. Another tree approximately two weeks later undergoing bud flush. Note three lower auxiliary buds produced normal branches in 1986. Several flushing lateral buds of the three branches produced in 1986 were killed by a 1987 spring frost (photo June 17, 1987).


A


B


C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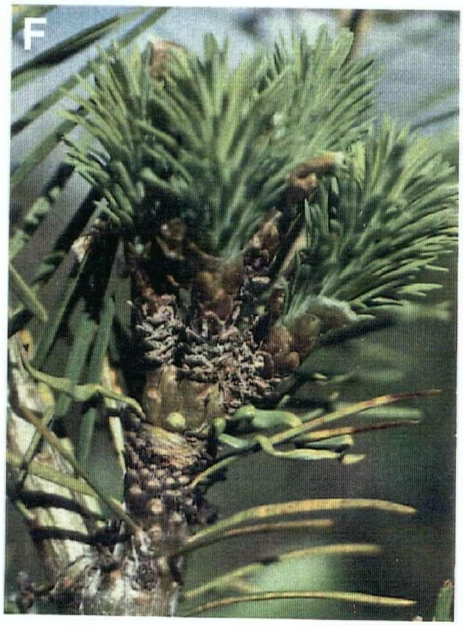

F


G


H

The first type of frost damage, (Figure 2 A-D) appears to occur at an early stage of bud development. The manner in which the bud scales of Douglas-fir open on different trees could be important here. Some Douglas-fir burst their buds through the tip of the bud scale sheath, whereas in other trees, the bud scales slowly separate from each other and the shoot carries the bud scale cap off the bud. In one particular tree, as the shoots began flushing through the tips of the buds, a severe frost (May 16, 1987) appeared to kill the expanding shoots. Several weeks later, the bases of the frost-killed shoots elongated, producing structures similar to those shown in Figure 2 A - D. The green shoots were crowned with a dead tip. Later on in the summer, several buds which formed near the end of the live portion of the shoot began to form lammas growth.

Inadequately winter chilled buds of Douglas-fir also appear very similar to this symptom (pers. comm. Dr. Worrall). It has been proposed that fall frosts could disrupt the bud's normal development of frost hardiness. These buds would then be unable to acquire sufficient chilling to meet the bud burst requirement in the spring. Cannell (1989) discusses this condition, and it's converse; budburst occurring earlier as a result of climatic warming leading to an increased risk to spring frost damage.

The short swollen shoot base and the well developed needles (Figure 2 F) indicate that a different process is involved in the malformation of the second symptom. The lack of frost-killed immature needles at the tip of the shoot and the distribution of the buds, which set and do not flush the same season, indicate that the damage occurs very early in the development of the bud. It appears that the stubby shoot is composed of a few basal scale segments which survive, elongate and set several buds in a row at the tip of

the short swollen axis (Figure 2 F). These buds appear to develop in leaf axils from bud primordia, which normally remain undeveloped. In a few Douglas-fir saplings, the terminal bud and several axiliary buds of every branch in the upper crown and the leader showed this symptom (Figure 2 E) as a result of a December 1984 frost.

Examination of 1989 winter damaged buds of Douglas-fir, spruce spp. and subalpine fir revealed a similar pattern of shoot development. The bud apices were killed leaving only a few preformed needle primordia to develop from the shoot base. From this live shoot base, shoots developed from leaf axils in a ring around the dead bud apice forming a rosette. In 1989 this symptom occurred on many buds of trees growing above the snow-line in the Prince George, Cariboo and Kamloops forest regions. In some cases the new shoots developed into buds immediately, and did not flush the same year.

Late spring frost-killed white spruce buds can develop a rossetting condition which appears somewhat similar to the previously described symptom in Douglas-fir. It can be easily distinguished from fall frost damage by the stage of development the shoot is in when it dies (Figure 2 G). In white spruce, a ring of buds developed beneath frost-killed flushing buds (Figure 2 G). These buds flushed the following year (Figure 2 H). The ring of buds appears to originate from the base of the current year's frost-killed shoot or bud.

Day and Peace (1934) made observations on a rossetting condition that resulted from spring frost damage to Japanese Larch [*Larix leptolepsis* (Sieb. and Zucc.) Gordon]. Experimental freezing conditions resulted in the formation of several buds immediately below a frost-killed bud.

A variation of this rossetting shoot formation occurring after frost damage was found on several spruce in a Horsefly plantation. The terminal end of the leader died apparently from fall frost damage. No new leader was formed since none of the numerous shoots which formed achieved dominance. The production of rossetting shoots appeared to be an annual phenomenon, judging by the different ages of the shoots that had formed and were still being formed each year from the same position. The overall effect looked something similar to a palm tree.

The degree of damage resulting from late spring and early fall frosts appears to depend not only on the stage of flushing, and the degree of freezing, but also on the rate of heating during the early morning. Alden and Hermann (1971) discuss the rate of thaw in relation to cold injury and desiccation. Parts of the crown that are exposed to the early morning sun while they are still frozen tend to be more severely damaged than sheltered parts. It seems as if a slow rise in temperature allows at least the less severely frosted tissues to recover. At several sites in the Cariboo, the damage to shoots on the southeast side of the crown was found to be facing the early morning sun of middle October. This would seem to indicate that frost damage could occur in the fall if the morning sun warmed tissues too rapidly. A gardener in the Gavin Lake area reported frost damage only to those cabbages that received early morning sun during a frost and not to those protected from the sun. Day (1946) mentions the importance of the rate of thaw with respect to severity of damage. Cannell (1985b) mentions that hardening can be reversed following warming trends in the fall. Alden and Herman (1971) state that, "the maintainance of the hardened condition appears to depend on the same environmental conditions that inhibit growth, induce dormancy, and develop cold resistance".

Internal Symptoms of Frost Damage to the Wood

Frosts can damage the cambium after it has become active. Mayr (1893) was the first to recognize and describe an abnormal zone of tissue resulting from freezing of the actively dividing cambium. Day and Peace (1934) divided the year into four periods and described the abnormal rings that resulted from frost during these periods. The periods were early spring, late spring and summer, autumn, and winter. In the early spring period, the damage to the wood in cross-section usually consists of one or two rows of parenchyma formed immediately adjacent to the fall wood of the previous annual ring. During the late spring and summer period, some wood is always laid down before the frost ring is formed (Figure 3 D). The earliest wood formed is usually mature and unaffected by the frost. They divided the frost ring that forms in this period into two parts. An inner zone of atypical cells which were not fully differentiated when killed by the frost, and a zone of atypical cells that are produced by the damaged cambium after the frost. This distinction between cells produced before and after a frost is very obvious for severe frosts (Figure 3 D).

The inner part of the frost ring consists of a zone of regularly shaped lignified cells, surrounded by a zone of collapsed cells, which would have been produced just prior to the frost. Often there is a visible directionality to the collapse, indicating torque rather than just plain compression. The walls of the inner part of the frost ring may or may not be lignified. This collapsed zone may not be very obvious in the case of a mild frost.

The cells formed after the frost constitute the outer part of the frost ring. Outside the zone of collapsed cells is the isodiametric parenchyma. The shape of these cells is very irregular. The degree of irregularity

Figure 3. Douglas-fir dieback symptoms, associated frost rings and sections through frost cankers of a 17 year old stem.

A. Severely affected 15 year old Douglas-fir with frost damaged foliage, terminal and lateral shoots, and buds. This tree did not grow out of the frost pool in time.

B. Healthy 15 year old Douglas-fir that shows severe lower crown damage only, whereas the upper crown is damage free. This tree is only meters away from the tree in A.

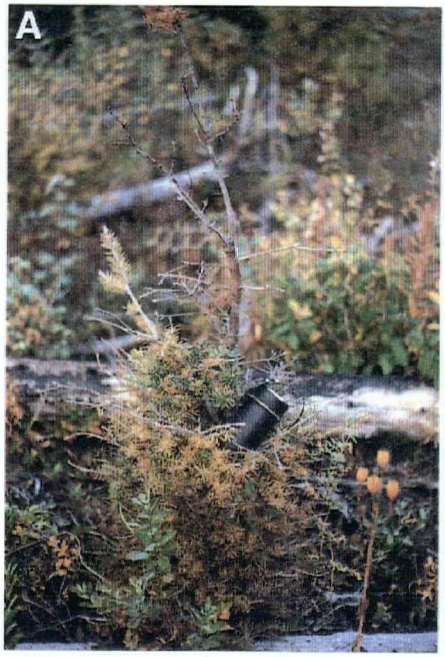
C. Douglas-fir showing damage to 1981 leader as a result of a 1982 spring frost.

D. Scanning electron micrograph (SEM) of a 1986 late spring frost (June). Note the amount of wood that has formed prior to the frost and the three zones of a frost ring described by Day.

E. SEM of two 1983 fall frosts, one being directly at the margin of the fall and spring wood, the other being well before the end of wood production.

F. Section through a frost canker showing the history of frost damage in the form of frost rings starting from 1975 to 1987. Note that prominent frost rings are present in most of the years.

G. Entire section of F.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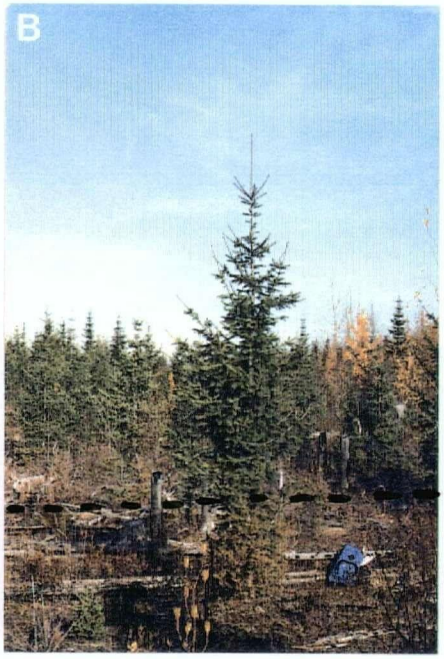
B


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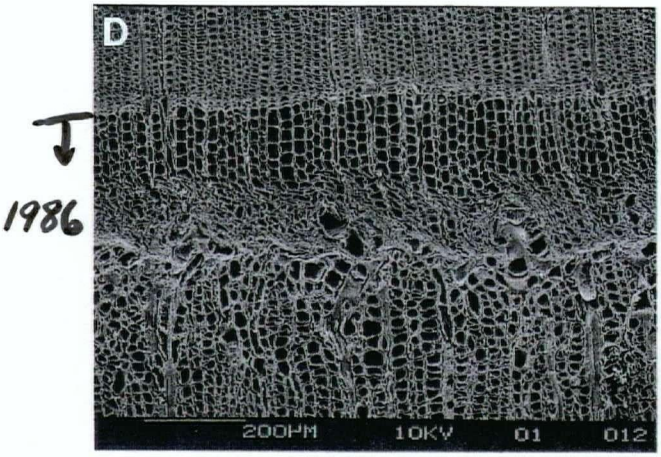
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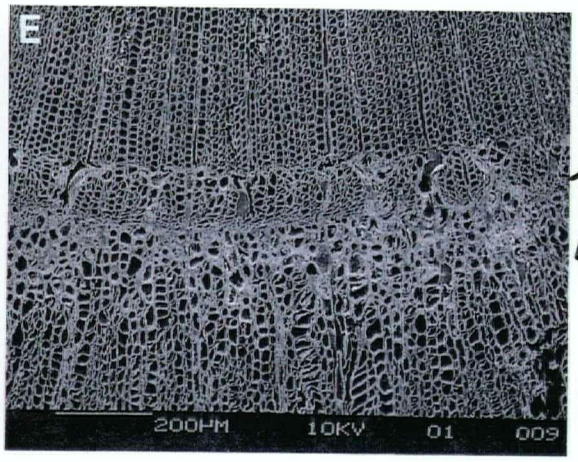
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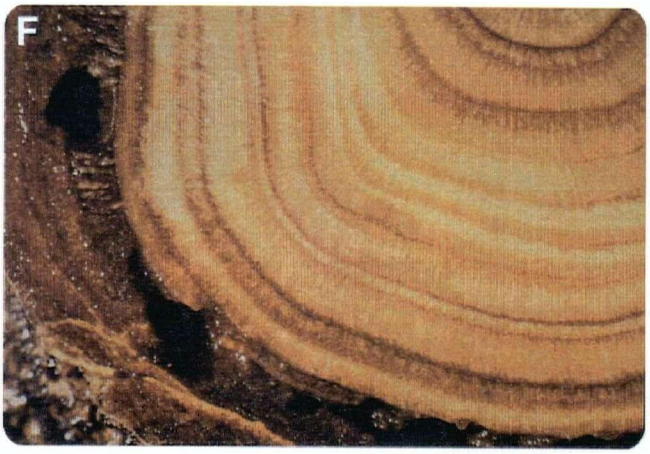
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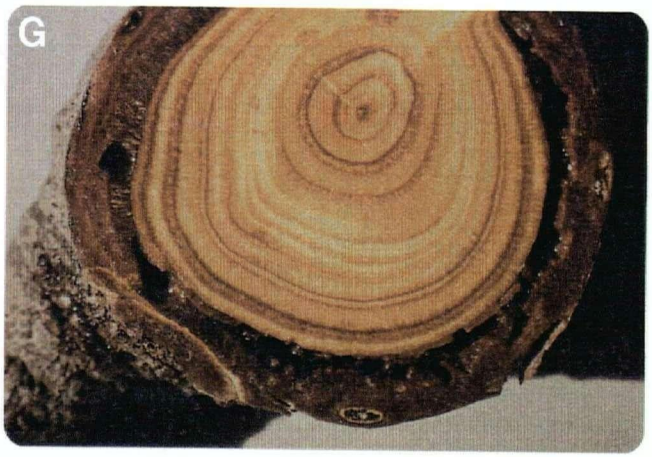

G

appears to be a function of the severity of the frost. The last zone of the outer part consists of the cells which form the gradation between parenchyma and normal wood cells during the recovery process. The tracheids in the outer part of the frost ring are usually irregularly shaped, but are still recognizable. The ray cells are kinked in the collapsed zone, but then become very enlarged as they enter the outer part of the frost ring (Figure 3 D). The rays resume their normal diameter as the newly formed tracheids become more regular in size and shape.

Often the wood tissue can be radially rent along the rays at the outer margin of the collapsed zone of the inner part of the frost ring. Frost rings that form very late in the developmental cycle as a result of late fall frosts, often complete their development in the following spring. Those which occur in the early fall typically are characterized by radial rents in the tissue along the rays (Figure 3 E). Sometimes these rents penetrate the wood already formed prior to the frost.

Sorauer's (1909) mechanical hypothesis of the frost ring development is based upon the unequal shrinking characteristic of the wood and bark during freezing. He believed that the cambium and phloem stretched over the wood during freezing. Upon thawing, the cambium and phloem swell again, but remain somewhat oversized, since they are not very elastic. Day and Peace (1934) refer to wrinkled bark as an example of this. Examples of "wrinkled" bark resulting from 1987 spring frost were found in this study also.

Artificial induction of frost rings

In order to confirm the nature and origin of the frost rings several Douglas-fir and other conifers were experimentally frozen overnight. By

simulating environmental conditions similar to radiation frost, frost dieback similar to that found in the field should result. Growing stems and leaders of several 17-year old Douglas-fir were subjected to experimentally induced freezing temperatures in order to simulate frost dieback and stem canker formation. Heavy duty ziplock freezer bags, filled with ice cubes or crushed ice and anti-freeze (to give a temperature of between -8 and -12°C), were either strapped to a bole (Figure 4 C), or hung by a support around a leader (Figure 4 A) overnight and then removed in the morning. The trees were observed at regular intervals until they were harvested at the end of either the first or second growing season.

The overnight freezing of tips caused the new shoots to yellow by morning (Figure 4 A) and resulted in death of the tip several days later. On several stems, the bark appeared sunken approximately two weeks after the freezing. One year later, a superficial layer of bark peeled away from the stem. Apparently, a layer of new bark had formed beneath the experimentally frost-killed bark. The dead bark was delineated by a necrophylactic periderm (Figure 4 D). Both dead leaders and the bark which peeled off the boles were invaded by a *Sclerophoma* sp. The frost rings which formed after experimental freezing (Figure 5 A-C) closely resembled those formed naturally (Figure 5 D). The characteristic enlarged and misformed ray cells, as well as distorted and weak tracheids were common. The bark tissues present at the time of freezing were eventually isolated by a periderm, but not until the cambium had recovered and produced new functional phloem. In some instances, the bark and the cambium were killed in localized places, resulting in the beginning of a

Figure 4. Simulation of natural frost events that produce frost dieback of leaders and frost cankers to the bole.

A. Morning after experimental freezing of a leader (photo June 4, 1987).

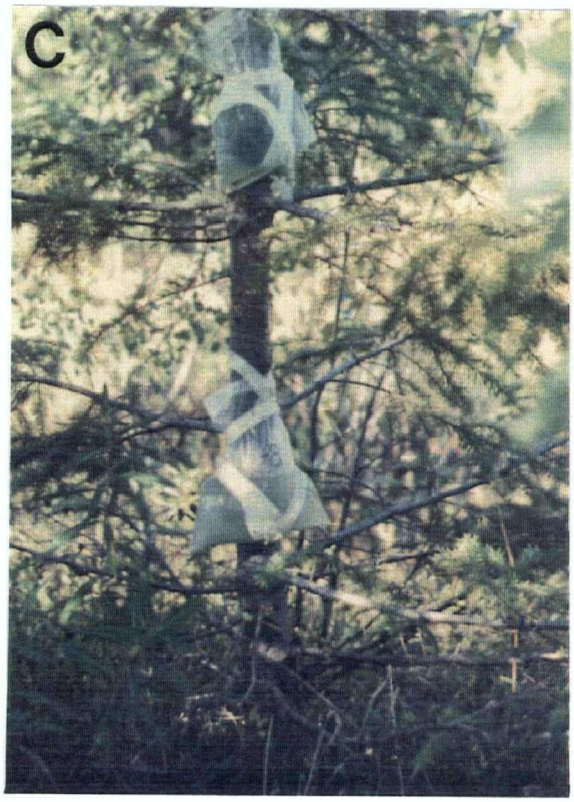
B. Four months later (photo October 10, 1987), immature fruiting bodies of *Sclerophoma* are found on the dead leader.

C. Freezing set-up on bole of 17 year old Douglas-fir (photo June 11, 1987).

D. Dead bark peeling off bole at site of freezing one year later (photo Oct. 8, 1988).


A


B


C


D
CENT
1988
CANADA

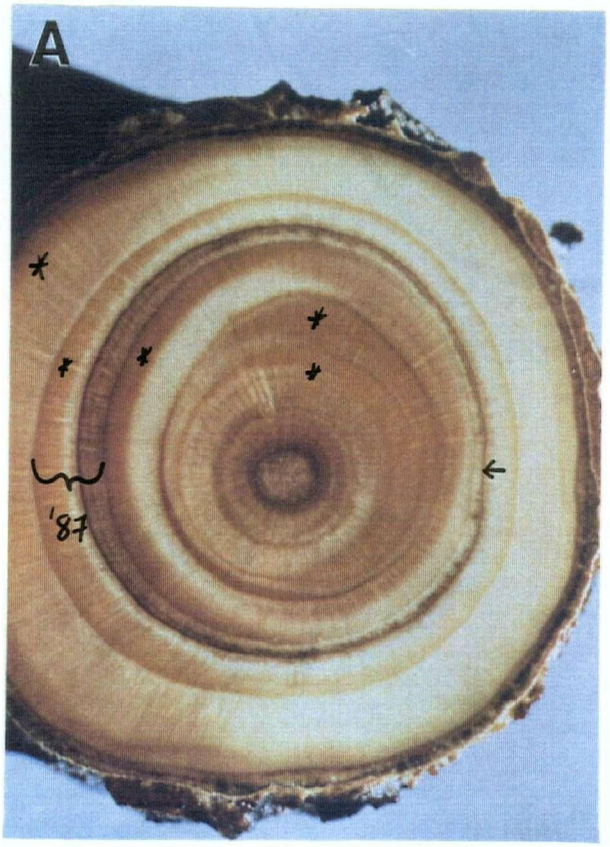
Figure 5. Naturally and artificially induced frost rings in early wood.

A. Frost ring (arrow) in 1987 spring wood (1987 annual ring indicated by the bracket). Note reaction wood in the left side of the section produced after the 1985 growing season. This tree was straightening itself upright after being knocked down in 1983. This tree was treated on June 11, 1987, and harvested on October 9, 1988. Note the irregular bands of reaction wood, marked with a star.

B. Same tree frozen at a lower position on the bole. Note the frost ring (arrow) is a semi-circle here, since the freezing treatment was only applied to one side of the stem (treated June 11, 1987, harvested Oct. 9, 1988).

C. Close-up of frost ring in A, showing the frost ring (dark line marked by an arrow) formed after the freezing.

D. Natural frost ring produced in the spring of 1985 (arrow). Note the strong similarity between artificial frost rings and a natural one. There is also a weak frost ring in the light-colored 1986 spring wood. 1985 annual ring indicated by a bracket.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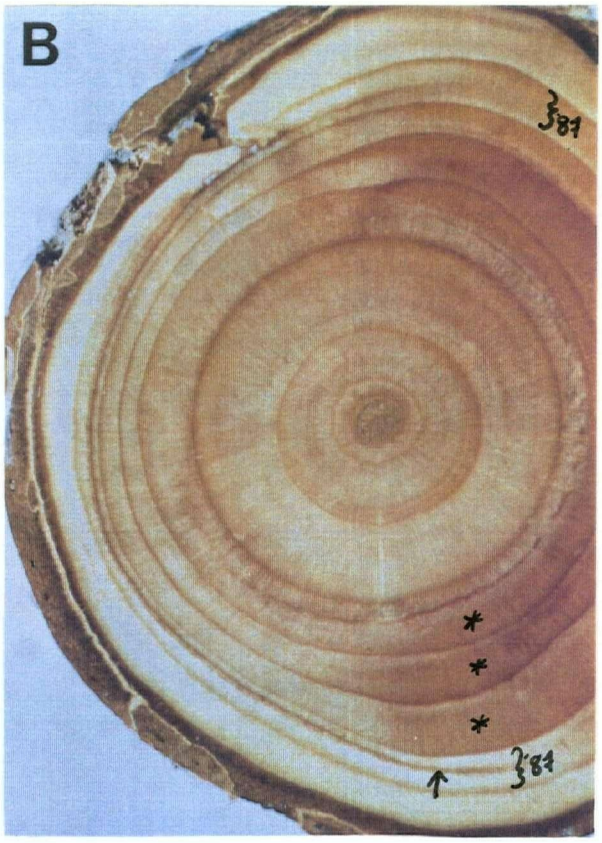
'87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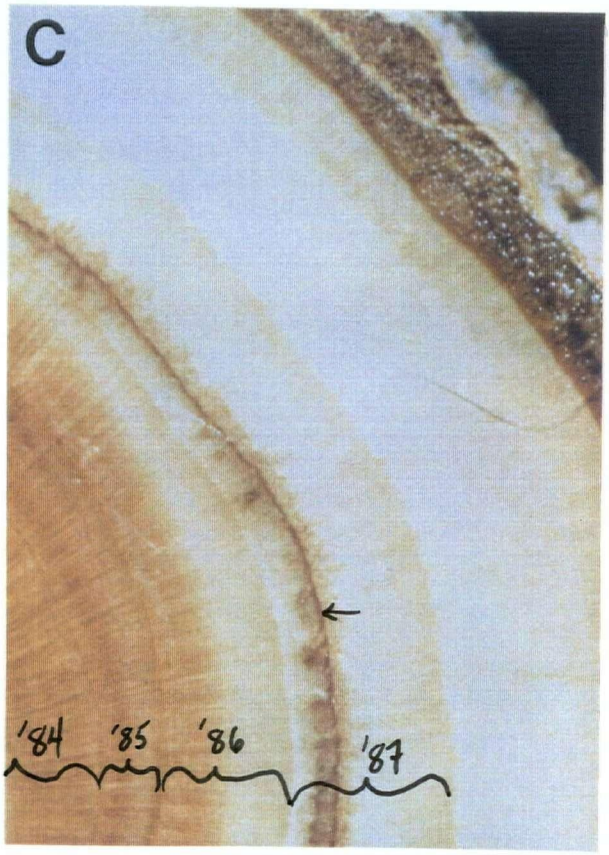
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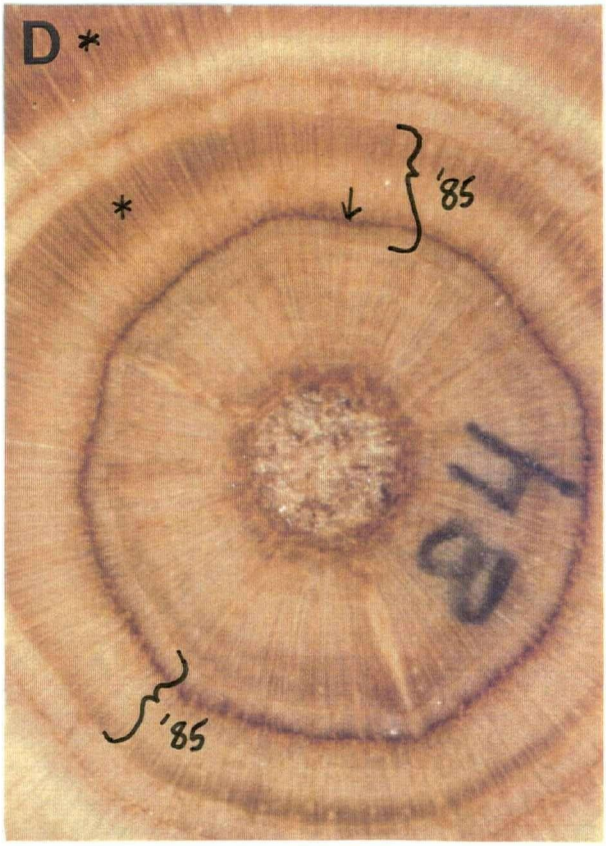
'87


D *
'85
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48

typical perennial canker (Figure 5 B, upper left). However, rapid callusing usually healed the cambium, making it continuous again. A necrophylactic periderm was always formed directly beneath the dead bark, and a new layer of bark was formed from the cambium. The following year (1988), a second necrophylactic periderm was formed in some discrete areas to isolate progressive pathogenic movement (Figure 5 B, upper left).

The frost rings that formed as a result of artificial freezing were similar to natural frost rings found in Douglas-fir and other species of conifers and deciduous trees. These artificially produced frost rings had very distinct inner and outer parts as described above. The wood formed prior to the frost became compressed, and the wood that formed afterwards was composed of weak parenchyma. The cambium produced healthy wood cells sometime later in the season.

The anatomical damage was always greatest wherever the bark was coldest as a result of freezing. At the edge of the area where the ice-pack was positioned, the gradient of damage was very noticeable even to the naked eye. This was most noticeable when only one side of a stem was frozen. The frost rings were most pronounced at the bottom and center of the ziplock bag where the temperature was lowest. This gradient of damage was similar to that produced naturally in a tree where the bark is thermally protected by branches in the middle and lower crown. In a natural frost event, damage occurs primarily to those tissues that are exposed directly to the cold night sky, or to the cold air layer that settles near the ground. Frost rings may form in the upper bole and at the ground line, while the mid-bole often remains unaffected.

In cross-sections of wood formed after both natural and artificial frosts, the rows of tracheids appear to be slightly curved instead of straight. This appears to be a result of reduced bark pressure, which does not force the cells to grow radially. Once the pressure resumes, the rows of tracheids and the rays become radially oriented as in normal wood. Often in experimental freezings, the freezing, and thus the damage, occurred on one side of the stem only. In such cases, the undamaged side of the tree produced wood faster, which resulted in a torsional shift of the bark and cambium on one side of the stem. The unequal tension resulting from faster growth of the undamaged side probably accounts for the tangential alignment of the rows of tracheids. In most cases the wounds caused by freezing healed by the end of the growing season.

Day and Peace (1934) and Manners (1957) successfully created frost cankers and dieback. Often when cankers formed and the tree survived, the cankers healed by the end of the growing season. Yde-Anderson (1980) took this to mean that these attempts to create cankers were unsuccessful, since in nature, cankers became perennial. However, in both of the above mentioned author's experiments and in this study, the trees were protected from subsequent frost events. Naturally formed cankers often show evidence of having experienced numerous frosts that occur after the original damaging frost event.

The expansion of bark wounds initially caused by frost requires either subsequent damaging frost events or pathogenic invasion. In the absence of these, callusing in young trees is often rapid. Temperature measurements in some adjacent areas showed that there were as many as 30 local frosts in the summer of 1988 in some frost pools (pers. comm. Ordell Steen). This

would suggest that the initial damage often is followed by many subsequent frosts, which would result in a more or less continuous stress to the tree.

Description of symptomatic trees

Dieback is most easily understood when it is viewed in the chronological fashion in which it develops. Two major forms can be distinguished. The first form results from the killing of leaders and upper laterals on a periodic basis and is non-progressive. Frost "prunes" leaders and laterals, and the tree responds by allowing new leaders to develop out of pre-existing laterals or buds. This can result in minor crooks, forks or multiple tops. If the frost events are frequent and damaging enough, the tree can easily become a bush through "frost pruning".

The second form of dieback is similar to the first, except that the frost-killed member becomes an entry court for weakly parasitic canker fungi that can progressively kill healthy stem tissues. In very frosty sites, trees that had progressive dieback originating from both leaders and branches were common. The dieback caused by these weakly parasitic fungi appears to be facilitated by repeated frost stress. Dieback of the upper crown, leaves the top of the live portion of the stem thermally unprotected, and subsequent frosts further stress the stem at the dieback margin.

At this point, it is important to introduce the damage symptoms of other trees that grow naturally on these sites. Western red cedar (*Thuja plicata* Donn.) was severely affected on frosty sites. The foliage appeared burnt, and frost rings were present in the wood. In some severely damaged trees, only the foliage that was sheltered by logs or dead overhead branches survived. Western hemlock [*Tsuga heterophylla* (Raf.) Sarg.] when present in frost pockets, was also frost damaged. Frost-damaged subalpine fir

closely resembled symptomatic Douglas-firs. Flushing buds were killed by frost, resulting in crooks and forks in many trees. Trees exhibiting progressive dieback as a result of frost and/or pathogens were not as common as in Douglas-fir. Planted and naturally regenerated spruce were susceptible to frost damage, but cases of frost dieback were infrequent. Dieback to the leader was found in a few trees of a Horsefly plantation, but very few cases of progressive dieback resulted. Lodgepole pine (*Pinus contorta* Dougl.) was apparently unaffected by frost, and no evidence of dieback was ever found. Some frost rings were present in pine, although only for those years where very severe frosts occurred which caused dieback in other conifers. The frost rings were very difficult to detect due to the very minor effect frost apparently has on lodgepole pine.

CHAPTER 4

THE FLUSHING DATE AND SEVERITY OF DIEBACK RELATIONSHIP

INTRODUCTION

Variation in flushing date between trees provided an important line of evidence indicating that frost was a major contributor to the development of progressive dieback. If late spring frosts result in frost dieback, then early flushing trees should be more frequently damaged by late spring frosts than late flushing trees. Since the range of flushing dates is quite large for interior Douglas-fir, one would expect to find a definite trend towards less dieback on late flushing trees than on early flushing trees.

METHODS

Three representative Douglas-fir plantations (Edney Creek-2, Hazeltine Creek, and Horsefly) were chosen, and areas of dieback were delineated within these. A plot consisting of about 100 trees was located within each of these dieback areas. All the living Douglas-fir in the plot were numbered and inspected at 2 or 3 day intervals, starting May 29 and ending when all trees were fully flushed on June 19. At each observation time, a separate rating was assigned to the top, middle and base of each tree. The tree rating was the average of these three observations. Ten stages of flushing were recognized. These started with 1, a fully dormant tight bud, through various degrees of swelling to 8, a bud with some green foliage showing between scales, 9, a bud in which the new foliage was protruding from the tip, and 10, a fully flushed bud with the new shoot in

active elongation. Several trees were at stage 5-8 or more at the time of first observation.

In October the trees were again inspected and rated according to the severity of dieback. The height of each tree was also measured at this time. Eleven damage classes were recognized, ranging from 1-healthy, to 11-severe dieback almost certain to lead to early death of the tree (Table 1). For classes 8-11, damage was judged to be so severe that trees in these classes would not make crop trees. Less severely damaged trees showed signs of recovery and will probably become crop trees although they have lost some height growth.

The data were analyzed in two ways. First, the trees at each location were divided into six groups, depending on the severity of dieback symptoms. The average date that each of these groups reached flushing stages 7 to 10 was then determined. Secondly, the trees in each plot were divided into groups, depending on the date at which stage 7 in flushing was reached. The average dieback damage rating for each group was then calculated. Correlation between the dates that certain flushing stages were reached and average damage rating, and between average flushing date for each damage class and severity of damage (class number) was then used to test the hypothesis that early flushing trees were more frequently damaged.

Table 1. Douglas-fir dieback damage rating classes.

Rating[1]	Description
1	Healthy.
2	Minor frost damage on branch tips; leader unaffected.
3	Minor forks or crooks resulting from frost kill of leaders or buds.
4	Forks resulting from frost kill.
5	Minor dieback that is not progressing any longer.
6	Forks plus minor dieback (4 + 5).
7	Frost damage to leaders and upper laterals in more than one year.
8	Progressive and active dieback involving canker fungi.
9	Severe recurrent frost damage and dieback, resulting in stunting.
10	Low stem cankers leading to branch lagging and girdling.
11	Severe dieback almost certain to lead to early death of the tree.

1 Rating classes 1-7 are considered to be potential crop trees, although they may sustain minor damage; rating classes 8-11 represent trees that will die or be severely stunted as a result of the damage observed.

RESULTS AND DISCUSSION

Table 2 shows the average date at which trees in six damage classes reached flushing stages 7 to 10. Lightly damaged trees flushed later than severely damaged trees at the Edney Creek-2, and Hazeltine Creek plantations. The correlation between date of flushing and degree of damage was significant ($P=0.05$) for flushing stages 7, 8 and 9 at Edney Creek-2: $r=$ -.8429, -.9197, -.8572; $n=6$, $df=4$ $r_{0.05}$ = .8114, and for flushing stages 8, 9, and 10 at Hazeltine Creek: $r=$ -.9488, -.9644, -.8333; $n=6$, $df=4$ $r_{0.05}$ = .8114. Dieback damage results in reduced average tree height: severely damaged trees were one half to one third as tall as healthy trees, $r=$ -.9724, -.8981 for the two sites respectively.

Table 3 groups trees by their date of flushing (date at which stage 7 was reached) and relates the amount of damage to flushing date. Again the average damage is seen to be less severe on later flushing trees at the two Gavin Lake plots. In this table the relationship between flushing date and height growth is not significant. Damage in the Horsefly plantation did not seem to be related to flushing date (Tables 2 and 3). The major frost damage in this plantation in recent years was associated with early fall frosts rather than spring frosts. Variation in damage by fall frosts is not related to flushing date.

Local residents in Horsefly also reported a severe frost in mid-June 1985 following an unusually warm spring, and evidence of such a frost exist in the 1985 annual ring of trees examined from the Horsefly site. Virtually all trees would have been in active growth by that time, and hence the damage would have been equally severe on early and late flushing trees.

Table 2. The relationship between dieback damage, flushing date[1] and tree height in three Douglas-fir plantations near Williams Lake, B.C. Damage was related to flushing date at plantations Edney Creek-2 and Hazeltine Creek, but not at the Horsefly plantation.

Dieback damage class[2]	Average date[1] of reaching flushing stage[3] 7-10				height	number of trees
	7	8	9	10	(m)	
location: Hazeltine Creek						
1- 2	2.81	4.02	6.44	7.65	3.46	48
3- 4	2.00	2.67	4.67	6.83	2.58	6
5- 6	1.24	2.29	5.29	6.71	2.72	17
7- 8	1.63	2.68	5.53	7.68	1.81	19
9-10	1.50	1.70	3.35	3.95	1.55	20
>10	1.00	1.00	2.33	6.67	.97	3
location: Horsefly						
1- 2	5.81	5.95	7.52	9.52	4.31	21
3- 4	5.47	5.87	7.20	8.93	3.11	15
5- 6	5.00	6.00	8.40	10.20	2.88	5
7- 8	5.67	6.33	9.67	10.33	2.67	3
9-10	5.97	6.32	7.16	8.97	1.61	37
>10	5.82	6.18	9.00	10.09	1.06	11
location: Edney Creek-2						
1- 2	4.81	9.65	12.27	10.00	4.00	26
3- 4	6.50	7.50	12.00	13.50	2.50	2
5- 6	5.83	7.83	10.92	11.33	3.11	12
7- 8	1.80	4.20	8.20	9.20	1.84	5
9-10	1.90	2.10	4.80	5.90	.99	10
>10	2.42	2.58	4.17	4.50	1.36	12

1 Dates: 1=May 30, 1987; 3-21=June 1-19, 1987.
2 Damage classes: 1-none, 11-severe (from Table 1).
3 Flushing stages: 7-swollen bud; 8-first green showing; 9-buds clearly broken; 10-fully flushed with elongating shoot.

Table 3. The effect of flushing date (the date at which trees reached flushing stage 7) on dieback damage and height of trees in Douglas-fir plantations near Williams Lake B.C. Damage was related to flushing date at Edney Creek-2, and Hazeltine Creek but not at the Horsefly plantation.

date (1987)	number of trees	damage rating[1]	average height (m)
location: Hazeltine Creek			
May 30	71	4.86	2.29
June 1	3	2.33	2.47
" 3	15	3.33	3.19
" 5	21	2.76	3.14
" 8	1	1.00	1.70
>" 9	2	1.00	3.35
location: Horsefly			
June 3	68	5.75	2.43
" 5	15	5.20	2.88
" 8	2	3.00	4.50
>" 9	7	6.57	1.93
location: Edney Creek-2			
May 30	25	6.88	1.74
June 1	7	4.57	2.31
" 3	7	4.86	3.43
" 5	11	3.00	3.32
" 8	12	3.33	3.71
>" 9	4	3.50	2.97

[1] Dieback damage rated on a scale from 1-none to 11-severe (from Table 1)

CONCLUSIONS

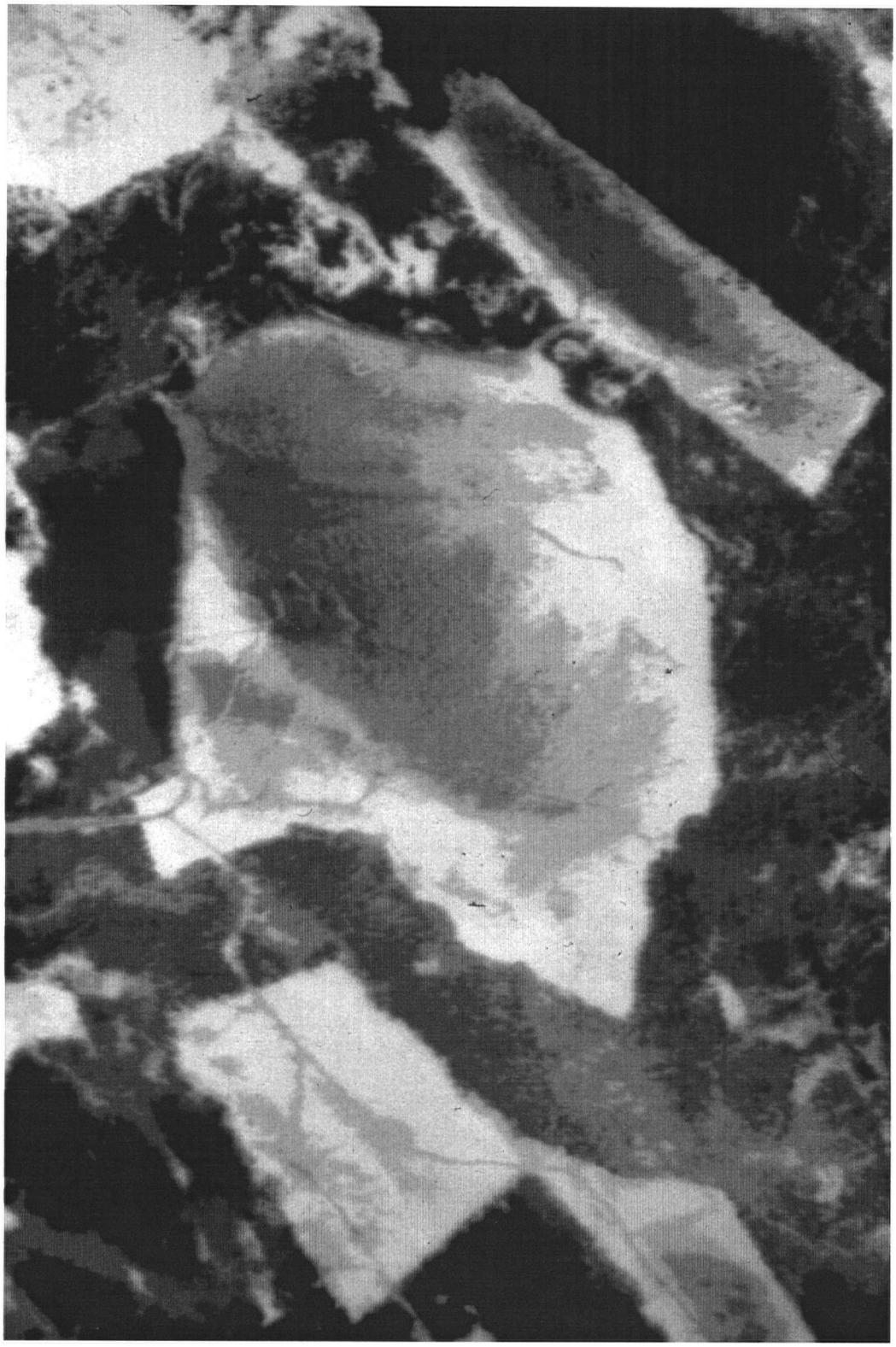
In summary, it appears that early flushing trees are more likely to develop dieback symptoms. This is interpreted as evidence that late spring frosts play a role in the development of the typical symptom complex. This is not a new discovery, but rather only a confirmation of what Munch (1928) and Day and Peace (1934) noted over 50 years ago.

CHAPTER 5
SPATIAL DISTRIBUTION OF DIEBACK

INTRODUCTION

Weather records at many weather stations in the high elevation areas of the Interior Cedar Hemlock (ICH) biogeoclimatic zone, such as Camille Lake, reveal that frost is common in all months of the year. At the Alex Fraser Research Forest, this was also the case in 1987. During each of the months of May, June, July and August, at least one major frost occurred near Gavin Lake. The daily maximum and minimum temperatures often differed by as much as 20°C. The lowest growing season nighttime temperatures occurred on calm clear nights with strong radiative cooling, resulting in temperature inversions and cold air drainage into frost hollows. In gently undulating or mountainous terrain, convex and midslope areas shed cool air, and thus do not get much colder than the general air mass. The cool air collects in valley bottoms, where it fills small depressions and then flows down valleys. Surface temperatures in such valley bottoms may drop many degrees below that of the general air mass, and frost damage is often severe in such areas. Figure 6 is an infra-red aerial thermograph of an area near the study sites described in this thesis. These normal patterns of growing season frosts were observed in all our study areas. Frost pockets were most obvious just before sunrise. Trees and shrubs were covered by frost in clearly delineated low-lying areas. Upper and midslope positions were relatively warm and the trees were generally free of damage. Such a distribution of frost showed that, whereas critical freezing temperatures were often reached in low-lying areas, upper and midslope areas escaped the worst effects of growing season frosts.

Figure 6. Infra-red aerial thermograph showing the variation in ground temperature. The cutblocks are considerably colder than the canopy of the mature forest. The different shades of grey represent 2°C thermal bands; dark grey represents the warmest thermal band with a 6°C maximum temperature. In the center of the photo, a large cutblock shows the coldest air pooling near the cutblock borders. The elevated center of the cutblock is warmer since the cold air formed here drains away. There are six shades of grey in the cutblock. This photo was made available from FRDA project 3.65 (Frost prone sites on the Interior Plateau; recognition and management) courtesy of Ordell Steen.



METHODS

An earlier survey for Douglas-fir dieback (Mussio, 1987) suggested that valley bottom plantations were more susceptible than those located on upper slope positions. This was to be expected if frost played a major role in symptom development. If so, then progressive dieback should be most severe in frost hollows, or almost entirely restricted to such landforms.

To demonstrate that phenomenon, two maps of typical frost pockets, were prepared showing the incidence of damage as related to microtopography. The procedure was as follows. Two typical frost pockets were located within a Douglas-fir plantation (Hazeltine Creek). Two rectangular blocks, 2.9 and 2.4 ha respectively, were then located to contain both the frost pockets and the surrounding slopes and gullies. Next a 10 by 10 m grid was laid out over each of the blocks. Each tree in each of the 100 m^2 grid squares was then rated on a scale of one to eleven (Table 1), and the proportion of trees in each grid square free of major frost or progressive dieback damage (rating <7, Table 1) was determined. More than 5000 trees were assessed in this manner. A 1 m contour map of each of the blocks was prepared.

RESULTS

Figures 7 and 8 illustrate the results. These figures demonstrate clearly that low-lying areas and gullies leading towards them are high risk areas in which at least half the trees are damaged. The center of the low-lying areas were not planted as indicated in Figure 7 and 8. In Figure 7 a

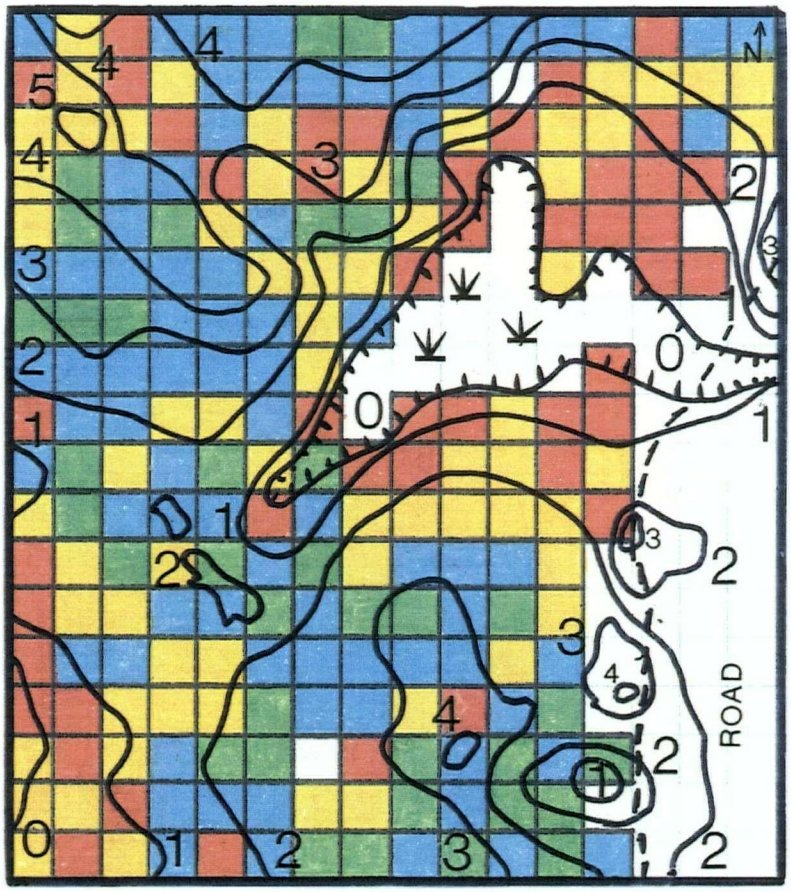


Figure 7. The distribution of dieback damage in frost hollows. Healthy trees (damage class 1-6) as a percentage of all planted trees is colour coded. Green...> 75%; blue...51 - 75 %; yellow...26 - 50 %; and red...0 - 25 %. The black lines represent one meter contours, with 0 being the lowest point in the landform.

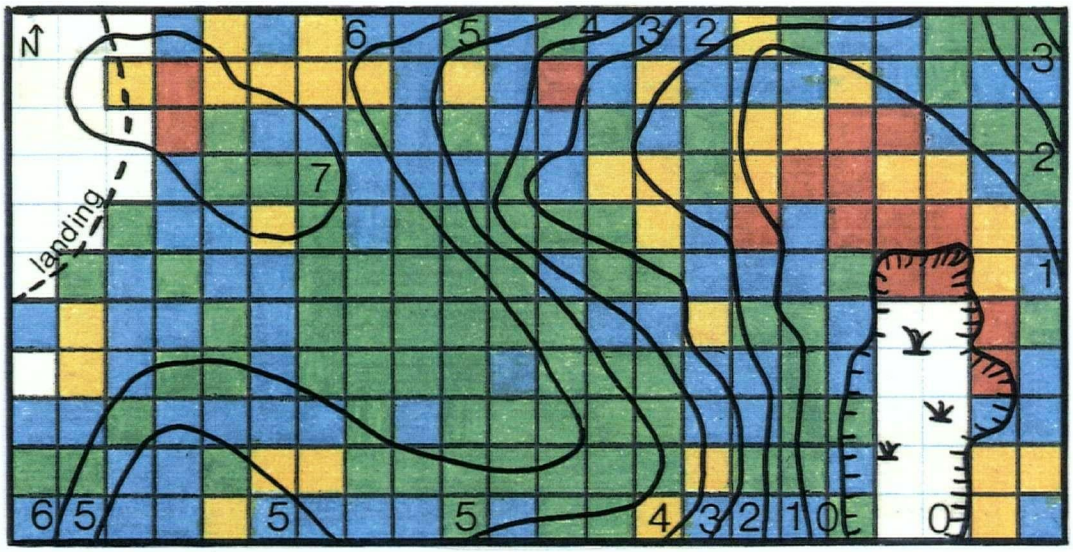


Figure 8. The distribution of dieback damage in frost hollows. Healthy trees (damage class 1-6) as a percentage of all planted trees is colour coded. Green...> 75%; blue...51 - 75 %; yellow...26 - 50 %; and red...0 - 25 %. The black lines represent one meter contours, with 0 being the lowest point in the landform.

large low-lying area in the center of the block drains over a low lip towards the southwest. A gully runs into the central frost pocket from the northwest. The worst damage occurs around the edges of the central low-lying area and in the gully. The lip of the frost pocket towards the southwest is less severely affected. The very southwest corner of the block is part of the next frost pocket, and damage again is severe.

In Figure 8, a wide gully runs east along the north side of the block, draining into a low-lying area near the southeast corner. A ridge lies across the southern and western parts of the block. The same patterns of distribution are evident. Damage is most severe around the low-lying area and along the center of the gully, while few trees on the ridge are affected. In both blocks, the surviving trees in the high risk areas were mostly late flushing trees.

DISCUSSION

Almost all trees in the two blocks exhibited some evidence of minor frost damage. Severe growing season frosts such as the one in June 1985 extend over large areas and would have affected all but the latest flushing trees in these two blocks. In the low-lying areas however, growing season frosts are common and occur perhaps as frequently as 30 times per year (pers. comm. Ordell Steen). This might suggest that progressive dieback involving weak canker parasites requires a continual stress by frequent frosts. The hypothesis that stress due to excessive soil moisture is a contributing factor cannot be wholly discarded however. Some of the severely damaged gully areas were not noticeably wet in 1987, but often the severe damage occurred near wet areas. However, moisture cannot be the

major factor. If it were, there should be little difference between early and late flushing trees. Also there would be no dieback on well drained slopes, and dieback would consistently be found in wet areas only, which is definitely not the case.

CONCLUSION

The spatial distribution of dieback clearly indicates that frost damage rather than excessive site moisture is the critical factor in the dieback process. The progressive dieback was restricted to flat and concave landforms that cause cold air pooling. The large variation in the degree of damage to individual trees growing adjacent to one another could not possibly have been caused by high water tables.

CHAPTER 6
DESCRIPTION OF THE DIEBACK PROCESS

INTRODUCTION

The presence of frost rings, distinct annual rings, and the determinate growth habit of Douglas-fir, enable one to determine the age of all xylem rings and the date of all major growing season frost events, thus unravelling the complex pattern of dieback. The position of frost rings within the annual ring allows dating of frost events. Spring, summer and fall frosts are easily distinguished. Thus, frost rings embedded in the wood provide a historical record of the frost events to which the tree has been exposed during its whole life. The combination of bud and shoot damage and frost rings can be used to reconstruct a detailed history of frost damage (Figure 9). The major purpose of such a detailed examination was to determine whether the initiation of progressive dieback and canker could be related to the occurrence of growing season frosts.

METHODS

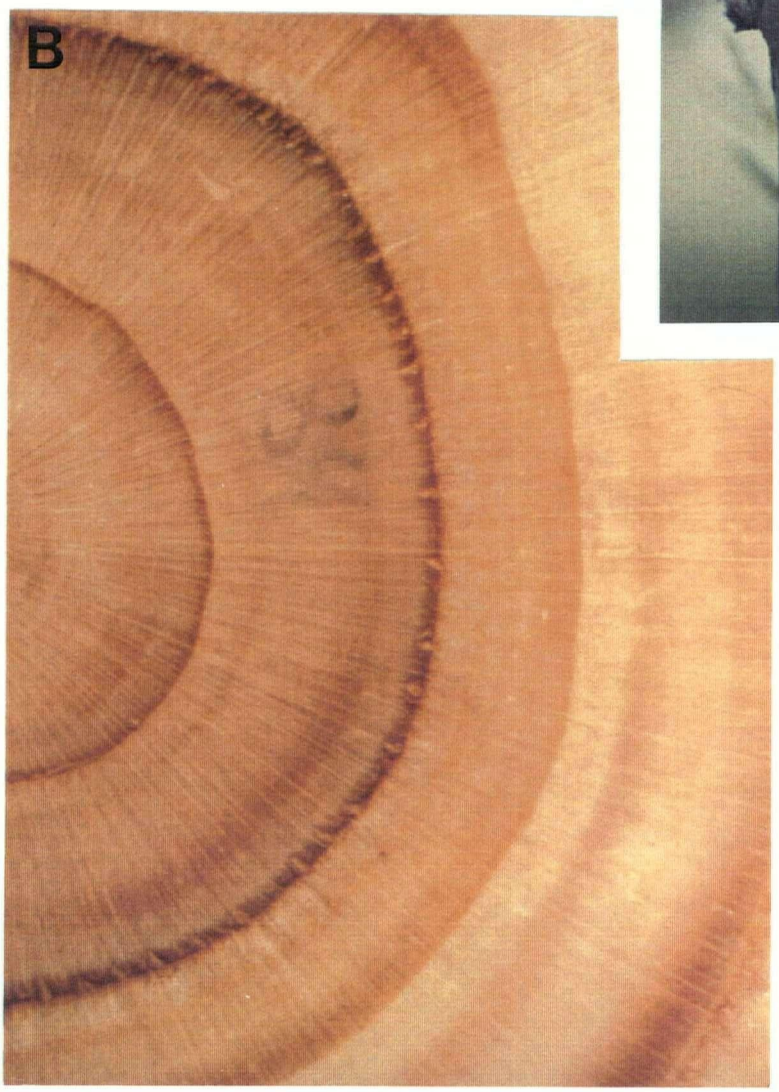
Twenty-three 15 to 17 year old Douglas-fir trees in the Horsefly district, and the Prince George district were carefully dissected to reconstruct the history of frost damage (both on shoots and as frost rings), to describe the location and identity of the various fungal parasites, and in order to determine the cause of dieback. Since trees could be found that had varying degrees of dieback, and a tree with a medium degree of dieback could take up to two days to dissect, only trees that had fairly recent damage were selected. Outbreaks of dieback were initiated at several

Figure 9. Analysis of frost rings in stems of trees showing dieback.

A. Douglas-fir stem with a frost-killed 1984 leader tip and recovery of single stem by a lateral bud. Although only the tip of this tree was killed, often the entire leader or more is killed by a single frost event. Although late spring frosts seldom kill one-year-old tissues, the damage will appear similar to this, when the frost-killed elongating shoots fall off several years later, and the shoot slowly dies.

B. Section through the stem at the 1982 internode showing a distinct frost ring at the end of the 1984 growing season (see Appendix VI for climate data).


A


B

different times during the history of many of the plantations. Leaders and tops that had been dead for over five years were too difficult to work with since the bark had usually deteriorated and the tops were often broken. Therefore, only trees with fairly recent damage and intact leaders were dissected. Often these trees had a fair degree of damage, sometimes over half the tree was dead.

Each tree was sectioned at almost every internode from the base to the tip in order to reconstruct the vertical profile of frost rings. The number of years between the onset of progressive dieback to the present was determined by aging the branch and stem internodes that had died using the age of the uppermost living branches as a reference point. The date of onset could often be matched with the number of annual necrophylactic periderms (ANP) found in the dead top. These ANPs were clearly visible as white boundary lines that formed between dead and live bark. If the tree had died back for three consecutive years, three periderms would delineate each section of stem that died. Often the bark was sunken above and below the periderm, and sometimes bark would separate and shrink away from the periderm. Stem sections were made between the annual periderms in order to age dead stem sections. By examining the foliage and determining when the buds and shoots were killed, a second line of evidence for dating frost damage could be reconstructed in order to compare with the frost rings found in the wood.

Trees with stem cankers were also dissected in a similar manner as above to determine the frost history record. A cross-section through a canker provided information on the exact time of canker initiation and how

the canker arose. Annual necrotic periderms were used as an indication of the initial size of the canker and the rate of spread of the disease.

RESULTS

Table 4 summarizes the results of the sample of 23 trees that were dissected to determine the frequency of frost rings in the seven major experimental areas. Frost rings were common in all trees examined. Certain years (eg. 1978, 1982 and 1984) were characterized by well developed frost rings at several of the locations studied. The degree of frost damage was always related the degree of freezing the tree was subjected to. Trees growing at the bottom of frosty depressions often had more numerous and prominent frost rings and more damage to the shoots and foliage than trees growing further upslope. It appears from Table 4 that almost all of the trees had similar frost histories, when looking at the frequency and number of frost rings detected alone. This impression, however, is misleading since severity of frost rings was not included in the table. Some trees were significantly more affected than others. Although a 1982 spring frost ring may have been present in two trees, in the one tree, the ring may have been much more prominent and have progressive dieback associated with it, whereas the second tree would have suffered minor bud kill, but escaped dieback.

Table 4. The occurrence of frost rings in Douglas-fir stems at seven B.C. Interior locations.

Year of ring	Location and tree number																						
	Prince George								Cariboo														
	Summit Lake[1]		Kenneth Creek			Shesta Lake			Horsefly Edney-2				Hazel. Cr.			Gavin Lake							
tree #	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23
1971	--[2]	--	--	--	--	--	--	--	00	00	00	00	00	00	00	00	10	00	00	00	00	00	00
1972	--	--	--	--	--	--	--	--	10	00	10	00	00	00	00	00	10	00	00	00	00	00	00
1973	--	--	--	--	--	--	--	--	00	00	10	00	00	00	00	00	10	00	00	00	00	00	00
1974	--	--	--	--	--	--	--	--	00	00	00	00	00	10	00	00	10	00	00	00	00	00	00
1975	--	--	--	--	--	--	--	--	10	00	00	00	00	10	00	00	00	00	00	00	00	00	00
1976	00[3]	00	00	00	00	00	00	00	10	00	10	00	10	10	10	00	10	00	10	10	00	00	00
1977	00	00	00	00	00	00	00	00	10	10	10	10	10	10	10	10	10	10	10	10	00	00	00
1978	00	00	10	10	00	01	01	00	20	30	10	10	10	10	10	10	20	10	10	10	00	00	10
1979	00	00	10	10	00	00	00	00	10	10	10	10	10	10	10	10	10	00	10	10	00	00	10
1980	00	00	00	00	00	00	00	00	11	21	00	10	00	00	00	00	00	00	11	11	11	00	10
1981	00	00	00	00	00	00	00	00	10	10	10	10	00	00	00	00	00	00	00	10	01	00	00
1982	00	00	11	10	11	00	00	00	10	10	11	10	11	21	11	10	10	10	11	10	11	11	10
1983	10	10	11	00	01	01	01	01	12	11	01	11	01	01	01	10	11	01	01	01	01	01	01
1984	00	00	11	10	01	01	01	01	01	--	11	01	01	11	11	10	10	01	01	01	11	11	11
1985	00	00	01	10	01	00	00	00	11	--	10	11	01	11	11	10	00	00	00	01	11	11	11
1986	00	00	00	00	00	00	00	00	12	--	10	10	00	11	10	10	10	00	10	00	02	12	11

[1] Trees growing in close proximity to each other and therefore exposed to the same frost events are identified by these lines.

[2] -- indicates that no observations were made for the years indicated.

[3] The two digits indicate the number of spring and fall frost rings respectively in the annual ring in question. Well developed frost rings are underlined.

All the trees described in Table 4 were taken from within or from the edges of frost pockets. Healthy trees surrounding these frost pockets had very few frost rings. These healthy trees had frost rings only in the years in which major growing season frosts occurred. The cambium appeared to be more sensitive to frost than the crown, since there were often more frost rings than identifiable frost events, as determined by crown symptoms.

Figures 10 and 11 show simplified patterns of dieback. They are taken from parts of two complete tree diagrams and demonstrate the two patterns of symptom development that were recognized. The width of the red lines representing frost rings indicate the relative severity of spring and fall frost periods. These show two trees with typical damage of the type illustrated in figures 3 C and A respectively.

The first tree (Figures 10 and 3 C) shows a typical case of simple frost damage. Two major frost events in the late spring of 1982 and fall 1984 resulted in death of the leader, and in both cases, a single lateral assumed dominance with little ensuing damage. *Sclerophoma* was found fruiting on the dead leaders. There was no progression of the dieback into the adjacent healthy bark. The frost rings found in this tree (Figure 10) showed that there were several frosts that did not result in major shoot damage. The frost rings that formed in association with the onset of dieback were more visually prominent and caused more anatomical disruption of the cambial tissues than the frosts that were not associated with much foliar or shoot damage. Climatological data, used as an independent check of the severity of individual frosts or frost periods also supported these findings (Appendix VI).

Figure 10. A: Schematic representation of simple frost dieback. A major frost in spring of 1982 killed the 1981 leader. A lower lateral branch turned up to become the new leader. In the fall of 1984, frost damage again resulted in death of the then current leader, and again recovery was by a single lower lateral shoot. B: Schematic representation of frost rings (red) in the longitudinal annual rings of the stem.

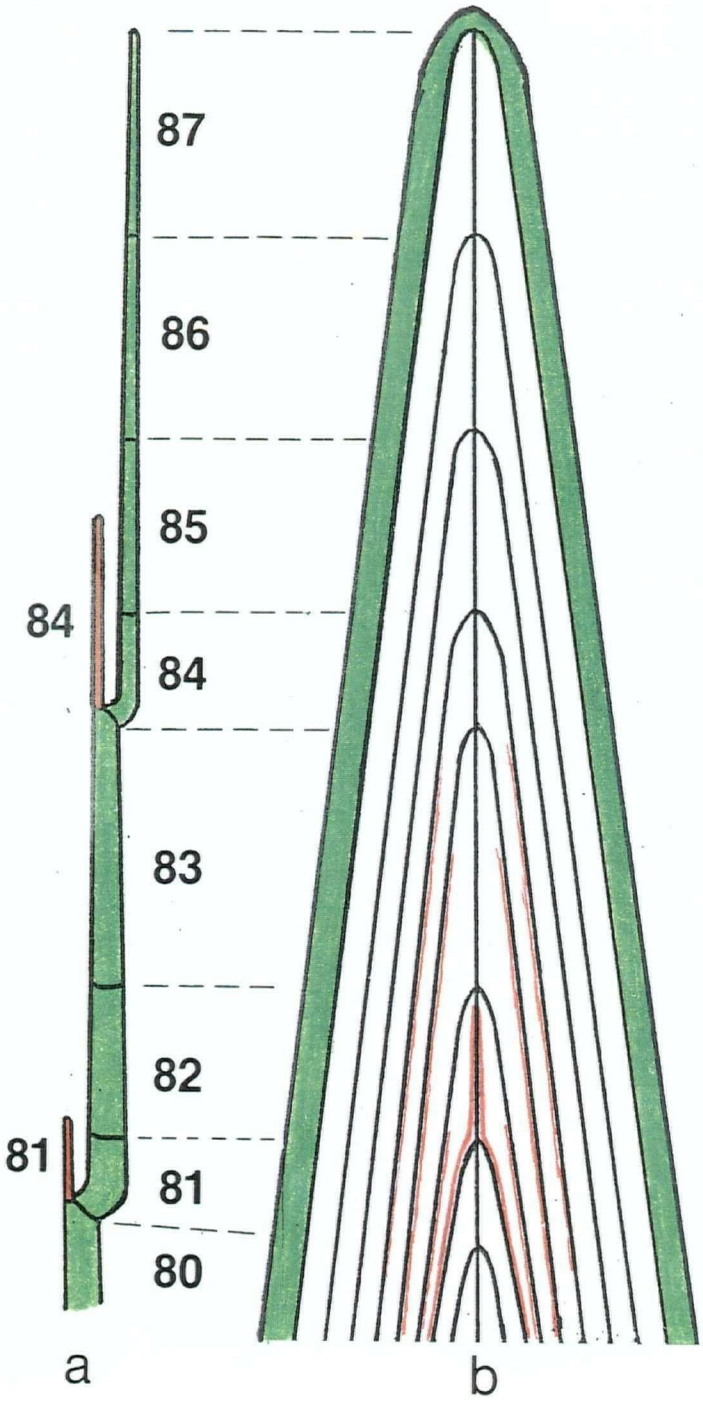

87
86
85
84
84
83
82
81
81
80
a
b

Figure 11. A: Schematic representation of progressive dieback drawn to scale. The color coded bark represents the years in which the bark and cambium has died: dark blue - '82; sky blue - '83; yellow - '84; orange - '85; red - '86; green - alive in '87. B and C: Schematic representation of the frost rings (red) in the original stem (B) and the major upturned branch (C). The straight horizontal lines in A correspond to the nodes in B and C. The dark oblique lines separating bark of different colors are the necrophylactic periderms laid down annually to halt the progression of the fungus each spring. (Note the 1987 ring is very narrow, since the tree was harvested early on in the 1987 growing season.)

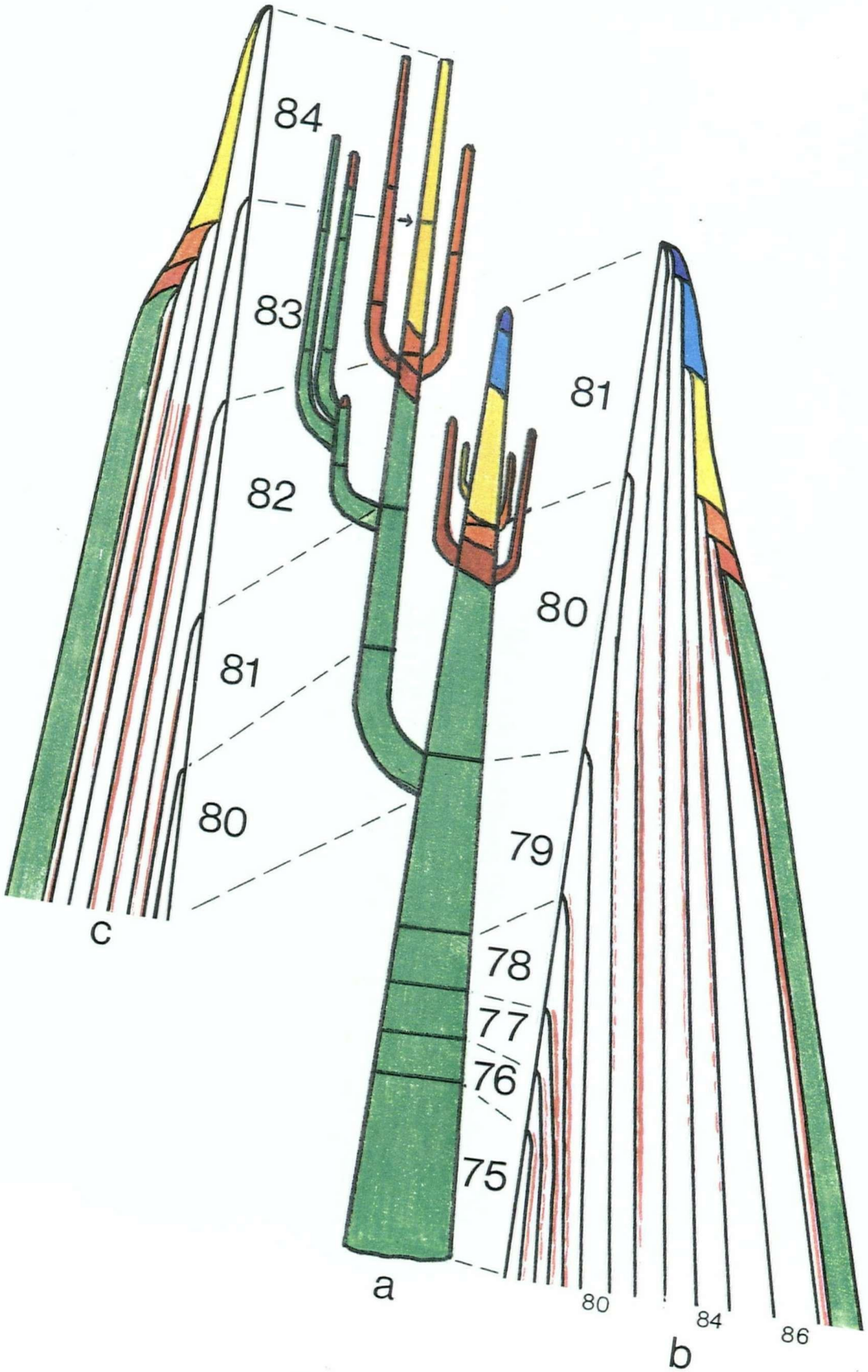
This diagram incorporates frost ring analysis information gathered at each internode through sectioning. Frost rings are evident in almost every year of the wood. There were few frost rings in the wood formed before 1976, but in 1976, '77, '78, and '79 there were major spring frosts.

In 1982, the next major spring frosts occurred causing extensive bud kill and some leader dieback. The buds at the tip of the 1981 leader died, although some wood was formed in 1982 before the leader died the following winter. This main stem was undergoing annual progressive dieback of the main stem at a steady rate until harvest.

Reaction wood began to form in the 1983 annual ring at the base of the upturned branch (C) indicating that recovery of the leading shoot occurred the year following the original dieback.

In 1984 fall frost killed the leader and another internode of the major upturned lateral (C). Slow progressive dieback was now occurring at two locations.

From this first upturned lateral (C) another branch (to the left of C) turned up to become the new leader. Frost damage was evident to the 1986 leader of one of it's forks. Although not included in the diagram, several other laterals were also undergoing progressive dieback.


84
83
82
81
80
c
81
80
79
78
77
76
75
a
80
84
86
b

The second tree (Figure 11) provides an example of progressive dieback. Two major frost events in 1982 and 1984 resulted in death of the tip of the original leader and the upper internode of the upturned branch (C) respectively. In both cases this initial damage was followed by progressive dieback over a period of several years. *Sclerophoma* was found fruiting on parts of the dead bark, but *Leucocytospora kunzei* was apparently responsible for the invasion of the healthy bark following the initial frost kill. Only *L. kunzei* was isolated near the lower edge of the recently killed tissues. In fact, progressive dieback, such as illustrated in Figure 3 A was always associated with fungal parasites other than *Sclerophoma*, with *L. kunzei* being the most common. Examination of the frost rings (Figure 11) showed that there were several frost events following the 1982 frost. These frost resulted in varying degrees of bud kill and presumably further weakened the tree.

The dissection of two trees near Gavin Lake which had large apparently frost-caused stem cankers yielded interesting results. Both trees had a history of frost dieback dating back to 1976. Both trees recovered from the initial events, but developed progressive dieback in 1984. A cross-section through the canker indicated that there was an increase in the level of frost damage that was occurring on the main stem near the ground (Figure 3 G). The stem was apparently no longer thermally protected after the upper crown had died. In 1987, the cambium and the phloem became so severely damaged by the late spring frost period in May, that the bark died. Since the cambium died while frost rings were forming it seem unlikely that anything else could have caused the death of the cambium. It would seem though, that the expansion of the canker, especially in non-frosty years, is mostly a result of progressive pathogenic activity. This

progressive movement occurs in the early spring, when the wound periderm loses it's ability to contain the pathogen. The pathogen kills the bark from the edge of the canker toward the healthy tissue (Figure 3 G), and the bark dies to the cambium at the canker margin. Then when the cambium becomes active later in the spring, a layer of callus tissue is laid down over the wood which was killed by the pathogen.

Engelmann spruce (*Picea engelmanni* Parry), subalpine fir and western red cedar, which were present in most of the study areas, all exhibited frost damage symptoms. Red cedar foliage appeared burnt when there was no overhead protection. On the other hand, lodgepole pine appeared to be remarkably resistant to frost damage. Individual lodgepole pine trees with actively elongating shoots and without any evidence of frost damage were often found in typical frost pockets in which all the other conifer species were seriously damaged. Several pine were selected for dissection from frost pools which were known to have severe conditions. Only a faint 1982 late spring frost ring could be detected in one tree.

DISCUSSION

It is clear that unusually severe frosts will cause frost dieback of leaders and the tips of branches in the upper crown. Such frosts occur on a periodic basis. Since the initiation of several progressive dieback events was always proceeded by severe growing season frosts it is clear that frost is the major initiating factor in the dieback process. No other abiotic or biotic factor or combination of factors can account for the frost and progressive dieback widely observed in the interior of B.C.

At least three other possible causes of dieback in general have been proposed. The first is that *Sclerophoma*, the organism found in association with the dead leaders, is the primary cause. The second is physiological stress due to excess moisture stress in depressions. The third is stress resulting either from micronutrient deficiencies or a lack of nutrient cycling.

All of these hypotheses can be discounted. On every site examined, there could always be found healthy trees growing amongst trees suffering from varying degrees of dieback. As was shown earlier, (Chapter 4) such healthy trees were usually late flushing trees which escaped many late spring frosts. The only difference between the affected and unaffected trees was the amount of frost damage that was sustained, and that, in turn, determined whether or not there was subsequent fungal invasion and progressive dieback of the healthy tissue adjacent to frost killed tissues. Trees on the healthy end of the scale had considerably less frost damage. The degree of damage over and above that caused by frost appeared to be the result of fungal invasion of healthy stem tissue. Frost damaged tissues were almost always colonized by weakly parasitic fungi, of which *Sclerophoma* was the most common. During years without major frosts, and on trees growing outside frost pockets there was no infection of leaders by *Sclerophoma*. There was no advance by *Sclerophoma* into healthy tissues of trees suffering some minor dieback. All of these factors combined indicate that *Sclerophoma* is a very weak pathogen that does not cause outbreaks.

The second alternative hypothesis that must be dealt with is the moisture stress hypothesis. Once again the interspersed distribution of affected trees, the type of damage and the way the damage arose did not

support this hypothesis. Affected trees were often growing on well drained soil on the slopes of a concave landform. There was no gradation of damage towards the wetter areas as would be expected if excess moisture was responsible. The large variation in the severity of damage to trees growing side by side indicates that the stress factor only affected some trees and not others. Outbreaks were sudden occurrences as determined by dissections of the dead members, with all members dying rapidly at the same time. Weather records did not indicate any abnormal levels of precipitation during or prior to the years when outbreaks occurred.

Finally the nutrient deficiencies and nutrient cycling problems were also discounted for the same reasons as the previous hypotheses can be discounted. The marked variation in degrees of dieback, the peculiar spatial distribution of the affected trees, and the nature of the damage all occurring within a short period of time for each individual outbreak does not support this view. Trees from a variety of sites representing different nutrient regimes all had the same symptoms.

An interesting question which has been posed by many people is why was the problem not recognized prior to 1985 and 1986. Table 1 shows that damaging frosts occurred in these plantations as early as 1975. Examination of very young plantations shows that frost damages young trees, and that progressive dieback can begin at a very young age. Examination of small dead trees in older plantations indicates that trees were dying over a long period of time.

The fall frost event in 1984 was unusually severe (see App. VI for climate data). During that event, widespread frost dieback occurred. The widespread dieback caused by this event was not restricted to frost pockets

which often are not visible from roads. Instead large sections of plantations were affected, and many casual onlookers became aware of dieback for the first time. However, an examination of trees in frost pockets reveals that individual trees were affected by dieback throughout the history of the plantations. Thus, it seems that both the scale of the damage and the fact that the trees were finally reaching a size that people could detect red tops accounted for the slow recognition of the problem.

What is the role of subsequent frosts in progressive dieback? It would appear from several trees that were dissected that once a tree has a dead top, the stem becomes somewhat unprotected from radiation cooling. Stems that showed somewhat minor frost rings prior to having a dead top became more frost damaged near the base of the dead top. This subsequent frost stress was thought to aid progressive dieback, since trees that were only subjected to periodic frosts recovered faster than trees that were under continuous frost stress. Progressive dieback usually consisted of the invasion of such frost-stressed upper bole tissues by weak fungal parasites.

A phenomenon which further supports the frost damage hypothesis is the discovery of a horizontal line in a frost pocket above which there was very little frost damage. This line indicated the level to which the frost pocket would fill with cold air on nights with strong radiation cooling. In the early morning frost was only visible on the portions of trees that were below this line. Pommerleau (1971) also pointed out a similar phenomenon in frost pools in Ontario. Infections caused by the disease *Gremmeniella abietina* were clearly restricted to the portions of the trees that were below the height of the frost pool (Pomerleau 1971).

Although previous authors have not reported on whole tree dissections, Day (1928, 1931) described amphitheater-like frost cankers on Douglas-fir and larch respectively. Day demonstrated how these cankers originated from the direct killing of the cambium as a result of severe frost. On European larch these cankers were often centered around a short shoot. In early spring the larch cambium becomes active around the short shoots first. This period of activity coincides with the time when the short shoots have flushed but the long shoots have not. Day stated that frost damage at this stage of cambial development results in the local death of the cambium and the bark at the site of the short shoots.

In Douglas-fir, Day showed how this type of amphitheater-like canker resulted from severe frost damage at the grass line, where the ambient temperature was lowest. Once cankers formed, they often expanded in the years following the initial damage, resulting in the amphitheater-like cankers or in the girdling of the tree. Since wounding was found to stimulate the cambium into activity even after it has ceased to divide in the autumn, and since in the spring, new wood is produced at the wound site earlier than elsewhere, this effect was proposed as a major contributing factor to canker expansion both in the fall and spring. This wider period of cambium activity would extend the cambium's period of susceptibility to frost earlier in the spring and later in the fall.

Dissections of Douglas-fir cankers in this study showed that frost can directly kill the cambium. In cross-section, pathogenic killing of the cambium at a canker margin, which is then followed by callus formation, can be confused with frost damage to the cambium at the margin of the canker. Day's explanation of the mechanism of frost canker enlargement

stated that subsequent frosts resulted in the enlargement. Although frost appears to initiate the canker, once a large enough piece of bark has been killed pathogens invade and are able to enlarge the area of dead bark. This point apparently caused Day some difficulty in interpreting the etiology of the larch canker. The fact that the expansion of the canker margin can occur without frost seems to partially discount Day's conclusions, at least in the case of larch. The grassline cankers on Douglas-fir seedlings described by Day (1928) were identical to the frost cankers near the ground-line on the 17 year old Douglas-fir at Gavin Lake. Although the amphitheater-like cankers in larch probably also resulted from frost, his conclusions about how the cankers enlarged were probably incorrect. His evidence, in the form of photographs of cankers with frost rings is not convincing since a callus formation over an area of dead cambium can resemble a frost ring.

CONCLUSION

Since the date of dieback initiation corresponds to specific prominent frost rings, and the leader damage was confirmed as frost damage, it is possible to conclude that severe frost is the major factor involved in causing dieback. The close relationship between the nature and anatomy of the frost ring and the type of external damage enables a detailed history of frost events and their relative magnitudes to be reconstructed. The various forms of damage always correlated well with frost rings and frost damage. Other damaging abiotic or biotic factors, such as soil moisture stress or a pathogenic outbreak could not have resulted in this type of damage. The horizontal frost damage lines in frost pools above which there is no damage and grassline frost cankers also support the frost-caused dieback hypothesis.

CHAPTER 7

PATHOGENS ASSOCIATED WITH DIEBACK AND CANKER OF DOUGLAS-FIR

INTRODUCTION

Most facultative parasites cause increasing amounts of damage as trees become stressed (Schoeneweiss 1986). Early on in the investigation, it became apparent that there were several species of facultative parasites involved in the dieback phenomenon. Also it became clear that the different pathogens occupied different niches in the dead and dying portions of the upper crowns. *Sclerophoma semenospora*, described by Funk in 1980, was reported by Lewis and Cozens (1987) and Mussio (1987) as being the major pathogen involved in the dieback phenomenon. However at the onset of the study it was recognized that *Sclerophoma* was probably not the most important pathogen involved with respect to damage caused to the tree. It became obvious that *Sclerophoma* was a rapid colonizer of frost-killed, or fire or mechanically injured tissues; it was frequently observed and restricted to such tissues. Several pathogens other than *Sclerophoma semenospora* could be found invading the bark at the margin of the frost dieback colonized by *Sclerophoma*. It was these other pathogens that were of greatest interest due to their apparent role in the progression of dieback following the initial frost event. Many of them could be identified from fruiting structures produced near the expanding canker margin. Since dieback of interior Douglas-fir had not received much attention in the past, much time was spent investigating the ecology and epidemiology of the pathogens. This chapter describes the isolation, identification, and the

frequency of the different pathogens and their ecological niches in relation to dieback.

Since pathogenic invasion of the main stem and branches was also shown to occur through *Cinara* (the giant conifer aphid) feeding damage sites, this chapter will also include work done to elucidate the relationship between *Cinara*, pathogens and frost. In this study, Douglas-fir, subalpine fir, lodgepole pine, white spruce and hemlock were frequently colonized by *Cinara* sp. (Figure 12). On Douglas-fir, *Cinara pseudotaxifoliae* Wilson was almost invariably found on the one year old main stem, just below the new growing shoot (Figure 13 A,B,E,F).

The nature of the *Cinara* feeding activity was thought to be of some importance because many trees with top-kill also had small to large, open cankers on either the one- or two-year-old dead main stem internodes. These unusual cankers commonly were in the shape of Cinara feeding colonies, and were always initiated on one year old leaders. The size of the cankers was variable, ranging from 1 - 2 cm long by 0.5 - 1 cm wide up to 10 - 15 cm long by 2 - 3 cm wide. The bark in the center of the canker was smooth without any signs of mechanical damage. Typically, one year after the damage occurred, the bark apparently peeled away from the wood in a well defined elliptical sheet, delineated by a necrophylactic periderm (Figure 13 C).

To test these interpretations, an experiment was set up to determine whether the fungal parasites isolated from active cankers were already present in small latent cankers. Furthermore some work on periderm formation at UBC (Mullick, pers. comm.) suggests that without living distal buds or elongating shoots, the rate of periderm formation is greatly reduced.

Figure 12. *Cinara* feeding site cankers on lodgepole pine and white spruce.

A. Fresh feeding site canker on lodgepole pine. Note the discrete patches of red bark.

B. Removal of the epidermis to show the dead brown outer bark in a discrete superficial patch.

C. Colony of *Cinara coloradensis* feeding on the stem of a planted 17 year old white spruce.

D. Stem canker on spruce, resulting from feeding activity and pathogenic invasion of the bark probably by *Leucocytospora*, judging by the copious resin flow.


A


B


C


D

Figure 13. *Cinara pseudotaxifoliae* feeding site damage and cankers on Douglas-fir stems.

A. *Cinara pseudotaxifoliae* feeding on one year old leading shoot of a seven year old Douglas-fir (June 1989).

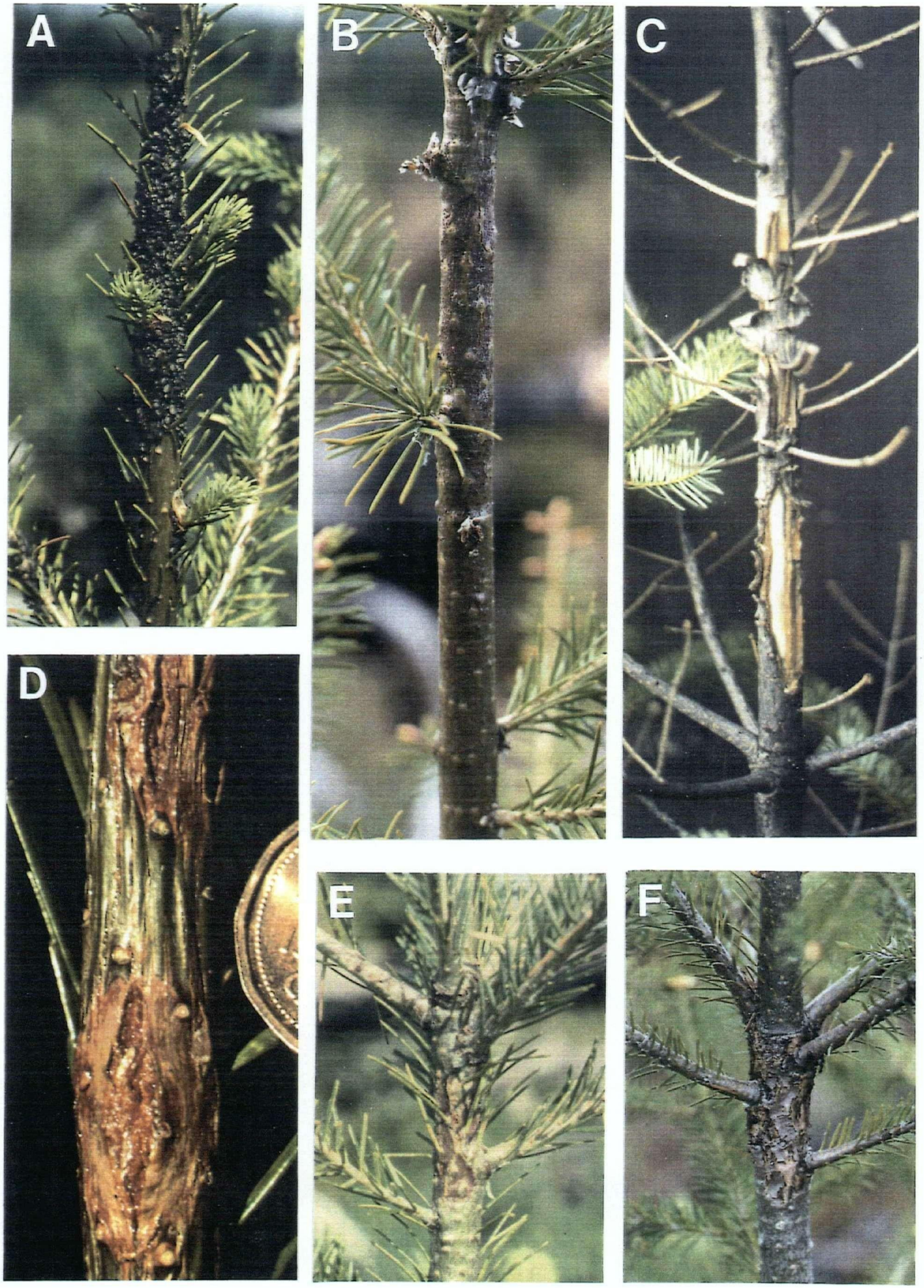
B. Sunken bark of feeding site on two year old leading shoot of a seven year old Douglas-fir (May 1989). Note that the damage occurred as a result of feeding activity on the one year old leading shoot during the 1988 growing season.

C. Combination of *Cinara* feeding damage in the 1984 growing season and fall frost in 1984.

D. Damage by *Cinara* to the current leader (1988) of a young vigorous Douglas-fir in a Chilliwack River plantation on a steep frost free slope.

E. Typical *Cinara* feeding site canker on the two year old main stem just below the node.

F. Four year old internode of the main stem with developing perennial canker at the same position as in E.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It was therefore postulated that one of the mechanisms by which frost might result in activation of small cankers would be through the death of distal buds. A second purpose of this experiment was to simulate the effect of frost by the removal of buds distal to latent cankers.

METHODS

A simple isolation procedure was used to isolate the pathogens living at the margin of the expanding canker of dead bark of the tops of 15 sapling Douglas-fir. Entire trees were brought into the lab, and a large area of bark at the progressive dieback margin was sterilized by swabbing with 95% EtOH and setting it aflame. Using a sterilized scalpel blade, three to four chips of diseased tissue were cut from the margin of healthy and diseased tissue and plated onto a 100 x 15 mm petri plate containing approximately 25 ml of 3% Difco malt extract agar (MEA). The summer periderms were recently formed, and thus the pathogens isolated from the margins were presumably the ones that caused the progressive dieback. The chips were observed daily until the mycelia that grew out of them were large enough to be tentatively identified and then subcultured. The subculturing consisted of removing a small piece of mycelium at the edge of a colony with a flame sterilized scalpel and transferring it to a fresh plate.

Twenty Douglas-fir affected by progressive dieback were selected in a Gavin Lake plantation. 75 instances of 1984 to 1987 aphid feeding damage on the branches of these trees were selected and marked. The selected branches usually had only one perennial canker. The 75 latent cankers were divided into 3 groups of 25 using a dice. Group 1 was treated by removing all buds distal to the canker. Group 2 was left as an untreated

control. The third group of branches was removed for isolation of the pathogens and sectioning. A fourth group of 25 branches without any obvious cankers was then chosen for bud removal from the same group of 20 trees. The three remaining groups of branches were observed throughout the summer. Adventitious buds were removed as they appeared. The treatments were applied on May 16-18, 1988.

The cankers on the 25 branches that were brought to the lab were surface sterilized by swabbing with 95% alcohol and flaming briefly. Small chips were then sliced out of the canker margin by cutting transverse sections of both healthy and diseased tissue. The chips were then placed onto a 100 x 15 mm petri dish containing approximately 20 ml of MEA. Two dishes containing three to four chips each were obtained for each of 25 cankers. The cultures were placed in a cabinet at room temperature and checked daily. As soon as the colonies growing out of the diseased bark were visible, they were subcultured.

All cultures were incubated at room temperature on 20 ml of MEA for as long as they remained free of contamination. The cultures were examined every two to three days to record their growth rate, color, and growth form, as well as the presence of fruiting structures.

Concurrent with the above isolation step, spore cultures were made from asexual and sexual fruiting bodies (Figure 14) for the purpose of comparing and identifying the cultures isolated from the cankers. Extensive spring 1988 field collections turned up several different pathogens fruiting in recently killed Douglas-fir bark of canker margins (Figure 15).

Figure 14. Vertical sections through fruiting bodies of major pathogens causing dieback.

A. *Leucostoma kunzei* (perithecia in stroma with black conceptacle)

B. *Leucocytospora kunzei* (pycnidia)

C. *Pragmopora pithya* (apothecia)

D. *Pragmopycnis pithya* (pycnidia)

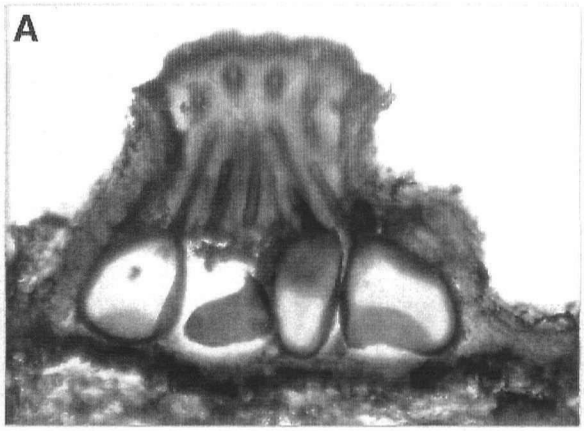
E. *Sclerophoma semenospora* (pycnidia)

F. *Sirodothis* species (pycnidia)

G. *Tympanis laricinia* (apothecia)

H. *Sirodothis species* (pycnidia)

I. *Endothiella aggregata* (pycnidia)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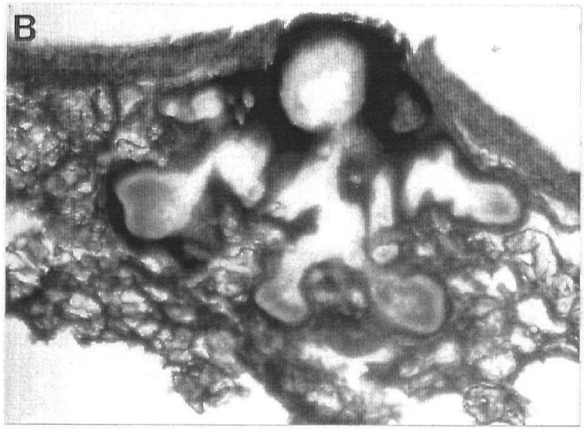
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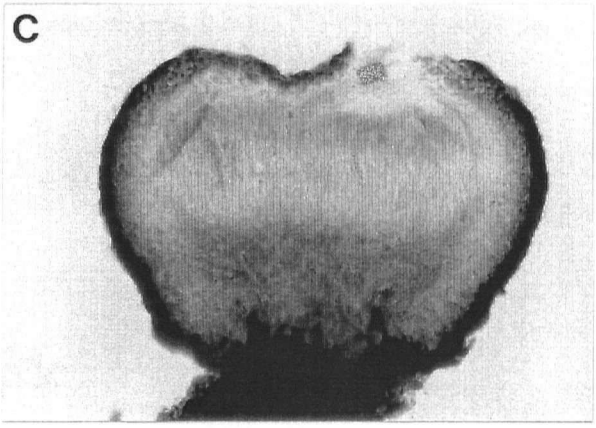
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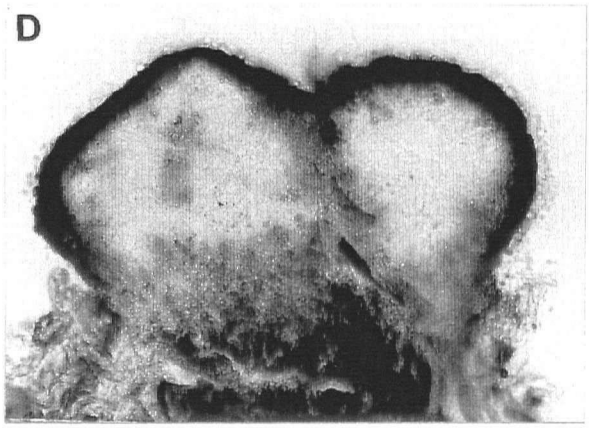
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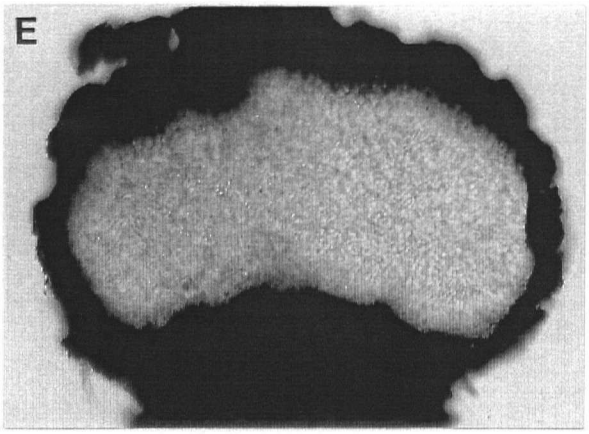
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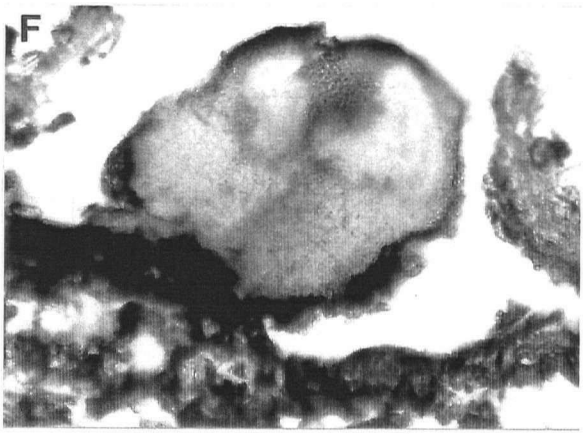
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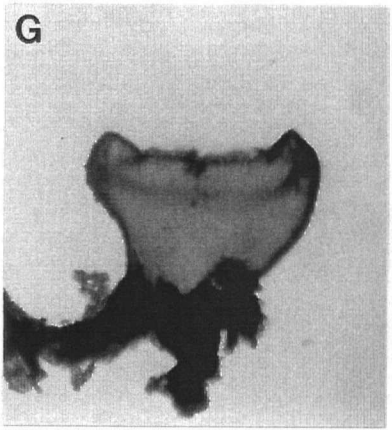
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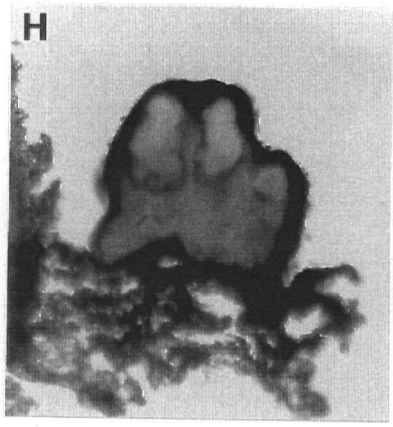
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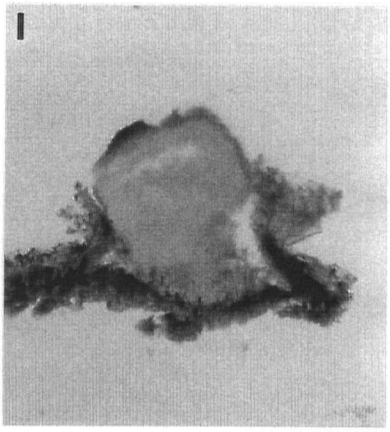
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Figure 15. Fruiting bodies of pathogens collected from recently killed Douglas-fir bark.

A. *Leucocytospora kunzei* x 0.5 (immersed erumpent conical pycnidia)

B. *Tympanis laricinia* x 1.25 (flat black apothecia)

C. *Pragmopora pithya* x 9 (black apothecia mixed with pycnidia)

D. *Sclerophoma semenospora* x 9 (immersed erumpent pycnidia)

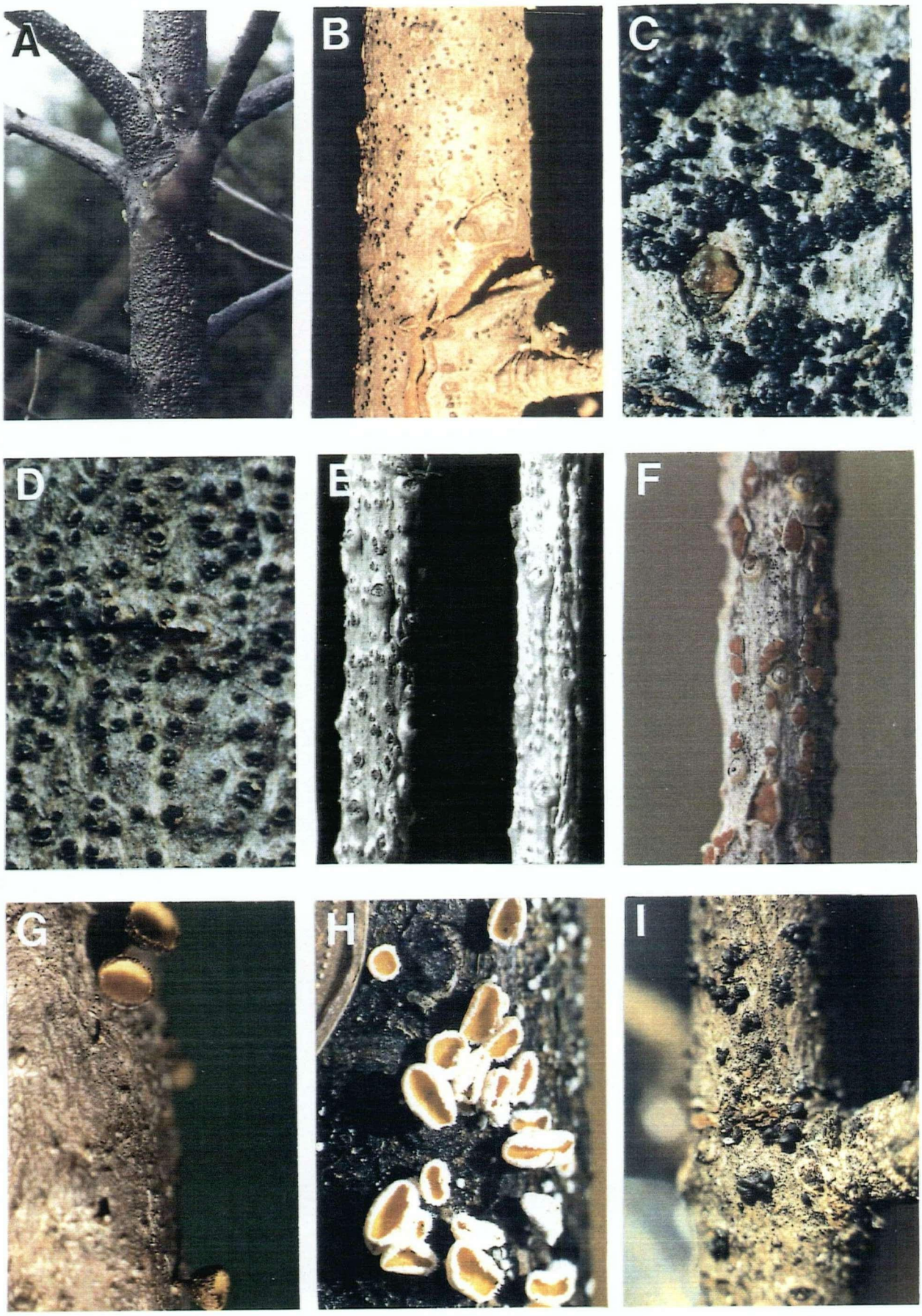
E. *S. semenospora* x 5 (immersed erumpent pycnidia)

F. *Endothiella aggregata* x 5 (red pycnidia)

G. *Lachnellula arida* x 5 (brown apothecia with yellow hymenium)

H. *L. suecica* x 5 (white apothecia with yellow hymenium)

I. *Lophium mytilinum* x 5 (black mussel shaped hysterothecia)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Using a light microscope, the fruiting structures were measured and drawn for several pathogens. Free hand sections were measured using a stage micrometer. Measurements of spores were made from the spore mass in each fruiting structure.

One isolate of *Leucocytospora kunzei* was grown on twigs of willow and Douglas-fir in 20 ml of MEA in test tubes and petri plates in order to positively identify the culture from its fruiting structures. The small twigs were placed in test tubes and glass petri plates along with the MEA prior to autoclaving. The tubes and plates were inoculated after the agar cooled.

RESULTS

In 1988, several fungi were identified from fruiting structures found on recently killed bark and by isolation from diseased tissues (Figure 15). These included: *Sclerophoma semenospora* Funk, *Leucostoma kunzei* (Fr.) Munk and its anamorph *Leucocytospora kunzei* (Sacc.) Urban, *Tympanis laricinia* (Fckl.) Sacc. and its anamorph (a *Sirodothis* species), *Endothiella aggregata* Funk, *Foveostroma boycei* (Dearn.) Funk, (the anamorph of *Dermea pseudotsugae* Funk), and *Pragmophora pithya* (Fr.) Groves and its anamorph *Pragmopycnis pithya* Sutton and Funk. Most of the pathogens were isolated from stem cankers, areas of progressive dieback on stems, and aphid induced cankers. In most cases they appeared to have invaded bark as secondary pathogens. All of these fungi are known as weak parasites of Douglas-fir and other conifers (Funk 1981).

Table 5 shows the pathogens isolated and identified from the 15 saplings with progressive dieback. *S. semenospora* was the most common

fungus encountered. It is not mentioned in Table 5 because although it was present on the distal portion of every stem exhibiting progressive dieback, it was never the pathogen found at the progressive margin of dieback. It was typically found on frost-killed internodes, dead branches and stems killed by rodent girdling or breakage, and on stem cankers associated with mechanical damage by machinery and cattle, or fire damage. *S. semenospora* pycnidia usually were evenly distributed over large parts of the dead bark of such tissues. *Leucocytospora kunzei* was the second most common isolate. It was most often associated with stem cankers and progressive dieback. It was the most common pathogen isolated from the progressive margin of dieback of 15 stems.

Table 6 lists the pathogens isolated from latent cankers by frequency of isolation. Most cankers yielded more than one pathogen. There was no discernable pattern to the combination of species isolated from a single latent canker. On the other hand, it was common to find trees in one area which had almost exclusively one canker pathogen, such as *Tympanis laricinia*, whereas most stems in another nearby area would be colonized by a different pathogen such as *Endothiella aggregata* or *Leucocytospora kunzei*. There seems to be no known explanation for this. It would appear from these and other observations that the various canker fungi involved in the progressive dieback have similar roles in producing the dieback. *Sclerophoma* was not isolated at all from small latent cankers. The fungal species isolated from latent cankers were nearly identical and of roughly the same relative frequency as the organisms isolated from active canker margins in 1987 (Tables 5 and 6).

Table 5. Pathogens isolated at the margin of progressive dieback on 15 Douglas-fir stems in the four Cariboo Region plantations listed in Table 4.

Tree #	fruiting bodies found at margin	pathogen isolated on MEA
1	*Leucocytospora kunzei*	*L. kunzei*
2	*L. kunzei*	*L. kunzei*
3	none	unidentified culture
4	*Sirodothis* sp.	*Sirodothis* sp.*Tympanis laricinia*
5	*L. kunzei*	*L. kunzei*\unidentified basidiomycete
6	no dieback\healthy tree	
7	none	unidentified basidiomycete
8	*L. kunzei*	*L. kunzei**T. lari.**Lophium mytilinum*
9	none	*L. kunzei*\unidentified basidiomycete
10	none	*Peniophora pithya*
11	*L. kunzei*	*L. kunzei*
12	*L. kunzei*	*L. kunzei*
13	none	*L. kunzei*
14	none	green furry*T. laricinia*
15	*T. laricinia*	*T. laricinia*
16	none	*Peniophora pithya*

Table 6. Frequency of pathogens isolated from 25 cankers.

isolate number*	frequency (25 attempts)	tentative identification
1	22	*Leucocytospora kunzei*
2	13	*Sirodothis* sp.
3	8	unidentified, (green)
4	5	*Endothiella aggregata*
5	3	*Lophium mytilinum*
6	2	unidentified (pink)
7	2	*Durandiella pseudotsugae*

* A description of the isolates in terms of cultural characteristics appears in Appendix I. Appendix II contains descriptions of reference cultures.

Giant Conifer Aphid feeding sites become cankers given the proper environmental conditions. Feeding begins prior to bud flush and ends well after the shoots have become dormant in the fall. Frost damage at the site of feeding is essential for the production of long lesions as seen in Figure 13 C. Feeding damage in the absence of frosty conditions was insignificant unless it was particularly heavy, and prolonged.

Shortly after feeding is initiated, sunken patches of bark often develop. On Douglas-fir these lesions are most often located on the 1 year old main stem, usually one or two cm below the current growing shoot (Figure 13 E), or slightly lower (Figure 13 A, B), and less frequently on the current growing shoot, (Figure 13 D).

From tree dissections (Chapter 6), it appears that if feeding damage is followed by frost in the same season, the bark often dies to the cambium, resulting in a long lesion in which the bark lifts from the wood (Figure 13 C). The dead bark harbors canker fungi, which can invade callus tissue formed in the subsequent summers, and cause the canker to enlarge (Figure 16 A, B, C). Typically, a wound periderm forms below the feeding site, walling off a superficial layer of dead bark. Aphid damage occurring later in the season usually results in a deeper necrotic area which can extend to the cambium. This tissue is not isolated by a periderm until the following growing season. Such necrotic areas become latent cankers (Figure 13 D and 16 A). Dieback was often seen to arise from the activation of such cankers on the lower bole or branches following a major frost event.

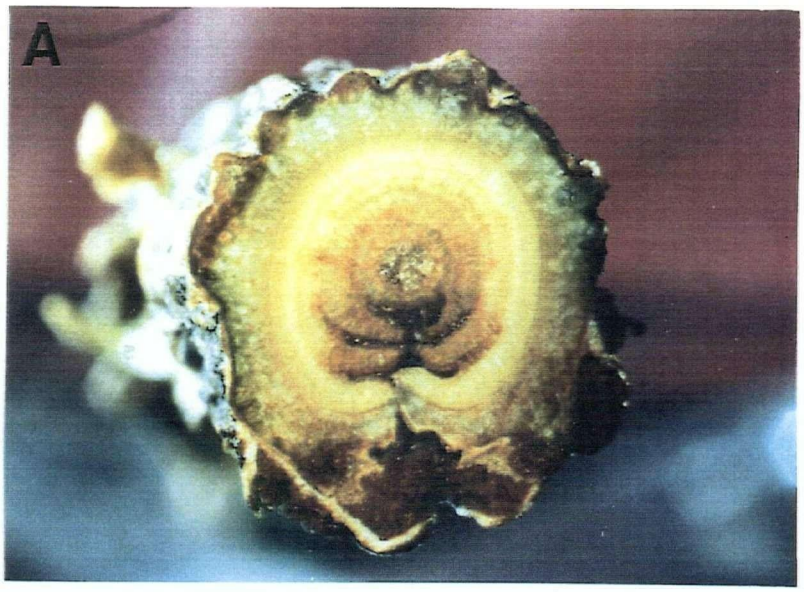
Of the 15 trees dissected in 1987 from the Horsefly forest district, eight had large open cankers on the killed top. Dissections showed that four of the tops had cankers on the one year old leader. These tops died during

Figure 16. Isolation and identification of pathogens from *Cinara pseudotaxifoliae* feeding site cankers

A. Branch canker in May 1988 showing repeated attempts by the tree to callus over a feeding site wound. In mid May (when this picture was taken) the healthy callus tissue was just being invaded from the adjacent canker margin by the spring progression of the pathogen.

B. Pathogen (*Leucocytospora kunzei*) isolated from the canker margin of recently invaded callus tissue.

C. Culture of *Leucostoma kunzei* grown from ascospores from freshly collected material.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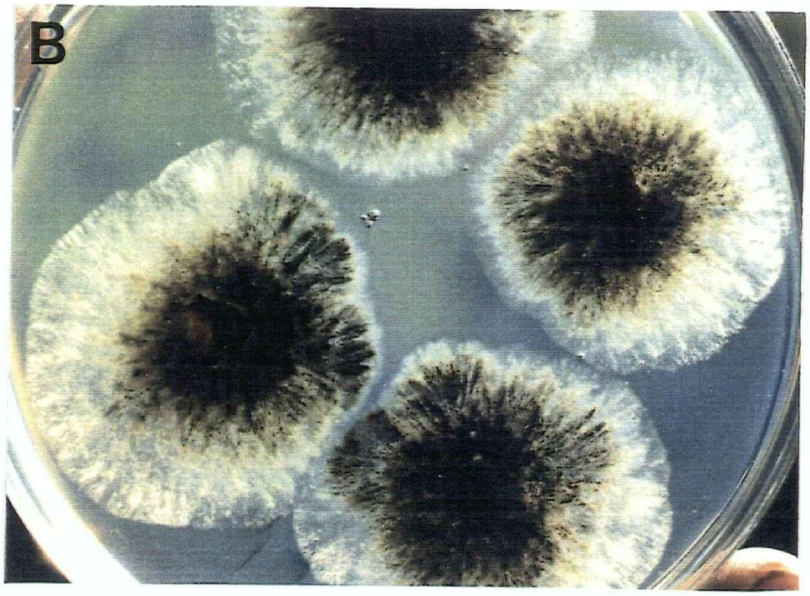
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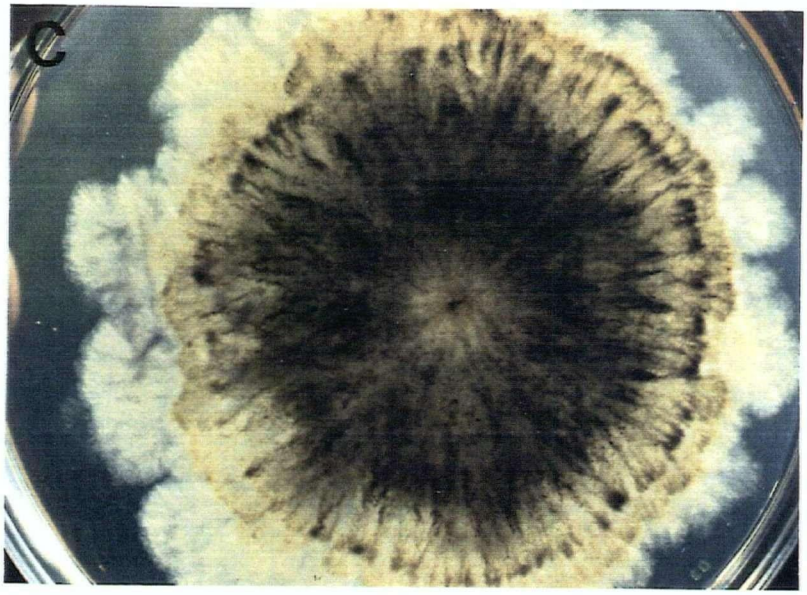
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the dormant season, as a result of a severe fall frost. The other four tops died the following dormant season, although the initial frost damage at the feeding site occurred at the same time for all eight tops. It appeared that although the second four tops survived the first dormant season that the pathogens which were present in the dead canker bark invaded and girdled the remaining stem tissue.

The bud removal treatment did not result in the aggressive movement of the pathogen. There was no difference between the treated and untreated groups with respect to gross external canker anatomy by the end of summer, mid October, or the end of December 1988, and also in mid August 1989. The healthy debudded branches remained healthy. This result suggests that the debudded branches were able to maintain successful barriers to fungal progression similar to those of the non-treated branches.

DISCUSSION

The canker parasites involved in dieback resulting from frost stress typically are secondary pathogens that normally grow on dead and dying plant parts. These fungi appear to fall into two classes: the first contains those that can rapidly colonize injured or recently killed tissues, but are generally restricted to those tissues, and the second consists of those that can cause disease by invading healthy bark either through dead bark colonized by *Sclerophoma*, or its disease margin, or through frost-stressed or frost-killed bark. The second group of pathogens succeed *Sclerophoma* and are of considerable importance because they cause progressive stem killing, often in association with repeated frost stress. Recovery of the main stem usually occurs after frost damage unless there is repeated frost stress that

kills the recovery leader(s), or when secondary pathogens become established and progress into the healthy adjacent tissue.

Even though *L. kunzei* or the other pathogens (other than *Sclerophoma*) do not often infect healthy bark, or even bark killed by frost, once established they can progress into healthy tissue probably by chemically killing the live bark ahead of the advancing mycelium. Langner (1936) suggested that the Larch canker fungus affected the healthy adjacent tissue with enzymes during periods of sufficiently high temperatures during winter creating a frost susceptible zone next to the cankered bark. Day (1958) considered this hypothesis attractive due to its simplicity, and because it united divergent views about the importance of both pathogen and frost in causing dieback. Yde-Anderson (1980) also thought it was probable that "the mycelium secretes one or more substances that cause the bark around the infected area to become particularly frost sensitive".

It appears therefore that the cause of death can be categorized as either abiotic or biotic in origin. Frost injured tissues would probably die to the extent that they are colonized by pathogens such as *Sclerophoma* or *Phomopsis* in Europe. The adjacent healthy tissue then becomes susceptible to frost damage or simply to pathogenic invasion. The second group of pathogens seem to require the abiotic factor to provide an entry court through which to affect the healthy tissue. Neither the first or the second group of secondary pathogens cause infection of healthy undamaged tissue.

The failure of 'debudding stress' to result in the extension of the latent cankers suggests that the breakdown of the trees ability to contain latent cankers, which appears to occur under frost stress, is a result of direct frost damage to the cambium and other functional cells which actively form

the necrophylactic periderm, rather than through the death of distal buds. Another interpretation is that successful periderms formed because the rest of the crown was not stressed, and therefore was able to direct periderm formation. The results may have been different if leaders rather than branches were tested.

CONCLUSION

In summary the various pathogens involved can be broken into two groups. The first group is a weak but fast colonizer of injured or dead tissues. It cannot compete well with the second group of pathogens which can progress into the bark adjacent to that colonized by the first group of pathogens. The pathogens found on stems exhibiting dieback are secondary pathogens incapable of causing disease in healthy, unstressed trees.

CHAPTER 8
BORON FERTILIZATION TRIAL

INTRODUCTION

On some sites in the interior of B.C., foliar content of boron has been reported as causing dieback of pine (R. Carter pers. comm.) and poor early growth of at least one Douglas-fir plantation (J. Revel pers. comm.). A foliar analysis of white spruce in 37 sampling strata within 20 plantations in the Cariboo and Prince George Forest Regions indicated that possible (but not necessarily definite) mild deficiencies of boron occur though not as commonly as nitrogen deficiencies (Ballard 1983). Also it has been shown that the addition of nitrogen can result in an induced or aggravated B deficiency. Boron deficiency symptoms can also appear rather similar to crown deformities caused by frost damage. It has also been postulated that B deficiency can lead to reduced frost resistance (R. Carter pers. comm.). Since some of the symptoms of frost damage could also be forms of B deficiency or at least in part be caused by a boron deficiency, two trials were established to determine if boron fertilization could alleviate frost stress symptoms and/or protect from frost damage.

METHODS

In May 1988 two plantations which exhibited typical dieback symptoms (Edney Cr. - 1, Hazeltine Cr. established in 1971 and 1973 respectively), were selected in the Gavin Lake area. The average height of healthy and moderately affected trees at Hazeltine Cr. was almost twice that at Edney Cr. - 1, the difference possibly resulting from a much hotter

burn at Edney Cr. - 1, which could have resulted in some site degradation and nutrient loss. Ten and nine sets of four trees were selected at the two sites respectively from locations in and around frost pools, such that the type of dieback damage, as well as the relative position in the frost pool, aspect, and the degree of brush competition, was uniform within each set. The trees were selected from a wide range of dieback damage classes, ranging from healthy to severely affected (Figure 17). Table 1 describes the damage classes that were recognized and sampled.

Two randomly selected trees within each set were treated with boron applied as 'Solubor' (partly dehydrated), at a rate of 12.5 kg/ha (=2.5kg B/ha) by watering within a 3 m radius circle around each tree with 34 g of 'Solubor' dissolved in 4 L of water. The control trees received water only. The treatment was conducted on May 11 and 12. Abundant precipitation in the following weeks ensured translocation of the solubor into the soil two weeks prior to budburst.

One of each of the treated and control trees in each set was used for foliar sampling, while the other was photographed at the time of boron application and again at the end of the growing season to record responses to the boron treatment. Foliage samples of 1987 needles, 1988 flushing buds, and 1988 mature foliage were collected in May 29, late June, and July 29 respectively. The flushing buds were collected when they were about 5 cm long, so that the exact date varied from tree to tree, depending on the time of flushing.

The foliage samples were prepared for analysis by drying in a conventional gas heat oven at 65°C. The needles were stripped off the branches prior to drying for the first and last collection, while the entire

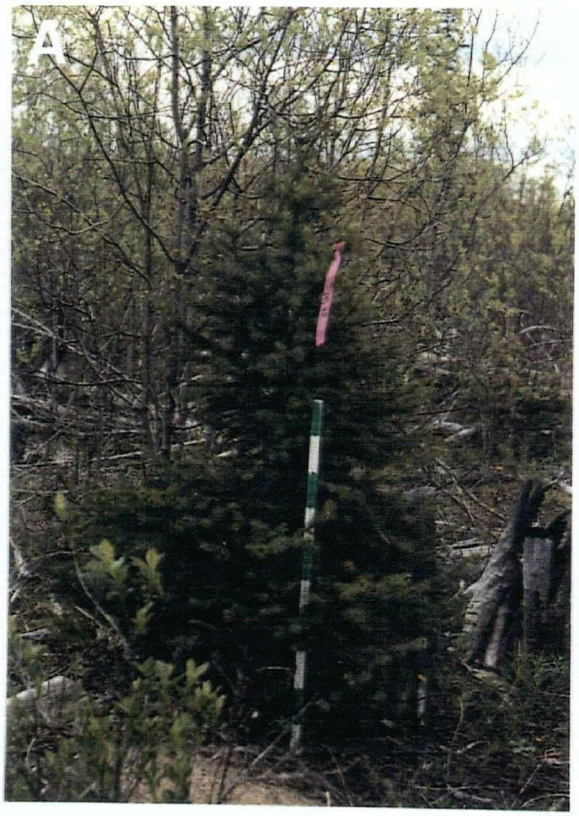
Figure 17. Boron fertilization trial showing range of treatment tree damage classes and tree form recovery in control tree.

A. Healthy 17 year old Douglas-fir at the Gavin Lake site.

B. Mildly affected 17 year old Douglas-fir at the Gavin Lake site.

C. Severely affected 15 year old Douglas-fir at the Hazeltine Creek site in May 1988. Note severe progressive dieback of the main stem and branches.

D. Same tree as in C in October of 1988. Note the recovery of branch form. Although this untreated tree is in a severe frost pocket, within one growing season of frost free conditions, remarkable recovery occurred.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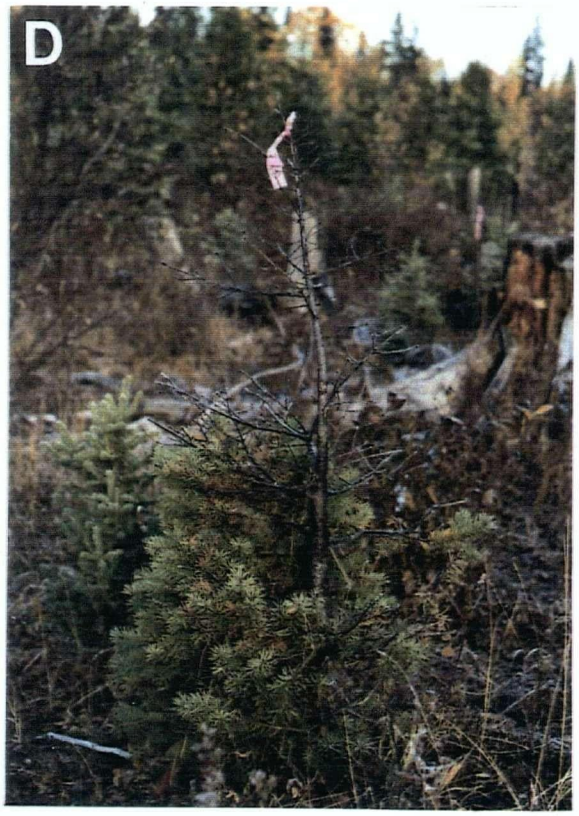
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immature shoot was dried for the second. The 100 needle weight was determined for all samples except the buds. The needles were ground for 30 seconds in a waring blender. Boron and nitrogen content was determined at Pacific Soils Analysis Inc. A complete foliar nutrient analysis of the 1989 needles collected on July 29 was made for one treatment and one control tree in each of a healthy and a diseased set at both locations.

RESULTS

Analysis of variance (Appendix III) showed that the location (the two plantations) and the degree of dieback was not related to N or B content of needles. Table 7 shows the mean nitrogen and boron content of treated and control trees at the three collection times. Appendix IV presents the total foliar nutrient analysis of the 1989 foliage collected in July. Boron concentrations ranged from 23 to 65 ppm in treated and 20 to 42 ppm in control trees. These figures are well within the range of satisfactory boron levels. Nitrogen content was also satisfactory (Table 7). The lack of frost during the 1988 growing season (late as well as early) precluded an immediate test of the effect of boron on frost resistance and recovery.

The marked recovery of many severely affected trees in the trial (Figure 17 C and D) during the frost free 1988 growing season is further evidence that frost plays a critical role in the initiation and progression of dieback. The recovery from dieback was equally pronounced in both the boron treated and control trees.

Table 7. Nitrogen (% dry weight), and boron (ppm) content of 1987 needles, 1988 buds, and 1988 mature needles collected in May, June and July respectively.

Treatment	Time of Collection					
	May		June		July	
	N	B	N	B	N	B
Boron	0.95	30	2.00	36	1.09	39
Control	1.00	28	1.93	33	1.08	24

CHAPTER 9
IDENTIFICATION OF HIGH RISK CUT-BLOCKS FROM AERIAL PHOTOGRAPHY

INTRODUCTION

If progressive dieback is frost-caused, and therefore usually restricted to frost conducive sites such as concave or flat landforms, then by simply identifying these sites on aerial photographs we should be able to predict areas at a high risk to dieback. A survey of sample plantations judged to be at either high or low risk to dieback through aerial photographs was conducted in order to test this method of predicting where dieback occurs. This survey was also used to roughly determine the incidence of dieback in selected high and low risk plantations. A final objective was to establish whether or not the symptoms and causes of dieback in the Kamloops region were similar to those described for the Gavin Lake area in Chapter 6. The Kamloops, Clearwater and Salmon Arm Districts were selected for the survey because they had numerous older Douglas-fir plantations in the Interior Cedar Hemlock zone, which was shown to be of highest risk to dieback by Mussio (1987).

METHODS

A list of ten year old and older Douglas-fir plantations, and aerial photographs was obtained from each of the three above mentioned districts in the Kamloops Forest Region. All plantations listed were classified into two strata (high or low risk) on the basis of topographical features using

aerial photographs. Five high and three low risk plantations were randomly selected within the Kamloops and Salmon Arm districts, and three high and one low risk opening within the Clearwater district. Prior to field inspections, areas suspected to be frost prone within the high and low risk blocks were delineated on aerial photograph overlays.

The extent of the damage was determined in the field by walking around the affected areas and drawing their location on the aerial photograph overlays. Since the topography of most plantations was usually either a uniform slope, basin or flat valley bottom, it was possible to determine the transition zone between affected and unaffected areas with minimal effort. The delineation of this zone was made where the proportion of severely affected trees rose to a level that would prevent Douglas-fir from becoming the major component of the new stand. The actual area of severe damage on the overlay was estimated using a dot grid, and the proportion of the area within high and low risk plantations that was severely affected was calculated.

The survey results were then used to rerank all of the plantations classified initially. This second ranking could be used for prioritizing the remaining high risk plantations for survey (Appendix V). Guidelines for avoiding planting Douglas-fir on high risk sites were the final product of the survey.

RESULTS

Of the 90 plantations classified using aerial photographs, 42 fell into the high risk category and 48 into the low risk category. 4 of the 13

surveyed high risk plantations were severely affected. All 7 low risk plantations were correctly classified; none had significant damage. Within the 13 high risk plantations, 10.1 percent of the total area exhibited severe dieback, resulting in plantation failure. Thus since 47% of all the openings were classified as high risk, an estimated 4.7 percent (10.1% of 47%) of the area in all 10 year old and older Douglas-fir plantations in the ICH in the Clearwater, Kamloops, and Salmon Arm districts has failed because of the dieback syndrome.

Damage prone sites can be identified as follows. Flat valley bottoms are very prone to frost pooling and dieback damage. Gentle slopes (< 10 %) are generally damage free, however, barriers to cold air drainage such as cut block edges (Figure 6), slash piles and roads can create local frost pools. Steep side slopes are generally frost free.

DISCUSSION

This method of identifying high risk sites using aerial photos is very useful in general, although some limitations exist. Plantations that are over 10 years old have regeneration that is visible on 1:10,000 scale photos with the naked eye. Frost prone areas can be identified by the relative density, size and distribution of planted trees using the topography to key in on obvious high risk (frost prone) areas. Often the regeneration is noticeably more sparse and smaller on high risk areas which contrasts with the low risk areas such as mid to upper slope positions, which are usually relatively frost free, and exhibit denser and larger trees. This is often apparent on a site with an undulating topography, where the rises are noticeably well treed, and the depressions have smaller, sparsely distributed trees. Often

the transition zone between the two areas is quite distinct, which on the ground relates to the height of a topographical feature such as a mature border of timber or a height of land. This method is therefore very useful for checking plantations with trees that are at least 4 to 5m in height when using 1:10,000 scale aerial photos.

This method could also supplement a Pre-Harvest Silvicultural Prescription (PHSP). Knowledge of air flow patterns in relation to cutblock layout and topography could help prevent unnecessary blockages to cold air flow which could result in cold air ponding and dieback damage. Examination of the topography using aerial photos prior to harvest could ensure proper cut block layout that would prevent creating cold air drainage problems. This would involve placement of cut block boundaries, landings, skidder trails and roads that would impede cold air flow as little as possible. The detection of frost conducive landforms is more effective on recently logged sites, since the mature canopies can hide the true topography. This method would definitely need more testing in order to evaluate it's effectiveness as an aid to PHSP assessments.

CONCLUSION

1. Using aerial photographs, frost prone landforms can be successfully identified after harvest and especially after the regeneration becomes visible on photos.

2. Frost prone sites include concave landforms, flat valley bottoms, flat hilltops or plateaus, and areas along cutblock boundaries that prevent drainage of cold air.

CONCLUSION

1. The major factor involved in the initiation of dieback is severe frost damage.

2. Topography was the major site factor that influenced frost hazard. Flat or concave landforms were the most conducive to frost pooling and dieback.

3. The pathogens associated with dieback of Douglas-fir do not normally affect unstressed trees. Progressive dieback involving weak canker pathogens occurred on sites with severe and repeated frost stress.

4. Frost tolerant species such as lodgepole pine should be planted on frost prone sites.

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APPENDIX I.

Description of isolates from latent cankers.

Isolate # 1 (*Leucocytospora kunzei*)

Colonies on 3% MEA growing rapidly, filling the plate in 7-10 days, colony hyaline for 3-4 days then becoming darker except at the margin for 5 mm. Hyphae with dark pigmentation 5-6 x 10-30 um, rectangular. Reverse black and bluing at the edges; mottled with immature fruiting bodies which grow onto the petri dish surface pushing the surrounding agar away from it, and in doing so creating a tiny air space. Aerial mycelium with numerous tufts of light grey mycelium (500 um in diam.), some tufts produce conidia, others are sterile.

Isolate # 2 [*Sirodothis* sp. (possible anamorph of *Tympanis laricinia*)]

Colonies on 3% MEA growing slowly, reaching 3 cm in 3 weeks; hyaline at first, then turning rust-colored in the center with cream-colored edges. Colony appearing smooth with tufts of white aerial hyphae evenly distributed over the surface. Can become scarlet red when growing with several other isolates. Submerged hyphae 1.5-2.0 x 40 um, sparsely branched, aerial hyphae hyaline or crimson red, 2-4 um diam., cells long, conidia hyaline or crimson red, 3-8 x 1um. Conidiophores 30-40 x 2-3 um.

Isolate # 3 (unidentified green)

Colonies on 3% MEA growing rapidly, filling the plate in 3 weeks with green mycelium. Aerial mycelium forming a thick velvety layer; reverse yellow, brown, and green; pigment in hyphae as well as in the agar, cells 2-6 um wide, very long; no conidia produced; after 2 weeks a green slime is produced in the center of the colony. This slime appears to be formed from the breakdown of hyphae.

Isolate # 5 (*Lophium mytilinum*)

Colonies on 3% MEA growing rapidly, filling the plate in 2 weeks, (4 cm in diameter in 10 days), light grey to white for the first 10 days, then becoming grey. Margin almost even. Hyphae of two types: (1) aerial, 3-4 x 50 um, forming dense grey stalks with white tufts (8 mm long with 2-3 mm wide tufts at the tip). The hyphae of the stalks conjugating in several places. The stalks form only in the central area of the plate, and, (2) superficial and immersed hyphae 2.5-5 x 60 um cells conjugating, hyaline, thinly branched at tip of cells with swollen end. At 9 days (4 cm diam. producing many light brown, spherical to kidney-shaped pycnidia, 100-225 um diam., single ostiole 25-30 um diam., finely toothed peristome, single celled phialids, conidia single-celled, 1-1.5 x 6-7 um, hyaline, smooth, rod-shaped or allantoid.

Isolate # 6 (unidentified pink)

Colonies on 3% MEA growing rapidly, filling the plate in two weeks; light cream-colored at first, then a pink, and salmon-colored spore mass covers the center and small randomly distributed patches; patches of yellow ropes of hyphae form as well. Hyphae of two different types: (1) submerged, hyaline, 1-2.5 um in diam., cells very long, and (2) black, 1.5 mm long x 60 um wide ropes of black hyphae 2.5 um wide, long cells, the tips of the stalks were pink and salmon in color with conidia 4-5 x 2-2.5 um.

Fruiting began after two weeks producing convoluted structures with rod-shaped protuberances. This structure had a flat black base (70 um thick) on top of a 200um deep root.

Isolates # 4 and # 7 were easily identified by comparison with the reference cultures and therefore were not described.

APPENDIX II.

Description of reference cultures from fruiting bodies.

1) *Leucostoma kunzei* (Fr.) Munk

Colonies on 3% MEA growing very rapidly, filling the plate in 7-10 days; colony hyaline for the first 4-5 days reaching about 2.0 cm before becoming darker: after two weeks, only 5 mm of the outer margin remains hyaline. Aerial mycelium is white and cottony; cells 5-6 um x 15-20 um, pigmented brown. Large sterile, spherical fruiting bodies produced in most plates.

2) *Sirodothis* sp.

Colonies on 3% MEA growing moderately fast, reaching 25-35 mm diam.; light cream colored colony, as well as some red hyphae in others; brown fruiting bodies produced in 2-3 weeks, tall, narrow, with a white conidial droplet exuding from the ostiole.

3) *Tympanis laricinia* (Fckl) Sacc.

Colonies on 3% MEA growing slowly, 2 cm in 3 weeks; mycelium yellow with bayed margin; reverse same color; hyphae 2-3 um with short pegs bearing numerous rod-shaped conidia, conidia also grow directly from hyphae. Pycnidia produced after 3-4 weeks; these tall brown columns with a drop at the tip, are similar to those from *Sirodothis* cultures.

4) *Endothiella aggregata* Funk

Colonies on 3% MEA growing rapidly, filling the plate in 2 weeks with white mycelium; hyphae 2-3 um diam. mostly submerged; red fruiting bodies produced after 2 weeks, characteristically fruiting high on the walls; in 3-4 weeks they reach 2-3 mm in diam. and height,the lower portion is composed of woven hyphae, the upper layer is composed of multilocular cavities, textura angularis. conidiophores branched, 15-20 x 1-1.5 um; conidia 1-1.5 x 3-5 um.

5) *Lophium mytilinum* (Pers.) Fr.

Colonies on 3% MEA growing slowly, reaching 35 mm diam. in 4 weeks; mycelium dark grey, margin entire; reverse same color; hyphae 2-3 um in diam.; Colony furry with appressed hyphae at the margin.

6) *Durandiella pseudotsugae* Funk

Colonies on 3% MEA growing extremely rapidly, filling the plate in less than one week, with a low felt-like mycelium, which becomes darkened in concentric rings after 2-3 weeks, reverse same color as front. Hyphae 2-10 um wide. Black fruiting bodies develop after two weeks; conidiophores 15-20 x 1 um, conidia 5 x 1 um, slightly allantoid.

7) *Pragmopora pithya* (Fr. Groves)

Colonies on 3% MEA growing very slowly, reaching 3 cm in 8 weeks; Mycelium yellow and brown in alternating concentric rings, reverse same color; hyphae small in diam. (3-4 um); concentric rings of dark aerial tufts of hyphae, no fruiting bodies.

8) *Lachnellula suecica* (de bary ex Fckl.) Nannf.

Colonies on 3% MEA growing very slowly, reaching two cm in 4 weeks; mycelium cream-colored with felt-like aerial hyphae. Reverse deep orange with many sectors of lighter orange. Hyphal cells 3-8 um diam. No fruiting bodies present.

9) *Lachnellula arida* (Phill) Dennis

Colonies on 3% MEA growing rapidly, filling the plate in 3 weeks; Mycelium dark grey, cottony aerial mycelium 2-10 um diam.; no fruiting bodies produced.

10) *Sclerophoma* sp.

Colonies on 3% MEA growing rapidly, filling the plate in 10 days. Colony black and appressed with a metallic sheen, agar blued. Cells of hyphae rectangular to ovoid, dark olive green. Conidia produced basipetally from a single locus of a single cell. Conidia hyaline, nonseptate, ellipsoid, 3.5 x 5-13 um.

APPENDIX III

Analysis of variance of nitrogen and boron content of Douglas-fir foliage as affected by location, time of foliage collection, boron application and dieback severity class.

ANALYSIS OF VARIANCE/COVARIANCE FOR VARIABLE NITROGEN

SRC. NO.	SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARE	F VALUE	F PROB	TESTED AGAINST
1	A	1	3.392134E-01	3.392134E-01	9.5509	0.0050	15
2	B	2	18.343532	9.171765	258.2410	0.0000	15
3	AB	2	1.184941E-01	5.924705E-02	1.6682	0.2084	15
4	C	1	7.641239E-02	7.641238E-02	2.1515	0.1519	15
5	AC	1	3.293093E-02	3.293093E-02	0.9272	0.3476	15
6	BC	2	-2.204872E-02	-1.102436E-02	-0.3104	1.0000	15
7	ABC	2	-4.509589E-02	-2.254794E-02	-0.6349	1.0000	15
8	D	7	3.191260E-01	4.558943E-02	1.2836	0.2998	15
9	AD	5	3.722503E-01	7.445002E-02	2.0962	0.1004	15
10	BD	11	2.126038	1.932761E-01	5.4419	0.0003	15
11	ABD	8	9.377005E-01	1.172125E-01	3.3002	0.0111	15
12	CD	7	1.041347	1.487638E-01	4.1886	0.0039	15
13	ACD	5	7.524654E-01	1.504930E-01	4.2373	0.0067	15
14	BCD	11	4.663725E-01	4.239750E-02	1.1937	0.3421	15
15	ERROR	24	8.523912E-01	3.551630E-02			
16	TOTAL	89	25.711129				

ANALYSIS OF VARIANCE/COVARIANCE FOR VARIABLE BORON

SRC. NO.	SOURCE	D.F.	SUM OF SQUARES	MEAN SQUARE	F VALUE	F PROB	TESTED AGAINST
1	A	1	88.002222	88.002213	2.2290	0.1449	15
2	B	2	429.797572	214.898773	5.4430	0.0112	15
3	AB	2	72.096001	36.047989	0.9130	0.4173	15
4	C	1	1140.554436	1140.554199	28.8884	0.0000	15
5	AC	1	18.811718	18.811707	0.4765	0.5033	15
6	BC	2	786.480018	393.239990	9.9601	0.0008	15
7	ABC	2	20.933826	10.466912	0.2651	0.7717	15
8	D	7	372.448846	53.206970	1.3476	0.2717	15
9	AD	5	723.254488	144.650894	3.6638	0.0133	15
10	BD	11	11.992471	1.090224	0.0276	1.0000	15
11	ABD	8	32.230623	4.028828	0.1020	0.9984	15
12	CD	7	286.248703	40.892670	1.0357	0.4332	15
13	ACD	5	476.785143	95.357025	2.4152	0.0653	15
14	BCD	11	100.633510	9.148500	0.2317	0.9921	15
15	ERROR	24	947.552648	39.481354			
16	TOTAL	89	5507.822222				

INDEX	RANGE	IDENTIFICATION
A INDEX	2	LOCATION
B INDEX	3	TIME OF COLLECTION
C INDEX	2	TREATMENT: 1=CONTROL 2=BORON
D INDEX	8	SYMPTOM SEVERITY CLASS

APPENDIX IV

Foliar nutrient content of lightly and severely damaged trees at two locations.

PSAI Pacific Soil Analysis Incorporated

Dr. Vandercamp -- Richard Reich Dec/88

Tissue			TOTAL (%)						TOTAL (ppm)							
LOCATION	TRMT.	DAMAGE RATING	Nitrogen N	Phosphorus P	Calcium Ca	Magnesium Mg	Potassium K	Sulphur S	Copper Cu	Zinc Zn	Iron Fe	Manganese Mn	Boron B	Aluminum (ppm) AL	Available Sulphate-Sulphur (ppm) S	Active Iron (ppm) Fe
1611	NB	6	1.11	0.25	0.29	0.085	0.81	.077	4	22	37	595	24	126	38	
1611	NB	1	0.95	.20	.22	.086	.74	.053	4	16	53	423	25	184	19	
1621	NB	1	1.20	.24	.30	.091	.60	.078	5	31	42	262	32	151	45	
1621	B	1	1.02	.20	.34	.057	.64	.066	5	21	42	255	41	207	45	
1621	B	6	0.97	.22	.32	.092	.64	.070	4	16	37	327	35	144	71	
1621	NB	6	1.16	.24	.29	.072	.88	.079	5	20	37	304	21	144	97	
1611	B	1	0.99	.20	.20	.078	.64	.063	4	17	43	600	42	152	26	
1611	B	6	1.07	.27	.24	.088	.70	.077	4	24	43	579	36	200	45	

1. Moisture determined by drying tissue at 105°C overnight.
2. Total Nitrogen, Phosphorus, Calcium, Magnesium and Potassium were determined on a peroxide-sulfuric acid digest. Total Nitrogen and Phosphorus were measured colorimetrically, and the remaining cations were measured on an Atomic Absorption Spectrophotometer.
3. Total Copper, Zinc, Iron and Manganese were determined on a dry ash.
4. Total Boron was determined on a dry ash using Azomethine-H.

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APPENDIX V.

Summary of plantation survey in 3 districts

JUN 20, 1988

ONETIME REPORT #75
LIST OF FIR PLANTATIONS DONE
ON OR BEFORE 1977
SORTED BY DISTRICT, OPENING

LAST UPDATE: 88.06.20
KAMLOOPS SALMON ARM

OPENING	LOCATION	ELEV (M)	REFOR YEAR COMP	REFOR TREES (K)	REFOR (HA)	1ST SPP REFOR	HISTORY RECORD KEY	Rating Before	Rating After	Site Description
82L06600 216	DALL C	1075	1972	51	64	FDI	K018518	L	L	HILLY
82L06600 219	DALL C	1330	1972	26	40	EDI	K018521	H	M	SLOPE, POOL
82L06700 205	KINGFISHER	1060	1970	10	8	FDI	K010660	H	L	V. GENT. SLOPE
	KINGFISHER	1060	1971	41	40	FDI	K010660	H	L	" "
82L06700 206 -	KINGFISHER C	600	1971	100	75	FDI	1▶ K010128	H	L	GENTLE SLOPE
82L07500 73 -	MARA MEADOWS	730	1968	44	43	FDI	K025963	H	M	ROLLING HILLS, 1 POSS. POOL
	MARA MEADOWS	730	1969		5	FDI	K025963	H	L	" "
82L07500 74	MARA MEADOWS	760	1969	7	4	FDI	K025942	H	L	GENTLE SLOPE
	MARA MEADOWS	760	1973	13	12	FDI	K025942	H	L	"
82L07600 17	LAFORGE RD	1175	1970	120	107	FDI	▶ K018324	H	L	STEEP, 1 SLOPED BASIN
	LAFORGE RD	1175	1972	20	15	FDI	K018324	H	L	" "
82L07700 206	NOISY C	914	1971	70	72	FDI	K090444	L	L	GENTLE SLOPE
82L07700 211	NOISY BASIN	760	1971	186	131	FDI	K010111	H	L	GENTLE SLOPE, 1 POSS. POOL
	NOISY BASIN	760	1971		11	EDI	K010111	H	M	SMALL POOL
82L07700 213 -	NOISY BASIN	880	1972	161	185	FDI	▶ K090433	H	L	POOR DRAINAGE
82L07700 249	KINGFISHER C	853	1976	44	37	FDI	K071030	L	L	STEEP
82L07700 259 -	KINGFISHER C	610	1975	49	46	FDI	▷ K010101	L	M	SOME DEPRESSIONS
	KINGFISHER C	610	1975	33	23	EDI	K010101	L	M	" "
82L07700 260 -	KINGFISHER C	610	1977	51	35	FDI	▷ K010129	-	M	FLAT, SLIGHT DEPRESSIONS
82L07700 267	NOREEN M	1219	1976	40	40	FDI	K018236	L	L	STEEP HILLS, 1 OR 2 SM. POOLS
	NOREEN M	1219	1977	4	2	FDI	K018236	L	L	" "
82L07700 268	NOREEN M	1311	1974	18	22	EDI	K018230	L	L	" "
	NOREEN M	1311	1977	13	9	EDI	K018230	L	L	" "
82L07800 200	WAP VALLEY	550	1971	95	70	FDI	K010133	H	L	" "
82L07800 202	WAP VALLEY	525	1971	71	64	FDI	K010140	-	-	
82L07800 203	WAP VALLEY	500	1972	178	137	EDI	K010669	-	-	
82L08500 13 -	4 M N SICAMOUS	1158	1971	34	27	FDI	▶ K070543	H	L	STEEP SLOPE
*82L08600 17 -	2 M N SICAMOUS	914	1971	3	2	EDI	▷ K070594	H	H	LARGE DEPRESSION/BASIN
	2 M N SICAMOUS	914	1971	0	5	FDI	K070594	H	H	" "

1▶ surveyed
▷ visited

JUN 20, 1988

ONETIME REPORT #75
LIST OF FIR PLANTATIONS DONE
ON OR BEFORE 1977
SORTED BY DISTRICT, OPENING

LAST UPDATE: 88.06.20
KAMLOOPS KAMLOOPS

OPENING	LOCATION	ELEV (M)	REFOR YEAR COMP	REFOR TREES (K)	REFOR (HA)	1ST SPP REFOR	HISTORY RECORD KEY	Rating Before	Rating After	Site Description
82M01100 40	DIXON L	1372	1974	21	30	FDI	[1] ▶ K032060	L	L	STEEP
82M01200 8	S BARRIERE L	1220	1974	8	8	FDI	▶ K032256	H	L	SLOPE
*82M03100 51	BARRIERE RIDGE	1250	1977	23	14	FDI	▶ K032062	H	H	STEEP
82M03100 53	BARRIERE RDG	1400	1971	43	36	FDI	▷ K102273	M	L	"
	BARRIERE RDG	1400	1974	15	11	FDI	K102273	?	-	
82M03100 54	BARRIERE RIDGE	1220	1974	81	60	FDI	▷ K032152	M	L	"
82M03100 55	BARRIERE RIDGE	1219	1971		91	FDI	▷ K027402	M	L	"
82M03100 56	BARRIERE RIDGE	1418	1971	14	12	FDI	▶ K032377	L	L	"
82M03100 58	BARRIERE RIDGE	1067	1971	92	97	FDI	K032247	-	-	
82M04200 24	FENNELL C	1581	1974		37	FDI	▶ K032289	L	L	"
82M04200 25	FENNELL C	1143	1974	17	28	FDI	K032302	L	L	SLOPE
	FENNELL C	1143	1977	28	28	FDI	▶ K032302	H	L	"
92I08300 29	NW MCLEAN L	1220	1976	17	52	FDI	K038617	?	L	"
92P01000 20	BARTLETT C	1220	1975	8	6	FDI	▶ K027447	H	L	HILLY
92P01000 21	BARTLETT C	1220	1975	7	6	FDI	▶ K027351	H	L	GENTLE SLOPE

1 ▶ surveyed
▷ visited

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JUN 20, 1988

ONETIME REPORT #75
LIST OF FIR PLANTATIONS DONE
ON OR BEFORE 1977
SORTED BY DISTRICT, OPENING

LAST UPDATE: 88.06.20
KAMLOOPS CLEARWATER

OPENING	LOCATION	ELEV (M)	REFOR YEAR COMP	REFOR TREES (K)	REFOR (HA)	1ST SPP REFOR	HISTORY RECORD KEY	Rating Before	Rating After	Site Description
82M023008428	HONEYMOON C	909	1974	33	25	EDI	K090790	L	L	STEEP
82M023008431	HONEYMOON C	909	1975	19	17	FDI	K087651	L	L	"
82M023008434	ADAMS L	763	1975		155	EDI	K087669	L	L	"
82M023008435	HONEYMOON C	758	1975	19	16	FDI	K032390	L	L	"
82M033008223	NW OF ADAMS L	610	1973	41	40	FDI	K002827	-	-	---
82M033008224	NW ADAMS L	610	1973	47	34	FDI	K002822	-	-	---
82M043008304 -	TELFER	1005	1974	21	13	FDI	1▶K071126	L	L	"
82M043008315 -	2MS BARTON C	457	1973	17	18	EDI	▶K002777	H	L	FLAT, DEPRESSIONS
82M043008318	MARJORY C	1432	1975	18	14	FDI	K002768	H	L	SLOPE
82M043008343	MOSQUITO C	1219	1977	50	33	FDI	K002586	L	L	"
82M044008012	TELEEK L	1067	1977	71	49	FDI	K002592	L	L	STEEP
82M045008210	MEADOW C	854	1977	63	38	FDI	K103797	H	L	"
82M052005361	3 MI E VAVENBY	914	1972	117	57	FDI	K002427	L	L	GENTLE
82M053005116	GOAT C	1677	1973	18	10	FDI	K002776	L	L	STEEP
82M053008053	8M SE VAVENBY	1036	1973	42	24	FDI	K002708	L	L	"
82M053008054	8M SE VAVENBY	1067	1973	41	36	FDI	K002710	L	L	"
82M055008043	MEADOW C	914	1977	15	10	FDI	K002600	H	L	SLOPE
82M061005028	MCCORVIE C	914	1975	17	20	FDI	K002713	H	H	FLAT, VALLEY BOTTOM
82M063002060	LONG EIRE	1212	1972	140	80	EDI	K002972	H	L	SLOPE
82M064005046	OTTER CR	1219	1973	91	67	FDI	K002440	L	L	STEEP
82M064005057 -	OTTER CR	909	1968		43	FDI	▷K002434	L	L	HILLY
-	OTTER CR	909	1973		40	FD	▷K002434	L	L	"
82M064005061 -	OTTER L	914	1968	50	22	EDI	▶K003089	H	L	FLAT, VALLEY BOTTOM
82M064006051 -	OTTER C	1068	1973	53	56	FD	▷K002601	L	L	SLOPE
82M064006054 -	OTTER CREEK	1068	1968		3	FD	▷K002599	H	L	GENTLE SLOPE
	OTTER CREEK	1068	1973	133	23	FD	K002599	-	-	---
82M072004012 -	BAEI R	780	1975	52	44	EDI	▶K002574	H	H	FLAT, VALLEY BOTTOM
82M072005036	MOILLIET C	762	1975	15	17	FDI	K002540	H	H	"
82M094006048	FINN C	715	1976	23	18	FDI	K031839	H	L	STEEP

1▶ surveyed
▷ visited

JUN 20, 1988

ONETIME REPORT #75
LIST OF FIR PLANTATIONS DONE
ON OR BEFORE 1977
SORTED BY DISTRICT, OPENING

LAST UPDATE: 88.06.20
KAMLOOPS SALMON ARM

OPENING	LOCATION	ELEV (M)	REFOR YEAR COMP	REFOR TREES (K)	REFOR (HA)	1ST SPP REFOR	HISTORY RECORD KEY	Rating Before	Rating After	Site Description
82L08600 39	WUEEST M	1220	1973	1	1	FDI	K070675	-	-	
82L09300 3	RUBY	1550	1963	58	48	FDI	K001772	H	L	STEEP, NARROW DRAINED GULLIES
82L09500 12	EAGLE BAY	680	1970	241	175	FDI	K090446	H	L	GENTLE SLOPE
82L09500 22	WHITE LAKE	670	1973	14	22	FDI	K026036	L	L	SLOPE
82L09500 25	EAGLE BAY	700	1973		29	FDI	K090448	H	L	"
82L09700 11	1 MI N CRAIGELL	700	1971	41	11	FDI	K070557	H	L	STEEP
82L09700 14	1MI NW CRAIGELL	640	1971	36	30	FDI	K070556	H	L	"
82M00400 36	VEGETATION C	1067	1972	45	40	FDI	K090401	L	L	"
82M00400 42	VEGETATION C	1524	1968		8	FDI	K010876	L	L	UNDULATING, TINY POCKETS
82M00400 85	W OF ROSS C	1371	1976	40	50	FDI	K010843	L	L	" "
82M00500 1	- NW OF ROSS CR	1060	1971	40	35	FDI	1 ▷K104175	L	L	STEEP
82M00500 3	- NW OF ROSS CR	1213	1971	91	82	FDI	▷K104174	H	L	"
82M00500 19	- SE ROSS C	916	1971	4	4	FDI	▷K071024	-	L	"
82M00500 33	- N OF ROSS C	991	1968		167	FDI	▶K010893	H	L	LG. SLOPED BASIN
	N OF ROSS C	991	1971	25	21	FDI	K010893	H	L	STEEP
82M00500 35	- N OF ROSS C	762	1968	77	53	FDI	▶K010894	H	L	"
	N OF ROSS C	762	1971	21	17	FDI	K010894	H	L	"
82M00500 39	- NW OF RUCKELL P	976	1971	196	158	FDI	▷K010854	L	L	"
82M00500 42	NW OF RUCKELL P	732	1968		55	FDI	K010833	H	L	STEEP, 1 SMALL DEPRESSION
82M00500 43	- NW OF RUCKELL P	488	1968		25	FDI	▶K104761	L	L	STEEP
82M00500 44	- NW OF RUCKELL P	610	1968		50	FDI	▷K010830	L	L	"
82M00500 45	- NW OF RUCKELL P	1220	1977	23	19	FDI	▷K010840	L	L	"
82M00500 51	- NW OF RUCKELL P	1067	1977	13	9	FDI	▷K010839	L	L	"
82M00700 3	PERRY R	750	1973	90	105	FDI	K025810	L	L	"
82M01500 45	- FIVE MILE CR.	915	1972	250	207	FDI	▷K010880	L	L	SLOPED
82M01700 37	PERRY R	600	1973	23	48	FDI	K025808	-	-	
82M02500 22	HUMAMILT L	1067	1977	82	56	FDI	K001736	H	L	GENTLE SLOPES
82M02500 46	4 M W ALBAS	1219	1975	59	71	FDI	K001716	L	L	STEEP
82M02600 1	- HUMAMILT L	762	1977	2	2	FDI	▷K001477	L	L	"
82M02600 2	- HUMAMILT L	762	1977	37	34	FDI	▶K001478	L	L	"

1▶ surveyed
▷ visited

CAMILLE LAKE CLIMATE SUMMARY FOR OCTOBER 1984
B.C. REGIONAL ID : 11301
AES HEADQUARTERS ID : 1091235

DAY	TEMPERATURE MAX	MIN	MEAN	TOTAL RAIN	SNOW	PCP	THRA	SNOW GND
1	13.5	-4.0	4.8	2.4		2.4		0
2	17.0	6.0	11.5	T		T		0
3	18.0	6.5	12.3					0
4	19.0	12.0	15.5					0
5	14.5	-2.0	6.3					0
6	16.0	2.0	9.0					0
7	19.0	12.0	15.5					0
8	21.0	15.0	18.0	5.6		5.6		0
9	17.0	3.0	10.0					0
10	11.0	9.0	10.0	8.2		8.2		0
11	8.0	3.5	5.8	T		T		0
12	10.0	-2.0	4.0	T		T		0
13	11.0	3.0	7.0					0
14	9.0	-2.0	3.5		T	T		0
15	7.0	-2.0	2.5					0
16	6.0	-10.0	-2.0		T	T		0
17	2.0	-3.5	-.8					M
18	2.5	-12.0	-4.8					M
19	3.0	-11.5	-4.3					0
20	7.5	-9.5	-1.0					0
21	8.0	-8.0	.0					0
22	8.0	-8.0	.0	T	T	T		0
23	5.0	-4.5	.3	1.0	T	1.0		0
24	3.5	-3.0	.3	4.0E	1.0E	5.0E		0
25	6.5	-1.0	2.8	T		T		0
26	2.5	-3.0	-.3		2.6	2.6		0
27	-7.5	-8.5	-8.0		6.0	6.0		2.
28	-11.5	-15.5	-13.5		T	T		8.
29	-8.0	-26.0	-17.0		5.0	5.0		8.
30	-17.0	-18.0	-17.5					13.
31	-12.5	-31.5	-22.0		T	T		13.
TOTAL	209.0	-113.5		21.2	14.6	35.8	0 0 0	
MEAN	6.7	-3.7	1.5					

MONTHLY MAXIMUM TEMPERATURE WAS 21.0 ON DAY 8
MONTHLY MINIMUM TEMPERATURE WAS -31.5 ON DAY 31
HIGHEST RAINFALL WAS 8.2 ON DAY 10
HIGHEST SNOWFALL WAS 6.0 ON DAY 27
MAX TOTAL PRECIP WAS 8.2 ON DAY 10
NUMBER OF DAYS WITH MEASUREABLE PRECIPITATION IS 8
NUMBER OF DAYS WITH MEASUREABLE RAINFALL IS 5
NUMBER OF DAYS WITH MEASUREABLE SNOWFALL IS 4

NUMBER OF HEATING DEGREE-DAYS FOR MONTH IS 510.1

LIST OF CODES USED IS AVAILABLE ON REQUEST

CAMILLE LAKE CLIMATE SUMMARY FOR NOVEMBER 1984
B.C. REGIONAL ID : 1130I
AES HEADQUARTERS ID : 1091235

DAY	TEMPERATURE MAX	MIN	MEAN	TOTAL RAIN	SNOW	PCP	THRA	SNOW GND
1	-3.5	-25.0	-14.3		7.0	7.0		17.
2	-4.0	-16.0	-10.0		7.2	7.2		19.
3	-2.0	-9.0	-5.5		2.2	2.2		21.
4	6.0	-6.0	.0					21.
5	5.0	-10.0	-2.5		2.0	2.0		20.
6	2.5	-2.5	.0	T	1.0	1.0		20.
7	6.5	-3.0	1.8					18.
8	5.0	-10.5	-2.8		T	T		18.
9	5.5	-3.0	1.3					18.
10	3.0	-10.5	-3.8		3.6	3.6		18.
11	0.0	-3.0	-1.5	T	T	T		21.
12	-9.5	-10.5	-10.0		8.0	8.0		21.
13	-9.0	-11.0	-10.0		1.0	1.0		25.
14	-6.0	-18.5	-12.3		9.4	9.4		25.
15	-1.0	-21.0	-11.0					32.
16	1.0	-12.0	-5.5					30.
17	3.0	-6.0	-1.5		1.0	1.0		29.
18	5.5	-3.0	1.3					28.
19	5.0	-4.5	.3					27.
20	3.0	-10.5	-3.8					25.
21	2.0	-12.0	-5.0					25.
22	5.0	-2.0	1.5	1.0		1.0		24.
23	3.0	0.0	1.5	4.4	T	4.4		23.
24	0.0	-5.5	-2.8					22.
25	-2.5	-13.0	-7.8		T	T		21.
26	-2.0	-18.0	-10.0		4.0	4.0		21.
27	-2.0	-7.0	-4.5		5.4	5.4		25.
28	-1.0	-3.5	-2.3		1.6	1.6		30.
29	-4.5	-20.0	-12.3		1.0	1.0		31.
30	-6.5	-11.0	-8.8		T	T		31.
TOTAL	7.5	-287.5		5.4	54.4	59.8	0 0 0	
MEAN	.3	-9.6	-4.7					

MONTHLY MAXIMUM TEMPERATURE WAS 6.5 ON DAY 7
MONTHLY MINIMUM TEMPERATURE WAS -25.0 ON DAY 1
HIGHEST RAINFALL WAS 4.4 ON DAY 23
HIGHEST SNOWFALL WAS 9.4 ON DAY 14
MAX TOTAL PRECIP WAS 9.4 ON DAY 14
NUMBER OF DAYS WITH MEASUREABLE PRECIPITATION IS 16
NUMBER OF DAYS WITH MEASUREABLE RAINFALL IS 2
NUMBER OF DAYS WITH MEASUREABLE SNOWFALL IS 14

NUMBER OF HEATING DEGREE-DAYS FOR MONTH IS 680.3

LIST OF CODES USED IS AVAILABLE ON REQUEST

COMPARATIVE RECORDS AT: RELEVÉS COMPARATIFS À: PRINCE GEORGE AIRPORT, BRITISH COLUMBIA — MONTH MOIS: OCTOBER / OCTOBRE 19 89

Temperature/Température — ° Celsius
Precipitation/Précipitation — Millimetres/millimètres (mm)
Rainfall/Hauteur de pluie — Millimetres/millimètres (mm)
Snowfall/Hauteur de neige — Centimetres/centimètres (cm)
Wind speed/Vitesse du vent — Kilometres/h / kilomètres/h (km/h)
Station pressure/Pression à la station — Kilopascals (kPa)

	THIS MONTH CE MOIS-CI		PREVIOUS YEAR ANNÉE PRÉCÉDENTE		NORMAL NORMALE	RECORD FOR THE MONTH RECORD POUR LE MOIS: HIGHEST EVER MAXIMUM ABSOLU			LOWEST EVER MINIMUM ABSOLU			NO OF YEARS NB D'ANNÉES
	VALUE RELEVÉ	DAY JOUR	VALUE RELEVÉ	DAY JOUR		VALUE RELEVÉ	DAY JOUR	YEAR ANNÉE	VALUE RELEVÉ	DAY JOUR	YEAR ANNÉE	
HIGHEST TEMPERATURE (MAXIMUM) TEMPÉRATURE MAXIMALE	19.1	9	24.1	4		25.2	1	1987				48
LOWEST TEMPERATURE (MINIMUM) TEMPÉRATURE MINIMALE	-9.8	31	-12.1	28					-26.5	31	1984	48
MEAN MONTHLY TEMPERATURE TEMPÉRATURE MENSUELLE MOYENNE	5.1		6.2		4.8	7.1		1944	1.5		1984	48
TOTAL MONTHLY RAINFALL HAUTEUR TOTALE MENSUELLE DE PLUIE	33.1		47.6		49.9	117.6		1962	16.3		1945	48
TOTAL MONTHLY SNOWFALL HAUTEUR TOTALE MENSUELLE DE NEIGE	.8		TR		9.1	40.9		1945	.0		1987 **	48
TOTAL MONTHLY PRECIPITATION PRÉCIPITATION TOTALE MENSUELLE	33.7		47.6		59.2	122.3		1962	23.9		1970	48
NO OF DAYS WITH MEASURABLE PRECIPITATION NOMBRE DE JOURS AVEC PRÉCIPITATION MESURABLE	14		14		14	24		1973	7		1970	48
GREATEST RAINFALL IN ONE DAY HAUTEUR DE PLUIE MAXIMALE EN UNE JOURNÉE	12.8	18	10.4	29		38.9	3	1962				48
GREATEST SNOWFALL IN ONE DAY HAUTEUR DE NEIGE MAXIMALE EN UNE JOURNÉE	.8	29	TR	26*		22.1	25	1971				48
GREATEST PRECIPITATION IN ONE DAY PRÉCIPITATION MAXIMALE EN UNE JOURNÉE	12.8	18	10.4	29		38.9	3	1962				48
MAXIMUM RAINFALL RECORDED IN: HAUTEUR DE PLUIE MAXIMALE ENREGISTRÉE EN:												
5 MINUTES	.8	21	1.2	15		2.5	1	1974				30
10 MINUTES	1.2	18	1.6	15		4.3	1	1974				30
15 MINUTES	1.6	18	2.4	15		4.6	1	1974				30
30 MINUTES	2.6	18	5.1	15		5.1	15	1988				30
60 MINUTES	5.0	18	6.7	15		6.7	15	1988				30
24 CONSECUTIVE HOURS HEURES CONSÉCUTIVES	12.8	18	10.4	29		38.9	3	1962				48
MEAN WIND SPEED (km/h) VITESSE MOYENNE DU VENT (km/h)	9.7		7.7		12.5	18.8		1961	5.2		1969	48
MAXIMUM SPEED (2 min. mean) (km/h) VITESSE MAXIMALE (moyenne sur 2 min.) (km/h)	W* 30	9	W 41	21		S 93	24	1955				48
MAXIMUM GUST SPEED (km/h) POINTE DU VENT MAXIMALE (km/h)	SW 56	9	W 72	21		W 118	31	1961				48
TOTAL HOURS OF SUNSHINE TOTAL DES HEURES INSOLATION	97.8		133.8		109.7	157.0		1987	72.5		1968	42
MEAN STATION PRESSURE (kPa) PRESSION MOYENNE À LA STATION (kPa)	93.36		93.49		93.42	94.02		1951	92.83		1947	48
GREATEST STATION PRESSURE (kPa) PRESSION MAXIMALE À LA STATION (kPa)	94.93	31	94.94	8		95.86	21	1953				48
LEAST STATION PRESSURE (kPa) PRESSION MINIMALE À LA STATION (kPa)	91.30	21	91.37	21					90.05	27	1950	48

CLIMATOLOGICAL DATA THIS MONTH FOR THE PAST TEN YEARS
DONNÉES CLIMATOLOGIQUES CE MOIS-CI POUR LES DERNIÈRE ANNÉES

YEAR ANNÉE	MAXIMUM TEMP. TEMP. MAXIMALE	MINIMUM TEMP. TEMP. MINIMALE	MEAN TEMP. TEMP. MOYENNE	RAINFALL HAUTEUR DE PLUIE	SNOWFALL HAUTEUR DE NEIGE	TOTAL PRECIP PRÉCIP. TOTALE	MEAN WIND SPEED VITESSE MOYENNE DES VENTS	MAXIMUM WIND SPEED VITESSE MAXIMALE DES VENTS	SUNSHINE HOURS HEURES INSOLATION	HEATING DEGREE-DAYS DEGRÉS-JOURS DE CHAUFFE	GROWING DEGREE-DAYS DEGRÉS-JOURS DE CROISSANCE	COOLING DEGREE-DAYS DEGRÉS-JOURS DE REFROIDISSEMENT	
1980	23.1	-5.9	6.2	32.0	TR	32.0	7.0	S 43	111.2	367.5	70.5		
1981	15.5	-8.2	5.0	42.6	1.8	44.4	8.6	S 48	118.2	403.2	39.6		
1982	19.6	-7.0	5.7	39.1	3.8	44.0	11.4	S 50	112.2	382.1	64.5		
1983	14.5	-6.1	4.8	47.8	.2	48.0	11.1	S 50	95.1	406.4	27.5		
1984	19.9	-26.5	1.5	93.4	13.0	103.2	10.5	S 48	87.7	510.9	58.9		
1985	14.5	-11.3	3.5	98.1	15.5	112.6	12.5	S 41	103.5	449.9	12.6		
1986	18.9	-12.5	6.7	26.0	.0	26.0	9.2	S 31	144.9	352.0	78.6		
1987	25.2	-9.5	5.2	39.8	.0	39.8	9.1	W 46	157.0	397.7	54.5		
1988	24.1	-12.1	6.2	47.6	TR	47.6	7.7	W 41	133.8	365.0	80.6		
1989	19.1	-9.8	5.1	33.1	.8	33.7	9.7	SW 56	97.8	402.0	47.1		

Notes/Avis
1. Climatological Day/Journée climatologique 22 01 P S.T. – 22 00 P S.T.
2. Normal/Normale 1951-1980
3. Extremes for period of record/Extrêmes pour la période de registre
4. Maximum rainfall recorded in: may overlap calendar days/Hauteur de pluie maximale enregistrée en: peut-être pour plus d'une journée du calendrier
5. ** Indicates most recent occurrence/Indique le plus récent
6. * Indicates first of more than one occurrence/Indique le premier de plusieurs

COMPARATIVE RECORDS
RELEVÉS COMPARATIFS

PRINCE GEORGE AIRPORT, B.C.

MONTH / MOIS: OCTOBER

1984

Temperature/Température – °Celsius
Precipitation/Précipitation – Millimetres (mm)
Rainfall/Hauteur de pluie – Millimetres (mm)
Snowfall/Hauteur de neige – Centimetres (cm)
Wind speed/Vitesse du vent – Kilometres per hour/par heure (km/h)
Station pressure/Pression à la station – Kilopascals (kPa)

	THIS MONTH CE MOIS-CI		PREVIOUS YEAR ANNÉE PRÉCÉDENTE		NORMAL NORMALE	RECORD FOR THE MONTH RECORD POUR LE MOIS						YEARS OF RECORD ANNÉES D'OBSERVATIONS
						HIGHEST MAXIMUM ABSOLU			LOWEST MINIMUM ABSOLU			
	VALUE RELEVÉ	DATE	VALUE RELEVÉ	DATE		VALUE RELEVÉ	DAY JOUR	YEAR ANNÉE	VALUE RELEVÉ	DAY JOUR	YEAR ANNÉE	
HIGHEST TEMPERATURE / TEMPÉRATURE LA PLUS ÉLEVÉE	19.9	4	14.5	11		25.0	2	1943	11.1	22	1973	43
LOWEST TEMPERATURE / TEMPÉRATURE LA PLUS BASSE	-26.5	31	-6.1	24		-3.3	26	1967	-26.5	31	1984	43
MEAN MONTHLY TEMPERATURE / TEMPÉRATURE MENSUELLE MOYENNE	1.5		4.9		4.8	7.1		1944	1.5		1984	43
TOTAL MONTHLY RAINFALL / HAUTEUR TOTALE MENSUELLE DE PLUIE	93.4		47.8		49.9	117.6		1962	16.3		1945	43
TOTAL MONTHLY SNOWFALL / HAUTEUR TOTALE MENSUELLE DE NEIGE	13.0		0.2		9.1	40.9		1945	0.0		1969	43
TOTAL MONTHLY PRECIPITATION / PRÉCIPITATION TOTALE MENSUELLE	103.2		48.0		59.2	122.3		1962	23.9		1970	43
NO OF DAYS WITH MEASURABLE PRECIPITATION / NOMBRE DE JOURS AVEC PRÉCIPITATION MESURABLE	18		18		14	24		1973	7		1970	42
GREATEST RAINFALL IN ONE DAY / HAUTEUR DE PLUIE MAXIMALE EN UNE JOURNÉE	36.9	8	10.2	26		38.9	3	1962				43
GREATEST SNOWFALL IN ONE DAY / HAUTEUR DE NEIGE MAXIMALE EN UNE JOURNÉE	4.4	27,29	0.2	24		22.1	25	1971				42
GREATEST PRECIPITATION IN ONE DAY / PRÉCIPITATION MAXIMALE EN UNE JOURNÉE	36.9	8	10.2	26		38.9	3	1962				43
MAXIMUM RAINFALL RECORDED IN: / HAUTEUR DE PLUIE MAXIMALE ENREGISTRÉE EN:												
5 MINUTES	1.0	8	0.9	3		2.5	1	1974				25
10 MINUTES	1.6	8	0.9	avr1		4.3	1	1974				25
15 MINUTES	2.1	8	1.3	3		4.6	1	1974				25
30 MINUTES	3.3	8	2.0	3		5.1	1	1974				25
60 MINUTES	4.3	8	2.4	18		5.8	5	1975				25
24 CONSECUTIVE HOURS / HEURES CONSECUTIVES	38.8	7-8	11.2	26-27		38.9	3	1962				43
MEAN WIND SPEED (km/h) / VITESSE MOYENNE DU VENT (km/h)	10.5		11.1		12.5	18.8		1961	5.2		1969	43
MAXIMUM SPEED (1 min.) (km/h) / VITESSE MAXIMALE (1 min.) (km/h)	S 48	12	S 50	24		S 93	24	1955				43
MAXIMUM GUST SPEED (km/h) / POINTE DU VENT MAXIMALE (km/h)	S 76	9	S 80	24		WNW 148	31	1961				42
TOTAL HOURS OF SUNSHINE / TOTAL DES HEURES INSOLATION	87.7		95.1		109.7	151.8		1972	63.8		1960	37
MEAN STATION PRESSURE (kPa) / PRESSION MOYENNE A LA STATION (kPa)	93.15		93.30		93.42	94.02		1951	92.83		1947	43
GREATEST STATION PRESSURE (kPa) / PRESSION MAXIMALE A LA STATION (kPa)	94.60	18	94.70	11		95.86	21	1953				43
LEAST STATION PRESSURE (kPa) / PRESSION MINIMALE A LA STATION (kPa)	91.08	12	92.16	21					90.05	27	1950	42

CLIMATOLOGICAL DATA FOR THE PAST TEN YEARS
DONNÉES CLIMATOLOGIQUES POUR LES DERNIÈRE ANNÉES

YEAR ANNÉE	MAXIMUM TEMP. TEMP. MAXIMALE	MINIMUM TEMP. TEMP. MINIMALE	MEAN TEMP. TEMP. MOYENNE	RAINFALL HAUTEUR DE PLUIE	SNOWFALL HAUTEUR DE NEIGE	TOTAL PRECIP. PRECIP. TOTALE	MEAN WINDSPEED VITESSE MOYENNE DES VENTS	MAXIMUM WINDSPEED VITESSE MAXIMALE DES VENTS	HOURS SUNSHINE HEURES INSOLATION	HEATING DEGREE-DAYS DEGRÉS-JOURS DE CHAUFFE	GROWING DEGREE-DAYS DEGRÉS-JOURS DE CROISSANCE		
1975	21.2	-4.3	4.9	31.6	6.2	37.8	6.2	SSW 35	87.2	406	45		
1976	15.1	-7.1	4.4	52.9	TR	52.9	10.1	S 51	106.4	423	36		
1977	16.2	-8.4	5.2	21.2	2.6	23.8	13.3	S 67	108.3	398	35		
1978	18.8	-5.5	6.3	35.8	11.2	48.6	13.0	S 48	140.6	363	73		
1979	22.0	-4.9	6.1	51.1	2.4	53.3	7.9	S 37	115.8	370	74		
1980	23.1	-5.9	6.2	32.0	TR	32.0	7.0	S 43	111.2	368	71		
1981	15.5	-8.2	5.0	42.6	1.8	44.4	8.6	S 48	118.2	403	40		
1982	19.6	-7.0	5.7	39.1	3.8	44.0	11.4	S 50	112.2	382	65		
1983	14.5	-6.1	4.8	47.8	0.2	48.0	11.1	S 50	95.1	406	28		
* 1984	19.9	-26.5	1.5	93.4	13.0	103.2	10.5	S 48	87.7	511	59		

* = latest occurrence