Branch and Leader Dieback of Young Douglas-fir in the Prince George Forest Region

by

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TABLE OF CONTENTS

Title Page	i
Table of Contents	ii
List of Figures	iii
List of Tables	iv
Abstract	1
Introduction	1
Symptoms	4
Survey of Douglas-fir Plantations	6
Materials and Methods	6
Results	10
Discussion	13
Acknowledgement	14
References	15

LIST OF FIGURES

Figure		Page
1	The Prince George Forest Region	2
2	Complete top-kill on immature Douglas-fir due to *Sclerophoma* spp.	5
3	Multiple leaders on immature Douglas-fir resulting from attack by *Sclerophoma* spp. - note dead original leader (stub) killed by the disease.	7
4	Crook in stem of immature Douglas-fir as a result of *Sclerophoma* spp. Note original leader killed by the disease.	8
5	Approximate locations of Douglas-fir stands sampled for incidence of *Sclerophoma* spp.	9

LIST OF TABLES

Table		Page
I	Young stands in the Prince George Forest Region sampled for Douglas-fir leader dieback, and the percent of surveyed trees with the disease.	11

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ABSTRACT

Surveys of 10 to 15 year-old Douglas-fir plantations in the Prince George Forest Region indicated varying incidences of a branch and leader dieback. This report details the occurrence of the dieback as recorded in limited surveys. Disease symptomology is described. The significance of this disease with respect to Douglas-fir management and necessary future work is discussed.

INTRODUCTION

Douglas-fir, *Pseudotsuga menziesii* Mirb. (Franco), in the Prince George Forest Region is at the northern-most portion of its range. It occurs

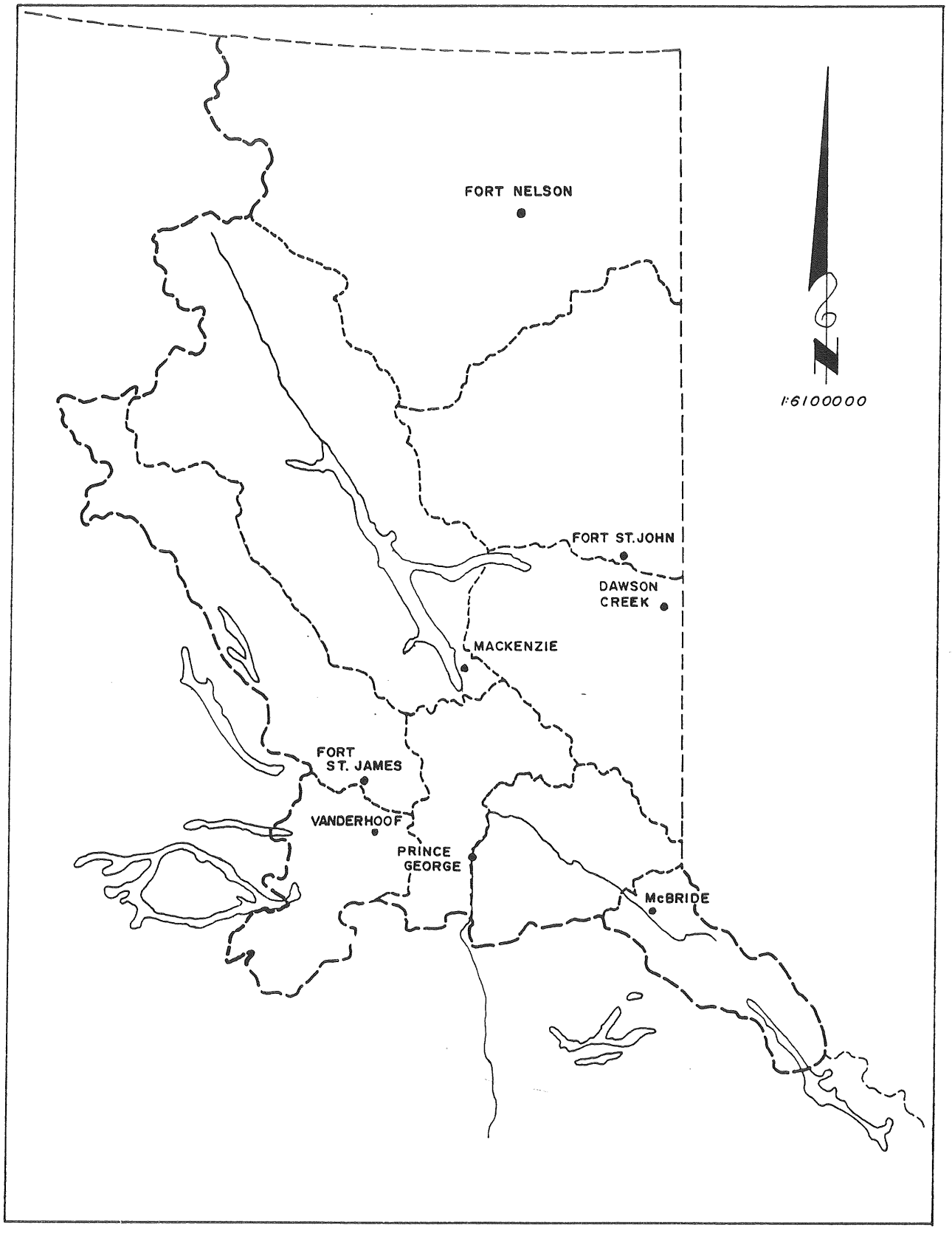

Figure 1- The Prince George Forest Region

naturally in five Forest Districts (Fig. 1): Prince George West, Prince George East, McBride, Vanderhoof and Fort St. James (Hosie 1975).

Douglas-fir prefers mild, humid climates and well-drained soils. In the Prince George Forest Region, Douglas-fir is usually found on southern aspects at elevations of less than 830 metres (m) (Fowells 1965) in the Sub Boreal Spruce biogeoclimatic zone.

Since 1963, 242 plantings of Douglas-fir have been recorded throughout the Region. Some of these are small areas within larger spruce or pine blocks. The high value of Douglas-fir compared to other species has resulted in some off-site plantations where performance of the trees is less than optimal and susceptibility to disease is increased. However, there exists in the Prince George Forest Region an opportunity to grow productive stands of Douglas-fir. This is especially attractive in view of the fact that *Phellinus weirii*, a root rot which has a major impact on the management of Douglas-fir on the coast and southern interior, has not been reported to be a problem in the Prince George Forest Region.

Branch flagging and leader dieback were first observed by the authors during the summer of 1986 in several Douglas-fir plantations, approximately 15 years old. Further enquiries revealed other occurences of the disease, including reports from homeowners with Douglas-fir trees on their property.

The purpose of this report is to provide some background information on

this disease and to report the results of a survey of selected Douglas-fir plantations. There are four objectives in this study:

1- to describe the symptoms and determine the causal agent;

2- to survey a representative sample of Douglas-fir plantations to determine the percent affected and the severity of attack;

3- to estimate the impact of the dieback on the economic worth of the future Douglas-fir crop; and,

4- to make recommendations regarding the management of the disease.

SYMPTOMS

The disease affects both leaders and individual branches. Leader and top dieback has been observed to extend down 75% of the stem. In the case of branch flagging, a single dead branch is seen, usually there is a slight swelling or "rippling" of the bark around the node. Frequently the dead tissue is limited to the branch only (including the portion within the stem), the remainder of the node appearing healthy. In some cases, the disease has progressed into the main stem causing typical canker symptoms: areas of discolored, sunken bark where the cambium has been killed. Open cankers are sometimes observed along the branch or stem. These resemble cankers caused by *Pragmopora pithya* (Fr.) Groves. Superficial cankers are frequently observed where the bark has cracked but the wood beneath is not affected.

Infection in the stem may result in complete top kill (Fig. 2), multiple

Figure 2- Complete top-kill on immature Douglas-fir due to *Sclerophoma* spp.

leaders (Fig. 3), or a crook in the stem (Fig. 4). Where top kill occurs, the disease has invaded the cambium in the node around the base of the leader. Sometimes the leader only is killed and one or more laterals takeover. Areas of discolored, sunken bark are seen around the base of the leader, open cankers may be observed at the node or up the stem.

SURVEY OF DOUGLAS-FIR PLANTATIONS

Materials and Methods

All of the Forest Districts within the Prince George Forest Region with Douglas-fir plantations were contacted and asked to supply a listing of those plantations, complete with maps and planting reports. A sampling of these plantations were surveyed (Fig. 5) in this initial assessment of the potential problem.

A strip survey was conducted in each plantation. Continuous strips, five metres in width, were located to sample each area uniformly. All trees within the strips were recorded in one of the following categories:

a- branch dieback;

b- leader dieback (including top kill originating further down the stem);

c- both branch and leader dieback; or,

d- asymptomatic.

General, subjective observations were made on overall site productivity,

Figure 3– Multiple leaders on immature Douglas-fir resulting from attack by *Sclerophoma* spp. -- note dead original leader (stub) killed by the disease.

Figure 4- Crook in stem of immature Douglas-fir as a result of *Sclerophoma* spp. Note original leader killed by the disease

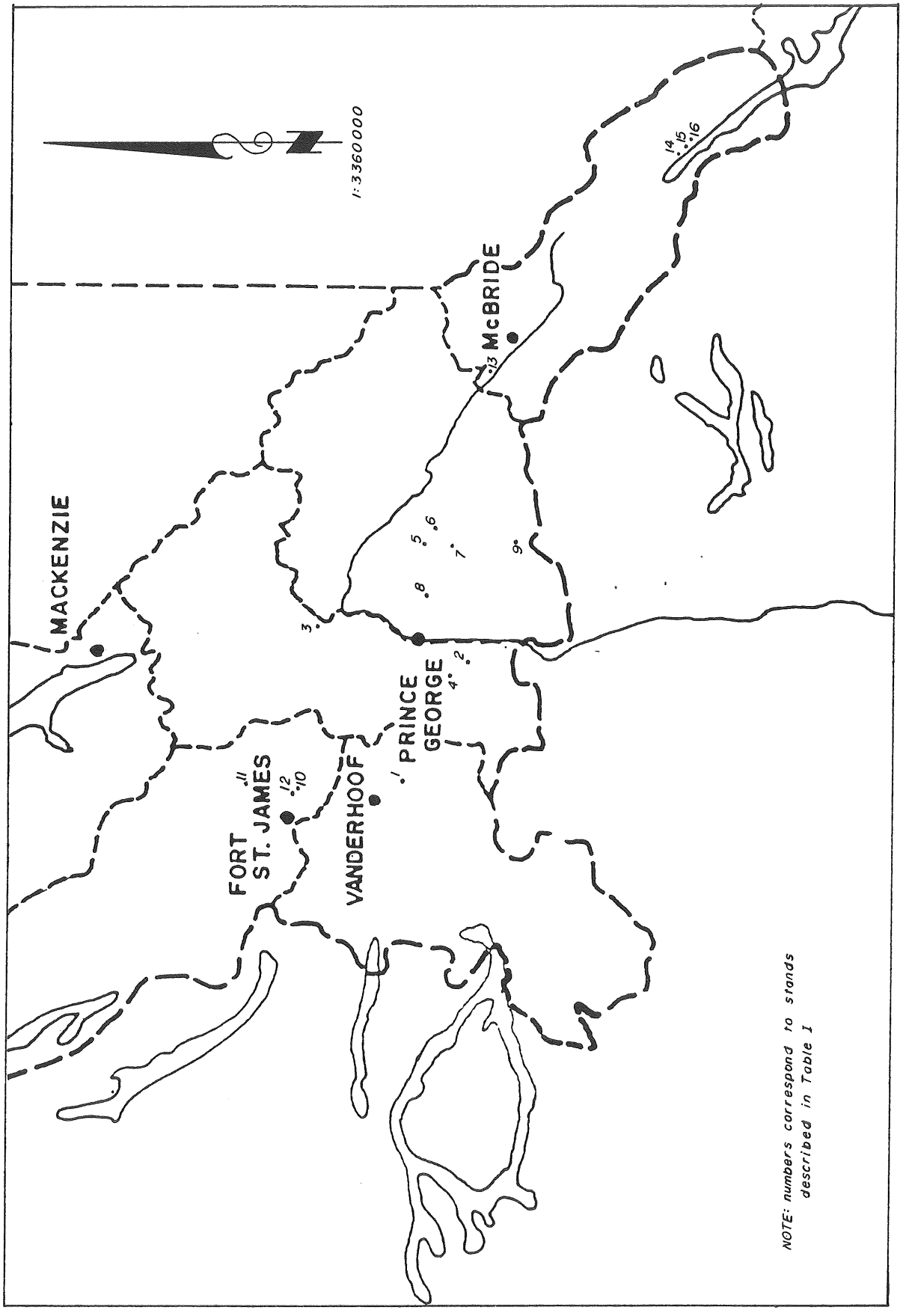

Figure 5- Approximate locations of Douglas-fir stands sampled for incidence of *Sclerophoma* spp.

vigour of the Douglas-fir trees and other problems occuring in the Douglas-fir were recorded. The percent of trees in each damage category were calculated.

Samples were taken from various plantations for microscopic examination and isolation onto malt extract agar (MEA). Samples were surface sterilized by swabbing with 95% EtOH and flaming briefly. Subsamples of bark or wood tissue were aseptically removed from canker margins and plated on 1.5% MEA. The plates were incubated at room temperature ($18^{o}C$-$20^{o}C$) for ten days. Cultures were examined microscopically and identified.

Results

The survey results are summarized in Table I. The percent of trees affected by the disease ranged from zero to 70 (Fig. 6). Based on the data and observarions collected during the course of this study, the following observations were made:

(i) Damage to individual trees was much more severe, and the number of trees attacked much greater in plantations where growth of the asymptomatic Douglas-fir was poor such as in off-site plantations. Vigorous trees appeared to be able to confine the infection to a branch or resist infection completely.

(ii) Natural trees 25-35 years old with the disease had little top

Table I- Young stands in the Prince George Forest Region sampled for Douglas-fir leader dieback, and the percent of surveyed trees with the disease.

		Stand Description		Percent Attack				
No.[1]	District	Establishment Year	Identification	Leader	Branch	Both	Overall	Comments
1	Vanderhoof	1967	EP 660	35	-	-	-	moderate site
2	PG West	1981	93G10D-7	-	-	-	1.1	nutrient problem suspected
3	PG West	1971	93J7A-8	33.0	12.8	10.3	44.3	good growth
4	PG West	1963	93G10E-13	11.9	20.9	37.3	70.1	poor site
5	PG East	1973	93G10O-15	9.1	9.1	3.0	21.2	med-poor growth
6	PG East	1973	93H082-10	-	-	-	22.0	spotty attack
7	PG East	1971N[2]	Beaver Rd - 83km	25.0	3.6	0	28.6	med site
8	PG East	1961N	Tabor Mtn.	-	-	-	33.0	some dieback
9	PG East	1976	93-804-132	45.0	-	-	45.0	good growth
10	FS James	1974	93J5F-4	25.0	3.6	14.3	42.9	good growth
11	FS James	1973	93J13D-8	23.8	0	0	23.8	poor site
12	FS James	1956N	adjacent to Harry Lake	-	-	-	30.0	no top dieback multiple leader and crooks
13	McBride	1975	93H10-9	12.0	6.0	18.0	64	med-poor site
14	McBride	1971	83D11-3	LT 1[3]	0	0	LT 1	good growth
15	McBride	1975	83D11-14	LT 1	0	0	LT 1	good growth some Armillaria
16	McBride	1971	83D11-2	0	0	0	0	excellent growth

[1] Stands can be located on Figure 5
[2] N = Natural Stand
[3] LT 1 = less than 1

kill; the main damage being stem crooks and multiple leaders.

(iii) Leaders were more commonly attacked than branches. The infection always appeared to originate at a branch node.

(iv) Trees in young plantations were not as susceptible to the disease. The critical age for susceptibility as determined in this brief study lies between 10 and 20 years.

Fruiting bodies were seen on dead leader and branch tissue. These are described as small black pycnidia (approximately 0.5 mm in diameter), erumpent and sessile but wih an immersed stem. They are irregular in shape. Conidiogenous cells line the inner wall, short globose to cylindric, phialidic. Phialospores ovate. Samples of the disease were sent to the Pacific Forestry Center in Victoria for identification; the major pathogen was identified as *Sclerophoma semenospora*. The open, superficial cankers were not thought important and their cause was not identified.

There are three species of *Sclerophoma* which are involved in dieback diseases: *Sclerophoma semenospora*, *S. pithyophila*, and *S. xenomeris* (*Xenomeria abietis* Barr). Funk (1980) documents some of the morphological and cultural differences between these three fungi.

Cultures made from diseased tissue consistently yielded a black colony with *Hormonema* characteristics. This identification was confirmed by Al

Funk[1] as the *Hormonema* state of *Sclerophoma*.

DISCUSSION

Consistent association of pycnidia on diseased tissue and isolation of the *Hormonema* state confirm *Sclerophoma spp.* as the causal agent for the leader and branch dieback on young Douglas-fir. There may be a species complex involved and it is certain that site and climatic influences are pertinent to disease development. Healthy, 'on-site' trees appear to be able to withstand or confine infection whereas 'off-site' trees are often damaged to the point of no longer being a potential crop tree. Therefore, the effect that this disease may have on Douglas-fir management may be influenced by silvicultural practices (such as planting prescriptions) and climate. The disease may add pathological constraints to the sites suitable for growing Douglas-fir. Established plantations of Douglas-fir are susceptible to major losses particularily if they are off-site or have been exposed to severe drought or frost conditions.

Previous studies of natural infections have indicated that *Xenomeris abietis (S. xenomeria)* is a weak pathogen, associated with other pathogens and weakened trees. Pathogenicity tests with this fungus have

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1 Dr. A. Funk. Forest Pathologist. Canadian Forestry Service. Pacific Forestry Centre, 506 West Burnside Rd., Victoria, B.C. personal communication.

been inconclusive (Funk and Shoemaker 1971) but do show that the fungus is capable of killing phloem tissue. The other two *Sclerophomas* are also found in association with other pathogens (Funk 1980; Funk and Shoemaker 1971). The status of *Sclerophoma* as a pathogen remains unclear but this study shows it to be a definite threat to Douglas-fir management, especially in conjunction with site related stress.

Further work is needed to:

1- establish the incidence of the disease in established Douglas-fir plantations;

2- correlate site with disease severity;

3- accurately determine the age of susceptibility; and,

4- define the characteristics which make a tree less (or more) susceptible to infection or damage.

Once this definitive work has been completed, the results can be incorporated into silvicultural practices to enhance the management of Douglas-fir in the Prince George Forest Region.

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