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Mortality and basal area growth following precommercial thinning in stands affected by Armillaria, Laminated and Tomentosus root diseases in southern British Columbia

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Abstract

Precommercial thinning aims to reduce the density of immature stands to stimulate growth of well-spaced crop trees of preferred species and free from defects and disease. The chronic persistence of Armillaria, Laminated and Tomentosus root diseases in coniferous forests of British Columbia, Canada may offset potential gains in timber yield of commercially important tree species by creating stumps that the fungi utilize as an energy source to infect neighbouring trees. In juvenile plantations and naturally regenerated stands in six biogeoclimatic (BEC) zones with evidence of root disease caused by *Armillaria ostoyae* (8 sites), *Coniferiporia sulphurascens* (2 sites) or *Onnia tomentosa* (1 site), five of ten 20 m square plots per site were randomly selected for thinning to British Columbia Ministry of Forests specifications. Crop tree diameter at breast height and mortality from all causes were recorded at establishment and periodically thereafter up to 19 years post-thinning. Logistic regression analysis of mortality rates showed significant differences among root disease pathogens, between planted and natural stands, and among ecological zones. Yet over all sites, differences between thinned and control plots were not significant. At the final assessment, crop tree basal area was higher in thinned than in control plots at 10 of 11 sites. Root disease, including infected and dead trees and other lethal biotic and abiotic agents, reduced potential yield in both treatments (thinned and control). At several Armillaria sites, mortality was slightly to substantially higher in thinned than in control plots, suggesting that thinning can increase the amount and potential of inoculum which may continue to adversely impact productivity of those stands. Recommendations for silvicultural management of the three root diseases are discussed.

KEYWORDS
growth loss, mortality, root disease, thinning

1 | INTRODUCTION

Precommercial thinning, synonymous with 'juvenile thinning', 'juvenile spacing' and 'spacing' is a silviculture treatment applied to immature stands to reduce the density to an optimum level for stand growth. Preferred species are selected as crop trees (based on growth traits), and others are cut. The resulting reduction in inter-tree competition should boost vigour of the remaining trees and enhance resistance to abiotic and biotic stressors (Wargo & Harrington, 1991). Such a treatment may exacerbate damage by root diseases. A prime example is spore infection by *Heterobasidion annosum* sensu *lato* on fresh stumps, subsequent transfer of mycelium to crop trees via root contacts, followed by extensive decay and mortality (Gunulf et al., 2013; Rishbeth, 1951). Yet the proximity of the primary inoculum source (infected stump) to crop trees may influence the effect following thinning. For example, in New Zealand, Hood et al. (2002) found that thinning increased the incidence of infection by *Armillaria* species in Radiata pine (*Pinus radiata* D. Don) compared with unthinned controls, and the incidence was significantly greater among crop trees within 5 m of stumps from asymptomatic trees already infected before thinning (i.e. harbouring non-lethal infections on roots). Some other factors may also affect the incidence of root diseases.

In the southern half of British Columbia (B.C.), root diseases caused by *Armillaria ostoyae* (Romagn.) Herink (Morrison, 1981; Morrison et al., 1991), *Coniferiporia sulphurascens* Pilat [syn. *Phellinus sulphurascens* (Pilat) L.W. Zhou & Y.C. Dai or *Phellinus weirii* (Murr.) Gilb.] (Wallis, 1976) and *Onnia tomentosa* (Fr.) Karst. [*Inonotus tomentosus* (Fr.) Teng] (Lewis & Hansen, 1991a) cause mortality in both plantations and naturally regenerated stands of conifers. In the coastal and interior regions of B.C., *C. sulphurascens* predominantly affects Douglas-fir (*Pseudotsuga menziesii* Mirb. Franco) across the species' range (Thies & Sturrock, 1995). Yet in southern B.C., *Onnia tomentosa* is generally restricted to the northern regions (boreal zone), and among higher elevation forests (Anon. 1995; Reich et al., 2013) where it has a host preference for spruce (*Picea* spp.). By contrast, the geographical, ecological and host ranges of *A. ostoyae* are much greater than those of the other pathogens (Cleary et al., 2008; Morrison et al., 1991).

Only a few trials examined the long-term effect of precommercial thinning on infection and mortality by root disease in western North America. They dealt primarily with Armillaria root disease and have provided variable and mostly inconclusive results. For example, 20 years after precommercial thinning in a 30-year-old ponderosa pine (*Pinus ponderosa* Laws.) stand in central Oregon, crop tree mortality caused by *A. ostoyae* in unthinned plots exceeded that in thinned plots, but differences were not significant (Filip et al., 1989). After 40 years, differences were significant (Filip et al., 2009). In contrast, 20 years after thinning in a dense western redcedar (*Thuja plicata* Donn.) stand in Idaho, 40% of trees in thinned plots had *Armillaria* root disease while only 12% in the unthinned plots were diseased (Koenigs, 1969). A 30-year study of precommercial thinning in four mixed-species plantations affected by *Armillaria* and *Heterobasidion* root diseases showed no significant differences in leave-tree survival in thinned versus control plots at three sites and significantly higher survival in thinned than control plots at the fourth (Filip et al., 2015).

The biology of pathogenic *Armillaria* species and *C. sulphurascens* suggests that incidence of infection should increase following precommercial thinning, as it does following selective cutting in older stands (Hagle, 2009; Morrison et al., 2001). With the additional food sources provided by colonized stumps after precommercial thinning, the inoculum potential of the fungi, and hence their capacity to cause disease, could increase. The roots of residual trees adjacent to colonized stumps are exposed to inoculum, and infection and mortality among crop trees could follow. Based on those hypotheses, the effect of precommercial thinning on crop tree infection and mortality by root diseases was identified as a high-priority research need for the interior of British Columbia (Morrison, 1991). To that end, the objective of this project was to determine the effect of precommercial thinning on (i) the incidence of crop tree infection and mortality by Armillaria, Laminated and Tomentosus root diseases; and (ii) the level of post-thinning basal area growth of crop trees as influenced by the interaction of disease, stand origin, tree species and biogeoclimatic zone. Findings would suggest whether management recommendations for precommercial thinning need to be revised and identify other silviculture considerations for managing root disease-infested stands.

2 | METHODS

2.1 | Site selection

In British Columbia, *A. ostoyae* occurs primarily in the forest regions of Kootenay-Boundary (formerly Nelson), Thompson-Okanagan (formerly Kamloops) and Cariboo in the southern interior and in the South Coast (formerly Vancouver) region. *Coniferiporia sulphurascens* primarily occurs in the South Coast, West Coast and Thompson-Okanagan Forest regions, with *O. tomentosa* primarily in the Omineca (formerly Prince George) and Skeena (formerly Prince Rupert) regions in the north. For each region, the Ministry of Forests pathologist and District Forest health officers identified potential sites to assess post-thinning effects of these diseases based on the following criteria: (1) a planted or natural stand of precommercial thinning age; (2) stocking, species composition, and incidence of one root disease uniform throughout; and (3) the site would accommodate ten 20- by 20-m treatment plots. Eight sites were selected for *A. ostoyae*, two for *C. sulphurascens*, and one for *O. tomentosa*. *Armillaria ostoyae* was emphasized, recognizing its wider occurrence in several biogeoclimatic (BEC) zones, thereby allowing an opportunity to evaluate the effects of stand origin and BEC zone on disease incidence following precommercial thinning.

2.2 | Site attributes

Table 1 lists all attributes for sites selected in the study. The BEC zone for each site was determined using the field guide for each

TABLE 1 Site attributes of the 11 precommercial thinning sites in southern British Columbia

Root disease pathogen	Installation name	Year established	Location N lat./W long.	BEC[a] subzone	Stand origin	Age at thinning	Species[b,c]	Conifer stems/ha pre-trt	Conifer stems/ha post-trt	Disease centres/plot (mean)
A. ostoyae	Young	1991	51°18′; 121°02′	IDFdk	Natural	30	Fd Pl	6600	1382	2.8
	Hornet	1991	49°38′; 121°50′	CWHds1	Planted	11	Fd Hw (Cw)	3880	662	2.3
	Kidd	1992	49°15′; 116°08′	ICHmw1	Natural	22	Bl Hw Cw Se	11,120	810	nr[d]
	Mag A	1992	51°07′; 119°03′	ICHmw3	Natural	20	Fd Hw (Cw)	16,600	1017	nr
	Nusatsum	1993	52°22′; 126°28′	CWHmm2	Natural	15	Hw Ba (Fd Cw)	11,840	596	4.5
	Talchako	1993	52°20′; 126°03′	CWHds	Planted	20	Fd	2440	825	4
	Lussier	1994	50°05′; 115°40′	MSdk	Natural	19	Pl (Lw Fd)	28,100	1270	4.8
	Fitzstubbs	1994	50°14′; 117°35′	ICHmw2	Planted	19	Fd (Cw Hw)	2280	632	2.5
C. sulphurascens	Mag P	1992	51°07′; 119°03′	ICHmw3	Natural	20	Fd Hw Cw	6500	985	nr
	Chilliwack	1993	49°05′; 122°00′	CWHdm	Planted	10	Fd (alder)	2520	617	2.7
O. tomentosa	Pelican	1990	53°35′; 123°05′	SBSdw3	Natural	35	Sw (Pl Bl)	6280	1335	nr

[a]Zone names: SBS, sub-boreal spruce; ICH, interior cedar hemlock; CWH, coastal western hemlock; MS, montane spruce; IDF, interior Douglas-fir; subzone unit names: dk (dry cool), ds (dry submaritime), mw (moist warm), mm (moist maritime), dm (dry maritime), dw (dry warm); numbers indicate variants (locations).

[b]Fd: *Pseudotsuga menziesii* (Mirb.) Franco (CWH) *P. menziesii* var. *glauca* (Beissn.) Franco (IDF, ICH); Hw: *Tsuga heterophylla* (Raf.) Sarg.; Cw: *Thuja plicata* Donn ex D. Don; Bl: *Abies lasiocarpa* (Hook.) Nutt.; Se: *Picea engelmannii* Parry; Sw: *Picea glauca* (Moench) Voss; Pl: *Pinus contorta* var. latifolia Engelm.; Ba: *Abies amabilis* (Dougl.) Forbes; Lw: *Larix occidentalis* Nutt.; alder, *Alnus rubra* Bong.

[c]() brackets indicate <10%.

[d]nr, not recorded.

region (Braumandl & Curran, 1992; Green & Klinka, 1994; Lloyd et al., 1990). The sites regenerated after either a diameter-limit harvest (Young and Pelican), wildfire (Mag A and P) or clearcutting (Kidd, Lussier, Nusatsum, Fitzstubbs, Talchako, Hornet and Chilliwack). The species and number of trees per hectare (ha) prior to precommercial thinning was estimated with a 0.01 ha sub-plot in each plot and averaged for each site (Table 1). Douglas-fir was planted at the four plantation sites. All other species regenerated naturally from seed. Stand age at thinning ranged from 10 to 35 years (Table 1).

2.3 | Plot establishment and experimental design

Each site was surveyed to locate 10 areas with uniform stocking and having trees killed or infected by one of the root diseases. A 20- by 20-m plot with a 5 m wide buffer was located in each area. Crop trees were selected according to B.C. Ministry of Forests specifications for species preference and post-thinning inter-tree distance. Crop trees within plots were tagged, and their species, diameter at breast height (dbh), and coordinates were recorded. In plots at seven sites, disease centres, as indicated by one or two adjacent dead or living symptomatic trees were tallied, marked with metal posts, and their coordinates measured. Mean numbers of centres per plot were calculated (Table 2). At those seven sites, one of two treatments (thinned and unthinned control) was randomly assigned to pairs of plots with a similar number of root disease centres. At the other four sites, treatment was randomly assigned to the plots. Non-crop trees in plots and buffers designated for thinning were cut with chainsaws. At Lussier, a third treatment known as pop-up spacing (see Morrison & Mallett, 1996), a technique by which a small excavator with a clamshell attachment is used to pull symptomatic crop trees and non-crop trees from the soil with their roots attached, was applied to an additional 5 plots.

2.4 | Monitoring

Each installation was assessed biennially for 10 years after establishment. That allowed year of tree death to be accurately determined. Thereafter, assessments were done at 4–5 years intervals, and year of death was estimated based on the degree of crown deterioration. Each crop (tagged) tree was examined for symptoms of root disease in the crown, as was the root collar for trees with crown symptoms. Disease signs at the root collar were resinosis and sub-cortical mycelial fans for *A. ostoyae*, and ectotrophic mycelium for *C. sulphurascens and O. tomentosa*. Causes of other biotic or biotic damage were also recorded. To ascertain the long-term change in basal area between thinned and control plots, the experimental design aimed for similar number of tagged trees for each treatment with similar starting diameters. Crop tree dbh was measured at the time of plot establishment, and at 10–13 and 15–19 years, depending on the site. Access to the Kidd and Hornet sites was lost after years 12 and 13, respectively, when access roads were decommissioned. Time from establishment to the last assessment for the other nine sites ranged from 15 to 19 years after thinning.

2.5 | Spatial distribution of root disease mortality within plots

At the seven sites where the location of root disease centres was recorded, the percentage of centres with crop tree mortality within 5 m of each disease centre post was determined from plot maps (Table 2). Five meters is the estimated distance *C. sulphurascens* and *A. ostoyae* could spread on root systems over 15 years based on measurements of spread rate (Bloomberg & Reynolds, 1982; Fournier, 1997; Nelson & Hartman, 1975; van der Kamp, 1993). Also, the mean percentages of crop trees killed within 5 m of centre posts in plots were determined for each treatment (Table 2).

2.6 | Statistical analysis

The data were analysed by logistic regression. The logistic model is given as $\log(p/1-p) = \beta_0 + \beta_1 x_1 + \cdots + \beta_m x_m$, whereby p = Prob (trees are alive and infected or dead due root disease at the end of an interval period divided by trees that were alive and uninfected at the beginning of interval) and $x_1,\ldots,x_m$ = indicator (0,1) or continuous variables; where the tested variables include the pathogen (*A. ostoyae*, *C. sulphurascens*), stand origin (natural, planted), BEC zone (CWH, ICH, IDF, MS) and treatment (thinned, control). Results for the added treatment (pop-up spacing) at Lussier were not included in the logistic regression analysis, nor were data from the single *O. tomentosa* site (Pelican).

The time interval is defined as one of three observation periods to assess the effects of variables fitted to the logistic regression model: (i) the 10- to 13-year interval between the first and second assessments; (ii) the 4- to 7-year interval between the second and third assessments; and (iii) the total observation period (12- to 19 years) between the first and last assessments.

Variations in dbh among trees (plots and sites) were modelled by including initial diameter (DBH0) in all models. The calculated odds ratio gives the probability (risk) of two different explanatory variables' contribution to infection or mortality of crop trees.

Crop tree basal area (for control vs. thinned treatment) and the magnitude of change (from establishment to the end of the experiment) were compared using 95% confidence intervals. In all analyses, a probability (p value) of .05 or less was used to declare significant differences between and among factors.

3 | RESULTS

3.1 | Treatment

Over all 11 sites, precommercial thinning did not have a significant effect (p = .415) on crop tree mortality caused by root disease (Table 3). Average values for control and thinned plots, respectively, were 6.8% and 8.9% for *A. ostoyae*, and 20.3% and 19.3%, respectively, for *C. sulphurascens* (Figure 1). The cumulative mortality for

TABLE 2 Plot attributes: Mean numbers of crop trees and disease centres per plot, and percentage of crop trees killed by root disease in thinned and unthinned plots from establishment to the end of the study. Brackets indicate standard deviation around the mean

Root disease pathogen	Site	BEC zone[a]	Stand origin	Crop trees/plot (#)		Centres/plot (#)		Root disease mortality (%)	
				Control	Thinned	Control	Thinned	Control	Thinned
A. ostoyae	Young	IDFdk	Natural	55 (8.6)	55 (4.5)	2.8 (0.5)	2.8 (0.8)	6.7 (4.7)	9.3 (5.6)
	Kidd	ICHmw1	Natural	32 (1.7)	32 (1.5)	nr[b]	nr	2.4 (4.1)	1.3 (1.7)
	Mag A	ICHmw3	Natural	41 (1.1)	40 (2.2)	nr	nr	3.5 (2.2)	1.4 (3.0)
	Lussier	MSdk1	Natural	52 (3.0)	50 (2.6)	4 (1.0)	5.4 (0.9)	14.2 (7.6)	11.7 (10.3)
	Nusatsum	CWHds	Natural	25 (2.9)	23 (1.9)	4.7 (0.5)	4.4 (1.2)	3.9 (5.6)	7.8 (4.6)
	Fitzstubbs	ICHmw2	Planted	25 (1.1)	25 (2.2)	2.4 (0.5)	2.6 (0.8)	4.7 (1.7)	18.1 (15.1)
	Talchako	CWHds	Planted	33 (1.9)	32 (4.3)	4.2 (0.4)	3.8 (1.0)	8.9 (7.3)	12.6 (9.0)
	Hornet	CWHds1	Planted	22 (0.5)	22 (1.1)	3 (0.9)	1.6 (0.5)	11.2 (15.2)	9.2 (6.5)
C. sulphurascens	Mag P	ICHmw3	Natural	40 (2.2)	39 (2.4)	nr	nr	10.7 (10.4)	8.2 (3.7)
	Chilliwack	CWHdm	Planted	25 (0.7)	24 (0.5)	2.8 (0.4)	2.6 (0.6)	27.9 (12.3)	32.7 (12.4)
O. tomentosa	Pelican	SBSdw3	Natural	50 (8.3)	57 (14.6)	nr	nr	12.4	13.7

[a]SBS, sub-boreal spruce; ICH, interior cedar hemlock; CWH, coastal western hemlock; MS, montane spruce; IDF, interior Douglas-fir.
[b]nr, not recorded.

the *O. tomentosa* site was 12.4% for control plots and 13.7% for thinned plots. The percentages of crop trees killed by root disease fungi in thinned and non-thinned plots for the individual sites are shown in Table 2.

The interactions between treatment and fungus, treatment and stand origin and treatment and BEC zone were also not significant (Table 3). Yet the *p*-values for treatment×fungus and treatment×BEC zone interactions reflect the differences between treatments for mortality caused by *A. ostoyae* at some sites and both *A. ostoyae* and *C. sulphurascens* among BEC zones. Odds ratios showed that across the whole time period, the probability of trees being becoming infected or killed is higher for thinned than for control plots located in the CWH (planted) and ICH (planted and natural regeneration) zones. At the Lussier *Armillaria* site, where pop-up spacing with an excavator pulled dead and symptomatic trees from the soil with roots attached, no mortality was recorded for the 19 years post-treatment.

3.2 | Fungus

There were significant differences between the root disease fungi in the level of crop tree infection and mortality over the observation period ($p < .0001$ Table 3). *Armillaria ostoyae* had a significantly smaller probability ($p = .029$) than *C. sulphurascens* (Table 4) for both control and thinned plots.

3.3 | Stand origin

Crop tree infection and mortality were significantly lower at naturally regenerated sites than in planted ones ($p < .0001$ Tables 3 and 4), regardless of treatment.

3.4 | Biogeoclimatic zone

There were significant differences in root disease infection and mortality among BEC zones ($p < .0001$ Table 3) with a significantly smaller probability of infected or dead trees in the CWH zone than the MS zone (.034) and in the ICH zone than in the MS zone (.05) (Table 4). Odds ratios for control plots showed a higher probability of infected or dead trees in the MS zone compared to other BEC zones, suggesting that the background level of disease was high relative to the other study sites.

3.5 | Other effects

The time from establishment of the experiment to final assessment varied from 12 to 19 years. The near-significant value for the effect ($p = .054$ Table 3) likely reflects the varying time for fungal spread within and between root systems and differences in fungal virulence.

Effect	df	X^2	Prob ≥ X^2
Fungus	1	18.71	<.0001
Origin	1	45.64	<.0000
BEC zone	3	21.27	<.0001
Treatment	1	0.67	.415
Fungus × Treatment	1	3.02	.082
Origin × Treatment	1	1.48	.223
BEC zone × Treatment	3	7.01	.072
DBH0 (initial DBH)	1	3.12	.077
Length of time interval (years)	1	3.7	.054
Tests for lack of fit			
Hosmer-Lemeshow	8	14.36	.073
Pearson	3432	3452.6	.399

TABLE 3 Logistic regression analysis tests for the effects of treatment, fungus, BEC zone, stand origin, initial tree DBH and time

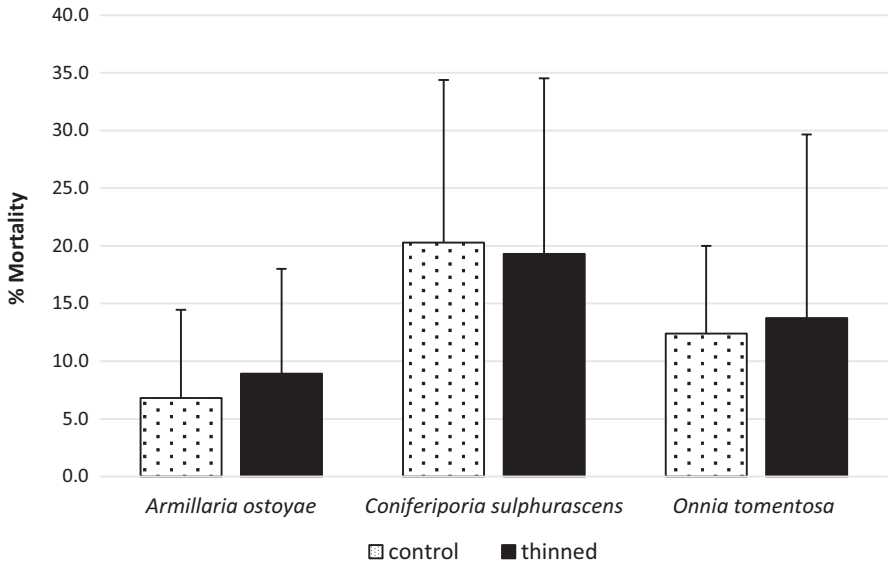

FIGURE 1 Average mortality of crop trees across all 11 sites caused by *Armillaria ostoyae*, *Coniferiporia sulphurascens* and *Onnia tomentosa* through 19 years after precommercial thinning.

The probability value for initial dbh was .077 (DBH0, Table 3), reflecting the considerable variation in DBH of crop trees at the time of establishment among *A. ostoyae* sites.

3.6 | Spatial distribution of root disease mortality within plots

In the Chilliwack Douglas-fir plantation, more than 90% of the disease centres identified at establishment in both treatments had mortality caused by *C. sulphurascens* 15 years after thinning (Table 5). More than 95% of mortality in plots occurred within 5 m of a disease centre post. Lapse time until death increased with distance from the centre post. These values indicate that nearly all *C. sulphurascens* inoculum was expressed by stand age 10, that post-thinning mortality occurred around centres of existing infections, and that thinning did not create new inoculum centres.

At the six *A. ostoyae* sites with recorded disease centre posts, the percentage of centres with crop tree mortality within 5 m of the centre post, and the percentage of total plot mortality within 5 m of centre posts depended on stand origin, treatment and site or some combination of these (Table 5). In the three Douglas-fir plantations infected by *A. ostoyae*, the percentage of centres with mortality was 1.3- to 2-fold higher in thinned (average 67%) than in control (average 42%) plots, suggesting that thinning of asymptomatic infected trees within 5 m of the centre post created primary inoculum that served as a source of infection and spread to residual trees. In contrast, at the three natural stands, the percentages were consistently higher in control than thinned plots, with the values for sites varying with BEC zone (Table 5). This suggests that thinning did not increase inoculum potential due to the small size of stumps at Young and Lussier or the effective response of western hemlock and amabilis fir to contain *A. ostoyae* infection at Nusatsum. The percentages of plot mortality within 5 m of centre posts were similar for both treatments at two of the three planted sites (Table 5) suggesting that most inoculum was expressed at the time of thinning. The low values for both treatments at Hornet reflect the younger stand age (11 years)

TABLE 4 Parameter estimates

Effect (xi)	Estimate	SE	Wald X^2	Prob ≥ X^2
Intercept (β0)	−1.35	0.894	2.28	.131
A. ostoyae	−0.413	0.19	4.74	.029
C. sulphurascens	0			
Natural	−1.583	0.247	40.95	<.0001
Planted	0			
CWH	−0.687	0.324	4.49	.034
ICH	−0.492	0.253	3.8	.051
IDF	−0.194	0.298	0.42	.516
MS	0			
Control	0.27	0.543	0.25	.619
Thinned	0			
A. ostoyae × Control	−0.454	0.261	3.02	.082
A. ostoyae × Thinned	0			
C. sulphurascens × Control	0			
C. sulphurascens × Thinned	0			
Natural × Control	0.428	0.351	1.48	.223
Natural × Thinned	0			
Planted × Control	0			
Planted × Thinned	0			
CWH × Control	−0.22	0.472	0.22	.642
CWH × Thinned	0			
ICH × Control	−0.772	0.361	4.57	.032
ICH × Thinned	0			
IDF × Control	−0.583	0.409	2.03	.154
IDF × Thinned	0			
MS × Control	0			
MS × Thinned	0			
DBH0	−0.0327	0.0185	3.12	.077
Time interval	0.0952	0.0495	3.7	.054

compared with 19 and 20 years at the other two sites. In control plots at Nusatsum and Young, most mortality occurred within 5 m of disease centre posts. However, at Lussier only half of plot mortality was associated with symptomatic trees present at the time of thinning. This difference may be due to less obvious signs of infection in lodgepole pine than on Douglas-fir, western hemlock and amabilis fir.

3.7 | Other causes of crop tree mortality

In addition to root disease, other biotic causes of crop tree mortality were mountain pine beetle (*Dendroctonus ponderosae* Hopkins) and Warren root weevil (*Hylobius warreni* Wood [Coleoptera: Curculionidae]) at Young and Pelican, western gall rust (*Endocronartium harknessii* [J.P. Moore] Y. Hiratsuka) at Lussier and animal damage at several sites. Small amounts of abiotic mortality were due to suppression, windthrow, snow-press and snow-breakage and some unknown causes (Table 6).

3.8 | Crop tree basal area growth

At the time of plot establishment, mean crop tree dbh and therefore crop tree basal area were similar across treatments (Table 6). At the final assessment, the mean basal area of living, root disease-free trees were higher in thinned than control plots, except at Fitzstubbs. At Young and Pelican, the change in basal area was negative in control plots and slightly positive in thinned plots, due to losses from mountain pine beetle, trees infected and killed by root disease, and snow-press and snow-breakage at Pelican. The largest post-establishment increases in disease-free basal area for both treatments were recorded at the naturally regenerated Armillaria sites (Lussier and Nusatsum). There, the increase was also higher in thinned versus control plots ($p < .05$) (Table 6). Modest increases occurred in both treatments at naturally regenerated sites (Kidd and Mag A) and at the Talchako plantation. For the *C. sulphurascens* sites, the increase in basal area at the naturally regenerated, interior site (Mag P) was one-half that at the coastal plantation (Chilliwack) probably due to poorer site quality. With the exception of Young and

TABLE 5 Percentage of root disease (RD) centres with RD mortality ≤5 m of the plot centre and the percentage of RD trees in plots that were ≤5 m from the centre tree at the 7 sites where dead root disease (RD) trees were recorded at establishment

			RD Centres with crop tree RD mortality ≤5 m from plot centre post (%)		Mean % of dead RD trees ≤5 m from a centre tree (%)	
Root disease	Site[a]	Stand origin	Control	Thinned	Control	Thinned
A. ostoyae	Hornet	Planted	46	62	50	58
	Fitzstubbs	Planted	38	75	87	78
	Talchako	Planted	43	63	93	100
	Nusatsum	Natural	29	10	100	29
	Young	Natural	71	57	88	61
	Lussier	Natural	54	45	50	74
C. sulphurascens	Chilliwack	Planted	93	100	96	95

[a]Root disease centres were not recorded at Kidd, Mag A and P and Pelican.

Pelican, the basal area on live, uninfected trees increased ($p < .05$), regardless of treatment.

4 | DISCUSSION

This project contributed to a long-term effort to understand how precommercial thinning influences the behaviour of three major root disease pathogens in coniferous forests of southern B.C, where root disease is considered a 'disease of the site', fungal mycelia essentially become endemic in colonized stumps and persist from one rotation to the next (Lewis & Hansen, 1991a; Morrison et al., 1991; Wallis & Reynolds, 1965). This study demonstrates the complexity of factors affecting the incidence of mortality caused by root disease fungi that are largely governed by pathogen virulence, the amount and distribution of fungal inoculum, host traits including resistance and environmental factors (e.g. soil moisture) that affect both the host and pathogen. These factors, in connection to the tested variables in this study (fungal pathogen, stand origin, BEC zone and treatment) are discussed below.

4.1 | Biology of the root disease fungi, disease epidemiology and host susceptibility

For the three fungi, ectotrophic and/or intrabark mycelium are necessary for mycelial transfer from an inoculum source to a suscept root (Bloomberg & Reynolds, 1982; Cruickshank et al., 1997; Lewis et al., 1992). Growth of *Armillaria* spp. rhizomorphs through soil and on roots is dependent on a film of water over the growing tip (Smith & Griffin, 1971). Spread of the fungi within root systems, and in soil in the case of Armillaria, will be affected by soil moisture regime.

Occurrence of multiple genotypes of *O. tomentosa* within discrete disease centres (Lewis & Hansen, 1991b), colonized thinning stumps containing different mycelial types of *A. mellea* (sensu *lato*) (Rishbeth, 1978) and an increase in the number of *A. novae-zelandiae* genets post-thinning (Hood et al., 2002) suggest a role for basidiospores in disease spread and intensification for these species. However, the large areas occupied by individual genets (Ferguson et al., 2003; Smith et al., 1994) suggest a limited role for spores of *A. ostoyae*, and spores of *C. sulphurascens* are considered unimportant in disease spread (Nelson & Hartman, 1975). Given the time frame of the study (up to 19 years), it is unlikely that spore colonization of the newly created thinning stumps would have contributed to killing adjacent crop trees.

The time from harvest to infection from stumps and subsequent appearance of disease symptoms and mortality varies with the root disease species and site conditions. *Coniferiporia sulphurascens* is the most virulent of the three root disease fungi, as evidenced by killing of more than 30% of Douglas-fir in 15 years and 54% during 25 years at the Chilliwack plantation in the CWH zone, and by mortality levels in Douglas-fir at a long-term trial in the ICH zone (Morrison et al., 2014). These observations indicate that mortality will be substantial where the fungus occurs with a susceptible host on sites favourable for fungal spread. The lower mortality at the interior Mag P site is likely attributable to natural regeneration of western red cedar and western hemlock, in addition to Douglas-fir. Both species are less susceptible to killing by *C. sulphurascens* (Cleary et al., 2011; Sturrock et al., 2006). All species of trees and many of the shrubs occurring in the range of *A. ostoyae* in B.C. are susceptible to killing by the fungus. However, tree species vary in susceptibility to killing (Cleary et al., 2008), notably, western larch (Robinson & Morrison, 2001) and western red cedar and western hemlock (Cleary et al., 2012, 2021).

Results from long-term monitoring plots (Morrison, 2011; Morrison & Pellow, 1994) and studies of juvenile plantations (Cruickshank et al., 2011) in the ICH zone show mortality in Douglas-fir caused by *A. ostoyae* becomes evident 5–7 years after harvest/planting, and its incidence peaks between ages 20 and 25 (Morrison, 2011). This peak in mortality typically occurs well after the time period of the free-growing declaration—a milestone used by licensees based on health and stocking where they declare that a

TABLE 6 Crop tree growth at establishment (Est) and end of the experiment

Root disease Pathogen	Site	Years Est. - end	Treatment	Mean crop tree dbh (cm): (se)		Crop tree basal area (m^2/ha): (se)			BA (m^2/ha)	Dead crop tree BA (m^2/ha)		
				Est.	End[a]	Est.	End[a]	Change End/Est.	RD[b] infected @ end	RD[b]	Other biotic[c]	Abiotic[d]
A. ostoyae	Young	17	cont.	2.8 (0.5)	11.3 (0.7)	14.2 (1.0)	13.7 (1.5)	0.96	0.26	0.52	6.86	0.02
			thin.	2.8 (0.8)	12.7 (0.8)	13.4 (1.7)	17.1 (2.2)	1.27	0.04	0.81	4.39	0.02
	Kidd	12	cont.	5.8 (0.5)	9.2 (0.6)	2.6 (0.3)	6.1 (0.5)	2.34	0	0.02	0.14	0.03
			thin.	5.5 (0.4)	10.2 (0.8)	2.6 (0.3)	9.0 (1.1)	3.46	0.22	0.02	0.11	0
	Mag A	18	cont.	5.4 (0.5)	9.7 (0.7)	2.6 (0.5)	7.3 (1.2)	2.81	0.26	0.03	0	0.18
			thin.	5.4 (0.4)	11.0 (0.7)	2.6 (0.4)	9.5 (1.3)	3.65	0.40	0.17	0	0.05
	Lussier	16	cont.	2.9 (0.0)	8.1 (0.2)	1.0 (0.0)	6.2 (0.3)	6.20	0.09	0.25	0.02	0.03
			thin.	3.0 (0.0)	11.6 (0.3)	1.0 (0.0)	11.8 (1.4)	11.8	0.06	0.20	0	0.06
	Nusatsum	15	cont.	7.1 (0.4)	15.5 (1.0)	2.7 (0.2)	11.6 (0.9)	4.29	0.31	0.06	0.03	0.20
			thin.	7.5 (0.6)	25.0 (1.0)	2.9 (0.5)	24.5 (2.1)	8.45	1.77	0.20	0	0.22
	Hornet	13	cont.	5.8 (0.6)	12.4 (0.8)	2.0 (0.4)	6.6 (1.0)	3.30	1.04	0.29	0.01	0
			thin.	6.7 (0.4)	15.9 (0.5)	2.6 (0.3)	12.3 (1.1)	4.73	0.38	0.40	0	0
	Talchako	15	cont.	10.1 (0.2)	17.9 (0.2)	7.2 (0.2)	20.0 (0.8)	2.78	0.20	1.12	0	0
			thin.	10.2 (0.3)	20.7 (0.4)	6.8 (0.6)	23.1 (1.1)	3.39	0.50	1.39	0	0
	Fitzstubbs	16	cont.	12.9 (0.1)	20.1 (0.3)	8.6 (0.3)	17.0 (1.4)	1.97	1.12	0.54	0	0.55
			thin.	12.9 (0.4)	21.4 (0.7)	8.4 (0.5)	15.7 (2.8)	1.87	2.88	1.97	0	0.31
C. sulphurascens	Mag P	18	cont.	5.9 (0.4)	10.4 (0.5)	2.9 (0.3)	6.9 (0.3)	2.38	0.07	0.48	0.01	0.15
			thin.	5.5 (0.7)	11.8 (1.0)	2.7 (0.6)	10.3 (1.7)	3.81	0.12	0.60	0	0.04
	Chilliwack	15	cont.	6.7 (0.5)	19.6 (1.5)	2.3 (0.3)	13.7 (1.7)	5.95	0.19	2.35	0	0
			thin.	7.6 (0.7)	25.4 (1.4)	3.0 (0.6)	21.9 (3.8)	7.30	0.12	2.57	0	0
O. tomentosa	Pelican	19	cont.	14.0 (1.2)	16.8 (1.7)	21.3 (2.4)	19.0 (3.8	0.89	2.37	2.23	4.86	0.74
			thin.	13.3 (0.9)	17.0 (0.8)	20.9 (1.6)	22.3 (3.6)	1.06	2.07	2.15	4.29	0.82

[a]Of live uninfected trees.

[b]RD, root disease.

[c]Namely mountain pine beetle, animal.

[d]Snow press, breakage, unknown.

stand can reliably be expected to provide a merchantable crop of timber at the next rotation without significant intervention. Mortality will continue as stands age, though typically after 25–30 years the mortality will slowly but steadily decline (Morrison, 2011). However, as Cruickshank et al. (2011) showed non-lethal root infections accumulate, resulting in accumulative growth loss without recovery.

4.2 | Treatment

Pelican, the single site with Tomentosus and only one located in the SBS zone, was the oldest stand (35 years) and presumably had well-established root contacts with neighbouring trees. Our results for 19 years after thinning showed no effect on crop tree infection and mortality at that site. Elsewhere, at 30 years, Whitney (1993) found lower Tomentosus-caused mortality among thinned plots. Overall, precommercial thinning did not significantly increase the percentage of crop trees killed by *O. tomentosa* or *C. sulphurascens* (Table 2), suggesting that treatment did not promote the spread of inoculum for these fungi. At four of the eight *Armillaria* sites, mortality was 1.4 to 3.8 times greater in thinned plots, probably due to post-treatment colonization in stumps of infected trees and subsequent mycelial transfer at root contacts or by rhizomorphs to adjacent crop trees (Cruickshank et al., 1997). Consistent with that, Hood et al. (2002) found that by 5 years after thinning, the incidence of Armillaria infection had increased particularly among trees within close proximity to cut stumps. At Kidd, Hornet and Lussier, no common factor seems to explain why thinning did not increase mortality due to root diseases. Earlier, Cruickshank et al. (1997) found that effective host response to thinning limits fungal advance at moist sites, like Hornet and Kidd. Consistent with this, Blenis (2000) suggested that thinning may initially cause an increase in tree vigour that delays mortality. Yet since only one quarter to one half of trees with belowground infection by *A. ostoyae* displayed above ground symptoms, depending on the climate region (Morrison et al., 2000), it can be presumed that many of the residual and seemingly healthy trees harbour non-lethal infections on their roots which may expand to become lethal with more time, or if trees become stressed. Lussier (in the MS zone) is the driest site and the colonization of thinned stumps, the viability of inoculum, and spread by mycelium or rhizomorphs could thereby be limited (Cruickshank et al., 1997; Morrison et al., 2000). Also, a large variation in crop tree infection and mortality occurred at some sites like Pelican (in the SBS zone) where mortality caused by *O. tomentosa* ranged between 0% and 5% in three of the five thinned plots, but 22% and 38% in the other two thinned plots. Although the number of root disease centres per plot in thinned and non-thinned treatments was rather similar at all sites, except Hornet, large differences in plot mortality were also evident. For example, Fitzstubbs had consistently low mortality in all control plots, versus losses in three of the five thinned plots where *A. ostoyae* killed between 17% and 40% of the trees. Blenis (2000) suggested that such an irregular spatial distribution of mortality reflects an uneven distribution of inoculum resulting in patches with high or low mortality.

4.3 | Stand origin

Prior to thinning, the seven naturally regenerated stands had between 6000 and 28,000 live stems/ha, and most sites had a homogeneous mixture of two or more species with varying susceptibility to infection and killing by the root disease species present. Cutting large numbers of mostly small-diameter trees with limited rooting radius probably provided only short-lived inoculum sources due to desiccation and colonization by insects and saprophytic fungi.

In marked contrast, the four Douglas-fir plantations with 2500 stems/ha (2 m × 2 m spacing) had little natural infill of other species and thus the number, size (DBH) and regular spacing of susceptible Douglas-fir enabled tree to tree spread of *C. sulphurascens* at Chilliwack, and *A. ostoyae* at Talchako, Fitzstubbs and Hornet. That seems consistent with Byler et al. (1985) who reported three times higher mortality by *A. ostoyae* in planted than naturally regenerated stands.

4.4 | Biogeoclimatic zone

The sites with *O. tomentosa* and *C. sulphurascens* are in moist BEC zones where colonization of stumps and transfer of mycelium from stump to suscept roots by these fungi would not likely be limited by low soil moisture. In contrast, soil moisture varies among the geographically dispersed BEC subzone sites with *A. ostoyae*. Cruickshank et al. (1997) evaluated the host–pathogen interaction at contacts between roots of Douglas-fir crop trees and precommercial thinning stumps colonized by *A. ostoyae* in the CWH, ICH and IDF zones. There, *A. ostoyae* colonized more thinning stumps in the ICH than IDF or CWH, crop tree bole volume in CWH was three to four times that of CDF and ICH, callus formation at root lesions was associated with bole volume and was significantly greater in CWH than the other zones. At Nusatsum and Hornet in CWH, all mortality occurred within 10 years following thinning. However, in the transitional zone between CWH and ICH (e.g. the plantation at Talchako), Cruickshank et al. (1997) had observed a trend towards less frequent callusing where mortality was similar to that in ICH plantations.

Based on a 30-year study in western Cascade plantations, Filip et al. (2015) concluded that precommercial thinning increases basal area growth significantly but did not increase mortality caused by Armillaria root disease. Similarly, our study showed that, with the exception of one site (Fitzstubbs), at the final assessment the mean basal area was higher in thinned than control plots (Table 6). In addition, mortality did not differ between thinned and control plots for *C. sulphurascens* and *O. tomentosa* (Table 2), but the large variability among and within the eight *A. ostoyae* sites did not allow for a clear effect from treatment to be discerned. Citing Cruickshank et al. (1997), Filip et al. (2015) suggested their recommendations for west-side plantations on moist sites may not be appropriate on the dry east side of the Cascade crest. Koenigs' (1969) results support that suggestion.

4.5 | Implications for management of diseased sites

The British Columbia Ministry of Forests published field guidelines for the selection of stands for precommercial thinning of Coastal (Anon., 2012a) and Interior (Anon., 2012b) sites. The guidelines address biological (tree species, age, site productivity), financial (yield) and forest health factors that could affect growth, yield and value of a thinned stand. A survey of candidate stands for disease and insect occurrence is required. To be considered for treatment, coastal stands must have fewer than 10 trees per hectare infected with *C. sulphurascens* or *A. ostoyae* and interior stands fewer than 10 with *A. ostoyae* or 5 with *C. sulphurascens* or *O. tomentosa* or a combined total of 12 per hectare.

All plots at the 11 sites in this study contained symptomatic or dead regeneration infected by one of the three fungi. At the seven sites where root disease centres were mapped at establishment, the number per hectare containing one or more symptomatic trees varied from about 60 to 130. Those numbers reflect the amount of disease in the previous stand. Disease management strategies post-harvest for *C. sulphurascens* (Thies & Sturrock, 1995; Wallis, 1976) and *A. ostoyae* (Morrison et al., 1991) include inoculum removal (sometimes referred to as 'destumping') and regeneration with tolerant, resistant or immune species (Cleary et al., 2008, 2011; Sturrock et al., 2006). No remedial treatment had been applied to any of the sites in this study.

Previous studies have shown stump root transfer of Armillaria mycelium to crop trees following precommercial thinning (Cruickshank et al., 1997; Rosso & Hansen, 1998). They suggest that thinning can increase the amount and potential of inoculum on site, causing mortality or growth loss on crop trees when their roots contact the fungus. Based on such information, the B.C. Forest Service does not currently recommend precommercial thinning for sites with Armillaria root disease (Anon., 2018). In this study, we found that precommercial thinning did not necessarily lead to mortality beyond the background level of root disease impacting productivity on those sites. Even so, the number of dead trees in thinned and unthinned plots suggests that stocking and timber productivity expectations for the stands may not be realized at rotation age since trees with non-lethal infections on their roots suffer an accumulating growth reduction (Cruickshank et al., 2011). However, the lack of replication and the complex interaction observed between root disease fungi, stand origin, tree species and biogeoclimatic zone, confounds conclusions about effects of the thinning particularly since mortality had doubled and nearly tripled, at Nusatsum and Fitzstubbs, respectively, following thinning.

In B.C., the geographic and ecological distributions of the three root disease fungi are well known (Anon., 2018; Morrison et al., 1991; Reich et al., 2013). Based on that information, the occurrence and distribution of root disease in mature stands selected for harvest should be accounted for in the pre-harvest silviculture prescription. This should be guided by published stand establishment decision aids for Laminated (Cleary et al., 2011; Sturrock et al., 2006), Armillaria (Cleary et al., 2008) and Tomentosus (Reich et al., 2013) root diseases and the hazard rating by BEC zone/subzone. Practitioners should use these stand establishment decision aids to guide forest management in stands where root disease seems likely to be a significant issue.

The first step for minimizing root disease losses in forested stands begins in the planning and pre-harvest stages of forest management. Following harvest of diseased sites, the amount of fungal inoculum can be reduced, but not entirely eliminated, by removing infected stumps (Cleary et al., 2013; Morrison et al., 1988, 2014). That will increase survival of trees and improve their growth (Cleary et al., 2013; Morrison et al., 2014) because trees do not need to shift resource allocation from stem growth to active defence which they do when infected (Cruickshank et al., 2011) and because increases in ectomycorrhizal fungal associations following stump removal has been shown to be positively associated with tree productivity (Modi et al., 2020). It is notable that in the extra stump removal treatment at the Lussier site, no mortality occurred during 19 years following stump removal, compared with 12% and 14.3% losses in the thinned and control plots, respectively, where the stumps remained in place. Yet with or without inoculum reduction, at all diseased sites, susceptibility to infection is of utmost importance when selecting the tree species for regeneration. To aid these decisions, tree species response have been rated for *C. sulphurascens* (Cleary et al., 2011; Thies & Sturrock, 1995), *A. ostoyae* (Cleary et al., 2008) and *O. tomentosa* (Reich et al., 2013) in the BEC zones where the root disease fungi occur.

The four Douglas-fir plantations in this study were established following harvest of mature Douglas-fir. Douglas-fir and several species naturally regenerated the unplanted sites, except Pelican. Mortality caused by both *A. ostoyae* and *C. sulphurascens* was higher in plantations of Douglas-fir with small numbers of other naturally regenerated species than in natural stands dominated by species more resistant to both fungi. These findings suggest that planting of sites where Douglas-fir is preferred must include substantial numbers of less susceptible conifers like those listed by Cleary et al. (2008). Because of different rooting habits between species, a mixed planting will reduce root contact with the Douglas-fir. Where appropriate, broadleaved species, such as alder, maple, birch and aspen, could also be encouraged (Cleary et al., 2008, 2011; Sturrock et al., 2006).

Following Ministry of Forests guidelines for root disease in candidate stands (Anon., 1996a, 1996b), none of the 11 sites in this study would have been precommercially thinned. However, at Lussier, Nusatsum and Mag, the stand surrounding plots was thinned. This emphasizes the need for better recognition of cryptic root disease presence by forest management practitioners during Stand Risk Assessment and utilizing the best knowledge synthesis on risk management that is provided in stand establishment decision aids for Laminated (Cleary et al., 2011; Sturrock et al., 2006), Armillaria (Cleary et al., 2008) and Tomentosus (Reich et al., 2013) root diseases.

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author, MC, upon reasonable request.

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