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A root rot pathogen associates with changes in forest community composition and productivity during 100 years of Douglas-fir forest development

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ABSTRACT

Laminated root rot (LRR; caused by the fungus *Coniferiporia sulphurascens*) is the most damaging root disease of Douglas-fir (*Pseudotsuga menziesii*) in the Pacific Northwest, USA and southwestern Canada. LRR's influences on tree mortality and forest productivity have been well-documented in young commercial Douglas-fir forests (<50 years); however, much less is known about the role of LRR in the dynamics of older forests and how root disease might alter trajectories of forest succession. The objective of this study was to examine the association between LRR, and changes in forest community composition and productivity during succession in Douglas-fir forests of the Pacific Northwest. We used live tree density, basal area, and biomass measurements from 16 long-term permanent plots established in 1910–1940 (5–10 year measurement intervals) and distributed across northwestern Oregon and southwestern Washington State, USA. In 2019 and 2020, each plot was surveyed to assess dead trees for evidence of *C. sulphurascens*. Dead wood samples from LRR-suspected dead trees were cultured, and fungal isolates were identified using DNA sequencing methods. The LRR pathogen was confirmed in 13 of 16 plots with one or two genets occurring in each plot. The analyses showed that elevated *C. sulphurascens* incidence (% of tagged dead trees infected) was related to 1) greater decreases in Douglas-fir tree density; 2) increased accumulation of aboveground live tree biomass (AGB) in other species [e.g., western hemlock (*Tsuga heterophylla*) and western redcedar (*Thuja plicata*)], with a 20 % reduction in Douglas-fir AGB-weighted dominance; and 3) net primary productivity but not in ecosystem AGB accumulation rate. These results imply that LRR could accelerate late-seral species establishment, growth, and thus replacement of Douglas-fir through forest succession, but may not alter overall AGB dynamics from 50- to 150-year-old stands. This research provides information to natural resource managers about LRR's influence in second-growth, mature, and old-growth forests of Oregon and Washington, USA, and provides further testament to the need for long-term forest demography plots.

1. Introduction

Many factors influence successional trends in Douglas-fir (*Pseudotsuga menziesii*) forests (Franklin et al., 1987; 2002), but the role of specific biotic mortality agents, such as pathogens, has been understudied (Hansen and Goheen, 2000; Harmon and Bell, 2020). Long-term research has indicated a need to account for pathogens in predictions of forest successional dynamics (Payne and Peet, 2023), while forest pathologists have suggested pathogens, especially root pathogens, are integral to forest development and succession (e.g., Castello et al., 1995; Hansen and Goheen, 2000; Hansen and Stone, 2005). For example, when pathogenic effects are spatially aggregated, density dependent

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responses in host tree growth and survival will be succeeded by altering competition (Flores et al., 2023; Preston et al., 2016). Because biotic interactions can alter tree growth and mortality responses to other stressors, such as climate change (Agne et al., 2018; Allen et al., 2015; Bell et al., 2020; Harmon and Bell, 2020), it is imperative that forest ecology research explore how tree pathogens may alter forest development trajectories and interacting disturbances (Cobb and Metz, 2017).

In the coastal region of the Pacific Northwest of North America, laminated root rot (LRR), caused by a wood decay fungus *Coniferiporia sulphurascens*, influences forest structure and process due to its prevalence in the landscape (5–11 % of forest area) and its ability to kill trees (Agne et al., 2018; Lockman and Kearns, 2016; Washington Academy of Sciences, 2013). *Coniferiporia sulphurascens* kills susceptible hosts such as Douglas-fir, grand fir (*Abies grandis*), and mountain hemlock (*Tsuga mertensiana*), but will also infect most other conifers, allowing it to persist on the site as long as conifers are present. Impacts of infection vary with host, which can be classified as highly susceptible, intermediately susceptible, tolerant, resistant, and immune. In highly and intermediately susceptible species, LRR can cause tree mortality, reduced growth rates, heart rot of large roots and the lower trunk, which can lead to windthrow due to reduced root mass and root stability; and it increases susceptibility of infected trees to bark beetles (Agne et al., 2018; Lockman and Kearns, 2016; Sturrock et al., 2018). Only hardwoods are immune, but several coniferous genera are tolerant or resistant (e.g., *Pinus* spp., *Libocedrus* spp., *Thuja* spp., *Callitropsis* spp., *Chaemaecyparis* spp.) (Thies and Sturrock, 1995). However, the disease can persist in these stand types without causing significant mortality until species composition changes to more susceptible hosts (as might occur after a major disturbance). *Coniferiporia sulphurascens* spreads primarily by root-to-root contact rather than spores, and often occurs in spatially explicit root disease centers occupied by one or a few genets (vegetative clones) (Hansen and Goheen, 2000).

Coniferiporia sulphurascens persists in tree roots and butts (lower portion of tree stem near forest floor), but not without wood in the soil. After disturbances such as fire or clearcut logging, the pathogen can remain alive in snags and old stumps for more than 50 years (Hansen 1979a). As new trees develop around the old, infected stump or snag, the fungus spreads onto susceptible host trees via root contact or growth over short distances through soil. In this way, young stands of Douglas-fir can form spatially distinct root disease centers around old stumps or snags occupied by a single genet (Bae et al., 1994). If the stand is uniformly Douglas-fir, the disease spreads readily (~30 cm/yr) (Hansen and Goheen, 2000) and the disease centers may coalesce depending on abundance of infected trees in the previous stand. A large number of dispersed, individual genets allows for the site to be occupied more rapidly than if there is a single disease center (Tkacz and Hansen, 1982).

Douglas-fir is a long-lived, early-successional species that persists into the late-successional stages of stand development (Franklin et al., 2002). In western Oregon and Washington State, USA, Douglas-fir is typically succeeded by western hemlock (*Tsuga heterophylla*) and western redcedar (*Thuja plicata*), which steadily begin replacing Douglas-fir after 150 years (Franklin and Dyrness, 1988; Franklin et al., 2002; Henderson et al., 1989). The initial Douglas-fir cohort is generally lost between 800–1300 years (Franklin and Dyrness, 1988). In natural settings, fire is the primary disturbance that initiates new stand development. Depending on environmental setting and intensity of fire, the new tree strata may be dominated by Douglas-fir in the early seral stages. This dominance of young stands by Douglas-fir positively influences the ability of *C. sulphurascens* to cause mortality and spread. As the Douglas-fir stand matures and density of Douglas-fir is reduced, the rate of spread by the fungus slows. This is due to multiple factors, including wider spacing of trees, reduced Douglas-fir root contacts, increasing abundance of tolerant and intermediately susceptible hosts, and the tendency for root-diseased trees to fall over and pull root crowns out of the soil, thus reducing inoculum (Sturrock et al., 2018; Thies and Sturrock, 1995).

Coniferiporia sulphurascens contributes to productivity losses in commercial Douglas-fir forests (Morrison et al., 2022; Sturrock et al., 2018; Washington Academy of Sciences, 2013). Sturrock et al. (2018) estimate that laminated root rot reduces fiber production in the northwestern United States and British Columbia, Canada by 4.4 million m^3 per year. Growth losses and reduced yields due to mortality were the focus of much research in the past century (Washington Academy of Sciences, 2013). For example, Thies (1983) noted that growth of individual trees infected with *C. sulphurascens* varied widely in a 40-year-old Douglas-fir stand, but that on average, 10-year growth reductions on live trees infected with *C. sulpurascens* were 7 %-13 %. Oren et al. (1985) investigated growth reduction and vigor of Douglas-fir infected by *C. sulphurascens*, concluding that sapwood basal area was reduced about 30 % in the stand compared to a nearby healthy stand, but that healthy trees in the infected stand increased sapwood basal area growth due to reduced competition. Therefore, they recommended taking the increased growth of residual trees into account when determining losses from LRR.

There has been relatively little published on how LRR affects natural Douglas-fir forest stand development and succession and even less on how the *C. sulphurascens* might influence rates of mortality, carbon dynamics and net primary productivity (NPP) in mature forests. Holah et al. (1993) and Holah et al. (1997) investigated the effect of LRR on forest composition and successional development inside and outside infection centers at five sites in old-growth Douglas-fir forests of western Oregon. They concluded that LRR created conditions for distinct understory vegetation communities to develop, mostly in response to increased sunlight from removal of over-story Douglas-fir. In addition, they found that succession to western hemlock and western redcedar was accelerated in some sites within infection centers, but in other sites, the high density of shrubs in the root disease centers prevented some colonization of the infection center by other tree species.

Additionally, *C. sulphurascens* is well-known to cause significant changes to forest composition and succession in natural high-elevation mountain hemlock forests in the Central Oregon Cascades where the disease causes a “wave-form” type of dieback in relatively pure mountain hemlock (Boone et al., 1988; Dickman and Cook, 1989; Matson and Boone, 1984; McCauley and Cook, 1980). Succession normally follows stand replacing fire in this system, which begins with mixed conifer forests dominated by lodgepole pine (*Pinus contorta*), with western white pine (*P. monticola*), whitebark pine (*P. albicaulis*), Shasta red fir (*Abies magnifica*), and mountain hemlock trees also occurring. Succession eventually leads to dominance of shade tolerant mountain hemlock after several hundred years (Boone et al., 1988; Dickman and Cook, 1989; McCauley and Cook, 1980). However, LRR persists after fire in large woody roots and selectively kills mountain hemlock trees as they move back into the site. When the forests become dominated by mountain hemlock, distinct root disease centers emerge (Dickman and Cook, 1989). The disease slowly migrates through stands of pure mountain hemlock trees [estimated at 34 cm/year (Nelson and Hartman, 1975)] leaving a zone of bare earth and coarse woody debris which is then colonized by western white pine, lodgepole pine, whitebark pine, Shasta red fir, as well as mountain hemlock.

In Douglas-fir forests natural succession following canopy closure includes maturation, vertical diversification, and horizontal diversification, stages that are necessary for the development and maintenance of old-growth Douglas-fir ecosystems (Franklin et al., 2002), which are the focus of regional and national forest conservation efforts (Spies et al., 2019). Because the effects of pathogens on forest development play out over decades, our understanding of the process and how it informs successional theory can be enhanced through long-term, direct observation (Harmon and Pabst, 2015; Lindenmayer et al., 2010). Long-term research plots in natural forest settings can allow for validation of the role of pathogens in forest development and productivity, but opportunities are quite rare. Among these opportunities are growth-and-yield

plots established by the US Forest Service in the early 1900s (Williamson, 1963). These plots were established in young, post-fire forest stands and now document forest development from canopy closure to vertical diversification stages of forest succession. The plots provide an unparalleled data record of stand development in this region and one of the few century-scale forest datasets suitable for testing successional theory *in situ* (Harmon and Pabst, 2015). In this study, we documented the contemporary (2019–2020) abundance and spatial extent of *C. sulphurascens* on 16 long-term plots in western Oregon and Washington, USA, to examine how the pathogen's incidence relates to multi-decade patterns of tree growth, tree mortality, forest succession and ecosystem productivity. The goals of this study were to: (1) explore the diversity of *C. sulphurascens* genets and (2) examine the relationships between LRR and changes in mature Douglas-fir forest productivity, composition, and biomass.

2. Methods

2.1. Field sites

The research sites are located in the Douglas-fir region of northwestern Oregon and western Washington. The climate is maritime, with dry summers and wet winters, and mild winter temperatures (Franklin and Dyrness, 1988). Rainfall varies from 1200 mm to 2500 mm per year across the study area (Williamson, 1963). Sixteen permanent plots across five sites were selected from the PNW-PSP forest plot network (https://pnwpsp.forestry.oregonstate.edu/) where data are publicly accessible (Franklin et al., 2022) (Fig. 1). The sixteen plots were chosen from the larger plot network based on stand composition and reported or suspected presence of LRR. The plots were established between 1910 and 1940 in Douglas-fir forests (Table 1). All trees at least 5.0 cm in diameter were measured every 5–10 years for diameter and assessed for mortality, constituting a record of 75–105 years (Table 1). The stands were between 45–98 years old at plot establishment and were selected for dominance of Douglas-fir for the purpose of growth and yield mensuration, but LRR appeared in several of the plots and was noted in descriptions (Williamson, 1963). The dominant trees in the sampled stands are now between 130 and 173 years old. Plots with significant mortality had down trees scattered across the site (Fig. 2 A) and characteristic tree tips up with well decayed roots (Fig. 2B).

2.2. Fungal confirmation and isolation

In October 2019 and August 2020, we surveyed three or four plots at each of five sites (16 plots in total) from North to South including Olympic (OL hereafter), Gifford Pinchot (GP hereafter), Wind River (WR hereafter), Mt Hood (MH hereafter), and Willamette (WI hereafter) (Fig. 1). At each plot, we assessed the root systems of all standing dead trees, down wood, and stumps by digging away duff and soil to expose the lower trunk and major lateral roots and chopping into suspect roots to examine the decay. We confirmed the presence of *C. sulphurascens* using visible signs, including ectotrophic mycelium (white crust on surface of roots) and advanced pitted decay (white rot) with setal hyphae (Fig. 2 C; fungus structures that look like reddish whiskers) (Goheen and Willhite, 2021). For dead trees with *C. sulphurascens* presence, we recorded tree number if the tag was still present to later extract year of death from the long-term tree data. We did not assess any living trees because these plots are used for long term vegetation dynamics research, and we wanted to avoid damage from digging and chopping roots that could cause negative impacts on tree growth.

For dead trees with *C. sulphurascens* presence, we collected samples (e.g., decayed wood) and isolated the fungus in the laboratory to

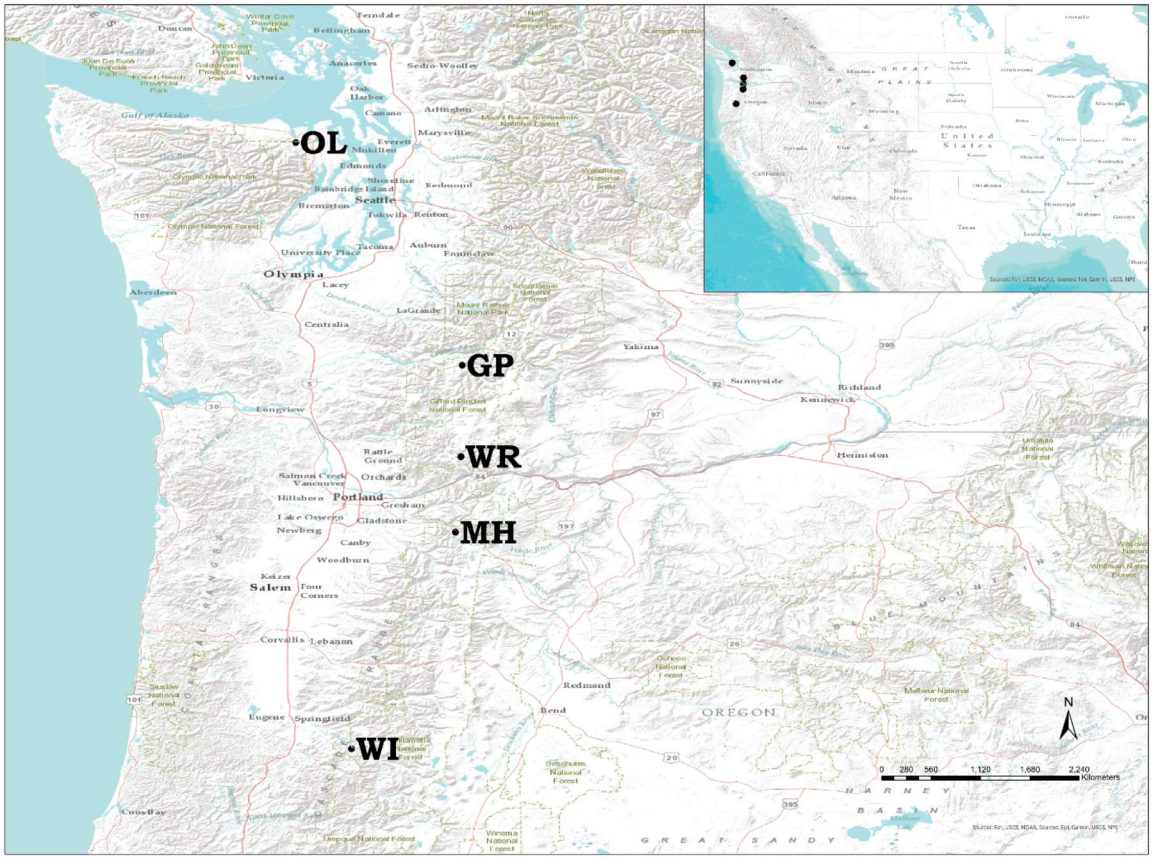

Fig. 1. The 16 long-term plots across 5 sites in western Washington and Oregon, USA. From North to South, the plots are located in Olympic peninsula (OL), Gifford Pinchot (GP), Wind River (WR), Mount Hood (MH), and Willamette Valley (WI).

Table 1
Plot establishment, measurement history, and current stand summary data for the sixteen plots, located at five locations in western Washington and Oregon States, USA.

Site	Plot ID	Plot establishment				Measurement history			Stand summary (2009-10)		
		Area (ha)	Year	Stand age	Trees per ha	# of meas.	Most recent	Age at last meas.	Trees per ha	Douglas-fir per ha	% Douglas-fir
Gifford Pinchot (Randle, WA)	GP01	0.4	1927	55	561	14	2015	143	909	111	12
	GP03	0.4	1927	50	632	13	2015	138	776	180	23
	GP05	0.4	1927	53	526	14	2015	141	277	143	52
Mt Hood (Zigzag, OR)	MH01	0.4	1930	45	899	14	2015	130	610	175	29
	MH02	0.4	1930	45	1218	14	2015	130	556	279	50
	MH03	0.4	1930	45	1188	14	2015	130	615	245	40
Olympic (Quilcene, Blyn)	OL01	0.4	1926	51	1112	13	2014	139	595	326	55
	OL02	0.4	1926	51	988	13	2014	139	464	168	36
	OL03	0.3036	1926	42	1860	10	2014	130	1157	286	25
	OL04	0.3036	1926	42	2467	10	2014	130	927	383	41
Willamette (Oakridge, OR)	WI01	0.4	1910	54	534	17	2015	159	254	188	74
	WI02	0.4	1910	54	548	17	2015	159	326	183	56
	WI03	0.4	1910	54	504	17	2015	159	361	198	55
Wind River (Carson, WA)	WR04	0.39	1914	72	462	18	2014	172	451	136	30
	WR05	0.36	1914	72	387	17	2014	172	469	203	43
	WR90	0.4	1939	98	343	14	2014	173	492	158	32

confirm species and identify genets of *C. sulphurascens*. If there was a disease pocket, an area where all dead trees were infected, then we only took one or two woody samples with setal hyphae from that pocket, instead of every single infection. Samples were transferred in coolers and were stored at 4°C before processing. In the laboratory, we first scraped setal hyphae from each sample and cultured on regular malt extract agar (MEA) medium (3 % malt, 3 % dextrose, 1 % peptone, 1.5 % agar) amended with streptomycin sulfate (100 ppm). After a couple of transfers, hyphal tips were transferred onto fresh MEA medium. The species identification of *C. sulphurascens* isolates were verified by the DNA sequencing of internal transcribed spacer (ITS) region following the methods of Leal et al. (2019). Following the isolations and species confirmation of *C. sulphurascens*, somatic incompatibility pairing tests were performed to identify individual genets among the isolates from each site (Hansen 1979b).

2.3. Data analysis

Because there were only a few western hemlock found with *C. sulphurascens* presence in the field, we only considered the infection of the major host, Douglas-fir, in all analyses. We characterized the plot-level intensity of the *C. sulphurascens* infections as the LRR incidence, which is defined as the percentage of the dead Douglas-fir trees sampled where *C. sulphurascens* was present (i.e., dead trees found with *C. sulphurascens* setal hyphae). Some Douglas-fir trees that were present at the time of plot establishment were not found when we surveyed in 2019 and 2020, either because they had died and were completely decayed or had missing tags and could not be successfully related to past tree measurements (Table 2). Because we could not determine if the missing trees died with *C. sulphurascens*, we excluded these trees and only focused on dead trees with tags (i.e., previously measured as live trees). Thus, our sampling for *C. sulphurascens* was restricted to tagged trees, which could bias our calculation of LRR incidence, though we assume that the bias would result in an underestimation within disease pockets.

Individual tree measurements were summarized for each plot and measurement year to characterize forest structure, composition, and biomass change at the ecosystem-level, which is most relevant to forest succession theory. Specifically, we calculated species-specific and total tree density (trees ha^{-1}), tree basal area (m^2 ha^{-1}), and aboveground live tree biomass (AGB; Mg ha^{-1}) for plot and year. We did not account for wood decay (i.e. cull) when estimating live tree biomass. The AGB was estimated with allometric equations for tree bole biomass from BioPak approach (Means, 1994). Additionally, we estimated aboveground NPP of tree boles (NPPB; Mg ha^{-1}) by summing the change in live tree bole-biomass with bole biomass of trees that died during the measurement interval (Acker et al., 2000, 2002; Kloeppel et al., 2007). We used NPPB as a proxy for forest NPP, which reflects the tree-dominated character of aboveground biomass.

To test whether NPPB was related to stand age and LRR incidence across 16 plots at five sites, we used a linear mixed model with three additive fixed effects (LRR incidence, sites, and stand age), with random effects for plots within sites (nested design). Data were analyzed using R (version 4.2.2, R Core Team 2017) using the packages dplyr (version 1.1.0, Wickham et al., 2017), ggplot2 (version 3.4.1, Wickham, 2009), and nlme (version 3.1–160, Pinheiro et al., 2017).

To quantify the long-term consequences of LRR incidence on forest structure and composition, we examined the changes in Douglas-fir and other species live tree density and AGB for all trees as well as the biomass-weighted relative abundance of Douglas-fir and the total live tree biomass. Changes were calculated as the differences between the first and last measurements and divided by the number of years between those measurements to generate rates of change. Therefore, this analysis does not examine short-term (5–10 year) changes related to, for example, tree mortality events, but rather focuses on long-term (>75 years) cumulative change. We then fit simple linear regressions of the change (y) as a function of the LRR incidence (x) with a mixed linear regression using the restricted maximum likelihood approach as implemented by the lme function of the nlme package (Pinheiro et al., 2017) in R. Random intercepts for each site incorporated in the models account for geographic variation in forest dynamics due to climate and tree population genetics. Additionally, plots within sites exhibited similar stand age both when plots were established and at the time of the last measurement (Table 1). To assess whether plots with particularly high LRR incidence drove the results, we fit the models excluding plots with LRR incidence greater than 50 %.

3. Results

3.1. LRR incidence in Douglas-fir

In this study, we confirmed LRR presence in 13 of 16 plots based on visual evidence (i.e. setal hyphae or mycelium), and did not find any presence at GP05, WI01, and WI03 (Table 2). Two plots at the OL site had the greatest LRR incidence (OL04 = 55.6 % and OL03 = 53.6 %), followed by WR04 (45.9 %). The remaining plots exhibited LRR incidence 0.0 %–33.3 %. In addition to tagged dead trees, we located evidence of LRR on 51 dead untagged trees across the 13 positive plots,

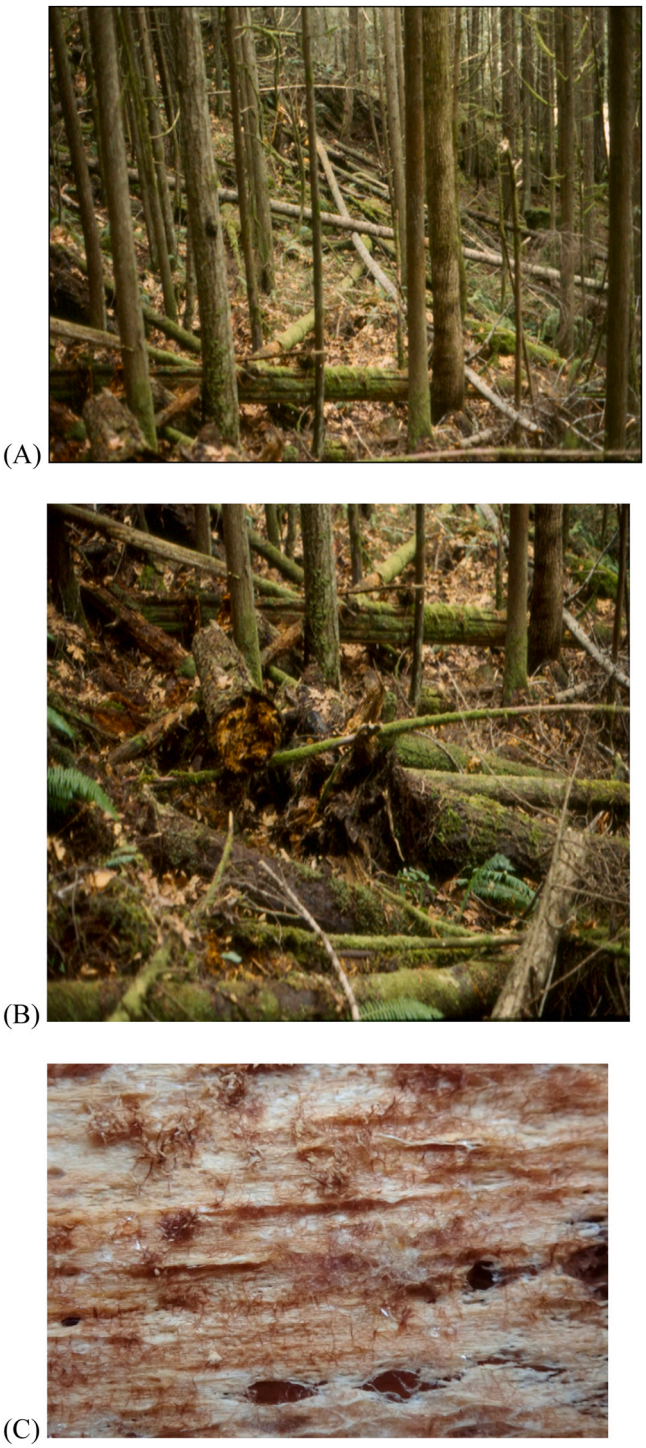

Fig. 2. (A) Forest near Blyn, Washington with heavy laminated root rot (LRR) presence, (B) down wood caused by LRR, (C) *Coniferiporia sulphurascens* setal hyphae.

which could not be directly related to our long-term tree data. For tagged dead trees with contemporary evidence of LRR, tree deaths ranged from 1934 to 2015 (Figure S1). Across plots, mean year of first observed tree death with LRR presence during the contemporary sampling was 1980.5 (95 % confidence interval 1972.3–1988.7).

We observed large variations in LRR incidence between and within sites. Overall, OL plots and one of the WR plots had the highest LRR incidence (17.6 %–55.6 % and 5.6 %–52.9 % respectively, Table 2). LRR incidence within WR plots varied substantially across plots (0 %, 5.6 %, and 52.9 %), even though LRR evidence (e.g. setal hyphae) was common in the area immediately outside of the plots. MH and GP sites had lower LRR incidence (0–21.9 %, Table 2). WI site had the lowest LRR incidence, as we only found one tree within one plot and one tree nearby but outside of the plot to have LRR presence (Table 2).

Table 2
Summary of dead Douglas-fir (*Pseudotsuga menziesii*) trees and laminated root rot (LRR) incidence in the plots. Summaries here do not include tree species other than Douglas-fir.

Plot	Alive trees (n)	Dead tagged trees (n)	Missing trees* (n)	Dead tree with LRR presence ** (n)	Dead tree percentage in the plot** (%)	Dead tree LRR Incidence ** (%)
GP01	43	74	112	7	63.2 %	9.5 %
GP03	71	49	147	1	40.8 %	2.0 %
GP05	53	57	30	0	51.8 %	0.0 %
WR04	40	34	95	18	45.9 %	52.9 %
WR05	67	8	78	0	10.7 %	0.0 %
WR90	54	18	32	1	25.0 %	5.6 %
MH01	70	28	229	1	28.6 %	3.6 %
MH02	104	64	274	14	38.1 %	21.9 %
MH03	92	63	214	4	40.6 %	6.3 %
WI01	73	20	99	0	21.5 %	0.0 %
WI02	71	3	140	1	4.1 %	33.3 %
WI03	73	13	100	0	15.1 %	0.0 %
OL01	112	57	210	13	33.7 %	22.8 %
OL02	47	102	202	18	68.5 %	17.6 %
OL03	56	28	388	15	33.3 %	53.6 %
OL04	87	54	548	30	38.3 %	55.6 %

* Missing tree refers to a tree previously measured in the plot but not found or not identified in 2019 and 2020.
** Includes only dead Douglas-fir with tags attached. Plots are Olympic peninsula (OL), Gifford Pinchot (GP), Wind River (WR), Mount Hood (MH), and Willamette Valley (WI), as described in Table 1.

3.2. *Coniferiporia sulphurascens* confirmation and genet identification

A total of 34 *C. sulphurascens* isolates were collected from 11 plots across five sites in Oregon and Washington. DNA sequencing of ITS region verified/confirmed *C. sulphurascens*. According to Lim et al. (2008), there are two types (1 and 2) of ITS sequences of *C. sulphurascens* in the Pacific Northwest, and both of them were discovered from our sites (GenBank submission #: ITS type 1 – PQ725603; ITS type 2 – PQ725610). Based on somatic pairing tests, one to two genets were

Table 3
Sample isolates identified as *Coniferiporia sulphurascens* based on internal transcribed spacer (ITS) sequences, and their assignment to genets based on somatic pairing tests. Plots are Olympic peninsula (OL), Gifford Pinchot (GP), Wind River (WR), Mount Hood (MH), and Willamette Valley (WI), as described in Table 1.

Plot	Number of isolates	Number of unique genet(s)
GP01	3	1
GP03	2	1
GP05	**	**
WR04	3	2
WR05	*	*
WR90	1	1
MH01	*	*
MH02	3	1
MH03	2	2
WI01	**	**
WI02	2	1
WI03	**	**
OL01	5	1
OL02	5	1
OL03	4	1
OL04	4	2

* *C. sulphurascens* confirmed in the field but failed to isolate in the laboratory.
** No *C. sulphurascens* evidence within the plot.

identified at each plot (Table 3).

3.3. Long-term changes in forest structure and composition

For most plots, average annual live tree basal area increased (69 % of plots; Fig. 3, Table S1), density decreased (56 % of plots; Figure S2, Table S2), and biomass increased (63 % of plots; Figure S3, Table S3) from plot establishment to the latest survey. For Douglas-fir specifically, average annual live tree basal area increased (50 % of plots; Fig. 3, Table S1), density decreased (100 % of plots; Figure S2, Table S2), and biomass increased (63 % of plots; Figure S3, Table S3) over time. The GP plots showed a large decline in Douglas-fir basal area around 1990–1995 (Fig. 3) that was primarily associated with bark beetles and windthrow. For most sites, we observed at least one measurement interval where basal area and biomass declined, reflecting mortality in excess of the growth during that interval. Most notably, live biomass at GP01 declined by approximately half between 112–117 years. Douglas-fir tree densities followed a relatively consistent decline, but total tree density often exhibited at least some increases at ages greater than 100 years, reflecting ingrowth of other tree species (Figure S2).

All 16 plots in this study were Douglas-fir dominated forests when long-term measurements began, with increasing contributions of western hemlock and western redcedar through time. By the end of our study period, shade-tolerant conifer trees other than Douglas-fir accounted for 5.1 % to 77.3 % of the tree density (Fig. 4A) and 0.7 % to 46.5 % of the tree basal area (Fig. 4B), although Douglas-fir still accounted for most of the live tree basal area in all plots. Western red cedar was the shade-tolerant dominant tree species at OL site, while other sites the shade-tolerant tree species was dominated by western hemlock (Fig. 4).

3.4. LRR vs Douglas-fir forest productivity, structure, and composition

The NPPB changed mostly through the time after stands established. We did not have LRR incidence data for each plot at establishment, but our model showed that NPPB was negatively associated with LRR incidence measured in 2019 and 2020 ($F=10.59$, $p = 0.009$) after accounting stand age and site effects, decreased 0.24 megagrams per hectare per year with 1 % LRR incidence increase. Compared to LRR incidence, the NPPB was more correlated to stand age and site effects. The NPPB was strongly negatively associated with stand age ($F=17.45$, $p < 0.0001$), decreased 0.01 megagrams per hectare per year for each additional year in stand age after accounting for LRR incidence and site effects. Also, NPPB had significant differences between sites after considering LRR incidence and stand age effects ($F=8.70$, $p = 0.003$), showed that GP plots had the highest productivity while OL plots had the least.

As stands aged and accumulated more live tree basal area, we observed declining dominance of Douglas-fir across all plots during the study period. OL plots exhibited stable or declining basal area after 100 years of age (Fig. 3) and the most dramatic decrease in Douglas-fir

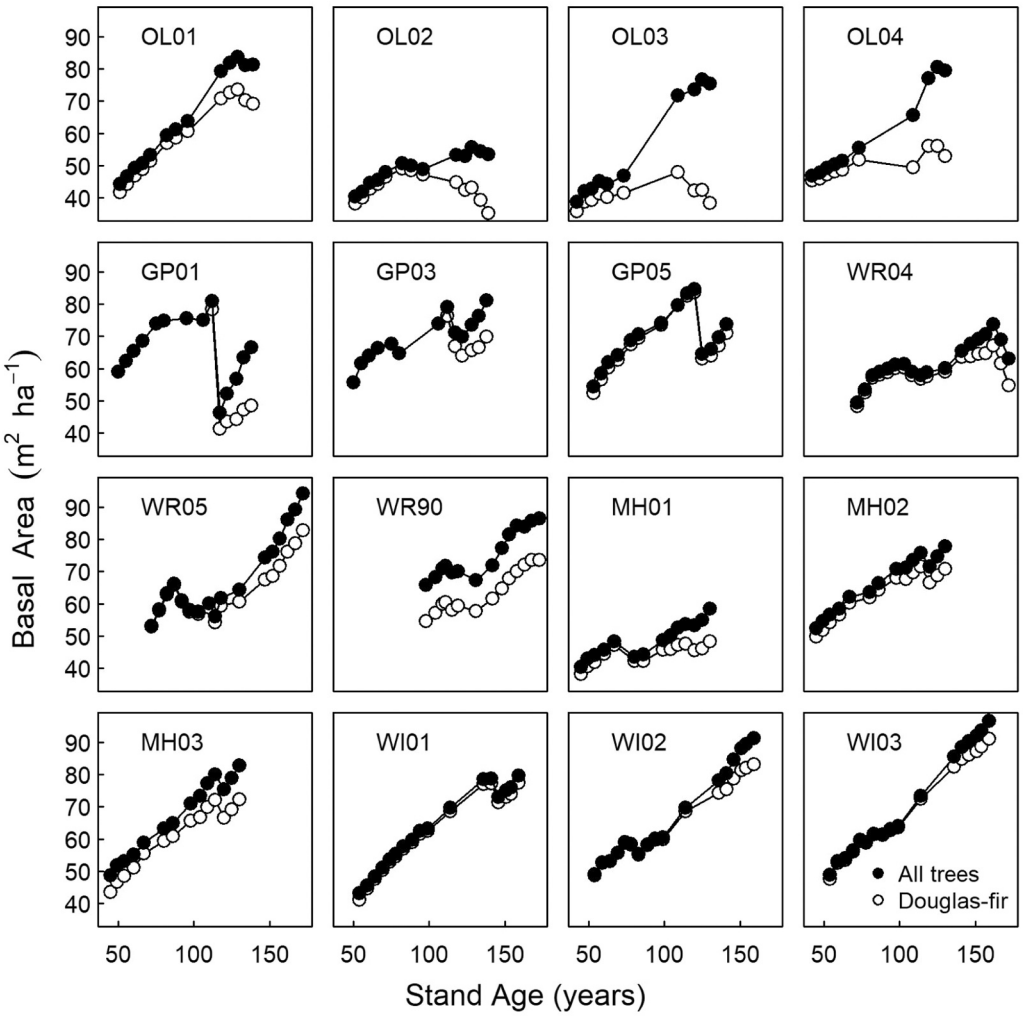

Fig. 3. Plot-level live tree basal area ($m^2\ ha^{-1}$) patterns as a function of stand age for all trees (including Douglas-fir) and Douglas-fir (*Pseudotsuga menziesii*) trees. Plots are Olympic peninsula (OL), Gifford Pinchot (GP), Wind River (WR), Mount Hood (MH), and Willamette Valley (WI), as described in Table 1.

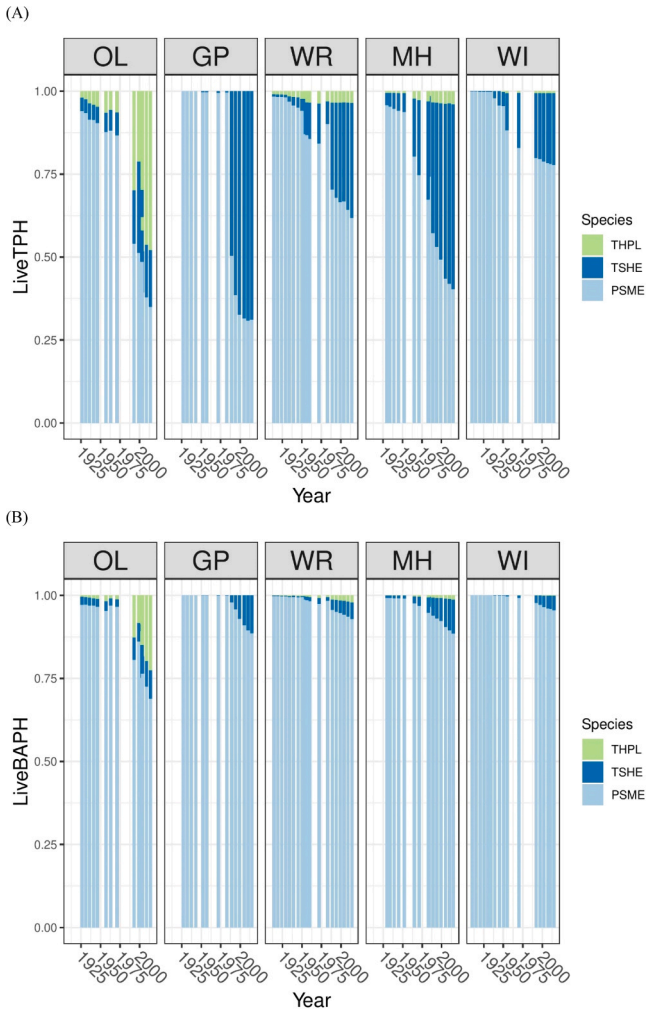

Fig. 4. Relative abundance (ratio 0–1) in terms of (A) living trees per hectare (LiveTPH) and (B) living tree basal area per hectare (LiveBAPH) from 1925 to 2015 of Douglas-fir (*Pseudotsuga menziesii*; PSME, light blue), western hemlock (*Tsuga heterophylla*; TSHE, dark blue) and western red cedar (*Thuja plicata*; THPL, green) across five sites. Plots located on the same site were pulled together to represent the vegetation changes in the site level. Sites from left to right were from North to South geographically. Sites are Olympic peninsula (OL), Gifford Pinchot (GP), Wind River (WR), Mount Hood (MH), and Willamette Valley (WI), as described in Table 1.

dominance in terms of basal area (Fig. 5). As the second highest LRR incidence plot, OL03 had almost a 50 % loss of percent of total basal area that was Douglas-fir. In contrast, plots without any evidence of LRR presence (GP05, WI01, and WI03), had less than a 10 % decline in Douglas-fir basal area dominance over time.

Examining changes in tree density and AGB from plot initiation to present indicated decreasing Douglas-fir tree density and biomass-weighted abundance and increasing biomass of other species as LRR incidence increased (Fig. 6, Table 4). The 95 % confidence intervals for regression intercepts, which estimate the annual rate of change for plots with LRR incidence equal 0 %, were positive (increasing through time) for other species density and biomass change, Douglas-fir biomass change, and total live tree biomass change, and did not differ from zero (no change through time) for biomass-weighted Douglas-fir relative abundance change and Douglas-fir tree density change. The 95 % confidence intervals for the effects of LRR on changes in Douglas-fir tree density and biomass-weighted Douglas-fir relative abundance were negative (Fig. 6a,c) and biomass of other tree species was positive (Fig. 6e). Fitting the same models excluding plots with LRR incidence $\geq$ 50 % indicated that only Douglas-fir density change declined with LRR incidence when severe LRR mortality centers were not considered (Fig. 6a; Table 4).

4. Discussion

Long-term studies, such as ours, are needed to support increased detection and improved understanding of forest pathogens (Flores et al., 2023), especially to prevent ecological surprises (Lindenmayer et al., 2010), as they relate to process like forest community and ecosystem dynamics that unfold over decades or centuries. Importantly, *C. sulphurascens* presence was noted for trees dying as early as 1934, with the average earliest year of dead trees with pathogen presence averaging 1980. This indicates that our contemporary sampling for *C. sulphurascens* is consistent with the long-term occurrence of the pathogen on these plots. Furthermore, past work indicates both a long resonance time for the pathogen in dead wood (>50 years, Hansen 1979a) and a slow rate of spread (0.3 m per year; Hansen and Goheen, 2000). It is possible that the pathogen infected the woody material after tree death, though all plots with contemporary *C. sulphurascens* presence in this study exhibited some tree mortality related to root rot and 85 % had records of either root rot pockets or referenced laminated root rot and/or the pathogen in the between 1990–2010 (Franklin et al., 2022). Therefore, our contemporary characterization of LRR incidence is likely a reasonable representation of plot-level differences in long-term pathogen presence, and thus its association with differences in forest dynamics, in this study.

Our results show that increased LRR incidence, indicating more frequent infection of trees by *C. sulphurascens*, was associated with changes in tree species composition, implying increases in the pace of successional tree species turnover during the transition from young (45–98 years) to mature (130–173 years) Douglas-fir forest. This is an important distinction, because it implies that in terms of ecological services, LRR may not reduce carbon sequestration. However, *C. sulphurascens* is still a primary pathogen of plantation-grown Douglas-fir in the PNW and is a major impediment to short/medium term rotation Douglas-fir forest stand productivity (Sturrock et al., 2018; Morrison et al., 2022).

4.1. Forest composition and successional changes

Changes in forest composition, structure, and succession at our long-term plots were associated with LRR incidence, with our results supporting the findings from old-growth forest studies of Holah et al. (1993);(1998) and Hansen and Goheen (2000) that succession to western hemlock and western redcedar was accelerated in some sites within infection centers in Douglas-fir forests of western Oregon and Washington. Douglas-fir mortality generally leads to increases in western hemlock and western redcedar, which are two shade-tolerant, late--successional species common in these forests (Franklin et al., 2002), and considered as intermediately susceptible and resistant to LRR respectively. The loss of Douglas-fir accelerates succession, but in some cases can lead to dominance of shrubs in root disease centers, which may slow western hemlock and western redcedar response to the opening (Holah et al., 1993).

Root diseases may increase tree diversity in a forest stand by killing the host trees and creating openings for the establishment of less susceptible tree species. In high elevation mountain hemlock (*Tsuga mertensiana*) forests where *C. sulphurascens* has a profound influence on tree species composition and succession, the root disease centers have increased tree species diversity (McCauley and Cook, 1980). Lodgepole pine, western white pine, subalpine fir (*Abies lasiocarpa*), Shasta red fir [*A. magnifica*, called *A. procera* (noble fir) in the publication], Pacific silver fir (*A. amabilis*), mountain hemlock, and Engelmann spruce (*Picea engelmannii*) occur in the root disease centers, while the intact forests are dominated by mountain hemlock with Pacific silver fir, Engelmann

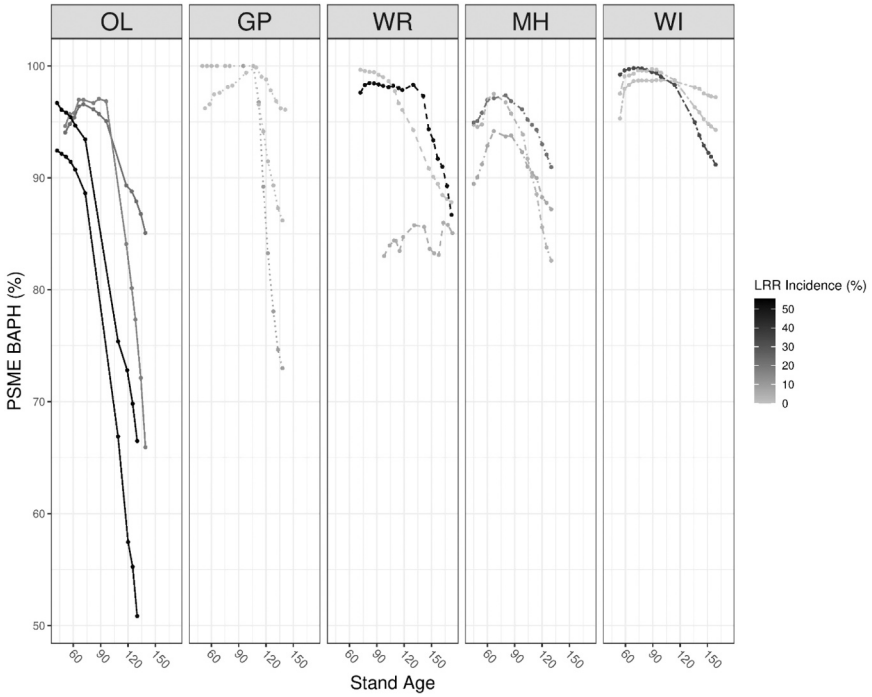

Fig. 5. The changes of percentage of basal area per hectare (BAPH) of Douglas-fir (*Pseudotsuga menziesii*, PSME) through time. The darkness represents the laminated root rot (LRR) incidence on the plot. Plots are Olympic peninsula (OL), Gifford Pinchot (GP), Wind River (WR), Mount Hood (MH), and Willamette Valley (WI), as described in Table 1.

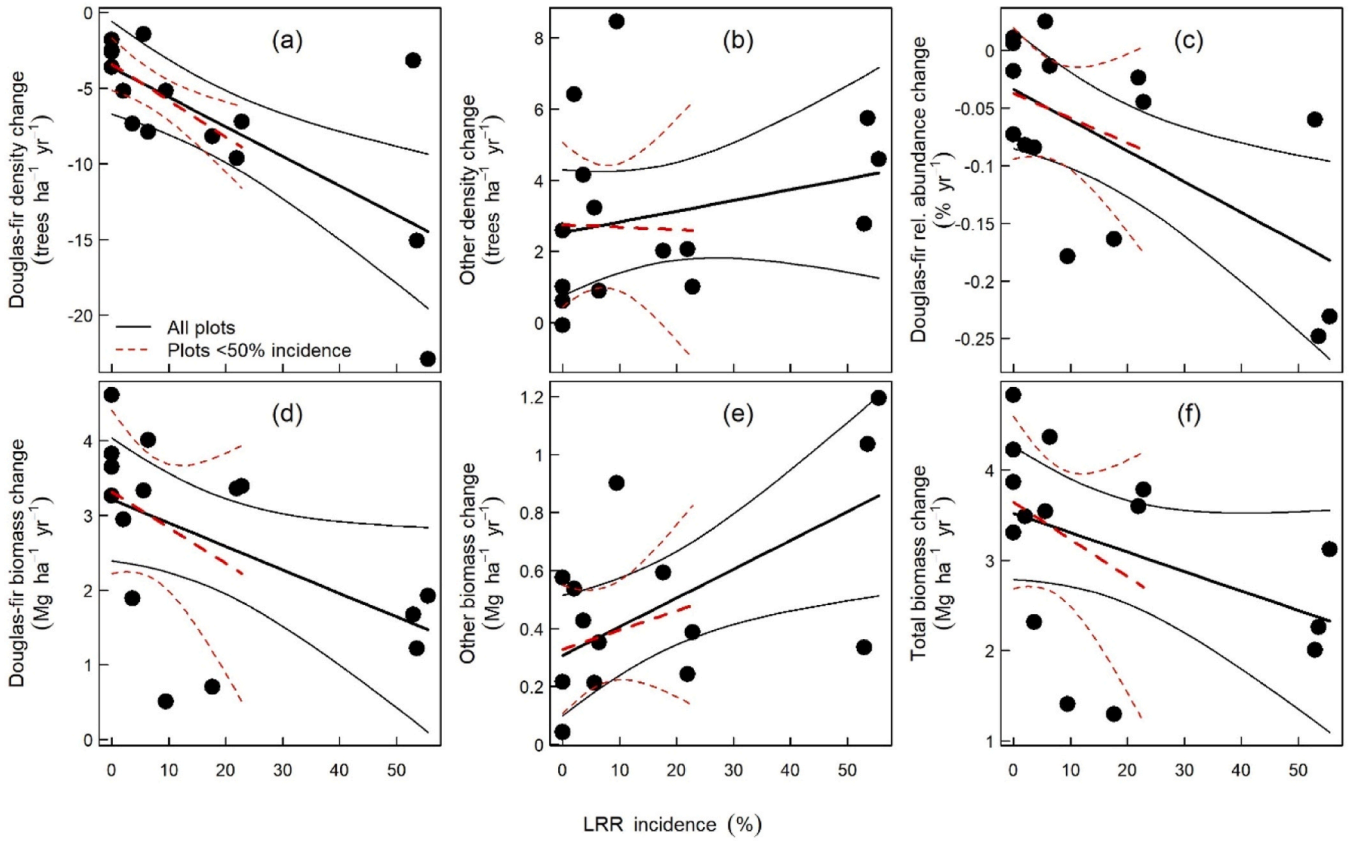

Fig. 6. Median and 95 % credible intervals for the relationship between (a) Douglas-fir (*Pseudotsuga menziesii*) live tree density change, (b) other live tree density, (c) Douglas-fir biomass-weighted relative abundance, (d) Douglas-fir live tree biomass change, (e) other live tree biomass change, and (f) total live tree biomass change. Solid black lines represent regressions based on all plots and dashed red lines represent regressions based on plots with less than 50 % laminated root rot (LRR) incidence. Intercept and slope coefficients are in Table 4.

Table 4
Fixed effects from models using all tree data and those where high laminated root rot (LRR) incidence (>50 %) were excluded. We present the mean parameter estimates and 95 % confidence intervals (in parentheses) for the intercept and the LRR effect. PSME refers to *Pseudotsuga menziesii* (Douglas-fir).

	All Data (n = 15)		LRR < 50 % (n = 12)	
	intercept	LRR effect	intercept	LRR effect
PSME tree density change	-3.90 (-7.82, 0.028)	-0.17* (-0.28, −0.05)	-4.14* (-6.79, −1.52)	-0.11* (-0.20, −0.02)
Non-PSME tree density change	2.42* (0.40, 4.44)	0.04 (-0.04, 0.11)	2.53 (-0.27, 5.33)	-0.01 (-0.23,0.25)
PSME Rel. Abun. change	-0.04 (-0.09, 0.02)	-0.0025* (-0.0046, −0.0003)	-0.04 (-0.10,0.03)	-0.0022 (-0.0078, 0.0035)
PSME AGB change	3.21* (2.35, 4.07)	-0.031 (-0.064, 0.001)	3.31* (2.11, 4.51)	-0.048 (-0.156, 0.060)
Non-PSME AGB change	0.31* (0.09, 0.52)	0.010* (0.002, 0.018)	0.33* (0.09, 0.57)	0.009 (-0.015, 0.028)
Total AGB change	3.52* (2.75, 4.29)	-0.02 (-0.05, 0.01)	3.64* (2.59, 4.69)	-0.04 (-0.14, 0.053)

* 95 % confidence interval does not include zero.

spruce, and lodgepole pine being minor components. Succession within the root disease centers is mediated by susceptibility to LRR but even susceptible hosts can take decades to die. McCauley and Cook (1980) consider western white and lodgepole pines tolerant to LRR, Pacific silver fir and Shasta red fir intermediately susceptible, and Engelmann spruce and mountain hemlock susceptible. Matson and Boone (1984) suggest that because nitrogen availability increases after mountain hemlock mortality, that tree vigor may also increase and lower susceptibility to disease.

Many other root diseases change forest composition by causing tree mortality of select tree species (Flores et al., 2023; Hawkins and Henkel, 2011; Hansen and Goheen, 2000). Hawkins and Henkel (2011) found that root rot fungi killed more white fir (*A. lowiana*) than Douglas-fir in mixed conifer forests of northern California, and therefore, in the absence of fire, root rot fungi provided for the persistence of Douglas-fir where white fir would have eventually dominated the site. Flores et al. (2023) used 63 long-term plots, from three plot networks, established in 1970–1972 to document changes associated with two species that cause Heterobasidion root disease, *H. irregulare* (pathogen of pines) and *H. occidentale* (pathogen of *Abies* species) over 48–49 years in pine and fir forests of California. They found that impacts varied with stand types. In the fir forests, there was a distinct effect, where *Abies* were killed and non-hosts became relatively more abundant. However, in the two pine forest types, the relative dominance of hosts remained unchanged through the study period although basal area and density were reduced.

There is an increasing appreciation that succession in the Douglas-fir dominated temperate coniferous forests of the Pacific Northwest can involve several distinct pathways determined, in part, by local variation in forest disturbance (Tepley et al., 2013; Reilly and Spies, 2015). Forest composition changes related to the presence of *C. sulphurascens* likely translate into long-term changes in forest successional trends by reducing density of Doulgas-fir and increasing density of less susceptible hosts. Our results show that LRR is associated with the shift from early successional species to late successional species, as was proposed by Holah et al., (1997). However, despite consistent reductions in Douglas-fir live tree density (Fig. 6a), greater decreases in Douglas-fir dominance with respect to live tree biomass associated with *C. sulphurascens* were supported when LRR incidence was greater than 50 %, but there was insufficient evidence to support a similar trend when high magnitude LRR plots were excluded (Fig. 6c). This result may reflect that major changes in Douglas-fir dominance not only require loss of Douglas-fir through mortality, but also opening of the forest canopy to increase growth rates in other tree species.

We did not measure non-tree vegetation changes, but composition within forest stands exposed to *C. sulphurascens* also changes, most likely due to increased sunlight reaching the forest floor (Holah et al., 1993; Goheen and Hansen, 2000). Average cover of all understory vegetation increased, within infections centers in old-growth Douglas-fir forests (Holah et al., 1993), while distance to forest edge of the infection center was also a significant factor in composition. In some sites, Holah et al. (1997), found that the abundance of dense shrub cover can prevent rapid establishment of western hemlock and western red cedar, which indicates that LRR may have slowed the transition of tree species composition.

Wildlife use of root disease infection centers vs closed forest habitat is also affected. For example, in British Columbia, Canada, the influence of canopy gaps on snowpack during winter has been shown to influence the distribution of mule deer within forest stands affected by two root diseases; Armillaria root disease and LRR (Carswell et al., 2021). Mule deer avoided root disease infection centers in winter, but wildlife diversity in general was higher within root disease caused canopy openings.

4.2. Net primary productivity

Similar to forest composition, we found statistical evidence that *C. sulphurascens* was associated with forest productivity over the 75–105 year period of observation in our study. LRR incidence, site effect, and stand age were all related to NPPB. Though increases in the AGB change of species other than Douglas-fir with LRR incidence were observed (Fig. 6e), those changes were small in comparison to the mean live tree biomass change observed across plots (Fig. 6f). Even in the most heavily impacted plots with > 50 % LRR incidence (e.g., OL03, OL04, and WR04), evidence of declining AGB was not evident (Figure S3). At the individual tree level, ecophysiological models indicate that growth and mortality are more sensitive to root disease pathogens than biotic agents attacking other plant organs (Dietze and Matthes, 2014). If individual infected trees are exhibiting substantial reductions in growth and increased mortality, then the lack of an effect on total live biomass change indicates that uninfected Douglas-fir and shade-tolerant tree species, which increased in density throughout the study period (Figure S2), may be increasing their growth rates in response to elevated resource availability (Oren et al., 1985). That compensatory response may degrade at more extreme levels of infection not observed in this study, as observed for the effects of emerald ash borer (Flower and Gonzalez-Meler, 2015).

Most assessments of root diseases suggest negative impacts on biomass and stand productivity (Healey et al., 2016; Lockman and Kearns, 2016). In this study, we found LRR reduces Douglas-fir density, but facilitates increases in aboveground live biomass of other tree species which were not as vulnerable to *C. sulphurascens*. Therefore, although vegetation composition changed with time and pathogen presence, NPPB and AGB indicated no changes in overall ecosystem productivity. This result may not be universal (Boone et al., 1988; Flores et al., 2023) and suggests resilience and adaptation in Douglas-fir forests to a native root disease. Boone et al. (1984) documented total ecosystem carbon (TEC) and net ecosystem production (NEP) changes along a 96-year regrowth sequence from mature mountain hemlock into a root disease center. TEC dropped after stand death and did not return to pre-stand death levels, while NEP was negative just after stand death and remained at zero thru 96 years after stand mortality. In this study, since decay (i.e., cull) was not accounted for in the analysis, we likely overestimated live biomass; therefore, detecting biomass change due to LRR may have been more difficult.

4.3. Species confirmation of C. sulphurascens and genet identification

We confirmed *C. sulphurascens* as the root disease pathogen present in our plot network using a DNA-based method. Two types (1 and 2) of ITS sequences (Lim et al., 2008) were discovered from our sites. Ecological differences (e.g., disease behavior) between the two ITS types are unknown, and this warrants further investigation. From our study, we did not see the associations between the type of ITS sequences and the pathogenicity. For each plot, one or two genets (clones) were identified based on somatic pairing tests (Table 3). This implies each plot only had one or two clones causing Douglas-fir mortality, which is consistent with the general paradigm that *C. sulphuracens* spreads by root-to-root contacts and grafts, and individual genets dominate local mortality centers (Bae et al., 1994; Hansen and Goheen, 2000; Sturrock et al., 2018). Identifying genets will help to understand genetic diversity of *C. sulphurascens*, how it spreads and how long the pathogen persists in the site.

4.4. Management implications

This study provides a rare opportunity to examine mature and old-growth forest ecosystem and community dynamics in the presence of an important tree pathogen. It has previously been recommended that intensive forest management aimed at minimizing Douglas-fir mortality, and thus timber volume losses, due to LRR may be supported by silvicultural solutions such as stump removal and planting alternative species less susceptible to *C. sulphurascens* (Morrison et al., 2014). Given our results indicating no changes in the rate of aboveground live biomass change and accelerated shift toward shade-tolerant, late-successional tree species with LRR incidence (Fig. 6, Table 4), carbon sequestration and old-growth forest management aimed at conservation may not benefit from silvicultural intervention to minimize negative effects of LRR.

Our results are unique to the Douglas-fir systems of western Oregon and Washington, USA and other forest types may not respond similarly to root disease caused mortality (Flores et al., 2023, Hansen and Goheen, 2000). Given the management objectives for a Douglas-fir stand impacted by LRR, our results suggest different potential approaches. If old-growth and complex forest structure development is desired, the presence of *C. sulphurascens* may relate to forest productivity, and more species and structurally diverse forests develop more rapidly in plots with greater LRR incidence (Fig. 3, 6, S2, S3). If short-rotation Douglas-fir timber production is the management objective, LRR should be considered and some well-known management responses need to be applied, including growing alternative species in root disease centers (Sturrock et al., 2018) but not thinning treatment (McMurtrey et al., 2024). Our results support the idea that *C. sulphurascens*, and likely other root disease pathogens, may play an important role in facilitating the development of old-growth forest structure and composition (Franklin et al., 2002).

Changing climate may interact with tree pathogens to accelerate forest change, but those effects are difficult to assess as they require a confluence of available hosts, presence of the pathogen, and environmental conditions appropriate for the disease, all of which may be changing as the climate changes (Hennon et al., 2021; Simler-Williamson et al., 2019). In the Douglas-fir forests of our study area, climate change complicates applying our findings when managing forests for future resilience (Agne et al., 2017). *Coniferiporia sulphurascens* appears to infect susceptible hosts regardless of individual tree vigor (Hansen and Goheen, 2000), therefore may not increase due to increased tree stress. Unlike *C. sulphurascens*, *Armillaria solidipes*, the pathogen that causes Armillaria root disease, which is linked to tree stress (Lockman and Kearns, 2016), has been modeled and suggests that *Armillaria* will increase significantly in the northwestern United States due to increasing tree stress (Kim et al., 2021). However, if hotter drought causes a decrease in stand density, there will likely be a decrease in root-to-root contacts that could result in less disease transmission (Kim et al., 2021). Drought can also slow the growth rate of roots which may also result in less root-to-root contacts and *C. sulphurascens* disease transmission, however, drought and water stress may increase susceptibility of trees to bark beetle-caused mortality (Kolb et al., 2019) and Douglas-fir beetle activity is associated with root disease during non-outbreak conditions (Goheen and Willhite, 2021). Hotter drought can exacerbate this effect, causing increased mortality of trees already compromised by root disease (Agne et al., 2017).

Extreme weather events like the Pacific Northwest heat dome in 2021 (Still et al., 2023, Sibley et al., 2025), may suppress tree vigor and make them more vulnerable to other pathogens (Allen et al., 2015). Even if the influences of LRR on Douglas-fir do not change with climate, species interactions with other biotic agents may occur. For example, western hemlock with moderate and heavy dwarf mistletoe infections have exhibited greater growth reductions and mortality than uninfected trees during hotter and drier conditions (Bell et al., 2020). If growth reductions and mortality associated with the joint effects of dwarf mistletoe and climate change lead to a general decline in western hemlock productivity, the apparent resilience of NPP to LRR in our study might also degrade. To inform future understanding of climate change and forest disturbance, maintaining long-term research plots is critical for predicting the interacting effects of biotic and abiotic disturbances on LRR and Douglas-fir forests.

CRediT authorship contribution statement

Bluhm Andrew A: Writing – review & editing, Data curation. **Chadwick Kristen L:** Writing – review & editing, Methodology, Investigation. **Shaw David C:** Writing – review & editing, Writing – original draft, Supervision, Methodology, Investigation, Conceptualization. **Pabst Robert J:** Writing – review & editing, Methodology, Investigation, Data curation, Conceptualization. **Holly S.J. Kearns:** Writing – review & editing, Methodology, Investigation, Conceptualization. **Yung-Hsiang Lan:** Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation. **Mee-Sook Kim:** Writing – review & editing, Writing – original draft, Supervision, Methodology, Investigation, Funding acquisition, Conceptualization. **Bell David M:** Writing – review & editing, Writing – original draft, Visualization, Supervision, Methodology, Formal analysis, Conceptualization.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.foreco.2025.123426.

Data availability

Data will be made available on request.

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