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ARTICLE

Effects of radial growth rate on outerwood properties of coastal Douglas-fir in healthy stands versus stands impacted by Swiss needle cast

B. Lachenbruch and R.G. Johnson

Abstract: A common belief in forestry is that rapid growth in coastal Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco var. *menziesii*) results in decreased outerwood quality. In Oregon, the opposite pattern has been reported for stands affected by Swiss needle cast, whereby a native fungus causes premature needle drop and an increase in latewood proportion (LW%), wood density, stiffness (MOE), and strength (MOR). Using a combination of new and existing data, we compared the properties of ~25-year-old outerwood from 18 healthy and 14 diseased stands using direct tests (6–8 beams from 7–12 trees per stand, 2614 beams total) and indirect SilviScanII tests (1 sample for each of the 366 trees). As seen before, diseased stands showed a decrease in wood quality with growth rate: ring count was strongly and positively correlated with density, MOE, and MOR (r^2 = 0.74, 0.65, and 0.63), and LW% was positively correlated with ring count, density, MOE, and MOR (r^2 = 0.50, 0.62, 0.30, and 0.44). In contrast, healthy stands showed no significant effect of ring count on density, MOE, or MOR. LW% was weakly and significantly correlated with MOE (r^2 = 0.25) but not with ring count, density, or MOR. Among healthy stands, growth acceleration had no adverse effects on outerwood properties.

Key words: growth rate, modulus of elasticity, modulus of rupture, density, Swiss needle cast.

Résumé : Selon une croyance populaire la croissance rapide du douglas de Menzies typique (*Pseudotsuga menziesii* (Mirb.) Franco var. *menziesii*) se solde par une détérioration de la qualité du bois mature. En Oregon, la tendance contraire a été rapportée dans les peuplements infestés par la rouille suisse des aiguilles, où un champignon indigène cause la chute prématurée des aiguilles et une augmentation de la proportion de bois final (%BF), de la densité, de la rigidité (MOE) et de la résistance (MOR) du bois. À l'aide d'une combinaison de données existantes et inédites, nous avons comparé les propriétés du bois mature de tiges âgées d'environ 25 ans provenant de peuplements sains (18) et malades (14) à l'aide de tests directs (6–8 éprouvettes prélevées sur 7–12 arbres par peuplement, soit au total 2614 éprouvettes) et de tests indirects effectués avec SilviScan II (un échantillon pour chacun des 366 arbres). Comme nous l'avions déjà constaté, la qualité du bois diminue avec le taux de croissance dans les peuplements malades : le décompte des cernes annuels était étroitement et positivement corrélé avec la densité, MOE et MOR (r^2 = 0,74, 0,65 et 0,63) et le %BF était positivement corrélé avec le décompte des cernes annuels, la densité, MOE et MOR (r^2 = 0,50, 0,62, 0,30 et 0,44). Par contre, dans les peuplements sains le décompte des cernes annuels n'a pas d'effet significatif sur la densité, MOE ou MOR. Le %BM était faiblement mais significativement corrélé avec MOE (r^2 = 0,25) mais pas avec le décompte des cernes annuels, la densité ou MOR. Parmi les peuplements sains, l'accélération de la croissance n'a pas d'effet négatif sur les propriétés du bois mature. [Traduit par la Rédaction]

Mots-clés : taux de croissance, module d'élasticité, module de rupture, densité, rouille suisse des aiguilles.

Introduction

Coastal Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco var. *menziesii*) is widely grown in plantations in its native range in western North America and across other parts of the world (Lavender and Hermann 2014). Highly valued for its use in structural applications, such as dimension lumber and components of glued lumber, the quality of Douglas-fir is judged largely by its mechanical and physical properties (Megraw 1986; WCLIB 2018). Because a frequent silvicultural goal for this species is the rapid production of a high volume of wood (Howe et al. 2006), it is important to understand the effects of growth rate on its mechanical and physical properties. Moreover, at least 24% of Douglas-fir timberland in the western United States is estimated to have very low growth rates owing to very high stocking levels (Green et al. 2005).

There is a widespread perception in forestry that rapidly grown Douglas-fir has inferior wood properties for structural uses (e.g., Markwardt and Wilson 1935), but that perception may come from the mistaken comparisons of logs of the same diameter but different growth rates. Faster growth results in a log that has a higher proportion of corewood (juvenile wood in some literature), which does have inferior properties (Kennedy 1995), and thus the properties of a rapidly grown young log are inferior to those of a slower-grown older log.

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We previously examined how Douglas-fir wood quality is affected by growth suppression due to swiss needle cast (SNC), a pathogenic needle disease caused by the native fungus *Nothophaeocryptopus gaeumannii*. SNC causes needles to drop pre-maturely. In diseased stands, radial growth rate was positively associated with the number of years of needle retention (NR, in years), meaning that the more impacted a stand was by SNC, the slower the trees grew. Radial growth rate in these diseased stands was negatively associated with latewood proportion (LW%), wood density, modulus of elasticity (MOE), and modulus of rupture (MOR) (Johnson et al. 2003, 2005). We observed these strong among-stand effects even though the proportion of total variation that occurred among stands was lower than that occurring between stands for density, LW%, MOE, and MOR (Johnson et al. 2005). In other words, in spite of large within-stand variation in properties, the more impacted a stand was by disease, the slower was its radial growth rate and the better was its wood quality for structural uses.

This pattern with diseased trees contrasts with studies showing that in healthy coastal Douglas-fir trees grown elsewhere, radial growth rate has no appreciable effect on wood quality (e.g., Pollet et al. 2017). Without a good estimate of the growth ring – wood property relationships for healthy stands in the Pacific Northwest, we could not deduce whether the observed relationships in the diseased trees were due to an inherent change in wood structure associated with the disease or were simply a function of slow growth. The current project was designed to characterize the effects of radial growth rate and NR in healthy stands in their native range in western Oregon, USA, to compare to our already-published dataset on diseased trees in order to infer whether the relationship of ring count to wood properties differs between healthy and diseased stands.

There are numerous challenges to using the literature to predict growth rate effects on wood properties in a particular region. Some studies do not specify which subspecies or provenance of Douglas-fir was studied (e.g., Pollet et al. 2017), but coastal and inland Douglas-fir (*Pseudotsuga menziesii* var. *glauca*) can differ greatly in characteristics such as growth habit (Silen 1978) and habitat (Lavender and Hermann 2014), which in turn can affect wood properties. Wood properties may vary by provenance (e.g., Drow 1957; Sergent et al. 2014), and the provenance effect can be larger than the environmental effects (Johnson and Gartner 2006).

Another challenge is that the wood properties studied here vary strongly over a specific range of cambial ages (Megraw 1986), but studies differ as to whether they report the cambial age (e.g., Rais et al. 2014), which cambial age is studied, and the threshold age used to separate corewood from outerwood (juvenile from mature wood in some literature) (e.g., 15 years in Henin et al. 2018; 18 years in Bendtsen et al. 1988; 20 years in Barrett and Kellogg 1989). Another difficulty with comparing studies occurs when the cambial age at which a treatment was imposed is not provided. For example, a study that compared wood properties from thinned and unthinned stands (Kranjc et al. 2019) would have different expected outcomes if the thinning occurred when the trees were producing corewood versus outerwood. Furthermore, tools such as acoustic velocity that measure the whole tree or whole log (e.g., Briggs et al. 2008) give values that include the effects of corewood and outerwood properties, and corewood proportion. In addition, different experimental methods can make it hard to compare results. Ring count (no. rings·cm^{-1} in a designated zone) is a common metric of growth rate for forest managers, and is included in some lumber- and log-grading rules (e.g., WCLIB 2018). Some studies use ring width instead, which emphasizes the rapidity of radial growth rather than a piece of lumber's characteristics. But because ring width is the inverse of ring count, these two growth rate metrics will have different statistical relationships with the properties that influence wood quality. Some studies report properties as a continuous variable (e.g., Drow 1957), while others report whether growth rate caused a value to fall below a threshold design value (e.g., Henin et al. 2018). Lastly, inferences may differ based on whether a study has included a relatively narrow (Green et al. 2005) or wide (Drow 1957) range of growth rates.

Even with these potential complicating factors, several patterns emerge for healthy Douglas-fir. Radial growth rate generally has a small negative effect or no effect on outerwood density, MOE, or MOR (e.g., Pollet et al. 2017). If a significant effect of growth rate is detected, it is usually very low or is caused by differences only at very rapid growth rates. For example, Drow (1957) reported on wood properties of ~4000 lumber specimens that came from four regions: Coast, Interior North, Interior West, and Interior South. Within a region, the author found no apparent effect of ring count between 30 and 3 rings·cm^{-1}, but he did find that values of MOE, MOR, and density declined between 3 and 1 rings·cm^{-1}. That study did not separate cambial age from growth rate effects, and the author stated that these very rapidly grown specimens were likely from corewood. Another pattern is that LW% is typically a strong driver of wood density (Smith 1956), and wood density is a stronger driver of MOE and MOR than is microfibril angle (MfA) (Lachenbruch et al. 2010).

SNC is locally severe in many coastal areas of Oregon and Washington (Lan et al. 2019) and planted forests elsewhere (Kimberley et al. 2011). The fungus responsible for SNC causes premature loss of needles. Shortly after budbreak, the fungus infects young needles (Stone et al. 2008) and grows within them before producing reproductive structures that plug the stomata and inhibit transpiration and carbon uptake (Manter et al. 2000). When ~50% of the stomata are blocked, trees abscise the needles (Hansen et al. 2000). Needle retention (NR) quantifies how many cohorts (years) of needles the tree retains at standardized mid-canopy positions (Ritokova et al. 2016). At NR values up to about 3 years, stand productivity is strongly and positively correlated with NR, and thus NR is often used as an index of disease impact (low NR = high impact). This strong effect of NR on productivity is attributed mainly to the effects of foliage area (Maguire et al. 2011).

The objectives of this study were to examine the effects of radial growth rate on outerwood properties in healthy versus diseased stands of coastal Douglas-fir grown for wood production in western Oregon. This research differs from our previously published work in several ways. First, Johnson et al. (2005) focused on growth rate effects of SNC disease on wood properties in 19 stands, only four of which were considered healthy. The current study contrasts relationships in 14 diseased stands (using some of the data from the Johnson et al. 2005 study) with relationships in 18 healthy stands (adding 14 stands that were not in Johnson et al. 2005). Secondly, the current work reassigned several diseased stands from Johnson et al. 2005 to healthy status based on current understanding, and eliminated one stand that was an outlier for age (see Materials and methods). With this larger sample size of healthy stands and the modified set of stands that are diseased, we were able to reevaluate the growth rate effects on wood properties in diseased stands, as well as evaluate de novo the growth rate effects in healthy stands.

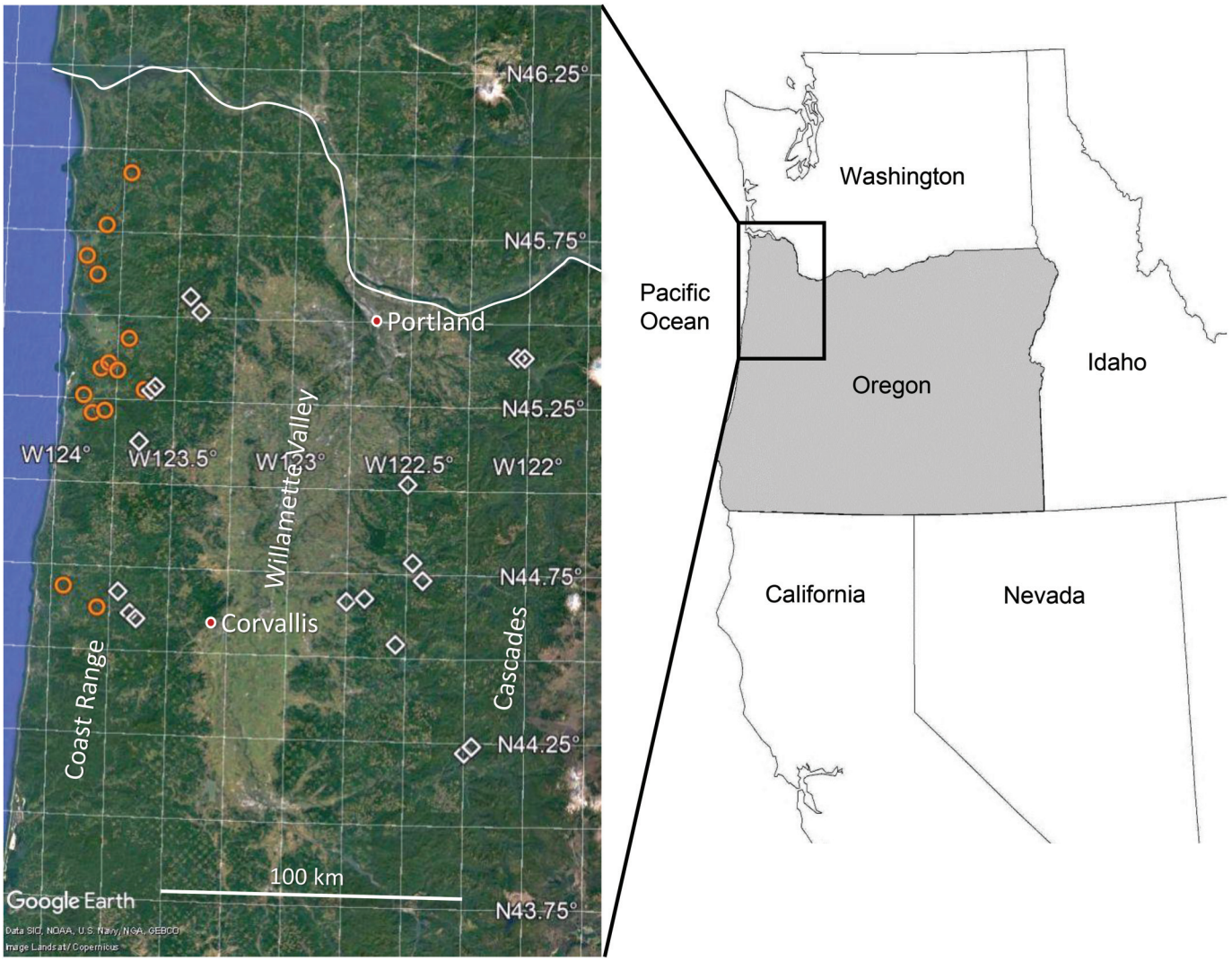
We formalized our hypotheses as follows: (*i*) There are no relationships between NR and either ring count or LW% in healthy stands, but in diseased stands both NR and ring count are negatively related to LW%; (*ii*) in both healthy and diseased stands, LW% has a positive relationship with wood density, MOE, and MOR, but MfA has no relationship or only weak negative relationships with these variables; (*iii*) in healthy stands, NR and ring count are not related to density, MOE, or MOR, but in diseased stands NR is negatively related to density, MOE, and MOR and ring count is positively related to density, MOE, and MOR.

Materials and methods

Stand and tree selection

This study compared wood properties of coastal Douglas-fir trees from healthy and diseased (affected by SNC) stands. The

Fig. 1. Locations of healthy (white diamonds) and diseased (orange circles) stands of coastal Douglas-fir sampled in Oregon, USA. Base map source: Google Earth. Software used to enhance the map and add words: PowerPoint. [Colour online.]



categories "healthy" and "diseased" are indicative of the impact, but not necessarily the presence, of the disease because SNC is present at very low levels throughout the area sampled (Ritokova 2016). However, growth impact studies (Maguire et al. 2011) and other research by the Oregon State University Swiss Needle Cast Cooperative (SNCC) (D.B. Mainwaring, personal communication, 2019) have showed that decreases in growth are seen only at values of NR $<$ 3.0 years. For that reason, we classified stands as healthy if NR $\geq$ 3.0 years and as diseased if NR $<$ 3.0 years.

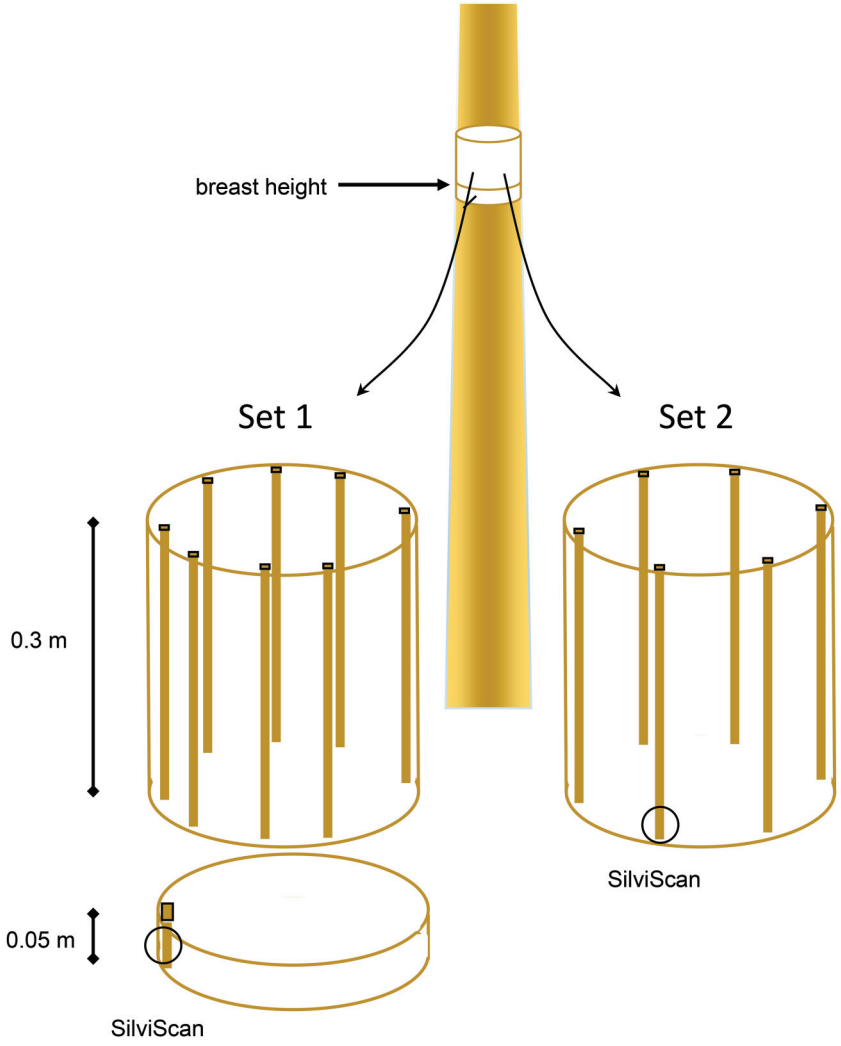
Our sampling was intended to provide a wide range of growth rates and disease severities in operational plantations rather than "mean" conditions. Our stand selection criteria were as follows: the stands were planted; breast-height age was >20 years (preferably >25 years); no precommercial thinning or fertilization had occurred in the previous 7 years; and site indices predicted 32–43 m height at 50 years. We gave these selection criteria to members of the SNCC and asked them to provide candidate sites. We were offered sites by five private timber companies and several state and federal agencies. From the offered stands, we chose those that, in aggregate, provided the full range of our target site indices and had adequate access for hauling out samples. The sampled stands were under the management of the US Bureau of Land Management, the US Forest Service, the Oregon Department of Forestry, Hampton Resources, Inc., Longview Fibre Co., Plum Creek Timber Co., and Simpson Timber Co.

For the first project (Johnson et al. 2005), we asked land managers for sites that fit the aforementioned criteria and had a range of SNC disease severities. They offered stands in the central or west side of the Oregon Coast Range (Fig. 1), typically within 50 km of the ocean, where SNC has the greatest impact. That project compared 15 diseased stands (NR $<$ 2.5 years) with 4 healthy stands. We called these our set 1 stands. In the current project, we eliminated a healthy stand (as an age outlier) and reclassified a diseased stand as healthy (following our revised disease threshold of NR $<$ 3.0 years), giving us 4 healthy and 14 diseased stands in set 1.

In 2003, we sampled a further 14 healthy stands (called set 2). These data have not been previously published. We again asked the land managers from the SNCC for stands that fit the same stand criteria but that were healthy. To find enough healthy stands, we had to expand the stand selection area eastward out of the high disease area throughout the Coast Range, the Willamette Valley, and the Western Cascades (Fig. 1). The 15 healthy stands in set 2 plus the 3 healthy stands in set 1 gave a total of 18 healthy and 14 diseased stands.

We sampled 7–12 dominant and codominant trees per stand, and included a range of diameters in each stand. The selected

Fig. 2. Bole positions of beams and SilviScanII samples from coastal Douglas-fir trees for set 1 and set 2. Beams (n = 8 for set 1; n = 6 for set 2) came from a disk above breast height. The SilviScanII sample (one per tree) came from immediately below the breast height disk for set 1 and from the base of a beam for set 2. [Colour online.]



trees were not leaning, forked, or injured; showed no disease other than SNC; and were located such that after their removal, the stand spacing was consistent with thinning, not a patch cut. The study used 366 trees, 203 from 18 healthy stands and 163 from 14 diseased stands.

Stand data

After trees were felled, we counted growth rings at breast height (1.4 m) and measured diameter at breast height (DBH, mm) and height (m). For many of the set 1 stands, NR was available from Maguire et al. (2002). For stands lacking those data, we estimated NR using the methods of Maguire et al. (2002) but for the current year only. Although the NR values came from different spans of years and different calendar years, both measures of NR gave the best estimate we had of a site's overall NR.

Beam properties

In the field, we removed a 30 cm tall disk from immediately above breast height for both the set 1 and set 2 trees. In the set 1 trees, we also removed a 5 cm tall disk from immediately below breast height for analysis by SilviScan (Fig. 2).

In the workshop, replicate vertical beams (1 cm radial × 1 cm tangential × ≥16 cm axial) were sawed green (at field moisture content) at regular spacing from the outermost xylem of the 30 cm tall disks. The beams' long dimension was in the axial direction of the tree, and one of the faces was in the tangential plane. We made 8 beams per tree for set 1 stands, but because of results of variance analysis (data not shown), we made only 6 beams per tree for set 2 stands (Fig. 2). In total, we sampled about 2614 beams (1278 from healthy trees, 1336 from diseased trees). Beams were air-dried to constant mass at 12% moisture content and then weighed and measured with calipers to obtain our estimate of wood density ($g \cdot cm^{-3}$). Ring count was estimated by counting the number of rings (including a decimal value for partial rings) on a beam's cross-sectional face.

The beams, still at 12% moisture content, were subjected to static bending tests on a universal testing machine. We used center loading on a 15 cm span (that is, supports were set 15 cm apart), and the load was applied at 5 $mm \cdot min^{-1}$. With the exception of the 140 beams that were tested in the longitudinal–tangential orientation (Grotta et al. 2005), the remaining beams (2474) were tested in the longitudinal–radial (L–R) orientation (L–R plane in active bending, with the load applied to the longitudinal–tangential surface) with the pith side up in compression, as specified by ASTM Standard D143 (ASTM 2003). We calculated the MOE in bending (stiffness) and the MOR in bending (the maximum force withstood

before breaking) for each beam according to the formulas in Markwardt and Wilson (1935), where MOE was the slope of the linear portion of the stress–strain curve and MOR was the stress applied at beam failure.

SilviScan data

After mechanical tests and density estimates were done, we prepared one sample per tree for analysis by SilviScanII (Melbourne, Australia) (Evans and Ilic 2001). The samples came from immediately below breast height for the set 1 trees and immediately above breast height for the set 2 trees. For set 1, this sample came from the outer 1 cm of xylem from the tree's lower disk. For set 2, the sample came from the lowermost portion of one of the tree's beams (Fig. 2). Samples were soaked in three successive baths of 100% ethanol for 1 week to extract resins and other materials. They were then air-dried and sent to SilviScan.

SilviScan sampled the material at 0.05 mm intervals for earlywood and latewood widths, and at 0.2 mm intervals for MfA. SilviScan provided ring-by-ring values for the earlywood width, latewood width, earlywood MfA, and latewood MfA for each growth ring in the sample. We calculated LW% as the percentage of the entire sample's width that was latewood, and calculated MfA as the width-weighted mean of MfA across the sample.

Data analysis

We used analysis of variance (ANOVA) to compare the mean values for healthy versus diseased stands for NR, age, height, DBH, height/DBH, ring count, ring width, density, MOE, MOR, LW%, and MfA. Because our focus was at the stand level, the input data consisted of the stand means. We previously reported on the distribution of variance among stands, within stands, and within trees (where applicable) in some of these stands (Johnson et al. 2005). Ring count was log-transformed to obtain equal variances. We removed one outlier stand value for height/DBH. After performing Student's t tests for the 12 comparisons, we made Bonferroni corrections (http://mathworld.wolfram.com/BonferroniCorrection.html) and report the adjusted P values for significance at $\alpha < 0.05$.

We performed linear regression analyses for pairs of variables separately for healthy and diseased stands, and plotted the data with the regression lines to help visualize the relationships. The coefficient of determination (R^2) and P value were used to indicate the strength of the relationship. The independent variables were NR, LW%, MfA, and ring count. Depending on the comparison, the dependent variables were LW%, MfA, ring count, ring width, density, MOE, or MOR.

For these same pairs of independent and dependent variables, we then did an analysis of covariance (ANCOVA) of the regression lines for the healthy versus diseased stands. The procedure first calculates coefficients (β_0, β_1, β_2, and β_3) for a linear regression of the dependent variable (y) and the independent variable (x), making use of an indicator variable ($I = 1$ if the stand is healthy, $I = 0$ if the stand is diseased): $y = \beta_0 + \beta_1 x + \beta_2 I + \beta_3 Ix$. This model gives the slope and intercept for the linear relationship between x and y for healthy and diseased stands combined. The procedure then uses ANOVA for the terms in the model, providing an F ratio and P value for the differences between the slopes, and the differences between intercepts, for the healthy versus diseased stands. The independent and dependent variables were the same as defined above. To show the range of data at the tree (rather than stand) level, we plotted density, MOE, and MOR versus ring count for healthy and diseased trees.

Analyses were performed using STATGRAPHICS Centurion 18 (v. 18.1.11; StatGraphics Technologies, The Plains, Virginia, USA), and graphing was done using SigmaPlot 14.0 (Systat Software, 2017, San Jose, California, USA). Results were considered significant at $\alpha < 0.05$ unless otherwise indicated.

Table 1. Comparisons of stand, beam, and SilviScanII values for healthy versus diseased stands of coastal Douglas-fir.

	Healthy (n = 18)	Diseased (n = 14)	P
Stands			
Breast-height age (years)	26.5 (19.6–33.6)	24.2 (19.0–30.4)	1.000
NR (years)	4.0 (3.0–5.1)	2.1 (1.5–2.8)	**<0.001**
Height (m)	23.6 (18.1–28.3)	21.3 (18.1–31.2)	1.000
DBH (mm)	316 (213–353)	271 (233–311)	**0.002**
Height/DBH ($m \cdot m^{-1}$)	75.0 (61.8–88.1)	75.0 (64.1–123.9)	1.000
Beams			
Ring count ($no. \cdot cm^{-1}$)	3.7 (2.7–4.9)	4.5 (2.5–7.0)	0.743
Ring width (mm)	2.8 (2.0–3.6)	2.4 (1.4–4.0)	1.000
Density ($g \cdot cm^{-3}$)	0.55 (0.51–0.61)	0.61 (0.55–0.67)	**<0.001**
MOE (GPa)	11.7 (10.2–13.0)	12.8 (11.5–14.4)	**0.020**
MOR (MPa)	106 (93–127)	107 (95–121)	1.000
SilviScanII			
LW% (%)	41 (35–46)	54.5 (44–60)	**<0.001**
MfA (°)	14.8 (12.5–17.6)	14.9 (12.5–16.3)	1.000

Note: Results are presented as means (range in parentheses). P values are derived from analysis of variance and adjusted by Bonferroni correction; $P < 0.05$ shown in bold. DBH, diameter at breast height; LW%, latewood percentage; MfA, microfibril angle; MOE, modulus of elasticity; MOR, modulus of rupture; NR, needle retention.

Results

Comparisons of means

The healthy and diseased stands did not differ significantly in breast-height age (Table 1). The healthy classes differed in mean NR because that was how we categorized the stands (Table 1). DBH was significantly higher in the healthy stands, but the stand types did not differ significantly in height or height/DBH (Table 1).

Neither ring count nor ring width differed significantly between healthy and diseased stands (Table 1). Healthy stands averaged ring counts from 2.7 to 4.9, whereas diseased stands averaged ring counts from 2.5 to 7.0 (Table 1). At the tree level, ring counts in healthy trees ranged from 1.6 to 9.0 (n = 203 trees) and diseased trees range from 1.6 to 9.4 (n = 163 trees) (see Regression analyses, tree-level data).

Wood density and MOE were significantly lower in healthy than in diseased stands, but there was no significant differences in MOR (Table 1). LW% was significantly lower in the healthy stands. MfA did not differ significantly between healthy and diseased stands (Table 1).

Regression analyses

In the healthy stands, very few of the studied relationships were statistically significant, and the largest of the R^2 values was 0.251 (Table 2; Figs. 3, 4, 5). Only two of the 19 relationships assessed were significant at $P < 0.05$ — MOE versus LW% and density versus MfA — but in both cases, the amount of variation explained by the dependent variable was very low (R^2 = 0.251 and 0.236, respectively; Table 2). Notably, NR was not significantly correlated with ring count, LW%, density, or MOR (Table 2; Figs. 3, 5). Additionally, LW% was not significantly correlated with wood density (although it was significant at P = 0.06, R^2 = 0.201) or MOR, but it was correlated with MOE. MfA was not correlated with MOE or MOR but it was positively correlated with density (Table 2; Fig. 4). Lastly, ring count had no appreciable effect on wood quality in healthy stands, as shown by the low values of R^2 for the variables (Table 2; Figs. 3, 5).

By contrast, in the diseased stands, 15 of the 19 relationships were significant, and in eight of these significant relationships, R^2 was 0.50 or above (Table 2; Figs. 3, 4, 5). Notably, NR was significantly correlated with ring count, LW%, density, MOE, and MOR (Table 2; Fig. 3, Fig. 5). Additionally, LW% was correlated with

Table 2. Coefficients of determination (R^2) for selected stand-level linear correlations of stand and xylem properties in healthy and diseased stands of coastal Douglas-fir.

		Healthy (n = 18)		Diseased (n = 14)	
Independent variable	Dependent variable	R^2	P	R^2	P
NR	Ring count	0.031	0.4840	0.656	**0.0004**
	Ring width	0.028	0.5101	0.524	**0.0034**
	LW%	0.144	0.1211	0.289	**0.0476**
	Density	0.085	0.2411	0.490	**0.0053**
	MOE	0.011	0.6770	0.477	**0.0063**
	MOR	0.187	0.0734	0.412	**0.0133**
LW%	Ring count	0.110	0.1791	0.500	**0.0048**
	Density	0.201	0.0622	0.618	**0.0009**
	MOE	0.251	**0.0340**	0.304	**0.0411**
	MOR	0.126	0.1480	0.440	**0.0098**
MfA	Ring count	0.010	0.6938	0.201	0.1075
	LW%	0.010	0.9013	0.088	0.3039
	Density	0.236	**0.0410**	0.192	0.1174
	MOE	0.109	0.1809	0.325	**0.0334**
	MOR	0.101	0.1987	0.223	0.0880
Ring count	LW%	0.110	0.1791	0.500	**0.0048**
	Density	0.012	0.6655	0.738	**0.0001**
	MOE	0.050	0.3720	0.652	**0.0005**
	MOR	0.014	0.6441	0.626	**0.0008**

Note: $P < 0.05$ are shown in bold. LW%, latewood percentage; MfA, microfibril angle; MOE, modulus of elasticity; MOR, modulus of rupture; NR, needle retention.

wood density, MOE, and MOR, and MfA was correlated with MOE but not density or MOR (Table 2; Fig. 4).

Comparisons of the slopes of regression lines between healthy and diseased stands showed no significant difference for any of the tested relationships with LW%, MfA, or ring count except for NR versus ring count (Table 3). In addition to ring count, the slope of the regression lines for NR in healthy versus diseased stands differed for all the comparisons except LW% (Table 2). About half of the relationships tested had intercepts that differed significantly between healthy and diseased stands (Table 3).

The tree-level data for ring count versus density, MOE, and MOR offers a picture of the samples' overall variability (Fig. 6). Although all the comparisons have statistically significant correlations (at $P < 0.05$, data not shown), the amount of variation in the response variable accounted for by ring count is operationally insignificant. The adjusted R^2 values for the tree-level correlations of ring count with density were 0.158 (healthy) and 0.386 (diseased). For ring count versus MOE, the adjusted R^2 values were 0.181 (healthy) and 0.216 (diseased). For ring count versus MOR, the adjusted R^2 values were 0.089 (healthy) and 0.223 (diseased).

The tree-level correlations of ring count with the three variables had adjusted R^2 values as follows: density (healthy, 0.158; diseased, 0.386), MOE (healthy, 0.181; diseased, 0.216), and MOR (healthy, 0.089, diseased, 0.223).

Discussion

Comparison of healthy and diseased stands

The healthy versus diseased stands in this study were sampled from different geographical regions. Short of using stands undergoing fungicide trials in the diseased region (e.g., Johnson et al. 2003), it would have been impossible to find neighboring healthy and diseased stands. Thus, the geographical separation of the sampled stands must be considered for the inferences. However, the healthy and diseased stands had no significant difference in two of the variables known to affect wood properties in coastal Douglas-fir, cambial age (e.g., Lachenbruch et al. 2011) and tree height/diameter (Johnson and Gartner 2006), suggesting that in some respects the healthy and diseased stands were similar.

As we reported previously using a subset of the data from this study (Johnson et al. 2005), stands that were heavily impacted with SNC had significantly higher LW%, wood density, MOE, and MOR than did the few healthy stands in that study, and did not differ significantly in MfA. In the current study, the lack of significant difference in ring count (a deliberate result of experimental design) and the similarity in age and height/diameter of the two samples gives further support to the inference that the differences in wood density, MOE, and LW% are linked to effects of the disease.

The inferences from the current study are only valid for the ring counts sampled in the healthy stands, which ranged from 2.7 to 4.9 at the stand level and 1.6 to 8.0 at the tree level (Figs. 3, 6). Our intention was to include the lowest ring counts likely to occur in 20- to 25-year-old managed stands of coastal Douglas-fir in western Oregon. We were unable to find direct data on what these ring counts are, so we turned to CIPSANON (http://cips.forestry.oregonstate.edu/cipsanon), a growth and yield model that was developed to model growth of intensively managed Douglas-fir plantations in western Oregon, Washington, and southwestern British Columbia. The model "grew" trees in two simulations, one at a site index (SI) typical of relatively good sites, and the second at an SI typical of the best sites (38 or 44 m height at age 50, respectively), at an initial planting density of 1075 tree·ha^{-1}, typical of the region. Of the 50 trees modeled in each simulation, the individual tree with the fastest growth had a ring count of 2.8 or 3.1 for the SI 38 or SI 44 sites, respectively (D.B. Mainwaring, personal communication). These are tree-level ring counts, which are much lower (representing faster growth) than stand ring counts would be. They provide strong evidence that the lowest ring counts (fastest growth) represented in the current study are sufficiently rapid to include the fastest growth likely to occur at the stand level in the region.

NR versus ring count

In support of the first hypothesis, there was a significant correlation between NR and ring count in diseased stands but not in healthy stands (Table 2; Fig. 3). The negative relationship of NR with ring count in diseased trees is consistent with reports on the negative growth impacts of SNC, both in recent decades (Maguire et al. 2011) and in previous centuries (Lee et al. 2013).

This inverse relationship between NR and ring count in diseased trees is likely a result of diseased trees having a reduced supply of carbon relative to healthy trees. Such a reduced supply has been shown with the seasonal carbohydrate dynamics of trees (Saffell et al. 2014) and can be inferred from a lower ectomycorrhizal fungal density and species richness in diseased stands (Luoma and Eberhart 2014). It can also be inferred from the lower foliage mass (Weiskittel et al. 2006) and lower levels of assimilation per leaf area of diseased trees. Using the mean NR values from healthy and diseased stands from the current study, a model by Weiskittel (2003) predicts that trees in the diseased stands would average 45%–47% of the foliage dry matter of trees in the healthy stands. Manter et al. (2003) found a progressive decline in maximum net CO_2 assimilation (A_{max}) of current-year, 1-year-old, and 2-year-old needles in diseased trees, but not in healthy trees, which would further depress the diseased trees' carbon economies. In the most severely impacted stands, Manter et al. (2003) showed that only the current-year needles had positive carbon budgets; the older needles were carbon sinks.

The above argument does not explain the lack of significant impact of NR on ring count in healthy trees stands. We hypothesize that the maintenance of multiple annual cohorts of needles provides a redundancy for a tree, such that if one or several cohorts of foliage become incapacitated through disease or injury,

Fig. 3. Relationship between (*a*) ring count and needle retention (NR), (*b*) latewood proportion and NR, and (*c*) latewood proportion and ring count in healthy (green circles, $n = 18$) versus diseased (orange boxes, $n = 14$) stands of coastal Douglas-fir. Values of R^2 are shown for linear regressions for healthy stands (solid green line) and diseased stands (broken orange line). *, slopes differ significantly at $P < 0.05$. [Colour online.]

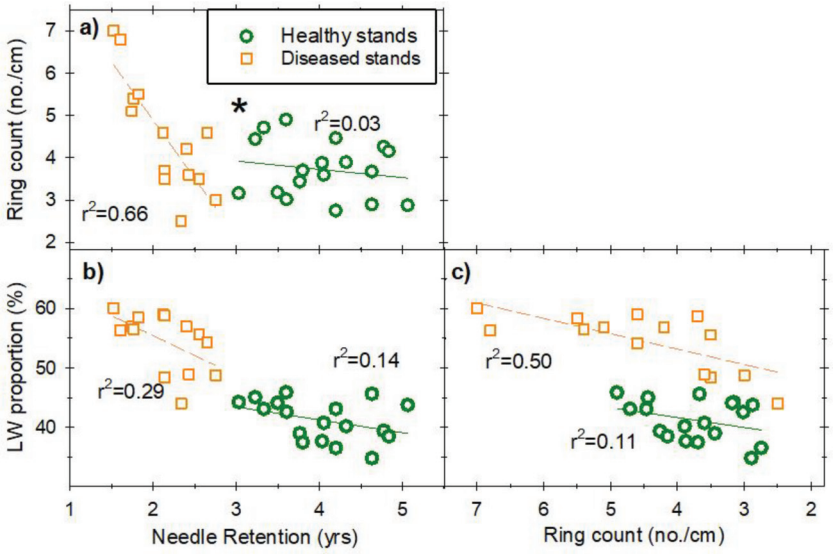
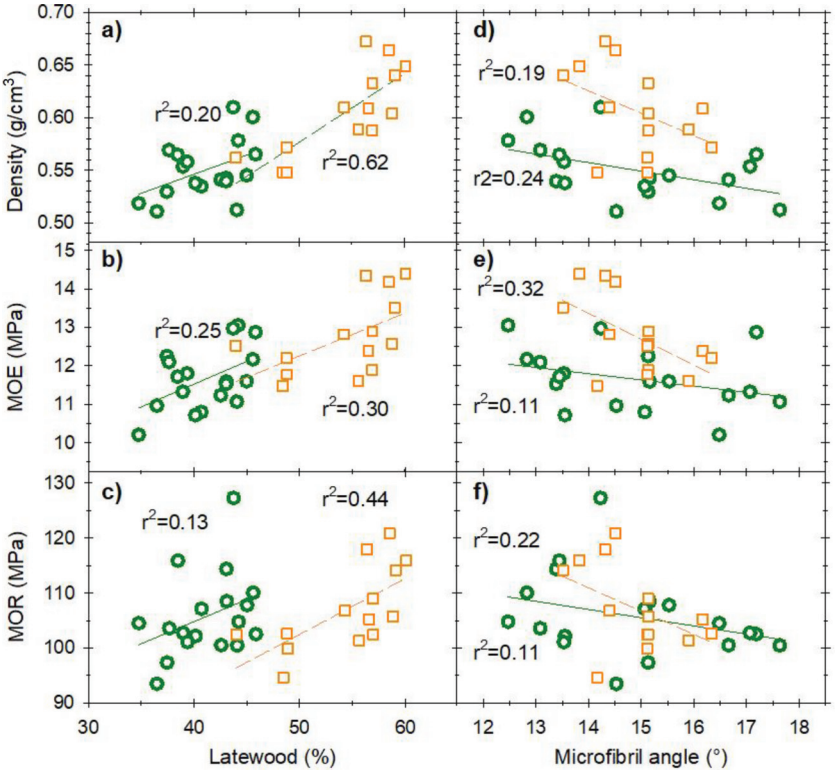


Fig. 4. Relationship between latewood percentage and (*a*) wood density, (*b*) modulus of elasticity (MOE), and (*c*) modulus of rupture (MOR) or between microfibril angle and (*d*) wood density, (*e*) MOE, and (*f*) MOR in healthy (green circles) versus diseased (orange squares) stands of coastal Douglas-fir. Values of R^2 are shown for linear regressions for healthy stands (solid green line) and diseased stands (broken orange line). No slopes differed significantly at $P < 0.05$. [Colour online.]



other cohorts may remain functional. We infer that the level of redundancy provided by NR above 3 years in the coastal Douglas-fir stands of western Oregon is sufficient to meet the species' evolved needs. That is, any growth reductions in stands with NR of at least 3 years are likely to be caused by factors other than needle retention, such as climate, exposure, stocking level, or soils.

NR and LW%

Also in support of the first hypothesis, there was a significant negative correlation between NR and LW% in diseased stands but not in healthy stands (Table 2; Fig. 3). The higher LW% of stands more heavily impacted by SNC is thought to be due to needle phenology (Johnson et al. 2003). In diseased trees, needles are mostly

Fig. 5. Relationship between needle retention (NR) and (*a*) wood density, (*b*) modulus of elasticity (MOE), and (*c*) modulus of rupture (MOR) or between ring count and (*d*) wood density, (*e*) MOE, and (*f*) MOR in healthy (green circles, $n = 18$) versus diseased (orange boxes, $n = 14$) stands of coastal Douglas-fir. Values of R^2 are shown for linear regressions for healthy stands (solid green line) and diseased stands (broken orange line). *, slopes differ significantly at $P < 0.05$. [Colour online.]

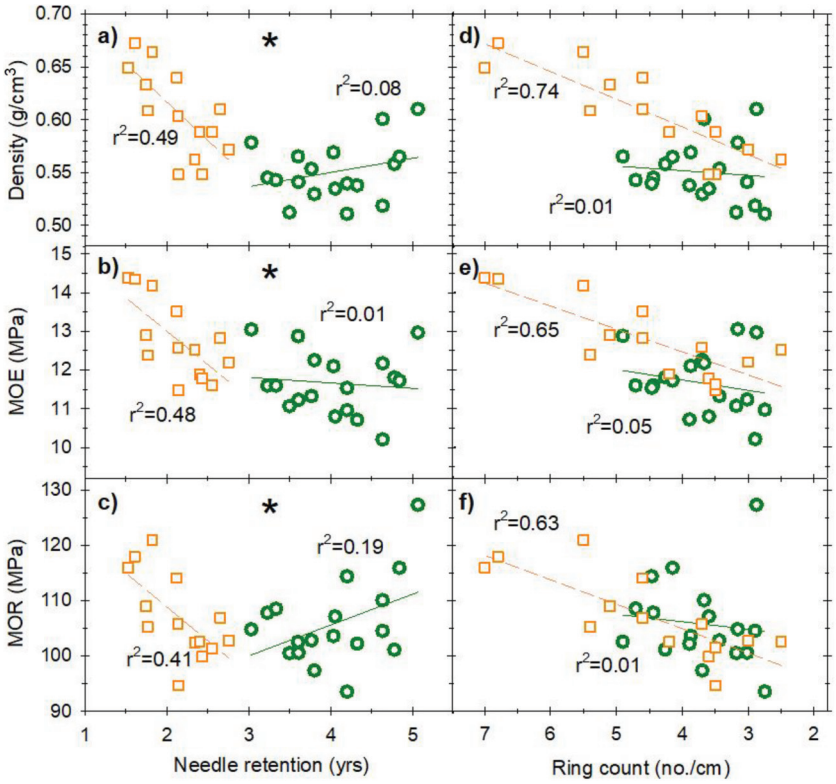


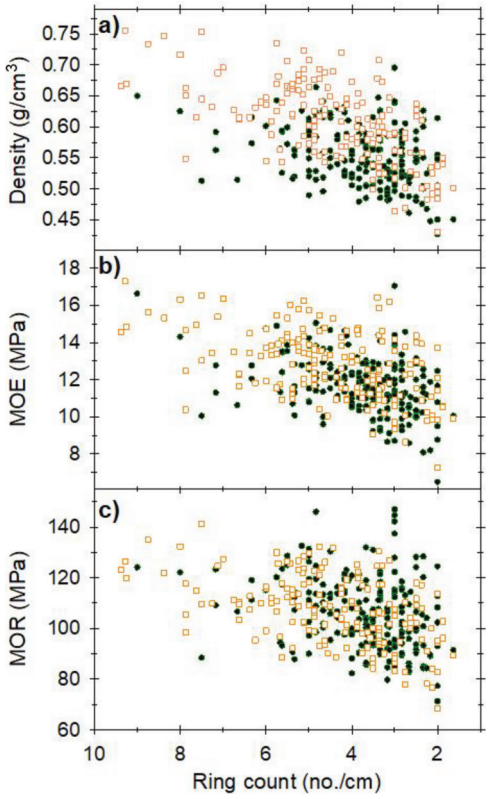
Table 3. Slopes and intercepts of regressions of selected stand-level linear correlations of stand and xylem properties in healthy ($n = 18$) and diseased ($n = 14$) stands of coastal Douglas-fir.

		Slope			Intercept		
Independent variable	Dependent variable	Healthy	Diseased	*P*	Healthy	Diseased	*P*
NR	Ring count	–0.200	–2.73	**0.0002**	4.51	10.3	0.1567
	Ring width	0.0139	0.130	**0.0085**	0.221	–0.0383	0.2379
	LW%	–2.16	–6.69	0.1456	49.9	68.9	**0.0185**
	Density	0.0134	–0.0725	**0.0009**	0.493	0.762	0.0753
	MOE	–0.142	–1.73	**0.0189**	12.2	16.5	0.9164
	MOR	5.58	–12.2	**0.0027**	83.3	133	0.5184
LW%	Ring count	0.065	0.191	0.0978	1.05	–5.92	0.0518
	Density	0.00363	0.00653	0.2275	0.401	0.250	0.3578
	MOE	0.118	0.111	0.9207	6.79	6.70	0.4400
	MOR	0.806	1.01	0.7405	72.6	51.8	**0.0242**
MfA	Ring count	–0.0405	–0.712	0.0636	4.32	15.1	**0.0296**
	LW%	–0.0653	–1.74	0.2401	40.2	80.4	**0.0000**
	Density	–0.00817	–0.0213	0.2543	0.672	0.924	**0.0000**
	MOE	–0.161	–0.673	0.0905	14.1	22.8	**0.0006**
	MOR	–1.50	–4.23	0.3502	128	170	0.5774
Ring count	LW%	1.69	2.61	0.5313	34.9	42.8	**0.0000**
	Density	0.00453	0.0264	0.0521	0.534	0.488	**0.0004**
	MOE	0.269	0.601	0.2855	10.7	10.1	**0.0194**
	MOR	1.35	4.47	0.2802	101	87.0	0.5469

Note: $P < 0.05$ are shown in bold. LW%, latewood percentage; MfA, microfibril angle; MOE, modulus of elasticity; MOR, modulus of rupture; NR, needle retention.

cast (dropped) in the winter, between December and May (Manter et al. 2003). Although new needles are produced in the spring, they are not mature until 11–15 weeks after budburst (Devine and Harrington 2009). Relative to healthy trees, therefore, diseased trees would have a lower functional needle area in the spring, when earlywood is made, than later in the season, when latewood is made. Then, assuming that current photosynthate production is linked to current xylogenesis (which is not necessarily

Fig. 6. Relationships between ring count and (*a*) density, (*b*) modulus of elasticity (MOE), and (*c*) modulus of rupture (MOR) in healthy (solid green circles, $n = 203$) versus diseased (open orange boxes, $n = 163$) stands of coastal Douglas-fir. [Colour online.]



the case; see Rossi et al. 2016), this needle demography and maturation pattern would result in diseased trees making xylem with relatively higher LW% compared with healthy trees.

The fact that in healthy stands LW% is not related to NR shows that variation in LW% must be related to other factors. This observation could be explained in at least two ways. First, if a plant has an evolved balance of earlywood and latewood production (following criteria we do not yet know), then we can infer that the redundancy supplied by NR above 3 years is sufficient to meet the plant's needs for that balance. Second, if a plant has no clear evolved balance, and if earlywood and latewood have distinct roles, then the nondependence of LW% on NR in healthy stands suggests that plants tend to have about the same needs for the earlywood functions and the latewood functions, regardless of whether their NR is 3 years or some value greater than 3 years. There is evidence for different roles of Douglas-fir earlywood and latewood in living trees, related to overall biomechanical functions, and water transport functions when water is abundant versus scarce (Domec and Gartner 2002; Dalla-Salda et al. 2014). However, we have little understanding of the requirements, controls, or timing of feedback in the production of the various tissues and organs involved in water transport, such as the amount and quality of earlywood and latewood, water storage and its accessibility rate, shoot length, sapwood area, root absorbing area, and leaf area (Meinzer et al. 2010).

LW% versus density, MOE, and MOR

Our second hypothesis started with an expectation that in both healthy and diseased stands, LW% is positively correlated with wood density, MOE, and MOR. This part of the hypothesis was partially supported (Fig. 4) in that we found these positive relationships, but they were significant only for the diseased stands, and for MOE in healthy stands. The coefficients of determination were much lower in the healthy stands than in the diseased stands for density and MOR but were similar for MOE (Table 2).

Although in diseased trees the relationship between LW% and wood density was strong ($R^2 = 0.62$), the relationship was weak in healthy stands ($R^2 = 0.20$). Dunham et al. (2007) showed no relationship between LW% and wood density in 32 mature coastal Douglas-fir trees, either for the data as a whole or for any of seven positions independently (three trunk, two branch, and one root position). One explanation for the large effect in diseased trees and the weak effect in healthy trees comes from work by Choi (1986) on healthy Douglas-fir outerwood (cambial age between 20 and 60 years). The author reported that earlywood percentage was so much higher than LW%, and the variability of density was sufficiently low within earlywood, that earlywood had a larger effect on sample density than latewood. This explanation may pertain to the healthy trees studied and could also explain the fact that diseased trees, with their much higher LW%, had a significant latewood effect. We were unable to test this explanation with our dataset because the earlywood and latewood densities provided by SilviScanII (at the same time that we obtained MfA and LW%) on one thin transect through the samples were not the measures we used here for wood density. Instead, we used a direct measurement of bulk wood density, which we expected to have a more direct relationship with the mechanical properties.

This explanation contrasts with the common expectation that because latewood has a greater density than earlywood (e.g., Domec and Gartner 2002), LW% must govern wood density (e.g., Smith 1956). Instead, this explanation requires that density values and variability across the whole growth ring be considered. Although we often use specific criteria to demarcate the earlywood–latewood boundary, wood density can vary in many ways across a growth ring (e.g., Ruiz Diaz Britez et al. 2014). Moreover, the wood properties within earlywood or latewood may differ among locations in an individual (e.g., by height or plant part Dunham et al. 2007); among growth rings in response to environment (e.g., Martinez Meier et al. 2008); and between individuals (e.g., Dalla-Salda et al. 2009). This variation is not surprising given that wood density can be determined by many factors, including lumen diameter, cell wall thickness, extractive content, cell wall material, and tracheid length (discussed in Rathgeber et al. 2006), and given that the different locations in the tree have different physiological and mechanical demands.

LW% had significant but weak relationships with MOE in both healthy and diseased stands ($R^2 = 0.25$ and 0.30, respectively; Table 2). We have no explanation for the similarity of this pattern in healthy versus diseased stands; we would have expected a lesser effect in healthy than in diseased stands if we used the same argument as above (i.e., that the lower LW% in healthy stands would decrease its effect on a property). However, some research suggests that the earlywood–latewood dichotomy is flawed for some functional interpretations. Rozenberg et al. (1999) found a stronger relationship of MOE to actual intra-ring density profiles than to the typical earlywood–latewood designations, which suggests the importance of within-ring variation. Todaro and Macchioni (2011) reported a stronger effect of bulk wood density than LW% on MOE.

LW% had no significant relationships with MOR in healthy stands, but a moderate and significant positive relationship in diseased stands ($R^2 = 0.13$ and 0.44, respectively; Table 2). The difference in the strength of the effect may relate to the relatively smaller influence of latewood on the total ring properties in healthy than diseased stands, using the argument from above (Choi 1986). MOR represents the stress at which a sample fails, which is expected to be related to the presence of microfractures in the sample. The likelihood of occurrence of microfractures

increases by probability alone in larger specimens (Bohannan 1966).

MfA versus density, MOE, and MOR

Our second hypothesis also predicted that MfA has no relationship or only weak negative relationships with density, MOE, and MOR (Fig. 4). We know of no causal reason that MfA should be related to wood density, although the relationship was weak, negative, and significant in healthy stands (Table 2). Lachenbruch et al. (2010) reported that the effect of density was higher than that of MfA in a study that used path analysis; but we note that this study made use of 17 stands, many of which were included in the current study, and so should not be considered independent of the current study. Vikram et al. (2011) found a weak negative effect of MfA on MOE, but their path analysis showed that the effect of MfA was lower than that of density. In diseased stands, the significant relationship of MfA with MOE may result from the LW% in SNC-affected stands representing the majority of the cross-section. Also, as discussed above, no causal relationship would be expected between MfA and MOR because MOR depends on microfractures, which should not be MfA dependent.

NR and ring count versus density, MOE, and MOR

The third hypothesis was that the correlations of NR and ring count with density, MOE, or MOR were not significant in healthy stands, but were significant in diseased stands. In diseased stands, we expected the relationships with NR to be negative and those with ring count to be positive. These hypotheses were supported (Table 2; Figs. 3, 5). Moreover, the values of R^2 were much lower for the healthy than for the diseased stands in these correlations (Table 2). These outcomes inform us that stands affected by SNC develop wood with different properties than healthy stands do, and the difference is not a direct result of ring count.

For stands with NR < 3 years, a decrease in NR resulted in an increase in LW%, which then increased density, MOE, and MOR. NR caused a higher ring count, and thus NR and ring count have similar correlations with other factors. If NR ≥ 3 years, the tree likely has sufficient photosynthetic capacity such that additional cohorts of needles have no further effect on LW%.

Our finding that ring count is not correlated with density, MOE, or MOR in outerwood has much practical significance. Using different methods, other studies have reported no or minor effects of the radial growth rate of Douglas-fir on wood density (Kimberley et al. 2017), MOE (Pollet et al. 2017), MOR (Lowell et al. 2014), or the proportion of outerwood boards that qualify for higher lumber grades (Bendtsen et al. 1988). Our plot of ring count versus mean tree values (rather than stand values) shows graphically the very large scatter in the data for the healthy stands (Fig. 6, filled green circles). The current study supports the previous research in this area, and suggests that within the range of inference of this study (wood near 20–25 years at breast height, in plantations in western Oregon), coastal Douglas-fir can be grown very slowly, at a moderate rate, or very rapidly, and all the lumber from the outerwood will have similar mechanical and physical properties.

Conclusions

Healthy coastal Douglas-fir stands and those impacted by Swiss needle cast differ in their relationships between radial growth rate and outerwood density, MOE, and MOR. We used the index of NR to estimate the disease impact on a stand, where a value of NR < 3 indicated that a stand was diseased. Stands with the lowest NR had the highest LW% and ring count. The biology of this system suggests that the low NR of heavily-impacted trees results especially in a decrease in earlywood, which would drive the higher LW% and ring count.

In diseased stands, there was a strong positive relationship between LW% and density ($R^2 = 0.62$) and a moderate positive relationship with MOE ($R^2 = 0.30$) and MOR ($R^2 = 0.44$). Needle retention had moderate negative correlations with density, MOE, and MOR ($R^2 = 0.49$, 0.48, and 0.41, respectively). Ring count had a strong negative relationship with density, MOE, and MOR ($R^2 = 0.74$, 0.65, and 0.63, respectively), meaning that at the slowest growth rates, these wood properties had their highest values.

In healthy stands, NR had a very weak effect on LW% and ring count, and ring count had a very weak effect on LW%. As in diseased stands, LW% was positively correlated with density, MOE, and MOR, but unlike diseased stands, these relationships in healthy stands were very weak. Thus, in contrast to the diseased stands, in healthy stands, LW% had very little effect on wood properties. Furthermore, in healthy stands, NR had almost no effect on the wood properties, and ring count also had no appreciable effect on density, MOE, or MOR ($R^2 = 0.01$, 0.05, and 0.01, respectively). This study supports the statement that the wood quality of outerwood in healthy stands is not affected appreciably by growth rate over the range of ring counts of healthy coastal Douglas-fir stands in western Oregon.

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References

ASTM. 2003. Standard test methods for small clear specimens of timber, D 143. Annual book of ASTM standards. Vol. 04.10 Wood. American Society for Testing and Materials, West Conshohocken, Pa., USA. pp. 25–55.

Barrett, J.D., and Kellogg, R.M. 1989. Bending strength and stiffness of second-growth Douglas-fir dimension lumber. For. Prod. J. **41**: 35–43.

Bendtsen, B.A., Plantinga, P.A., and Snellgrove, T.A. 1988. The influence of juvenile wood on the mechanical properties of 2x4's cut from Douglas-fir plantations. *In* Proceedings of the International Conference on Timber Engineering, Seattle, Wash. 19–22 Sept. 1988, Vol. 1, pp. 226–240.

Bohannan, B. 1966. Effect of size on bending strength of wood members. USDA Forest Service, Forest Products Laboratory, Madison, Wisconsin. US Forest Service Research Paper FPL. 56, May 1966. 30 pp.

Briggs, D.G., Thienel, G., Turnblom, E.C., Lowell, E., Dykstra, D., Ross, R.J., et al. 2008. Influence of thinning on acoustic velocity of Douglas-fir trees in western Washington and western Oregon. *In* Proceedings of the 15th International Symposium on Nondestructive Testing of Wood, Duluth, Minnesota. USDA Forest Products Society, Madison, pp. 113–123.

Choi, A.S.C. 1986. Correlation between mechanical strength of wood and annual ring characteristics of Douglas-fir juvenile and mature wood. M.S. thesis, Dept. of Forest Products, Oregon State University, Corvallis.

Dalla-Salda, G., Martinez-Meier, A., Cochard, H., and Rozenberg, P. 2009. Variation of wood density and hydraulic properties of Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) clones related to a heat and drought wave in France. For. Ecol. Manage. **257**: 182–189. doi:10.1016/j.foreco.2008.08.019.

Dalla-Salda, G., Fernández, M.E., Sergent, A.-S., Rozenberg, P., Badel, E., and Martinez-Meier, A. 2014. Dynamics of cavitation in a Douglas-fir tree-ring: transition-wood, the lord of the ring. J. Plant Hydraul. **1**: e-0005. doi:10.20870/jph.2014.e005.

Devine, W.D., and Harrington, C.A. 2009. Relationships among foliar phenology, radial growth rate, and xylem density in a young Douglas-fir plantation. Wood Fiber Sci. **41**: 300–312.

Domec, J.-C., and Gartner, B.L. 2002. How do water transport and water storage differ in coniferous earlywood and latewood? J. Exp. Bot. **53**: 2369–2379. doi:10.1093/jxb/erf100. PMID:12432029.

Drow, J.T. 1957. Relationship of locality and rate of growth to density and strength of Douglas-fir. U.S. Forest Products Lab. Report No. 2078.

Dunham, S.M., Lachenbruch, B., and Ganio, L.M. 2007. Bayesian analysis of Douglas-fir hydraulic architecture at multiple scales. Trees, **21**: 65–78. doi:10.1007/s00468-006-0097-8.

Evans, R., and Ilic, J. 2001. Rapid prediction of wood stiffness from microfibril angle and density. For. Prod. J. **51**(3): 53–57.

Green, D.W., Lowell, E.C., and Hernandez, R. 2005. Structural lumber from dense stands of small-diameter Douglas-fir trees. For. Prod. J. **55**(7/8): 42–50.

Grotta, A.T., Leichti, R.J., Gartner, B.L., and Johnson, G.R. 2005. Effect of growth ring orientation and placement of earlywood and latewood on MOE and MOR of very-small clear Douglas-fir beams. Wood Fiber Sci. **37**: 207–212.

Hansen, E.M., Stone, J.K., Capitano, B.R., Rosso, P., Sutton, W., Winton, L., et al. 2000. Incidence and impact of Swiss needle cast in forest plantations of Douglas-fir in coastal Oregon. Plant Dis. **84**: 773–778. doi:10.1094/PDIS.2000.84.7.773. PMID:30832107.

Henin, J.-M., Pollet, C., Jourez, B., and Hebert, J. 2018. Impact of tree growth rate on the mechanical properties of Douglas fir lumber in Belgium. Forests, **9**: 342. [Online.] doi:10.3390/f9060342.

Howe, G.T., Jayawickrama, K., Cherry, M., Johnson, G.R., and Wheeler, N.C. 2006. Breeding Douglas-fir. Plant Breed. Rev. 27: 245–353.

Johnson, G.R., and Gartner, B.L. 2006. Genetic variation in basic density and modulus of elasticity of coastal Douglas-fir. Tree Genet. Genomes, **3**: 25–33. doi:10.1007/s11295-006-0054-0.

Johnson, G.R., Gartner, B.L., Maguire, D., and Kanaskie, A. 2003. Influence of Bravo fungicide applications on wood density and moisture content of Swiss needle cast affected Douglas-fir trees. For. Ecol. Manage. **186**: 339–348. doi:10.1016/S0378-1127(03)00305-0.

Johnson, G.R., Grotta, A.T., Gartner, B.L., and Downes, G. 2005. Impact of the foliar pathogen Swiss needle cast on wood quality of Douglas-fir. Can. J. For. Res. **35**(2): 331–339. doi:10.1139/x04-170.

Kennedy, R.W. 1995. Coniferous wood quality in the future: concerns and strategies. Wood Sci. Technol. **29**: 321–338. doi:10.1007/BF00202581.

Kimberley, M.O., Hood, I.A., and Knowles, R.L. 2011. Impacts of Swiss Needle-Cast on growth of Douglas-fir. Phytopathology, **101**: 583–593. doi:10.1094/PHYTO-05-10-0129.

Kimberley, M.O., McKinley, R.B., Cown, D.J., and Moore, J.R. 2017. Modelling the variation in wood density of New Zealand-grown Douglas-fir. N.Z. J. For. Sci. **47**: 15. doi:10.1186/s40490-017-0096-0.

Kranjc, L., Farrelly, N., and Harte, A.M. 2019. The effect of thinning on mechanical properties of Douglas fir, Norway spruce, and Sitka spruce. Ann. For. Sci. **76**: 3. doi:10.1007/s13595-018-0787-6.

Lachenbruch, B., Johnson, G.R., Downes, G.M., and Evans, R. 2010. Relationships of density, microfibril angle, and sound velocity with stiffness and strength in mature wood of Douglas-fir. Can. J. For. Res. **40**(1): 55–64. doi:10.1139/X09-174.

Lachenbruch, B., Moore, J.R., and Evans, R. 2011. Radial variation in wood structure and function in woody plants, and hypotheses for its occurrence. *In* Age- and size-related changes in tree structure and function. *Edited by* F.C. Meinzer, B. Lachenbruch, and T.E. Dawson. Springer-Verlag. pp. 121–164.

Lan, H.-Y., Shaw, D.C., Beedlow, P.A., Lee, E.H., and Waschmann, R.S. 2019. Severity of Swiss needle cast in young and mature Douglas-fir forests in western Oregon, USA. For. Ecol. Manage. **442**: 79–95. doi:10.1016/j.foreco.2019.03.063.

Lavender, D.P., and Hermann, R.K. 2014. The genus Pseudotsuga. Forest Research Laboratory, Oregon State University, Corvallis. Available from https://ir.library.oregonstate.edu/concern/defaults/sf268560k.

Lee, H.E., Beedlow, P.A., Waschmann, R.S., Burdick, C.A., and Shaw, D.C. 2013. Tree-ring analysis of the fungal disease Swiss needle cast in western Oregon forests. Can. J. For. Res. **43**(8): 677–690. doi:10.1139/cjfr-2013-0062.

Lowell, E.C., Todoroki, C.L., Dykstra, D.P., and Briggs, D.G. 2014. Linking acoustic velocity of standing Douglas-fir trees to veneer stiffness: a tree-log-product study across thinning treatments. N.Z. J. For. Sci. **44**: 1. [Online.] Available from http://www.nzjforestryscience.com/content/44/1/1.

Luoma, D.L., and Eberhart, J.L. 2014. Relationships between Swiss needle cast and ectomycorrhizal fungus diversity. Mycologia, **106**: 666–675. doi:10.3852/12-180. PMID:24895426.

Maguire, D.A., Kanaskie, A., Voelker, W., Johnson, R., and Johnson, G. 2002. Growth of young Douglas-fir plantations across a gradient in Swiss needle cast severity. West. J. Appl. For. **17**: 86–95. doi:10.1093/wjaf/17.2.86.

Maguire, D.A., Mainwaring, D.B., and Kanaskie, A. 2011. Ten-year growth and mortality in young Douglas-fir stands experiencing a range in Swiss needle cast severity. Can. J. For. Res. **41**(10): 2064–2076. doi:10.1139/x11-114.

Manter, D.K., Bond, B.J., Kavanagh, K.L., Rosso, P.H., and Filip, G.M. 2000. Pseudothecia of Swiss needle cast fungus, *Phaeocryptopus gaeumannii*, physically block stomata of Douglas fir, reducing CO_2 assimilation. New Phytol. **148**: 481–491. doi:10.1046/j.1469-8137.2000.00779.x.

Manter, D.K., Winton, L.M., Filip, G.M., and Stone, J.K. 2003. Assessment of Swiss needle cast disease: temporal and spatial investigations of fungal colonization and symptom severity. J. Phytopathol. **151**: 344–351. doi:10.1046/j.1439-0434.2003.00730.x.

Markwardt, L.J., and Wilson, T.R.C. 1935. Strength and related properties of woods grown in the United States. USDA. Technical Bulletin No. 479.

Martinez Meier, A.G., Sanchez, L., Salda, G.D., Pastorino, M.J.M., Gautry, J.Y., Gallo, L.A., and Rozenberg, P. 2008. Genetic control of the tree-ring response of Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) to the 2003 drought and heat-wave in France. Ann. For. Sci. **65**(1): 102. [Online.] doi:10.1051/forest:2007074.

Megraw, R.A. 1986. Douglas-fir wood properties. *In* Douglas-fir: stand management for the future. *Edited by* C.D. Oliver, D.P. Hanley, and J.A. Johnson. University of Washington, Institute of Forest Resources, Contribution No. 55, Seattle, Wash. pp. 81–96.

Meinzer, F.C., McCulloh, K.A., Lachenbruch, B., Woodruff, D.R., and Johnson, D.M. 2010. The blind men and the elephant: the impact of context and scale in evaluating conflicts between plant hydraulic safety and efficiency. Oecologia, **164**: 287–296. doi:10.1007/s00442-010-1734-x. PMID:20668883.

Pollet, C., Henin, J.-M., Hebert, J., and Jourez, B. 2017. Effect of growth rate on the physical and mechanical properties of Douglas-fir in Western Europe. Can. J. For. Res. **47**(8): 1056–1065. doi:10.1139/cjfr-2016-0290.

Rais, A., Poschenrieder, W., Pretzsch, H., and van de Kuilen, J.-W.G. 2014. Influence of initial plant density on sawn timber properties for Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco). Ann. For. Sci. **71**: 617–626. doi:10.1007/s13595-014-0362-8.

Rathgeber, C.B.K., Decoux, V., and Leban, J.M. 2006. Linking intra-tree-ring wood density variations and tracheid anatomical characteristics in Douglas fir (*Pseudotsuga menziesii* (Mirb.) Franco). Ann. For. Sci. **63**(7): 699–706. doi:10.1051/forest:2006050.

Ritokova, G., Shaw, D.C., Filip, G., Kanaskie, A., Browning, J., and Norlander, D. 2016. Swiss Needle Cast in western Oregon Douglas-fir plantations: 20-year monitoring results. Forests, **7**(8): 155. [Online.] doi:10.3390/f7080155.

Rossi, S., Anfodillo, T., Cufar, K., Cuny, H.E., Deslauriers, A., Fonti, P., et al. 2016. Pattern of xylem phenology in conifers of cold ecosystems at the Northern Hemisphere. Global Change Biol. **22**: 3804–3813. doi:10.1111/gcb.13317. PMID:27082838.

Rozenberg, P., Franc, A., Mamdy, C., Launay, J., Schermann, N., and Bastien, J.C. 1999. Genetic control of stiffness of standing Douglas fir; from the standing stem to the standardised wood sample, relationships between modulus of elasticity and wood density parameters. Part II. Ann. For. Sci. **56**(2): 145–154. doi:10.1051/forest:19990206.

Ruiz Diaz Britez, M., Sergent, A.-S., Martinez Meier, A., Breda, N., and Rozenberg, P. 2014. Wood density proxies of adaptive traits linked with resistance to drought in Douglas- fir (*Pseudotsuga menziesii* (Mirb.) Franco). Trees, **28**: 1289–1304. doi:10.1007/s00468-014-1003-4.

Saffell, B.J., Meinzer, F.C., Woodruff, D.R., Shaw, D.C., Voelker, S.L., Lachenbruch, B., and Falk, K. 2014. Seasonal carbohydrate dynamics and growth in Douglas-fir trees experiencing chronic, fungal-mediated reduction in functional leaf area. Tree Physiol. **34**: 218–228. doi:10.1093/treephys/tpu002. PMID:24550088.

Sergent, A.S., Breda, N., Sanchez, L., Bastein, J.C., and Rozenberg, P. 2014. Coastal and interior Douglas-fir provenances differ in growth performance and response to drought episodes at adult age. Ann. For. Sci. **71**(6): 709–720. doi:10.1007/s13595-014-0393-1.

Silen, R.R. 1978. Genetics of Douglas-fir. USDA FS Research Paper WO-35. Washington, D.C. 34 pp.

Smith, D.M. 1956. Effect of growth zone on specific gravity and percentage of summerwood in wide-ringed Douglas-fir. Forest Products Laboratory, Issue 2057.

Stone, J.K., Capitano, B.R., and Kerrigan, J.L. 2008. The histopathology of *Phaeocryptopus gaeumannii* on Douglas-fir needles. Mycologia, **100**: 431–444. doi:10.3852/07-170R1. PMID:18751550.

Todaro, L., and Macchioni, N. 2011. Wood properties of young Douglas-fir in Southern Italy: results over a 12-year post-thinning period. Eur. J. For. Res. **130**: 251–261. doi:10.1007/s10342-010-0425-9.

Vikram, V., Cherry, M.L., Briggs, D., Cress, D.W., Evans, R., and Howe, G.T. 2011. Stiffness of Douglas-fir lumber: effects of wood properties and genetics. Can. J. For. Res. **41**(6): 1160–1173. doi:10.1139/x11-039.

Weiskittel, A.R. 2003. Alterations in Douglas-fir crown structure, morphology, and dynamics imposed by the Swiss needle cast disease in the Oregon Coast Range. M.S. thesis, Dept. of Forest Resources, Oregon State University, Corvallis, Oreg.

Weiskittel, A.R., Maguire, D.A., Garber, S.M., and Kanaskie, A. 2006. Influence of Swiss needle cast on foliage age-class structure and vertical foliage distribution in Douglas-fir plantations in north coastal Oregon. Can. J. For. Res. **36**(6): 1497–1508. doi:10.1139/x06-044.

West Coast Lumber Inspection Bureau (WCLIB). 2018. Rev. Sept. 1, 2018. Grading rules for west coast lumber. Standard no. 17. West Coast Lumber Inspection Bureau, Portland, Oreg. WWPA (Western Wood Products Association).