

Genetic Variation in Tolerance of Douglas-fir to Swiss Needle Cast as Assessed by Symptom Expression

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Summary

The incidence of Swiss needle cast on Douglas-fir has increased significantly in recent years on the Oregon coast. Genetic variation in symptoms of disease infection, as measured by foliage traits, was assessed in two series of progeny trials to determine whether these "crown health" indicators were under genetic control and correlated with tolerance; tolerance being continued growth in the presence of high disease pressure. Foliage traits generally had lower heritabilities than growth traits and were usually correlated with diameter growth. Foliage traits of crown density and color appeared to be reasonable indicators of disease tolerance. In the absence of basal-area data, assessing crown density and color can help screen for families that show tolerance to the disease.

Key words: resistance, *Phaeocryptopus gaeumannii*, *Pseudotsuga menziesii*, genetic gain.

Introduction

Since the late 1980s, Swiss needle cast (*Phaeocryptopus gaeumannii* (ROHDE) PETRAK) has become increasingly severe in plantations of Douglas-fir (*Pseudotsuga menziesii* (MIRB.) FRANCO) in coastal Oregon and Washington, USA, and, more recently, in older, naturally established stands. In severely impacted stands, symptoms of Swiss needle cast infection include yellowing and premature loss of older foliage. Annual growth lost by young Douglas-fir plantations from this disease on the north Oregon coast was estimated to be 23% in 1996 (MAGUIRE *et al.*, 2002). The disease is reaching epidemic proportions along the north coast of Oregon (Oregon Department of Forestry, 1998). Swiss needle cast is endemic to the Pacific Northwest, yet apparently caused no detectable injury to Douglas-fir in the region until the 1970s when Christmas tree growers began to report needle loss (MICHAELS and CHASTAGNER, 1984; HANSEN *et al.*, 2000). The disease was known to injure Douglas-fir in Europe (BOYCE, 1940; MERKEL, 1951; BONIFACIO *et al.*, 1970), New Zealand (HOOD *et al.*, 1990), Australia (PEDERICK and MARKS, 1975), the Lake States (MERRILL and LONGENECKER, 1973) and the east coast (MORTON and PATTON, 1970) of North America.

The disease impairs water relations in the needles by occluding stomates with fruiting bodies (pseudothecia). Typically, new needles become infected in the spring and early summer, shortly after they emerge from buds. The fungus grows inside the needle and eventually form pseudothecia which release spores in the spring and early summer. The amount of fungus and number of pseudothecia increase with age of the needle until the needle is dropped (HANSEN *et al.*, 2000). In heavily infected stands, first-year needles become chlorotic the following spring and drop from the tree during the second growing season. The loss of foliage and the impaired function of needles remaining on the tree combine to reduce tree growth and vigor. Typically, healthy Douglas-fir have at least 5 years of needles (HOOD, 1982); but stands on the north Oregon coast have 3 years or less (MAGUIRE *et al.*, 2002).

A likely hypothesis for the cause of increased disease severity in the Pacific Northwest is that recent forest management practices, combined with a climate conducive to the disease, have shifted conditions to favor the pathogen (KANASKIE *et al.*, 1996; ODF, 1998; HANSEN *et al.*, 2000). Douglas-fir plantations have often replaced natural mixed stands of Sitka spruce, western hemlock and Douglas-fir, or alder. Because Douglas-fir cone crops are not reliable in low elevation coastal areas, the seed sources used to establish these plantations tended to be from areas farther inland and at higher elevations – areas that may lack any natural resistance to the fungus. Also, the sources of seeds used to reforest the vast area of the Tillamook burns of the 1930s, 40s and 50s are largely unknown. The combination of a climate that favors the disease, increased amount and density of Douglas-fir in coastal areas, and off-site seed sources may have set the stage for rapid and efficient spread of the fungus. As a result, the pathogen population may have increased enough to overwhelm natural mechanisms of disease tolerance, resulting in loss of tree growth and health in impacted areas.

In Europe, where the disease was first observed, it is controlled by restricting Douglas-fir establishment to drier sites and using coastal (not inland) provenances. Early provenance studies in Europe showed coastal provenances were much more resistant than inland provenances (FERRE, 1955; ROHMEDE, 1956). Later studies showed that within the coastal variety of Douglas-fir, resistance increased as the seed source origin approaches the coast, with lower elevation, and northward from California (HOOD and WILCOX, 1971; PEDERICK and MARKS, 1975). McDERMOTT and Robinson (1989) found a positive correlation between resistance and rainfall in the parent location. Family variation was shown in a study of Christmas trees by NELSON *et al.* (1989) and BLADA's (1988) results suggest that genetic resistance could be increased through use of resistant clones.

Examination of heavily infected stands reveals that there can be considerable variation in the expressions of symptoms from tree to tree, but all trees are infected as evidenced by fruiting bodies. There is also limited data that indicates the amount of fungus per needle does not differ by family (TEMEL and JOHNSON, 2001), suggesting that any genetically controlled defense mechanism is a tolerance function as opposed to a resistance function (actually repelling the fungus). Tolerance to a forest manager is defined as continued growth in the presence of high disease pressure. This definition will also be used in this paper. Whether the growth rate is a function of inherent growth rate or some other resistance mechanism cannot be examined with the available data at present. Such a question requires a more complex study than is available.

The objectives of this study were to examine the within-provenance genetic variation in disease symptoms (i.e., foliage traits) and to determine whether they are correlated with past and/or subsequent growth (tolerance). Such data is useful in

designing testing programs for developing Douglas-fir tolerant to Swiss needle cast.

Methods and Materials

Two series of progeny trials were examined in this study. The most comprehensive set of data was from the Nehalem tree breeding cooperative, which is affiliated with the Northwest Tree Improvement Cooperative. This breeding program has 10 progeny test sites that encompass the northern Coast Range of Oregon from Hebo to the Columbia River; however, only five sites with the most severe disease symptoms were assessed for foliage health for this report. The second series of progeny tests was from the USDA Forest Service (USFS) breeding program on the Hebo Ranger District of the Siuslaw National Forest. From a series of six progeny test sites in the Hebo Ranger District, only two with the most severe symptoms were assessed for foliage health. At all seven of these progeny test sites, there was only three age classes of foliage remaining on the trees, and the remaining age classes did not have 100% retention (Table 1). Disease severity at these sites were not monitored prior to age-10. Disease severity has been relatively constant since age 10.

Nehalem

The Nehalem tests were established in a "reps-in-sets" design, where replications (blocks) are nested within family sets. Forty open-pollinated families were allocated to each of ten sets, for a total of 400 families. All replications of each individual set were planted together, resulting in essentially 10 separate progeny tests planted side-by-side at each site. The trials were established as three replications of four-tree noncontiguous plots per site. Height and diameter at breast height (DBH) were assessed on all trees in the winter of 1995/1996 at age 11 from seed. Height data from an age-5 assessment were also available. At the five progeny test sites judged to be most infected with Swiss needle cast, five separate foliage traits were assessed as potential indicators of tolerance to the disease:

- Needle retention on the 1994 primary laterals (NR94p) – scored 0 to 9, where 0 = 0% to 10% retention, and 9 = 91% to 100% retention.

- Needle retention on the 1994 secondary laterals (NR94s) – scored 0 to 9.

- Needle Retention on the 1993 secondary laterals (NR93s) – scored 0 to 9.

- Crown Density – a subjective score where 1 = sparse crown to 6 = dense crown. This score attempted to "quantify" the amount of foliage on a tree by examining how dense (or transparent) the live crown appeared.

- Color – a subjective score where 1 = yellow, 2 = green, 3 = dark green.

On 16 May 1996, bud burst was examined on four sets of families at one site (Coal Creek) to see if earlier or later flushing families were more resistant to Swiss needle cast. Bud burst was scored as: 1 = tight buds, 2 = swollen buds, 3 = bud beginning to burst through tip, 4 = extended branchlet, needles not reflexed, 5 = extended branchlet with needles reflexed.

In the spring of 1998 a single set (40 families) at three sites was assessed a second time for foliage scores to examine their repeatability over time. Traits assessed were crown density, color, and needle retention of the 1996 secondary foliage. Needle retention could only be assessed on two of three sites because the trees had become so tall on the third site that assessing retention was difficult.

In December of 1998, DBH was measured on four sets (160 families) on the same three progeny test sites that had repeated assessments for foliage scores. These data were used to determine three-year basal-area increment, which is a direct measure of tolerance per. se. Tolerance being defined as continued growth in the presence of disease pressure.

USFS – Hebo

Similar information was collected on two sites from the USFS series of progeny tests. These trials were established as a "sets-in-reps" design, where sets are nested within replica-

Table 1. – Needle retention percentages* for 2nd and 3rd year needles and narrow-sense heritabilities for color, crown density, needle retention (NR), height and DBH on seven progeny test sites of Douglas-fir (standard errors in parentheses).

Series	Site	% Needle Retention*		Narrow sense heritability									
		2 nd year foliage	3 rd year foliage	Color	Crown density	NR 94p	NR 94s	NR 93s	Age-5 height	Age-10/11 height	Age-11 DBH	Age-13/14 DBH	Budburst
Nehalem	Cole Mt.	78	55	0.10 (0.03)	0.16 (0.04)	0.21 (0.04)	0.15 (0.04)	0.34 (0.05)	0.31 (0.04)	0.30 (0.04)	0.27 (0.04)		
Nehalem	Davis Creek.	64	30	0.27 (0.04)	0.28 (0.04)	0.24 (0.04)	0.28 (0.04)	0.28 (0.04)	0.39 (0.05)	0.33 (0.04)	0.32 (0.04)		
Nehalem	Slick Rock	80	50	0.20 (0.04)	0.22 (0.04)	0.10 (0.03)	0.06 (0.03)	0.20 (0.04)	0.39 (0.05)	0.37 (0.05)	0.32 (0.04)	0.34 (0.05)	
Nehalem	Acey Creek.	80	56	0.04 (0.03)	0.23 (0.04)	0.07 (0.03)	0.03 (0.03)	0.16 (0.04)	0.36 (0.05)	0.38 (0.05)	0.40 (0.05)	0.36 (0.05)	
Nehalem	Coal Creek	74	45	0.10 (0.03)	0.19 (0.04)	0.07 (0.03)	0.05 (0.03)	0.13 (0.04)	0.30 (0.05)	0.32 (0.05)	0.32 (0.05)	0.36 (0.05)	0.96 (0.07)
USFS	Salal	65		0.30 (0.07)	0.44 (0.08)		0.27 (0.06)		0.08 (0.04)	0.12 (0.05)		0.18 (0.05)	0.62 (0.10)
USFS	Gordy	83		0.12 (0.05)	0.21 (0.06)		0.20 (0.06)		0.10 (0.05)	0.23 (0.07)		0.14 (0.06)	

* % needle retention represents the percentage of foliage as indicated by the average needle retention score on the secondary branches for second year foliage (NR94s) and third year foliage (NR93s).

tions. Each site had five replications of 4-tree noncontiguous plots. In these trials, each field replication included all three sets. Each of the three sets had 35 open-pollinated families, for a total of 105 families. Height at age 10 was assessed in the fall of 1995. In the spring of 1996, crown density, color and needle retention on 1994 secondary laterals was assessed on the two sites. Bud burst was assessed on one site with the same scoring procedure, except that no distinction was made between tight and swollen buds. In 1998, DBH at 13 years was measured on all trees in the USFS series of trials.

In order to examine the correlation of foliage traits with subsequent basal area increment (i.e., tolerance) for the USFS series, it was necessary to first estimate DBH at age 10. The age-11 Nehalem data were used to estimate a diameter-height equation. The first step was to limit the Nehalem data to only those trees in the height range of the USFS trees at age 10 (32,720 trees in total). Both height and diameter were log transformed because the residual variation increased with increasing height. The final equation for predicting diameter in millimeters with height in centimeters was:

$$\text{Age-10 DBH} = e^{(-21.29 + [6.8539 \cdot \text{Ln}(\text{height}) - 10.4435 \cdot (\text{Ln}(\text{height})^2)])} \quad (r^2=0.869)$$

Genetic calculations

The traits were first examined for normality. The Kolmogorov test, an extremely sensitive test for normality, indicated that the residuals for all foliage variables were not normal at any site, while the growth variables were not statistically normal at some sites ($\alpha=0.05$). Further examination of plot means (rep-by-family means) indicated that only the retention scores were skewed to a significant degree; a log transformation resulted in a more normal distribution and the transformed data were used for heritability estimates. It was unnecessary to transform the other variables for heritability estimates because the assumption of normality typically is not required for estimating components of variance (STEEL *et al.*, 1997, page 174). The F statistic is directly related to heritability and only in extreme circumstances does the normality assumption affect F tests (SNEDECOR and COCHRAN, 1967, page 276; NETER and WASSERMAN, 1974, page 513). Because there was very little change in correlations between transformed and untransformed data, the untransformed data were used for correlations. Results are more easily interpretable with untransformed data, and for testing the null hypothesis that $r = 0$, use of untransformed data may be used provided that one of the variables is normal (SNEDECOR and COCHRAN, 1967 p. 193). Because the variance of color and needle retention at a site was related to the site mean, data were standardized for all variables at each site for the computation of correlations. Standardization took the form of subtracting the site mean and dividing by the standard deviation of the trait.

Narrow-sense heritabilities were determined in each progeny test site by using the formula:

$$h^2 = (4 \sigma_{\text{family}}^2) / (\sigma_{\text{family}}^2 + \sigma_{\text{within family}}^2)$$

Standard errors for the heritability estimates were calculated according to BECKER (1984).

The heritability formula assumes that the open-pollinated families were truly half-sibs such that σ_{family}^2 = one quarter the additive genetic variance (σ_a^2), and ignores any bias from genotype-environment interaction. Variance components for the Nehalem data were obtained by using SAS Varcomp procedure (SAS, 1990) with the REML option from the following model:

$$y_{ijkl} = \mu + \text{set}_i + \text{rep}(\text{set})_{ij} + \text{family}(\text{set})_{ik} + \text{error}_{ijkl}$$

where y_{ijkl} is the response for the l^{th} tree in the k^{th} family in the j^{th} rep in the i^{th} set

μ is the population mean;

set_i is the effect of the i^{th} set;

$\text{rep}(\text{set})_{ij}$ is the effect of the j^{th} replication in the i^{th} set;

family_{ik} is the effect of the k^{th} family in the i^{th} set;

error_{ijkl} is the pooled effect of the replication-by-family interaction for the j^{th} replication and k^{th} family in the i^{th} set, and the effect of the $ijkl^{\text{th}}$ plot, i.e. the within plot variation. The rep*family component was pooled because no evidence was found of rep*family interactions.

Because the USFS trials utilized a "sets-in-reps" design, the model was modified to:

$$Y_{ijkl} = \mu + \text{rep}_j + \text{set}_i + (\text{rep-by-set})_{ij} + \text{family}(\text{set})_{ik} + \text{error}_{ijkl}$$

Type B genetic correlations examining the correlation of the same traits measured at different sites (r_b) assumed that the genetic variation, but not the environmental variation, was the same at each site, and used the YAMADA formula (1962) cited in BURDON (1977):

$$r_b = \sigma_{\text{family}}^2 / (\sigma_{\text{family}}^2 + \sigma_{\text{family} \times \text{site}}^2)$$

Variance components came from the analysis of variance for each combination of two sites. The analyses were done on the standardized data and used the model:

$$Y_{hijkl} = \mu + \text{site}_h + \text{set}_i + \text{rep}(\text{site-set})_{hij} + \text{family}(\text{set})_{ik} + \text{family} \times \text{site}(\text{set})_{hik} + \text{error}_{hijkl}$$

Genetic correlations among different traits (r_a) were calculated using the standardized data with the following equation:

$$r_a = \sigma_{\text{family-crossproduct A*B}}^2 / (\sigma_{\text{family-trait A}} \times \sigma_{\text{family-trait B}})$$

where:

$\sigma_{\text{family-crossproduct A*B}}^2$ is the family covariance component for traits A and B;

$\sigma_{\text{family-trait A}}^2$ is the family variance component for trait A; and

$\sigma_{\text{family-trait B}}^2$ is the family variance component for trait B.

In Nehalem, a separate r_a was calculated for each set so that it was possible to obtain a mean correlation and a standard error. Because the USFS trials were a sets-in-reps design and had only three sets, the correlations (variance and covariance components) were estimated for the model that included sets, and therefore gave only one estimate of the genetic correlation.

Family means for correlations were obtained by first getting family means at each site and then averaging the families over all sites, giving equal weight to each one.

When the same traits were assessed at different ages, age-age genetic correlations were estimated as:

$$r_a = \sigma_{\text{family crossproduct}}^2 / (\sigma_{\text{family-1996 assessment}} \times \sigma_{\text{family-1998 assessment}})$$

where the family variance components estimates come from a complete ANOVA model, including sites, sets, replications and families.

Results

Foliage traits were under less genetic control than were growth traits; that is, they had lower heritabilities in all instances but one, the USFS Salal site (Table 1). Trees on this site suffered more defoliation than did trees on any other site in either trial series.

For the Nehalem sites, the crown density and color scores showed significant positive correlations with age-11 DBH, and color was correlated with age 5 to 11 height increment (Table 2). None of the needle retention scores were significantly correlated with growth (Table 2). Family-mean correlations of color and crown density with DBH were larger for the five Nehalem sites chosen for the needle cast assessment (the most infected sites) than for the remaining five sites $r = 0.30$ vs 0.13 for color, and $r = 0.32$ vs 0.17 for crown density).

Table 2. - Family mean correlations (above diagonal) and genetic correlations (below diagonal) for 10 sets of families planted on 5 sites (standard errors in parenthesis). All family mean correlations are statistically significant at $\alpha = 0.01$ unless otherwise noted.

	Age-11 DBH	Age-14 DBH	Age-11 height	Height increment	Color	Crown density	Retention 93 sec.	Retention 94 prim	Retention 94 sec.	Bud burst ¹
DBH-11	1.0	0.971 (0.005)	0.750 (0.017)	0.689 (0.026)	0.411 (0.025)	0.445 (0.035)	0.038 ^{ns} (0.038)	0.068 ^{ns} (0.060)	0.114 ^{ns} (0.054)	-0.078 ^{ns} (0.160)
DBH-14	0.989 (0.006)	1.0	0.770 (0.027)	0.697 (0.056)	0.449 (0.052)	0.503 (0.040)	0.159* (0.041)	0.097 ^{ns} (0.060)	0.197 ^{ns} (0.127)	-0.085 ^{ns} (0.072)
Ht-11	0.749 (0.021)	0.774 (0.034)	1.0	0.961 (0.005)	0.223 (0.037)	0.167 (0.043)	0.041 ^{ns} (0.034)	-0.028 ^{ns} (0.041)	0.025 ^{ns} (0.036)	-0.137 ^{ns} (0.069)
Ht Inc	0.704 (0.032)	0.728 (0.074)	0.969 (0.005)	1.0	0.278 (0.038)	0.145 (0.049)	0.142* (0.033)	0.076 ^{ns} (0.041)	0.109 ^{ns} (0.039)	-0.164 ^{ns} (0.075)
Color	0.506 (0.045)	0.650 (0.161)	0.211 (0.049)	0.306 (0.052)	1.0	0.436 (0.051)	0.353 (0.033)	0.420 (0.028)	0.475 (0.050)	-0.010 ^{ns} (0.141)
Crn Den	0.478 (0.047)	0.570 (0.040)	0.162 (0.058)	0.156 (0.065)	0.546 (0.081)	1.0	0.160 (0.033)	0.225 (0.048)	0.263 (0.051)	-0.038 ^{ns} (0.146)
NR 93S	0.043 (0.052)	0.204 (0.064)	0.057 (0.047)	0.177 (0.047)	0.526 (0.054)	0.200 (0.051)	1.0	0.782 (0.016)	0.699 (0.029)	-0.196* (0.064)
NR 94P	0.075 (0.083)	-0.054 (0.310)	-0.060 (0.059)	0.073 (0.057)	0.626 (0.058)	0.269 (0.071)	0.898 (0.025)	1.0	0.823 (0.021)	-0.234 ^{ns} (0.114)
NR 94S	0.131 (0.075)	0.244 (0.240)	-0.007 (0.047)	0.109 (0.055)	0.687 (0.064)	0.314 (0.080)	0.854 (0.032)	0.966 (0.028)	1.0	-0.165 ^{ns} (0.319)
Bud burst	-0.050 (0.083)	-0.128 (0.091)	-0.164 (0.060)	-0.272 (0.124)	0.040 (0.092)	-0.153 (0.289)	-0.173 (0.069)	-0.249 (0.196)	-0.084 (0.098)	

¹) Assessed on only four sets on one site

*) Significant at $\alpha=0.05$.

^{ns}) Nonsignificant at $\alpha=0.05$.

Table 3. - Family mean (above diagonal) and genetic (below diagonal) correlations for traits averaged over two USFS sites. Family mean correlations are all significant at $\alpha = 0.01$ unless otherwise noted.

	Age-13 DBH	Age-10 height	Height inc.	Crown density	Color	Retention	Bud burst
Age-13 DBH	1.0	0.708	0.658	0.332	0.346	0.274*	0.096 ^{ns}
Age-10 height	0.564	1.0	0.956	-0.114 ^{ns}	-0.006 ^{ns}	-0.018 ^{ns}	-0.036 ^{ns}
Height increment	0.600	1.107	1.0	-0.151 ^{ns}	-0.030 ^{ns}	0.043 ^{ns}	-0.057 ^{ns}
Crown density	0.158	-0.570	-0.481	1.0	0.503	0.270*	0.086 ^{ns}
Color	0.365	-0.204	-0.156	0.569	1.0	0.470	0.301
Retention	0.563	-0.260	-0.293	0.330	0.500	1.0	-0.001 ^{ns}
Bud burst	0.132	0.072	-0.025	0.094	0.394	-0.077	1.0

^{ns}) Nonsignificant at $\alpha=0.05$

*) Significant at $\alpha=0.05$

Age-13 DBH was associated with higher foliage scores (greener crowns with more foliage) on the USFS sites (Table 3). The family mean correlations between foliage traits and height were nearly zero, but the genetic correlations were negative (Table 3). The genetic correlations for the comparison of foliage traits with height varied among the sets. In one of the three sets in this series there was no significant family variation for age-10 height; therefore one would not expect any significant correlations with height. In the other two sets, the genetic correlations of height with retention and color were positive in one set and negative in the other.

Examination of the three Nehalem sites with basal-area increment data showed that all the foliage traits were better

correlated with basal-area increment than with height or DBH (data not shown). As expected, age-11 DBH was well correlated with subsequent basal-area increment on all three sites measured in 1998 ($r_a \geq 0.93$) (Table 4). At two of the three Nehalem sites, the genetic correlation of crown density with basal-area increment was reasonably high ($r_a = 0.90$ and $r_a = 0.79$) and on the third site (Slick Rock) the genetic correlation of color with basal-area increment was 0.70 (Table 4). At the USFS Salal site, both crown density and color had strong genetic correlations with estimated basal-area increment ($r_a > 0.7$).

Symptom expression was associated with proportionally more loss of diameter growth than loss of height growth. For example, height of the yellow trees was 89% of the height of

Table 4. – Correlations of age-11 (Nehalem) or age-10 (USFS) variables with subsequent three-year basal-area increment (Nehalem) and estimated basal-area increment (USFS). All family mean correlations significant at $\alpha = 0.01$ unless noted otherwise.

Trait	Nehalem						USFS			
	Coal Creek		Acey Creek		Slick Rock		Salal		Gordy	
	Family mean	Genetic (r_a)	Family mean	Genetic (r_a)	Family mean	Genetic (r_a)	Family mean	Genetic (r_a)	Family mean	Genetic (r_a)
Height	0.26	0.83	0.72	0.91	0.60	0.63	0.48	0.40	-0.01 ^{ns}	-0.24
DBH	0.76	0.93	0.80	0.93	0.79	0.95				
Crown density	0.46	0.90	0.43	0.79	0.30	0.35	0.55	0.73	0.44	0.56
Color	0.35	0.53	0.26	0	0.48	0.70	0.49	0.78	0.36	0.46
Needle retention*	0.21	0.49	0.05 ^{ns}	-0.03	0.13 ^{ns}	0.27	0.31	0.41	0.29	0.64

*) Needle retention of 2-year-old needles on lateral branches.

^{ns}) Nonsignificant $\alpha=0.05$

the green trees, both in Nehalem and on the USFS sites; DBH of the yellow trees were 83% of the green trees in Nehalem and 80% on the USFS sites. For both test series, height for crown density class 2 was approximately 85% of that for class 4; DBH of crown density class 2 was about 74% of class 4.

Site-to-site genetic correlations for foliage traits were generally higher among the Nehalem sites than between the two USFS trials (Table 5). Growth traits tended to be better correlated across sites than did the foliage traits, with the exception of a poor correlation of age-13 DBH between the two USFS sites ($r=0.59$).

Table 5. – Site-to-site family mean and Type B genetic correlations for SNC traits.

Trait	Nehalem ¹		USFS	
	Family mean	Type B	Family mean	Type B
Height	0.48	0.92	0.43	0.80
DBH	0.43	0.90	0.33	0.59
Color	0.24	0.87	0.34	0.71
Crown density	0.32	0.84	0.51	0.64
Retention 94 primary	0.26	0.95		
Retention 94 secondary	0.23	0.83	0.12	0.10
Retention 93 secondary	0.40	0.86		

¹) Nehalem correlations are means of all combinations of 5 sites (10 pairs).

The phenotypic correlations among the foliage traits over time were relatively weak; however, the age-age family mean and genetic correlations calculated over the sites were moderate to high (Table 6).

Bud burst was highly heritable (Table 1), but showed no consistent patterns of correlation with the foliage traits (Table 2 and 4). A slight negative correlation was found with the needle retention scores in Nehalem and a positive correlation with color at the USFS sites.

Table 6. – Phenotypic, family mean and genetic correlations among 1996 and 1998 foliage traits for the Nehalem data.

Trait ¹	Nehalem		
	Phenotypic	Family Mean	Genetic
Crown density	0.28	0.49	0.68
Color	0.27	0.60	0.87
Retention	0.13	0.47	1.15

¹) Three sites were assessed for crown density and color, only two sites for retention.

Discussion

The foliage traits all appeared to be under genetic control, but to a lesser degree than for growth. Heritabilities reported for *Pinus radiata* foliage traits measuring resistance to *Dothistroma pini* (0.19 to 0.37 in CARSON and CARSON, 1986) and *Cyclaneusma* needle cast (0.32 in KING *et al.*, 1998) were both higher than the heritabilities for foliage scores in these trials. Neither color nor crown density are continuous variables; therefore, normality assumptions were violated, which could be one explanation for the lower heritabilities of the foliage traits. Another possible explanation is that all of the foliage scores were subjective and thus have more measurement error associated with them than do the directly measured growth traits. To better understand the repeatability of foliage scores, 150 trees were scored for crown density at the Slick Rock site in 1998 by two separate observers on consecutive days. One of the two observers used only categories 1 to 4 and the other 1 to 5. Of the 150 trees, 75 were given the same score by the two observers; of the remaining 75 trees, 69 differed only by 1. Six trees differed by 2 crown-density categories. Although this agreement appeared relatively close, its correlation coefficient of $r = 0.45$ was less than what was expected. This finding demonstrated how categorical variables may not give the same results as would be expected from continuous variables. Heritability estimates could also be expected to be less than those of an equally heritable continuous variable.

In a clonal test, BLADA (1988) reported broad-sense heritabilities for "resistance" larger than 0.75. This estimate could result from increased environmental uniformity, a different assessment trait, or a large portion of the resistance/tolerance arising from non-additive variation. If non-additive variation is the reason for increased heritability, then programs would need to reconsider using seed orchards to produce seed tolerant to Swiss needle cast. Non-additive variance cannot be captured in wind-pollinated orchards and only one-quarter of the dominance variation can be captured by using controlled-pollination.

The foliage traits showed reasonable repeatability over sites and years. Site-to-site correlations for foliage traits were somewhat smaller than for growth variables, but – except for USFS retention – they were still strong (Table 5). The age-age phenotypic correlations were low (typically < 0.5), but the genetic correlations were moderate to high (typically > 0.5). This difference between phenotypic and genetic correlations is partially due to the poor repeatability of individual scores.

For the Nehalem data, crown density and color scores had much larger correlations with growth data than did the needle retention scores. Heritabilities for crown density were also larger than those for color or retention when examined over all five Nehalem sites. These larger heritabilities imply that crown density may be the best single foliage score for examining Swiss needle cast tolerance. Crown density had the largest heritabilities in the USFS trials, but the correlation with growth traits were poorer than those for color and retention.

Needle retention was not apparently as good a trait as expected for assessing tolerance. MAGUIRE *et al.* (2002) found needle retention a good predictor of growth loss for a stand. Taller families were more exposed to wind and sun and may have lost more needles as a result. Another possible explanation is that the disease does not impact trees until they have reached a size where older needles contribute a significant proportion to the total crown biomass. Taller families would reach this point earlier than shorter families. These points could help explain why the foliage scores were negatively correlated with height in USFS. Note, however, that the correlations of foliage scores with DBH were positive.

Needle retention scores may not well represent total foliage on an individual tree basis. The crown density score, which attempted to estimate the relative amount of foliage on a tree, was poorly correlated with the needle retention scores. For the Nehalem data, the individual phenotypic correlation between crown density and needle retention (1994 secondary foliage) averaged only 0.15 over the five test sites. Family-mean correlations were 0.23, and the genetic correlation was 0.32. Many of the trees with relatively dense crowns did not have large retention scores, but appeared to have larger quantities of first-year foliage than those that were less dense.

Budburst was under strong genetic control (Table 1), but it was not correlated with growth and only correlated with foliage traits in two instances (Table 2 and 3). A slight negative correlation with one of three needle-retention scores was found in Nehalem and a positive correlation with color was found on the USFS sites. The color score in the USFS trials was correlated with growth, suggesting that budburst may be associated with disease tolerance; however, later-bursting families would not be expected to be any less susceptible because Swiss needle cast spore flight continues for weeks after budburst. These results suggest that date of bud burst did not affect tolerance.

Because defoliation associated with this disease affects diameter growth before height (MAGUIRE *et al.*, 2002), for foliage traits to be better correlated with DBH than height is not surprising. The data from this study also showed that greater

crown density and darker color scores correlated with improved diameter growth more than with height growth.

The most useful definition of tolerance to Swiss needle cast is continued tree growth in the presence of increased disease pressure, as measured by basal-area increment from age 11 to 14. Past growth on the diseased sites is one indicator of future growth, and the foliage traits may be other useful indicators. Because many of the foliage traits were correlated with past and subsequent diameter growth, some basis exists to suggest they may be indicators of tolerance in the future. For two of the Nehalem sites, the genetic correlation of crown density with subsequent basal-area increment (0.90 and 0.79) was nearly as high as the correlation of age-11 DBH and subsequent basal area (0.93).

The best single trait to select for would be a direct measure of basal-area increment. Using the Nehalem data, it was found that indirect selection for basal-area increment using DBH (age-11) was 92% as efficient as direct selection. Including crown density and color with DBH in an index only increased the efficiency of indirect selection to 93%. Indirect selection for basal-area increment using age-11 height was 72% as efficient as direct selection, and adding the above two foliage traits increased selection efficiency to 83%. The efficiency of selecting for basal-area increment with color and crown density in an index was 57%. Although the use of the foliage traits can be helpful in selecting for increased basal-area increment in the presence of the disease, the best indicator of future basal-area growth was past growth (i.e., age-11 DBH).

Not all test series have basal-area increment or even DBH data, and selecting for tolerance to Swiss needle cast is limited to using only height and foliage traits. Height was strongly correlated with subsequent basal-area increment in the Nehalem data, but not so for the USFS sites. This poor correlation may be an artifact of having to estimate early DBH with the height-DBH equation. If the poor correlation between height and basal-area increment is real, then the foliage scores will contribute to increasing gain in disease tolerance because they had larger genetic correlations with estimated basal-area increment.

The top 10% of the families selected for basal-area increment had 28% more basal area than the population average for the three Nehalem sites with age-14 DBH measurements and 26% more basal area for the two USFS sites (Table 7). Actual gains from using the parents in a seed orchard (assuming no pollen contamination) would be close to these differences (selection differentials) for most traits because gain is equal to the selection differential times twice the family mean heritability, and most family-mean heritabilities were near 0.5 (Table 7). This increased growth could offset the 27% reduction that MAGUIRE *et al.* (2002) estimated for stands with only two years of foliage, but it may not offset the growth reduction in stands with one-and-a-half years of foliage or less (37% volume reduction with only 1.5 years of foliage). The 49% reduction expected for stands with only one year of foliage probably could not be offset by a single generation of breeding. On severely affected sites with less than 1.5 years of foliage, avoiding Douglas-fir altogether would be best (FILIP *et al.*, 2000).

Conclusions

Based on this information, the simple foliage traits of crown density and color may be reasonable indicators of tolerance to Swiss needle cast. These traits were heritable and genetically correlated with basal-area growth. Use of these traits with DBH did little to improve gain over using DBH alone. Many breeding programs have little or no data for DBH and basal-

Table 7. — Selection differentials from choosing the best 10% of the families for basal-area increment on three Nehalem sites and two USFS sites, including family mean heritabilities.

	Population average	Average of top 10%	% increase	Family mean heritability
NEHALEM				
Basal-area increment (mm ²), age 11-14	9118	11640	28%	0.535
DBH (mm), age-11	111.4	122.5	10%	0.571
Height (cm), age-11	771.6	817.4	6%	0.606
Crown density, age-11	3.85	4.01	4%	0.541
Color, age-11	1.91	1.95	2%	0.329
Needle retention, age-11	7.77	7.87	1%	0.379
USFS-HEBO				
Estimated basal-area increment (mm ²), age 10-13	6504	8174	26%	0.519
Height (cm), age-10	502	543	8%	0.502
Crown density, age-10	3.57	3.74	5%	0.565
Color, age-10	1.97	2.08	6%	0.545
Needle retention, age-10	7.39	7.49	1%	0.099

area increment; many have only height data. The foliage traits would be useful in estimating tolerance in these situations. Multiple sites should be assessed to ensure good data are obtained that will be applicable over a wide variety of sites. Probably more important than assessing foliage traits is obtaining growth-increment data for families under disease pressure, but collecting these data is not always possible.

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