

Seed weight – seedling size correlation in coastal Douglas-fir: genetic and environmental components

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The effect of seed weight on nursery seedling height was analyzed in two experiments. In expt. 1, 16 seeds per family from 111 families were individually weighed and sown in autumn. In expt. 2, a second group of 16 seeds were individually weighed and stratified and sown in spring. Four-tree noncontiguous family plots were randomly assigned to two densities in two replications in each experiment. Date of emergence and duration and rate of shoot elongation were determined over 2 years of growth. Seedlings in expt. 1 were exposed to damaging frost after emergence; some seedlings in expt. 2 suffered *Lygus* bug damage to the terminal shoot. Developmental associations between seed weight, a maternally inherited trait, and seedling height and its components were examined using sets of path analyses with and without adjustment for planned and accidental treatment effects. Results suggested both “environmental” and “genetic” contributions of seed weight to seedling height. The weight (environmental) component, alone, decreased with time. The genetic component, which was indicated by lack of direct effect of seed weight on seedling height in the path analyses and by changing female:male variance ratios over time, was quite stable across treatment effects. Because of the genetic relation, seed weight adjustment is not recommended as a procedure for increasing precision in early selection of coastal Douglas-fir (*Pseudotsuga menziesii* var. *menziesii* (Mirb.) Franco) families.

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L'effet du poids des semences sur la hauteur des semis en pépinière a été évalué au sein de deux expériences. Dans l'expérience 1, 16 semences pour chacune de 111 familles ont été pesées individuellement et ensemencées à l'automne. Dans l'expérience 2, un deuxième groupe de 16 semences ont été pesées individuellement, puis stratifiées et ensemencées au printemps. Des parcelles familiales non contiguës de quatre arbres ont été distribuées aléatoirement parmi deux niveaux de densité au sein de deux répétitions par expérience. La date d'émergence ainsi que la durée et le taux d'élongation de la pousse ont été déterminés pendant 2 années de croissance. Suivant leur émergence, les semis de l'expérience 1 ont été exposés aux gelées d'automne potentiellement néfastes; certains semis de l'expérience 2 ont souffert de dommages de punaises à la pousse terminale. Au niveau du développement, les associations entre le poids des semences, un caractère transmis par le parent maternel, et la hauteur des semis ainsi que ses composantes ont été étudiées en utilisant des ensembles d'analyses de coefficients de direction en ajustant ou non pour les effets planifiés et les effets accidentels de traitement. Les résultats ont suggéré que le poids des semences avait des contributions à la fois “environnementale” et “génétique” au niveau de la hauteur des semis. La composante due exclusivement au poids (composante environnementale) diminuait avec le temps. La composante génétique a été identifiée par le manque d'effet direct du poids des semences sur la hauteur des semis au niveau des analyses de coefficients de direction, ainsi que par les rapports variables de variance femelle : mâle dans le temps. Cette composante génétique était relativement stable d'un effet de traitement à l'autre. En raison de la relation génétique, les ajustements en fonction du poids des semences ne sont pas recommandés en tant que procédure permettant d'augmenter la précision au niveau de la sélection précoce de familles de sapin de Douglas vert (*Pseudotsuga menziesii* var. *menziesii* (Mirb.) Franco).

[Traduit par la rédaction]

Introduction

To decrease the considerable costs of field testing Douglas-fir (*Pseudotsuga menziesii* var. *menziesii* (Mirb.) Franco) families, early testing in nurseries is receiving increased attention (Howe and Adams 1986). One proposal is to cull a percentage of families based on their nursery performance. A complicating factor is the relation between seed weight and seedling size, which has been examined in several studies with various results (Lavender 1958; Bell *et al.* 1979; Muhle *et al.* 1985; Sorensen and Campbell 1985). If seed weight affects growth in a manner that camouflages the genetic values of the families, some adjustment may be warranted. If, however, seed weight and plant size both respond to pleiotropic genes broadly affecting vigor, adjustments may be undesirable.

In an earlier effort to explicate the association of seed weight and seedling size (Sorensen and Campbell 1985), we eliminated the confounding factors of genotype and date of emergence by sowing pregerminated seeds of paired heavy-

and light-weight classes of several families. A 10% difference between seed-weight classes within families yielded a 3.5% difference in 2-year seedling height. The study therefore suggested an effect of seed weight on seedling size that was independent of genotype. It did not, however, rule out the possibility of additional seed-weight effects associated with genotype or the modification of seed-weight effects by the nursery environment. Because each of these potentialities may influence decisions on adjusting for seed weight, they deserve study. In the tests reported here, we specifically investigated genetic relations between seed weight and growth potential and the role of nursery spacing.

We had three objectives: (i) to evaluate the stability of seed weight effects when influenced by planned and accidental treatments including the contribution of females (large seed weight differences) and males (small seed weight differences); (ii) to evaluate effects of various adjustment procedures on the estimated genetic variation in seedling height

TABLE 1. Analysis of variance, degrees of freedom (df), and coefficients for expected mean squares in expt. 1

Sources of variation ^a	df	Expected mean squares and coefficients ^b						
		σ_E^2	$\sigma_{D \times M(F)}^2$	$\sigma_{D \times F(P)}^2$	$\sigma_{D \times P}^2$	$\sigma_{M(F)}^2$	$\sigma_{F(P)}^2$	σ_P^2
Replication	1							
D	1							
Main plot error	1							
P	3	1	6.60	17.32	146.26	12.98	34.40	291.35
F(P)	36	1	6.57	17.54	0.54	12.81	34.75	
M[F(P)]	71	1	6.35	0.17	0.08	12.36		
D × P	3	1	6.41	16.92	142.49			
D × F(P)	36	1	6.26	16.89				
D × M[F(P)]	71	1	5.99					
E	1167	1						
Total	1390							

^aD, density; P, populations; F(P), females (populations); M[F(P)], males (females (populations)); E, minor plot error.

^b σ_E^2 is variance of minor plot error effects including variance within plots; $\sigma_{D \times M(F)}^2$, $\sigma_{D \times F(P)}^2$, $\sigma_{D \times P}^2$ are variance of interaction effects; $\sigma_{M(F)}^2$ is variance of males-in-female effects; $\sigma_{F(P)}^2$ is variance of females-in-population effects; and σ_P^2 is variance of population effects.

and on rankings among family means; and (iii) to identify developmental mechanisms by which seed-weight effects persist or are lost during growth. In essence, we wished to examine further if adjustment of seedling size for seed weight is advisable in nursery progeny evaluations of coastal Douglas-fir. Our primary analytical tool was analysis of variance, but for the last two objectives and particularly for (iii) we also used path analysis, i.e., consideration of paths through seed emergence timing and through 1st- and 2nd-year growth rates and durations.

Materials and methods

Experiments

Two experiments of identical design and family composition were installed, expt. 1 in 1985 and expt. 2 in 1988. Each experiment included two replications, two spacings (main plots) of 7.5 and 10.0 cm (178 and 100 seedlings/m²), and 111 families. Families included 80 design I crosses, in which 40 "seed" parents (females) were mated with two males (males in females), plus open-pollination families from 31 of the females. We considered open pollination as representing a third male in crosses with seed parents. Four-seed noncontiguous plots represented each family in each main plot.

Seed parents were in natural stands near Marys Peak (44°28'N, 123°30'W; elevation 460 m; 5 seed parents), Corvallis (44°38'N, 123°12'W; elevation 75 m; 6 seed parents), Lacombe (44°35'N, 122°42'W; elevation 275 m; 21 seed parents), and Lyons (44°44'N, 122°23'W; elevation 500 m; 8 seed parents), Oregon. Males were in the same stands as females except for trees in the Lyons stand, whose seeds were crossed with males from the Lacombe stand. The four parental stands were treated as "populations" and seed trees as "females in populations" in the analysis of variance.

Cones were collected when mature, based on cone color and seed and seedwing color. All cones and seeds were handled similarly. Moisture content of seeds was not determined, but based on experience with comparable material it should have been 5 to 7% on a fresh weight basis. The megagametophyte plus the embryo constitute about 70% of the filled weight of coastal Douglas-fir seeds.

For each family and experiment, 16 filled, healthy seeds, based on x-ray examination, were individually weighed. For expt. 1, unstratified seeds were sown in early November. For expt. 2, seeds were individually stratified for 61 days starting on February 19 and were sown on April 20 and 21. Sowing depth was controlled by using a peg board to impress 0.5 cm deep holes in a smoothed nursery bed surface. The tests were surrounded by two rows of border plants; density plots were separated by six buffer rows, three at each spacing.

Measurements

The following traits were measured: seed weight to nearest 0.1 mg; the date of emergence above the ground surface, based on daily observations; hypocotyl length in mm; the date of setting of the terminal bud in years 1 and 2, based on twice weekly observations; the date of flushing of the terminal bud, based on observations made every other day; and final heights in years 1 and 2, measured in 0.5 cm units. Eleven traits were analyzed: weight of filled seed (SDWT); date of seedling emergence (EMG); hypocotyl length (HY); 1st year date of bud set minus EMG (DUR1); 2nd year date of bud set minus 2nd year date of bud burst (DUR2); 1st year stem elongation rate (RTE1), as 1st year height minus HY divided by DUR1; 2nd year stem elongation rate (RTE2), as 2nd year height minus 1st year height divided by DUR2; total height at 1 year (HT1) and 2 years (HT2); and heights adjusted for SDWT at 1 (AHT1) and 2 (AHT2) years.

Statistical methods

The SAS VARCOMP procedure was used with type 1 mean squares for analysis of variance (SAS Institute Inc. 1987) (see Table 1 for model and coefficients for expt. 1). Each experiment was analyzed twice, once including all families and once omitting open-pollination families. Because of the imbalance of the data sets, approximate *F*-tests were applied by constructing mean squares with appropriate expectations and degrees of freedom (Rawlings 1988). Individual-seedling heritabilities were calculated as

$$\frac{2\sigma_{F(P)}^2 + 2\sigma_{M[F(P)]}^2}{\sigma_E^2 + \sigma_{D \times M[F(P)]}^2 + \sigma_{D \times F(P)}^2 + \sigma_{F(P)}^2 + \sigma_{M[F(P)]}^2}$$

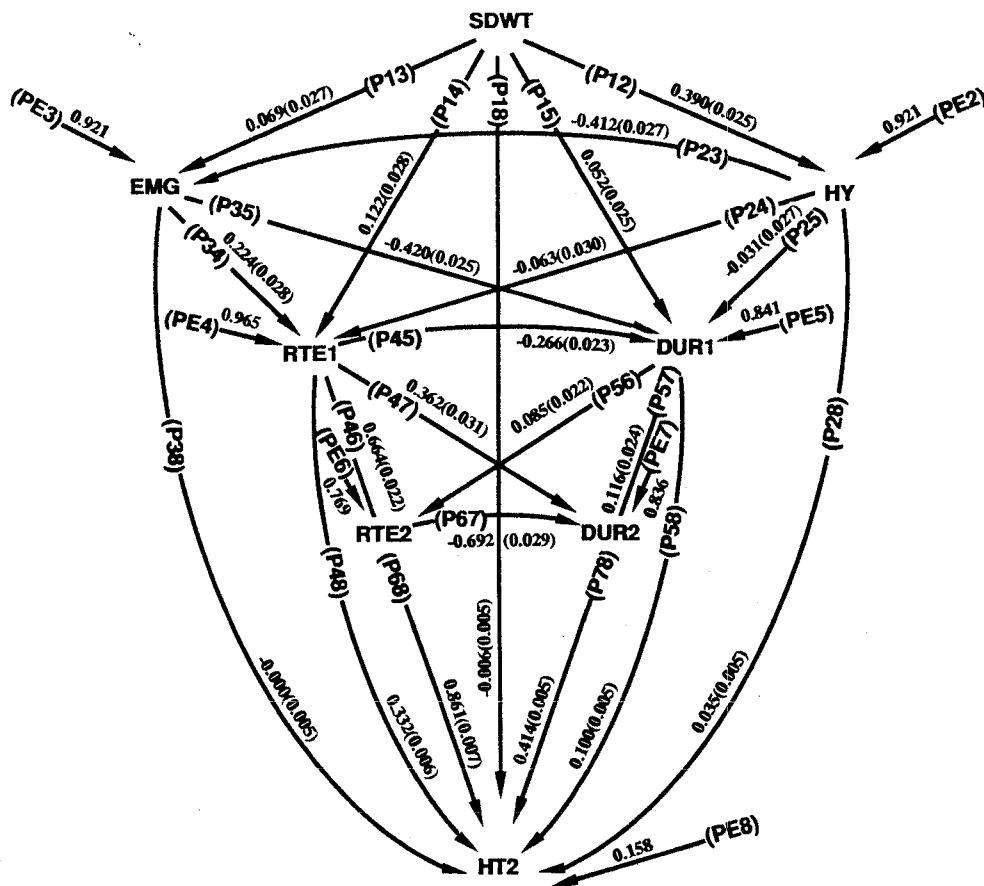


FIG. 1. Path correlation coefficients (with standard errors in parentheses) for expt. 1 (unstratified Douglas-fir seeds sown in autumn). SDWT, weight of filled seed; EMG, date of seedling emergence; HY, hypocotyl length; RTE, rate of stem elongation; DUR, duration of stem elongation; HT, total height; 1 and 2, years 1 and 2 in the nursery. P identifies the paths; PE_i are the path coefficients from exogenous variables, i.e., variables outside the pictured system.

where symbols are given in Table 1.

Heritabilities of seedling traits can be overestimated if there are maternal effects operating through SDWT and EMG. However, comparison of heritabilities based on both adjusted and unadjusted values indicated that the bias is small to moderate (St. Clair and Adams 1991; see also Table 2, this paper, HT1 vs. AHT1 and HT2 vs. AHT2).

Also, estimation of heritability using this equation assumes no dominance. Dominance, if present, will inflate the numerator through the term, $\sigma_{M(F(P))}^2$ (Becker 1967). We know of no published information on dominance deviations in nursery Douglas-fir, but some evidence comes from the present test. By definition, dominance variance = $4(\sigma_{M(F)}^2 - \sigma_F^2)$ (Becker 1967). For our expt. 2B (spring sowing, design I families only), $\sigma_{F(P)}^2$ is 109.1 (HT1), 51.9 (AHT1), 644.6 (HT2), and 325.2 (AHT2), and $\sigma_{M(F(P))}^2$ is 34.5, 39.3, 604.8, and 630.0, respectively. Without adjustment for SDWT, $\sigma_{F(P)}^2 > \sigma_{M(F(P))}^2$ in both years and dominance is estimated as zero. With adjustment there is dominance variance in year 2, but not year 1. On this basis, we assume that dominance variance is small compared with additive variance. We return to this point in the Discussion.

Five adjustments of HT1 and HT2 for SDWT were compared by using regression equations of different origins: (i) adjustment using the regression of all seedling heights on all seed weights, (ii) adjustment using regression family mean

seedling heights on family mean seed weights assuming a single population, (iii) adjustment using regression computed from the population line in the analysis of variance table (Snedecor and Cochran 1967, pp. 436-438), (iv) adjustment using regression based on females-in-populations line, and (v) adjustment using regression based on the "minor-plot error" line (Table 1). Regression coefficients came from an analysis of covariance analogous to the analysis of variance in Table 1. Because expt. 2 represented a standard procedure applicable to early testing in nurseries, adjustments were compared only for this experiment and by using data after deleting open-pollination families.

Path analysis

Sets of path analyses were used to examine developmental association of SDWT with seedling height growth. Path analysis is a method for decomposing and summarizing linear relations in a closed system. Because it is closed, the analysis assumes that it is a causal system. The system is described by a path diagram that represents the assumed causal structure (Fig. 1). The paths among variables (arrows) lead from hypothesized causes to effects. Because few systems in biology are closed in the sense that they are not influenced by an exogenous variables, the structure is mathematically closed by adding summary paths from residual causes to the system variables, the PE_i in Fig. 1.

For path analysis and the investigation of developmental relations, all families were treated as if coming from a single population. This was done for three reasons: (i) most importantly, preliminary analyses indicated that including populations had a negligible effect on the size of the path coefficients, but it did complicate the analysis, (ii) population effect was small and often nonsignificant, and (iii) the four populations were only slightly more dispersed than what normally would be within a single tree breeding unit, where operationally they would be treated as one population.

Our primary interest in using this analysis was to examine the direct and indirect influence of SDWT on HT1 and HT2 through the intermediate steps of EMG, HY, and the growth attributes, growth rate (RTE1, RTE2) and duration (DUR1, DUR2).

SDWT can influence seedling height (HT1 and HT2) by the nutrient and energy capital stored in endosperm reserves for germination and seedling emergence (P13) or for growth thereafter (P14, P15). Extension of the preformed embryo determines the HY (P12), from which all subsequent growth extends (P24, P25, P28). Numbers of cotyledons were counted and analyzed, but in this experiment other variables were not associated with number of cotyledons and they were not considered to be part of the system influencing HT2.

Given the same average depth of sowing, seedlings with longer HY may emerge before those with shorter ones, thus affecting EMG (P23). An early emerging seedling quickly adds photosynthates to early reserves, potentially contributing to growth (P34); its DUR1 is also potentially longer (P35). Both growth rate (RTE1, RTE2) and duration (DUR1, DUR2) contribute directly to total HT2 (P48, P58, P68, P78). A given extension can be produced either by a fast rate for a short duration (P46) or by a slow rate for a long duration (P57). The RTE1 and DUR1 may affect the RTE2 and DUR2 (P47, P56). If, for example, in any growing season, total extension is fixed by genotype, accumulated energy capital, or meristem potential, fast growth rates will result in short durations (P45, P67), thereby allowing more time for shoot meristem development in autumn.

In analyzing the closed system of paths, a coefficient was estimated by multiple regression for each path in the diagram. In succession, each lower order variable was used as a dependent variable, with all higher order variables as predictors (Kim and Kohout 1975). Because we wished to evaluate the relative effects of several variables in a common scale, path correlation rather than path regression coefficients were estimated. Data for all variables therefore were standardized to mean = 0 and standard deviation = 1, before regression analysis.

A correlation coefficient and a path correlation coefficient are fundamentally different. The former measures the absolute correlation between two variables; the latter measures the effect of one variable on another, given the causal system stated in the path diagram (Li 1975). A regression coefficient ordinarily describes a condition in a set of data, as does the correlation coefficient. The regression coefficient, b , calculated from standardized data becomes a path coefficient or a "causal effect coefficient", c , only if the causal system is closed, linear, additive, and unidirectional (Kim and Kohout 1975). The effect coefficient measures the change in the affected variable brought about by unit change in the causal variable. This is the change expected (in the affected variable)

in an experiment in which the causal variable is manipulated and all other potential causal variables are held constant unless affected by that causal variable.

Effect coefficients are equivalent to path coefficients only in a two-variable system (excluding exogenous variables). For example, SDWT affects HY only through P12 (Fig. 1). The effect coefficient, 0.390 (Table 3), is therefore the same as the path coefficient for P12. In contrast, EMG has both direct (P13) and indirect (P12 \times P23) causes, and the effect coefficient (-0.092, Table 3) is equal to P13 + P12 \times P23 (0.069 + 0.390(-0.412)). Effect coefficients through lower-order variables are constructed as the sum of direct and indirect effects through several paths.

The analyses provided not only the estimates of all path coefficients in our eight-variable system, but also the path coefficients from exogenous variables, the PE_{*i*} of Fig. 1. The PE_{*i*} represents all residual causes including error and lack of fit. The values for PE_{*i*} are mostly large. This suggests that local bed effects, perhaps subsurface, hidden pest effects, and observer error contribute much to the variation. Values were estimated as $(1 - R^2)^{0.5}$, where R^2 is the coefficient of determination from the regression solution.

Stability of the path coefficients was evaluated by using two sowing densities. Effect of SDWT and EMG might be influenced by competition. Seeds from different dams were included because much of the SDWT variation in Douglas-fir is among seed trees (Silen and Osterhaus 1979). In addition to planned experimental treatments, we had accidental ones. In expt. 1, a spring frost damaged or killed new needles on many seedlings, and in expt. 2, *Lygus* bugs damaged terminal meristems of leaders of some seedlings in late summer of the first growing season.

We examined the effects of treatments on stability of path coefficients by estimating coefficients for a complete experiment, then removing, for example, frost effects and reestimating coefficients, etc. In expt. 1, seedling trait, Y , is considered initially to be the sum of all effects

$$Y = F + R + D + R \times D + F + M(F) + D \times F + D \times M(F) + \text{error}$$

where F is frost, R is number of replications, D is density, F is female, and $M(F)$ is males in females.

For each experiment, nine sets of path coefficients were estimated by using data for all seedlings in each set. First, a set was estimated by ignoring all listed effects (set 0). This was the adjustment used in determining AHT1 and AHT2. Then injury effects (frost in expt. 1, *Lygus* bugs in expt. 2) were removed (set 1), that is, adjustment was made for the effects of injury and path analyses were repeated on deviation from that adjustment. Then other sets were created as follows: injuries and replications were removed (set 2), injuries, replications, and densities were removed (set 3), and injuries, replications, densities, and replications \times densities were removed (set 4). After each set was created the analyses were repeated. Subsequent sets removed the remaining effects sequentially until set 8, which analyzed effects of error only (microsite and within-family deviations). In each set, each trait was individually fitted to the appropriate general linear model (GLM) procedure of SAS (SAS Institute Inc. 1987). Residuals from GLM were then standardized and subjected to regression analyses to give the seven solutions required to

estimate all path coefficients and their standard errors (Kim and Kohout 1975). In sets 2 to 8, degrees of freedom for calculating standard errors of path coefficients were reduced by the degrees of freedom attached to the removed effects in the GLM procedure plus those for predictor variables in the regression solutions.

Results

In expt. 1, 191 seedlings (10.8%) did not emerge and an additional 194 seedlings died or were too badly damaged to include in final measurements. In expt. 2, 80 seedlings (4.5%) did not emerge and 119 died or were badly damaged. SDWTs of analyzed seedlings were greater than weights of seeds that did not produce emergent seedlings by 2% in expt. 1 and by 6% in expt. 2.

Two accidents (frost and insect damage), our planned treatments (autumn and spring sowing, sowing density), and genetic entries (cross- and open-pollination families) all affected seedling growth to some degree. Potentially different variations in growth patterns and potentially different correlations between seed weight and growth therefore were possible.

Autumn vs. spring sowing

Mean seed weights (and standard deviations) were 12.5 mg (2.4 mg) in expt. 1 and 12.5 mg (2.5 mg) in expt. 2. Averaged over the two experiments and four analyses, 59% of the variance was associated with families (females, males, and populations combined), and 41% with seeds within families. In expt. 1 (autumn sowing), first EMG was observed on February 20, last EMG was observed on April 19, mean EMG was March 11, and the standard deviation was 6.9 days. In expt. 2 (spring sowing), stratified seeds were sown on April 21 or 22. First EMG was observed on May 12, last EMG on June 10, mean EMG was May 17 (26 days after sowing), and the standard deviation was 2.9 days. In both experiments, a much higher proportion of the variance was associated with seeds in families for EMG than for SDWT (Table 2).

Each of the experiments suffered an accident that affected seedling growth. In expt. 1, a temperature of -2.8°C was recorded on April 24. Needles emerging from the center of the whorl of cotyledons were damaged on 51% of the seedlings. Short new needles were killed or damaged; needles longer than about 5 mm and cotyledons were not obviously injured. By about May 20, all damaged seedlings appeared to have formed adventitious buds 1-2 mm in diameter. Frosted seedlings came from seeds that were 5% heavier. They had emerged earlier (March 9 on the average), compared with the unfrosted seedlings (March 14). The HT2 for frosted seedlings was 16% less than for unfrosted seedlings, 61.4 cm vs. 71.5 cm. Thirteen and 15% of the variation among seedlings suffering frost damage was associated with parentage, specifically seed parents (Table 2). Frequency of frost-damaged seedlings ranged from 14 to 80% among seed parents. Component correlations ("genetic correlations") at the seed parent level were $r = -0.90$ between proportion of seedlings damaged and EMG, $r = 0.35$ between proportion damaged and SDWT, and $r = -0.08$ between EMG and SDWT.

In expt. 2, *Lygus* bugs (Schowalter *et al.* 1986) damaged leaders during 1st year elongation in 14% of the seedlings. The proportion of seedlings damaged in expt. 2A (year 2 including open-pollination families) was slightly associated with family ($\chi^2 = 68.4, P < 0.003$, uncertainty coefficient

TABLE 2. Percentages of variation among seedlings based on variance components but ignoring the variation due to replication, density, and replication x density

Sources of variation	SDWT		EMG		HY		FROST		RTE1		DURI		HT1		AHT1		RTE2		DUR2		HT2		AHT2	
	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B	A	B
P	5ns	10*	9*	12**	12**	12**	3*	1ns	3ns	1ns	1ns	0ns	2ns	2ns	2ns	2ns	2ns	2ns	1ns	1ns	3ns	1ns	3ns	4*
	4ns	8ns	6**	3ns	4ns	4ns	6**	9*	-0ns	-0ns	0ns	0ns	1ns	2ns	4ns	1ns	1ns	3ns	1ns	1ns	1ns	1ns	3ns	3ns
F(P)	32***	45***	19***	11***	10*	10*	6**	9*	11***	12***	15***	15***	11***	13***	5**	5**	5**	6*	3*	3*	2ns	7***	7*	7*
	39***	47***	3ns	18***	19***	19***	2ns	1ns	16***	17***	14***	14***	17***	18***	10**	10**	10**	3ns	9***	9***	8**	12***	10**	6ns
M(F)	1	23***	5*	14***	16***	16***	2ns	1ns	4*	5*	15***	15***	5*	5**	5**	5**	3ns	2ns	2ns	3ns	6**	6**	8***	8***
	17***	3**	2ns	8**	7*	7*	87	85	11***	10**	13***	22***	10**	6*	8**	11***	11***	90	97	98	3ns	11***	10**	12**
E	1	39	41	63	61	61	87	85	81	74	74	70	84	81	75	92	90	85	89	86	88	88	84	81
	2	41	43	70	68	68	87	85	74	73	74	72	72	72	75	84	85	89	86	77	77	78	78	81
Heritability																								
	1	0.95	0.56	0.52	0.52	0.52	0.21	0.35	0.60	0.36	0.36	0.18	0.10	0.30	0.30	0.18	0.18	0.10	0.10	0.23	0.23	0.41	0.41	0.36
	2	0.99	0.16	0.52	0.52	0.52	0.21	0.53	0.58	0.49	0.49	0.28	0.23	0.41	0.41	0.28	0.28	0.23	0.23	0.23	0.41	0.41	0.41	0.36

NOTE: Height adjustments for seed weight are on an individual-seedling basis. SDWT, weight of filled seed; EMG, date of seedling emergence; HY, hypocotyl length; RTE, rate of stem elongation; DUR, duration of stem elongation; HT, total seedling height; AHT, heights adjusted for SDWT; 1 and 2, 1st and 2nd year, respectively. Seedlings were grown in two experiments, 1 and 2. Data were analyzed (A) including wind-pollination families and (B) omitting wind-pollination families. Percentages for density x population, density x female, and density x male were all nonsignificant and near zero, and were not included in the table. Values have been rounded to the nearest percentage; columns may not total 100. Values between 0 and 0.5 are entered as 0, values between 0 and -0.5 are entered as -0. ns, $p > 0.05$; *, $p < 0.05$; **, $p < 0.01$; ***, $p < 0.001$.

TABLE 3. Correlations among individual seedlings in expt. 1 (above diagonal, $n = 1391$) and in expt. 2 (below diagonal, $n = 1576$)

	SDWT	EMG	HY	HT1	RTE1	DUR1	RTE2	DUR2	HT2
SDWT	—	-0.092	0.390	0.134	0.077	0.058	0.104	-0.018	0.121
EMG	-0.066	—	-0.385	0.041	0.237	-0.476	0.031	0.010	0.050
HY	0.483	-0.194	—	0.043	-0.102	0.178	-0.000	0.003	0.017
HT1	0.394	-0.325	0.303	—	0.923	0.011	0.622	-0.100	0.805
RTE1	0.356	-0.256	0.226	0.915	—	-0.359	0.634	-0.119	0.788
DUR1	-0.002	-0.165	-0.049	0.156	-0.213	—	-0.153	0.092	-0.107
RTE2	0.179	-0.164	0.057	0.514	0.543	-0.093	—	-0.481	0.856
DUR2	0.023	0.018	-0.015	0.072	-0.011	0.236	-0.268	—	-0.030
HT2	0.274	-0.218	0.126	0.741	0.713	0.049	0.878	0.145	—

NOTE: For identification of trait abbreviations see Table 2.

$C/R = 0.053 \pm 0.012$ (Theil 1972, pp. 115–120)), but was not correlated with SDWT, EMG, HY, or HT1. HT2 of damaged seedlings was less than HT2 of undamaged seedlings by 8% (undamaged 50.1 cm, damaged 45.9 cm).

Simple correlations between SDWT, EMG, HY, and seedling height (HT1, HT2) were larger in expt. 2 than in expt. 1 (Table 3). In expt. 1, SDWT, EMG, and HY were not associated with more than 2% of the variation in height (r^2 values calculated from Table 3). In expt. 2, SDWT was associated with about 16 and 8% of the variation in HT1 and HT2, respectively, EMG with 11 and 5%, and HY with 9 and 2%. These r^2 s exceeded even those for associations between durations (DUR1, DUR2) and heights (HT1, HT2).

Sowing density

Seed traits (SDWT and EMG) did not differ between density plots, but apparently density did affect growth; F -values were large for size traits, but because main-plot error had only one degree of freedom, the difference was significant ($p < 0.01$) only for HT2. Compared with seedlings in the low-density plots, seedlings in high-density plots were 95% as tall the 1st year and 86% as tall at the end of the 2nd year.

Genetic entries

We considered open pollination as representing a third male in crosses with seed parents. Seeds from open pollination weighed 12% less than those from cross pollination. Compared with seedlings from cross pollination, open-pollination seedlings emerged slightly later (1%) and were slightly shorter (HT2), by about 1% in expt. 1 and 4% in expt. 2. Eliminating open-pollination seedlings from analyses increased the proportion of variation attributed to females in SDWT and EMG but not in other traits (Table 2). Their elimination greatly decreased variation among males for SDWT, although male-in-female effects were still significant (Table 2) for SDWT in both experiments and for EMG in expt. 1 only (Table 2).

Distributions of trait variation between females and males in females were generally similar in the two experiments. The two experiments did differ in the way that females and males contributed to variation in EMG. Females accounted for five times more of the variation than did males in expt. 1 but not in expt. 2 (Table 2). Error, which includes variation among and within family plots, differed among experiments and traits. For EMG especially, and for HY, it was larger in expt. 2 than in expt. 1. In expt. 2B (omitting data from open-pollination

families, see Table 2), the ratio in variation of SDWT contributed by females vs. males was almost 16:1. This greater contribution by females was repeated in a lesser degree in RTE1, HT1, and DUR2, but the ratios decreased with age.

Adjustment for seed weight

Adjustments of HT1 and HT2 for SDWT required the calculation of regression coefficients. For HT1, coefficients ranged from 3.86 mm/mg (family-in-population means) to 4.24 mm/mg (female-in-population line), and for HT2, from 9.45 mm/mg (family-in-population means) to 10.77 mm/mg (error line). Coefficients were lower for the source lines, but not greatly so: 2.00 mm/mg for HT1 and 8.78 mm/mg for HT2. The similarity of regression coefficients for population and family-in-population lines, particularly for SDWT–HT2 regressions, further justified the deletion of population effects from the path analyses.

Rankings of family means derived from adjusted seedling heights (AH1, AH2) were minimally affected by the equations used; correlation among values adjusted by the several procedures ranged from 0.97 to 1.00. The various correlations among family mean heights at the two ages also were little affected, as is indicated by small standard deviations around the mean value, i.e., HT1:HT2 ($r = 0.849$), AHT1:AHT2 ($r = 0.785 \pm 0.001$), AHT1:HT2 ($r = 0.667 \pm 0.018$), and HT1:AHT2 ($r = 0.620 \pm 0.025$).

Adjusting for SDWT on an individual seedling basis reduced female parent contribution to variation in HT1 and HT2 by 52 and 50%, respectively; adjustment increased male contribution by 14% and 4%. As noted above, in expt. 2B (year 2, excluding wind-pollination families) females contributed almost 16 times more variation in SDWT than did males (Table 2), and this greater contribution by females also existed in RTE1, HT1, and DUR2. In AHT1 and HT2, female variance was greater than male variance, but the ratios were about 1:1. In DUR1, RTE2, and AHT2, males contributed significantly more to variation than did females. After adjustments for SDWT, the female:male ratio for HT1 decreased from 3.14:1 to 1.27:1, and the ratio for HT2 from 1.06:1 to 0.51:1. In both instances, the proportion of total variation accounted for by males increased and that by females decreased. Variation in AHT2 associated with females was statistically nonsignificant. Estimated heritabilities decreased with adjustment by 24% in HT1 and 12% in HT2. St. Clair and Adams (1991) estimated family heritabilities from plot means and open-pollination progenies. Heritabilities

TABLE 4. Decomposition of covariation of seed and seedling traits with 2-year seedling height in the original data set (set 0)

Covariation type	Covariation with HT2						
	SDWT	EMG	HY	RTE1	DUR1	RTE2	DUR2
Expt. 1							
Total phenotypic = r	0.121	0.049	0.018	0.788	-0.107	0.856	-0.030
Causal-direct	-0.006	-0.000	0.035	0.332	0.100	0.861	0.414
Causal-indirect	0.076	0.099	-0.098	0.464	0.096	-0.286	0
Total causal (effect coefficient)	0.070	0.099	-0.063	0.796	0.196	0.574	0.414
Noncausal	0.051	-0.050	0.081	-0.007	-0.303	0.282	0.444
Expt. 2							
Total phenotypic = r	0.274	-0.218	0.126	0.713	0.049	0.878	0.144
Causal-direct	0.006	0.006	0.023	0.287	0.107	0.823	0.343
Causal-indirect	0.278	-0.208	0.039	0.401	0.104	-0.130	0
Total causal (effect coefficient)	0.284	-0.202	0.062	0.688	0.212	0.693	0.343
Noncausal	-0.011	-0.016	0.064	0.025	-0.163	0.186	-0.198

NOTE: For identification of trait abbreviations see Table 2.

decreased with adjustment by 18% (HT1) and 4% (HT2) in their material.

Seed-effect pathways

Path analysis was used to explore the causal influences of SDWT on seedling height growth through direct and indirect pathways. Except for EMG and HY in expt. 1, the origin of seed parents (source) made small or nonsignificant contributions to the variation of traits (Table 2). For this reason, and others given earlier, we omitted source and density \times source interaction as categories in the model and analyzed the material as representing a sample of a single population.

Path coefficients differed substantially from correlation coefficients for only a few comparisons (compare Figs. 1 and 2 and Table 3), but in expt. 2 these differences involved the paths connecting the early traits, SDWT and EMG, to HT2. Total phenotypic $r = 0.274$ for the SDWT-HT2 relation (Table 4). The causal coefficient was even larger (Table 4, $r = 0.284$) indicating noncausal effects were small and negative. On the other hand, the direct path coefficient (Fig. 2) was very small (0.007) and nonsignificant. The differences in coefficients suggest that the SDWT effect on HT2 is not strictly a direct environmental influence, but is operating through an association between SDWT and other components of the growth process.

Sequential adjustments for the various planned and accidental treatment effects and genetic effects provided estimates of seed weight : growth relations freed from these effects. As different data sets (0-8) were created by consecutively adjusting for design variables, the path correlation coefficients among traits changed from set to set, but usually did not differ significantly from preceding ones or from those seen in Figs. 1 and 2.

Effects coefficients estimating causal effects of seed and growth traits on HT2 are shown before and after adjustments in Fig. 3. The greatest change in coefficients occurred in adjustment for frost (Fig. 3, expt. 1, data set 0 vs. set 4). But even in this case the initial covariation structures seen in Figs. 1 and 2 remain essentially unchanged after all adjustments. Because many parents were involved, as well as planned and unplanned treatment effects, the lack of change

implies considerable stability in the path structure, as outlined in Figs. 1 and 2. Stability of SDWT effects also is in accordance with the observations of St. Clair and Adams (1991) that competitive environment and sowing type did not greatly affect correlations between SDWT, EMG, and HT.

Discussion

Results of these tests indicated that in coastal Douglas-fir, if seed parents originate in a homogeneous physiographic region, larger seeds produce, on average, taller 2-year-old nursery seedlings. Our results also indicate that covariation of SDWT and HT2 seems to have two parts (hereafter referred to as "components"), one mainly "environmental" and one "genetic." Because of the genetic component particularly, we believe that adjustment for SDWT usually is not advisable.

Before discussing evidence for a genetic component, we emphasize that the interpretation applies to material and conditions comparable with ours. Seed-source effects were negligible, and in both tests seedlings grew vigorously. Undamaged expt. 1 and expt. 2 plants reached average heights of 72 and 50 cm, respectively, at the end of 2 years. The results and implications, therefore, apply to provenances capable of free growth and seedlings cultured under nursery conditions that promote free growth. The following discussion emphasizes results from expt. 2, as analyzed after deleting data from open-pollination families. These data should be representative of families grown in well-managed experimental nurseries and free of the confounding effects connected with unknown parentage, significant provenance variance, and accidental damage.

Two observations initially suggested that there was more to the SDWT influence than could be explained by weight (environment) alone. First, SDWT effect was not direct (Fig. 2, P18), but indirect and in association with other growth components. We surmised that a purely environmental influence would have shown a significant direct path correlation coefficient. Second, path structure from SDWT to HT2 was stable across treatments. Again, it seemed that if the correlation were purely environmental, the path structure would have

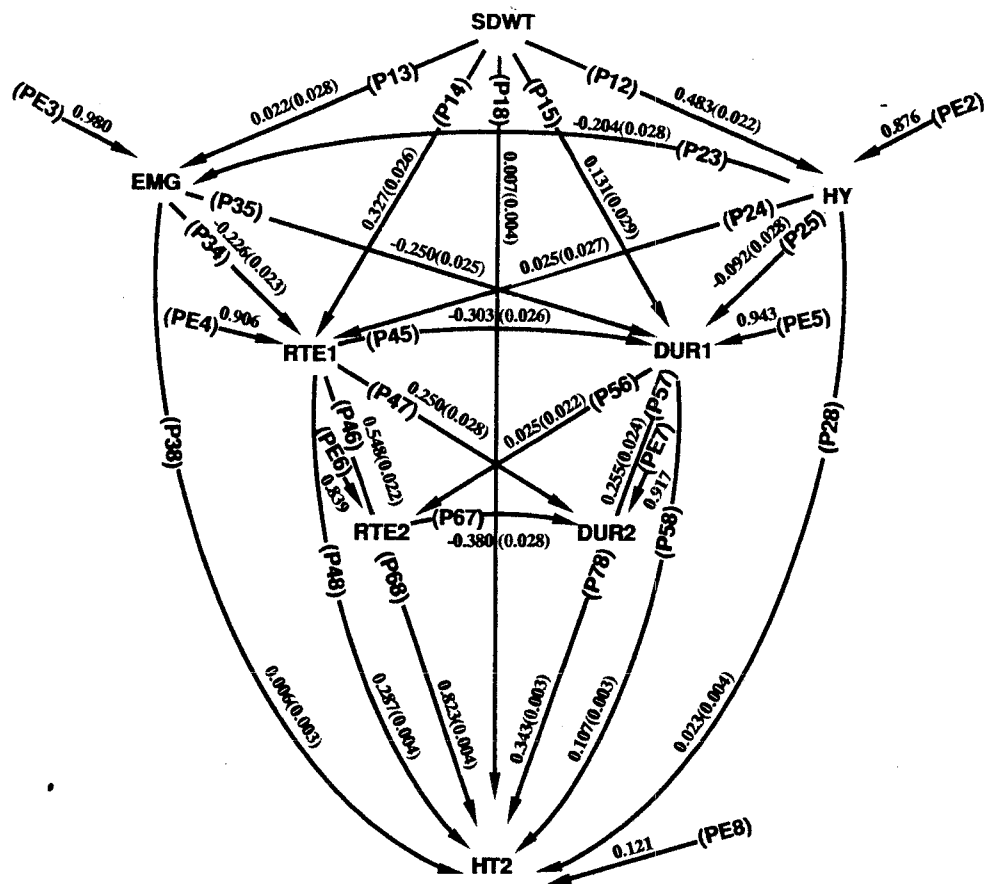


FIG. 2. Path correlation coefficients (with standard error in parentheses) for expt. 2 (stratified Douglas-fir seeds sown in spring). Abbreviations as in Fig. 1.

been more disturbed by planned and accidental treatment effects.

That the second component was genetic followed from the expectation that genotypes encouraging vigorous vegetative growth will also encourage vigorous growth of reproductive structures, i.e., larger cones and seeds. The expectation could arise in two complementary ways. First, genotypes differ in fruitfulness (Sarvas 1962; Eis *et al.* 1965; Griffith 1968; Schoen *et al.* 1986) and vegetative growth is impacted by fruitfulness (Morris 1951; Eis *et al.* 1965; Tappeiner 1969; Linder and Troeng 1981). This suggests that high-vigor or competitive genotypes may put less energy into reproductive structures, and because fewer cones and seeds are produced per unit crown, they may be large (Simak and Gustafsson 1954; Caron *et al.* 1990). Second, because the cone is a modified branch (Owens and Smith 1964), large cones (and seeds) might be a corollary effect of vigorous vegetative growth. Some evidence for this relation has been observed in nursery provenance tests, where significantly positive SDWT-HT correlations have been reported among families in sources (Campbell *et al.* 1989; F.C. Sorensen, to be published) even when the correlation at the source level has been negative (F.C. Sorensen, to be published).

The environmental component is a real effect of seed size on seedling height; the second component is an apparent effect. It is engendered by a positive correlation of SDWT and inherent vigor or growth potential. It may involve genetic correlation but is not truly amenable to genetic analysis

because relevant male genes are not expressed until the offspring of a cross bears seeds. Genetic contributions of male and female parents therefore cannot be directly evaluated in the same generation.

We postulate that the genetic component accounts for changes in variation among males and females during seedling growth and after adjustment for SDWT. If the SDWT-HT relation does reflect an association of large seeds with females whose seedling progeny have greater vigor, then adjustment for SDWT should do two things: it should reduce estimates of genetic variation among females and the reduction should be excessive, that is, the remaining variance among females should be less than the estimate for males. By the end of the second growth season, it appeared that both of these conditions existed in expt. 2B. In our nested mating design with open-pollination families deleted, the variation among females or among males estimates one-quarter of the additive genetic variation and minor genetic interactions. Variation among males-in-females estimates, in addition, one-fourth of the dominance variation. In the absence of dominance variation, the expected ratio, female variance : male variance, is 1:1. In expt. 2B, the estimated total genetic variation (females and males) in AHT1 was 37% less than in HT1, and in AHT2 it was 23% less than in HT2. All of the decrease occurred in the variation among females; in fact, variation among males in adjusted heights increased by 16% in AHT1 and 5% in AHT2. Variation among females in AHT2 was reduced by SDWT adjustment to the point that it became significantly

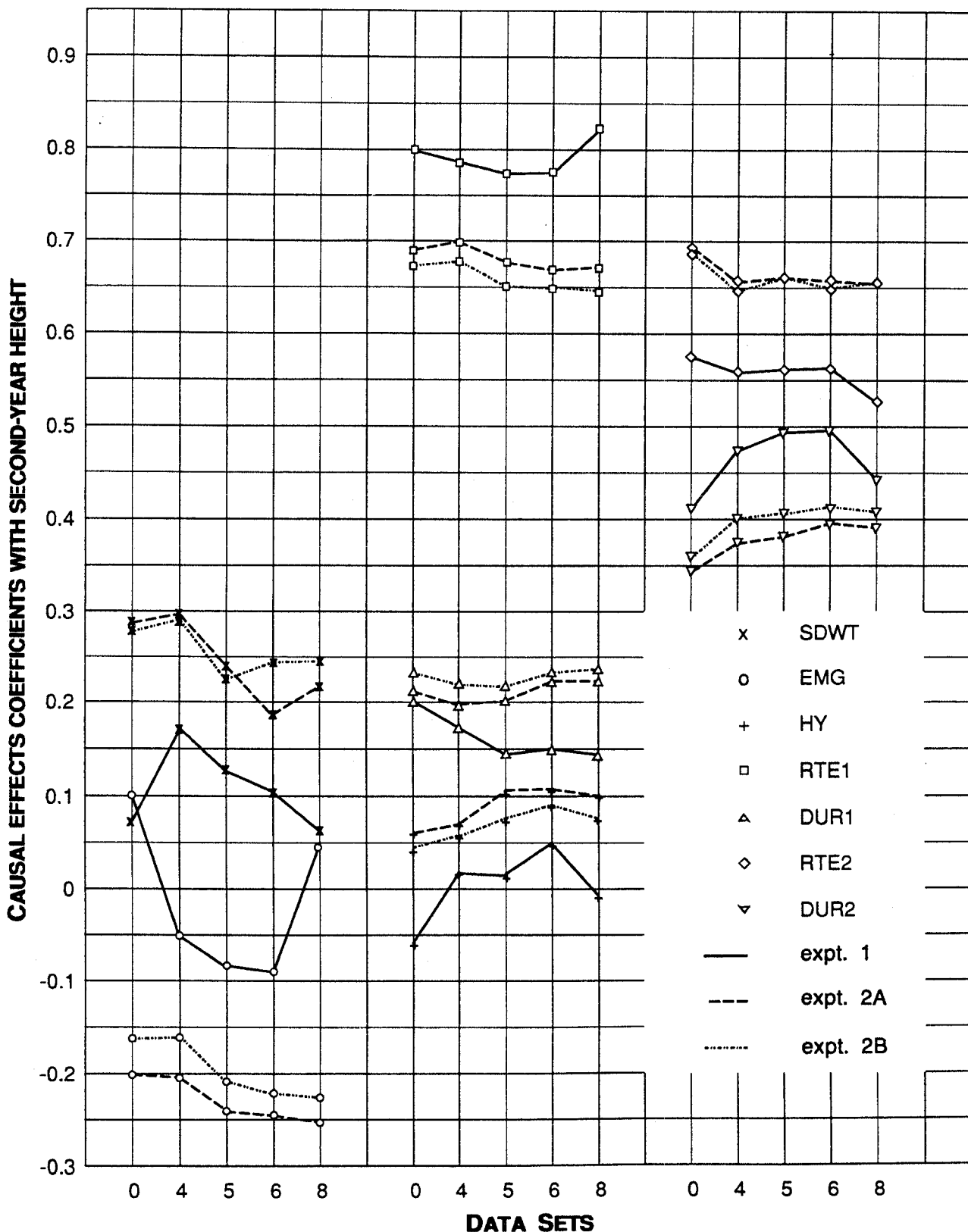


FIG. 3. Correlation coefficients (ordinate) estimating causal effects of seed and growth components on HT2 without adjustment and after adjustment for test variables. Data sets (abscissa): 0, all variables included; 4, data adjusted for frost (expt. 1) or Lygus bug damage (expt. 2), replication, density and replication \times density; 5 and 6, data adjusted additionally for female and male parent, respectively; 8, data adjusted for female parent \times density and male parent \times density. Abbreviations as in Fig. 1.

($p < 0.05$) less than variation among males and not significantly different from zero.

Over time, the female:male variance ratios in expt. 2B were 16:1 (SDWT), 3:1 (HT1), and 1:1 (HT2). After adjustment for SDWT, the same array of ratios was, respectively, 16:1, 1:1, and 0.5:1. In other words, adjusting for SDWT changed the female:male ratios in HT1 from 3:1 to 1:1 and in HT2 from 1:1 to 0.5:1. Therefore, we propose that, under the conditions of expt. 2, the genetic component contributed a substantial fraction to the SDWT-HT relation. A 3:1 ratio of female:male variance in HT1 decreasing to 1:1 for HT2 suggests some contribution in the first growing season, but more in the second.

It would be possible for the environmental component to account for all changes in female:male ratios if (i) by chance, females involved in the crosses truly differed far less in vigor than did males even though they sample the same population, (ii) SDWT variation among females did not have a genetic component, or (iii) genes affecting seedling height exhibited a high degree of dominance and little additivity.

The probability of *i* occurring is less than 1 in 20. The evidence for *ii*, that SDWT is under some genetic control, is indirect. The reported range of family mean SDWTs in Douglas-fir are from 1.75:1 to 2:1 (Olson and Silen 1975; Silen and Osterhaus 1979; St. Clair and Adams 1991; this paper). On the purely environmental side, year effect on SDWT in Douglas-fir averaged about 15% with a maximum of 40% for one tree (Silen and Osterhaus 1979), and 23% in noble fir (*Abies procera*) (Sorensen and Franklin 1977). Bagging cones during cone and seed development increased SDWT 12% (this paper) and 11% (Sorensen and Campbell 1985). Seed-weight differences between young seed orchard ramets and their natural stand ortets range from about 15% (Tak *et al.* 1985) to about 60% (Hadders 1963). The last contrast (seed orchard - natural stand) probably represents a near maximum environmental effect. Because the range among families in stands considerably exceeds most environmental effects, we assume that much of the variation in SDWT among females is genetic.

Evidence concerning the third possibility (*iii*), the degree of dominance, is also indirect. Results from seedlings tests will be biased by SDWT effects, but both this study and that of St. Clair and Adams (1991) gave large heritabilities for HT1 and HT2 with and without adjustment for SDWT. Dominance variance was estimated for our material from the term ($\sigma_{M(F)}^2 - \sigma_F^2$) and was negative for all heights except AHT2. For AHT2 ($\sigma_{M(F)}^2 - \sigma_F^2$) was approximately equal to σ_F^2 . Finally, even pure dominance would, in a statistical sense, have a large additive component (Falconer 1960, pp. 122-125). For these reasons, even though indirect, we believe that the assumption of additive genetic variance for growth and size traits is justified for this material.

The larger correlation coefficient that existed in expt. 2 at the female (r_F) compared with error (r_e) line (0.63 vs. 0.34 for HT1 and 0.55 vs. 0.26 for HT2) provides further indication of the existence of a genetic component in the SDWT-HT relationship. The covariance between SDWT and HT based on the female line, r_F , includes female environmental and female additive genetic effects. The covariance between SDWT and HT based on the error line is r_e . The error line does not include male effects or interactions, but does include error plus such genetic effects as are within full-sib families,

which in turn would include SDWT effects due to seed size variation within full-sib seedlots.

The proportion of covariation associated with the environmental component is estimated as r_e^2 , with the genetic component as ($r_F^2 - r_e^2$). In the 1st year, the female environmental component is estimated to have an effect 41% as large as the genetic component [$0.34^2 / (0.63^2 - 0.34^2) = 0.41$], and in the 2nd year the effect is 29% as large [$0.26^2 / (0.55^2 - 0.26^2) = 0.29$]. The proportions indicate that the female environmental effects are smaller than the female additive genetic effects and that the former decrease more with age. In comparison, the effects coefficients for SDWT in Fig. 3 (compare effects before and after adjustment for female parents, data set 4 vs. set 5) indicate that both SDWT components contribute about equally to the variation in HT2.

According to the path diagrams, SDWT contributes indirectly to HT2 through RTE1 and RTE2 and DUR1 and DUR2 (Figs. 1 and 2). The larger contribution of DUR2 compared with DUR1 may account for the reduction in the environmental component in the second growing season. A genetic influence on HT2 unaffected by SDWT is expected in the male contribution. Seed weight influences HT2 through the females and primarily through RTE1 (Fig. 2, Table 2). These conditions are reflected in the variability patterns among females and males. Variability among females is larger than among males for RTE1 (Table 2). Female contribution to variation is larger in RTE1 than in DUR1, and male contribution is larger in DUR1 than in RTE1 (Table 2). The latter suggests a rather strong heritable aspect of DUR1 that is uninfluenced by SDWT. The increased contribution of DUR2 over DUR1 (Fig. 2) may represent only a more prominent expression of the genetic control of growth, consequently relegating the environmental component to a lesser role in the second season. The decreasing year-to-year influences of SDWT on HT2 through the various developmental paths shown in Figs. 2 and 3 and the lack of direct effects suggest that a persistent effect based on a purely environmental component would be surprising.

Reports of long-term SDWT effects are unusual, but Robinson and van Buijtenen (1979) recorded small, but significant, SDWT - tree volume correlation in loblolly pine at 5 ($r = 0.36$), 10 ($r = 0.31$), and 15 ($r = 0.30$) years of age, where the parent trees were growing in a single seed production area. A correlation that persists this long indicates genetic covariance of SDWT and vigor.

Conclusions

Heavier seeds produced taller 2-year-old seedlings than did light seeds. Based on results of our experiments, however, we cannot recommend SDWT adjustment as a procedure for increasing precision in early selection of coastal Douglas-fir families. We calculated from SDWT-HT2 correlation coefficients that the environmental component of the SDWT effect was only 29% as large as the additive genetic component. Therefore, as a conservative estimate, we suggest that at least 50% of the SDWT effect on seedling nursery height in this test was due to an association of SDWT with inherent vigor of the seed parent. Any adjustment for SDWT penalizes some genetically vigorous families. Adjustment affects the ranking of families, as indicated by family-mean correlations of adjusted and unadjusted HT2s. At the same time, the SDWT

effect is small and would account for very little in, for example, phenotypic selection.

Treatments included two densities, early frost damage in one test and insect damage in the second. All impacted height growth. Both frost and insect damage had family components, with frost damage primarily associated with females. High seedbed density might be expected to exaggerate the influence of SDWT. In our tests, the covariation structure nevertheless seemed little affected by these factors. Undesirable consequences of adjustment appeared to hold under a variety of conditions.

In given situations, tree breeders may find it difficult to determine if a SDWT effect reflects predominantly the environmental or genetic component, but a guideline is suggested. If the effect persists through several years (Robinson and van Buijtenen 1979), it probably represents the action of the genetic component. With increasing age, as indicated in the path diagrams (Figs. 1 and 2), a SDWT effect operates through increasingly long chains of paths of RTEs and DURs. The environmental component will probably dissipate over time so that only those SDWT effects associated with the heredity of the female parent are likely to persist.

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