The Impact of Genetic Variation in Relative Growth Rates on Stem Volume Differentiation: a Simulation Study

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Abstract

Time trends in quantitative genetic parameters of trees may be severely confounded by the effects of inter-tree competition, the temporal autocorrelation of growth, and the significance of size itself on future growth. When growth is a process influenced by both genetic and non-genetic factors the isolation of true genetic effects may be insurmountable. Realistic simulation studies can illustrate how growth dynamics may distort the genetic parameters and thus caution against naive interpretations of field results. Using a single-tree distance-independent stochastic growth model for stem volume, the influence of additive genetic control of relative growth rate on several growth and yield components (stem density, mean volume, total volume, and mean annual increment) was quantified. Growth results for 2 rotations (150 years), 3 levels of initial spacing, and 4 levels of genetic control are presented. The second rotation consisted of offspring from crop trees at the end of the first generation. Simulation results indicated an upper limit of 20% of the additive genetic contribution to the phenotypic variation in stem-volume. Classic heritability estimates of stem volume became grossly inflated by inter-tree competition. The magnitude of inflation was mirrored by the intensity of the competition process. Nevertheless, a narrow spacing appears more efficient for screening genotypes with a high growth potential than wider spacings. Genetic gains achieved by elimination of trees with slow growth rates (self-thinning) were most pronounced during the juvenile phase of the second generation, and they could only be maintained at low levels of inter-tree competition. Volume and growth gains of about 5% per generation appears realistic when relative growth rates are under a fairly strong genetic control (heritability > 0.3).

Key words: heritability, competition, density, selection, genetic gain, bias.

FDC: 165.3; 165.5.

Introduction

Plant size in even-aged perennial monocultures is rarely uniform (HARPER, 1977; WEINER and THOMAS, 1985). Spatial differences in the supply and demand for plant growth resources, growth modifying events (for example insect damage), and genetic effects act to create a size hierarchy of dominance and suppression (GRIME, 1979; WEINER, 1988; WESTOBY, 1982). In long-lived perennials this hierarchy undergoes temporal shifts and changes that are accompanied by density induced thinning of the weakest elements (BRAND and MAGNUSSEN, 1988; FORD, 1975; HARA, 1984; WELLER, 1987). Studies of the shaping forces and the underlying spatial and temporal dynamics of the growth processes leading to these size hierarchies are important for our understanding of the functioning and evolution of species and plant communities (COOK, 1992; CHEPLICK, 1991; LEWONTIN, 1974; PIANKA, 1983; TILMAN, 1988).

Complex and nearly intractable confounding temporal and spatial interactions of plant growth processes have made quantification of the significance of the genetic growth component extremely difficult in non-controlled settings (HARI and KELLOMÄKI, 1981; VIA and LANDE, 1985; TAIT, 1988; WEST-EBERHARD, 1989). This is especially pertinent for forest trees growing for many decades in a heterogeneous environment (KUNG, 1993; NANCE et al., 1983; SWITZER and SHELTON, 1981). Based on empirical evidence and theoretical considerations, it has been argued that in an even-aged monoculture genetic factors may account for 5% to 20% of the observed phenotypic variation in quantitative traits of primary economic concern such as, for example, height, diameter, and stem volume (BALOCCHI et al., 1993; BIROT and CHRISTOPHE, 1983; MONSERUD and REHFELDT, 1990; MORGENSTERN et al., 1975; ZOBEL and TALBERT, 1984). Most tree improvement programs have been founded on the premise of exploiting this genetic component for economical reasons.

In this study, a stochastic growth model (MAGNUSSEN and BRAND, 1989), developed around data collected in a red pine (*Pinus resinosa* AIT.) spacing trial, is used to illustrate how known additive genetic effects in relative growth rate (growth per unit size and time) manifest themselves as phenotypic differences in size. Phenotypic size is the complex outcome of a growth process with deterministic (size and density) and stochastic elements. It will be shown how apparent heritability estimates of stem volume size derived from observed data can be grossly inflated by competition (MAGNUSSEN, 1989a), and how density management impacts on realized genetic gains. Time trends of these aspects are highlighted by following tree growth over two complete rotations (150 years per rotation). Trees not lost to self-thinning during the first rotation serve as parents of the second generation (rotation). These parent trees are considered 'plus' trees that have been spared a 'natural' selection against slow growing trees. Density effects on heritability estimates and genetic gains are illustrated by varying the initial spacing in 3 steps from 1.0 m^2 to 9.0 m^2. Although the model was intended to simulate the growth and development of even-aged red pine plantations it is considered ideal for the intended type of simulating the impact of genetic effects on growth and yield because the basic red pine model can be considered as 'free' of genetic effects (FOWLER and LESTER, 1970; FOWLER and MORRIS, 1977; MOSSELER et al., 1991).

The Growth Simulator

An overview

The growth simulator predicts for individual trees in a regularly spaced and even-aged plantation their stem volume (v_t) in year t from: i) the 'known' volume of the tree in the previous year (v_{t-1}), ii) its maximum growth rate given the size in the previous year (v_{t-1}), and iii) the 'vigor' (δ_{t-1}) of the tree at the end of the preceding year (t-1). A random component is added to these deterministic predictions. Tree mortality is a function of a tree's relative size, its vigor, and chance (random) events. Relatively small trees with a low vigor have a higher mortality rate than relatively larger and more vigorous trees.

Through the dynamic structure of the growth simulator it is clear that random fluctuations are integrated into and change

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the growth trajectories of individuals. A random effect in 1 year changes the size of the tree and thus its growth predictions for the next year, and so on. Random growth component are made up of additive genetic and an environmental contributions. The relative strength of the 2 components is governed by the heritability of growth vigor.

Tree vigor, a measure of how close a tree is to its maximum growth rate (RGR_{max}), is clearly a pivotal part of the growth simulator. The maximum growth rate is a ceiling on relative growth rates that no tree can exceed. Trees that grow at the maximum rate have a vigor of 1 while trees that do not grow at all have a vigor of 0 and all others have intermediate vigor ($0 \leq \delta \leq 1$). A tree's vigor (δ) is determined by its relative size (s) in the population, the overall population density (RDI), its genetic worth (g), and a residual random component u (reflecting the variation in tree growth that is not explained solely by current size and genetic worth). Tree vigor is promoted by low population densities and a prominent ranking in the size hierarchy, and reduced by high population densities and low ranking.

Competition effects are implicit in the growth model through the combined effects of the relative density index (RDI) and a tree's relative size (s) on vigor. There is no explicit allowance for localized competition effects. Hence, growth predictions are independent of local spatial patterns. At first this may appear to be an unrealistic simplification, especially when competition intrinsically act at the local level (AARSSEN, 1992; HARA, 1984; LAMBERS and POORTER, 1992). However, our interest lies in average population parameters obtained through several simulations of plantation growth trajectories, and not in local deviations. As long as the competition process is 'correct' for the average situation the obtained results will reflect realistic trends in the means. Hence, a tree's relative size will, in effect, mirror its average competitive situation and thus eliminate the need for a detailed specification of the local competitive environment.

The genetic worth (g) of an individual tree is assumed to act in an additive way on vigor. Genetic worth is, furthermore, assumed constant throughout the life of a tree, at least on a standardized scale, with a mean of zero and a variance of one. The magnitude of the genetic influence on growth vigor (δ), and thus growth via relative growth rates, was controlled by the heritability of vigor (h^2_δ). Heritabilities of 0 (no genetic effect), 0.1, 0.3, and 0.5 were imposed on the simulations in order to evaluate the effects of a weak (h^2_δ=0.1), a moderate (h^2_δ=0.3), and a strong (h^2_δ=0.5) genetic control of vigor (relative growth rate). Contributions to stem volume growth attributed to the genetic worth of an individual tree was added up through an entire rotation as the difference between the growth with the designated genetic worth and the growth corresponding to a genetic worth of zero. Knowledge of the genetic contributions to volume growth allows the calculations of both apparent and true heritability of stem volume growth.

After one rotation (i.e., 150 years) of simulated growth those trees remaining in the population were used as 'parents' for a second generation of growth simulations. The average genetic value of the trees left at the end of the first generation became the average genetic worth $\bar{g}_{150}$ of the second rotation. Growth, yield, and mortality in this 'improved' second generation population was generated and followed in the same way as the parental population.

The use of benchmark growth rates (RGR_{max}) and vigor (δ) in the growth simulator facilitated the formulation of a fairly simple model of the growth process (MAGNUSSEN and BRAND, 1989) that bypassed the complexities surrounding the complex shifts in the relationship between tree size and relative growth

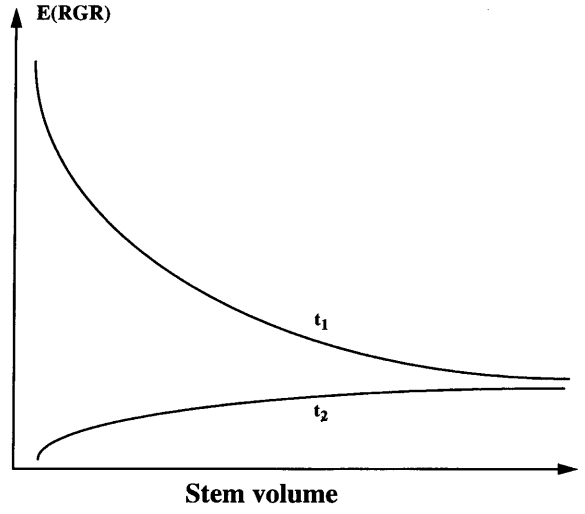

Figure 1. – Relationship between tree size (volume) and expected relative growth rate (E(RGR)) before (t_1) and after (t_2) the onset of intertree competition.

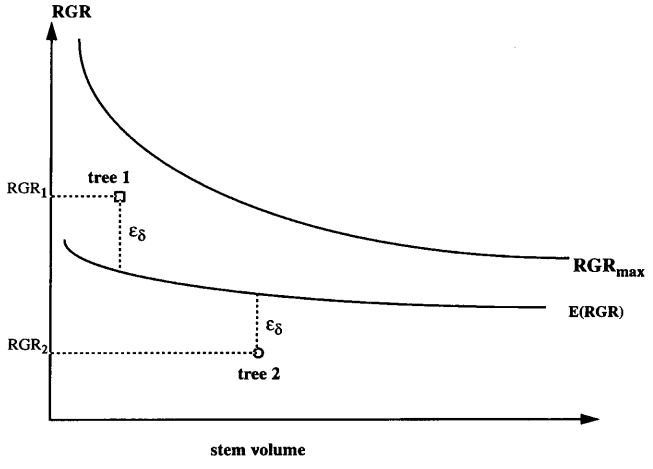

Figure 2. – Maximum (RGR_{max}) and expected (average) relative growth (E(RGR)) of all trees in a population. $E(RGR) = RGR_{max} \bullet \delta^0$ where δ^0 is the expected (population) average vigor (see text for further details). Actual relative growth rates of 2 trees (1 and 2) are denoted by RGR_1, and RGR_2, respectively. Deviations of these relative growth rates from the expected rates (ε_δ) are due to environmental and genetic effects (see text).

rate that occurs over time and in response to competition (PERRY, 1985). *Figure 1* illustrates how the relationship between tree size and relative growth rate can change over time while *figure 2* outlines the relationship between the maximum growth rate (RGR_{max}), the expected growth rate (no random effects), and the realized growth rate (including stochastic random effects).

Growth simulations are initialized at time t_0 (here age 3) by assigning each tree with a volume (v_0) drawn at random from a known distribution, and they are completed at age t = 150 years (rotation age). To study the effect of initial plantation spacing, the simulations were carried out with an initial tree population of 400 trees distributed over an area defined by a square spacing of either 1 m x 1 m, 2.0 m x 2.0 m, or 3 m x 3 m, which corresponds to densities of 10 000, 2 500, and 1 111 trees per hectare, respectively (these spacing are all represented in the data source, MAGNUSSEN and BRAND, 1989). Presented population results are means of 9 independent repetitions of 2

full rotations (150 years per rotation) of growth simulations. A brief outline of the simulation approach is given next. Further details on derivations are found in MAGNUSSEN and BRAND (1989).

A Stochastic Growth Model for Individual Trees

To prorate a tree's volume from year *t-1* to year *t* the following model was used:

$$v_t = v_{t-1} \times [1 + \delta_{t-1} \times RGR_{max}(v_{t-1})] \qquad (1)$$

where RGR_{max} is the maximum possible relative growth rate (growth per unit size, $0 \le RGR_{max} \le 1$) of a tree of size v_{t-1}, and δ_{t-1} is tree vigor ($0 \le \delta_{t-1} \le 1$). Vigor times the maximum growth rate is equal to the actual (realized) growth rate (RGR). An estimate of RGR_{max} was obtained from (2):

$$RGR_{max}(v_{t-1}) = \begin{cases} 0.94 - 57.39 \times v_{t-1} \text{ for } v_{t-1} < 0.01m^3 \\ exp(0.4188 - 0.1251 \times \log v_{t-1}) \text{ for } v_{t-1} \ge 0.01m^3 \end{cases} \qquad (2)$$

The general trend in the relationship between RGR_{max} and tree size (volume) is depicted in *figure 2*.

The growth vigor (δ) of an individual tree was determined as the sum of the expected vigor (δ^0) and a random component (ε_δ). Equations 3 to 9 provide the details (age subscripts have been omitted for convenience).

$$\delta = \delta^0 + \varepsilon_\delta \qquad (3)$$

where δ^0 is the expected growth vigor of a tree and ε_δ is the stochastic deviation due to individual environmental (u_δ) and genetic (g) effects. The expected vigor (δ^0) of a tree was determined via:

$$\delta^0 = a \times (1 - exp(b \times s)) \qquad (4)$$

where a and b are the parameters of 2-sided and 1-sided competition, respectively (BRAND and MAGNUSSEN, 1988; MAGNUSSEN and BRAND, 1989). Equations 8 and 9 show how a and b were determined. s in (4) is the relative size of the tree.

$$s = \frac{v}{\left(\bar{v} - \frac{v_{min}}{2}\right)} \qquad (5)$$

where $\bar{v}$ is the mean tree volume of all live trees in the population, and v_{min} is the minimum tree volume in the population.

The stochastic deviation of an individual's growth vigor, ε_δ in (3), from the expected value (δ^0), was computed as the weighted sum of 2 standardized (mean 0 and variance 1) random normal variates (u_δ for the environmental chance events, and g for the additive genetic effects) multiplied by the standard deviation of the expected growth vigor (σ_δ) at the appropriate age. Weights were determined by the heritability of growth vigor ($h^2_\delta = \sigma^2(g)/(\sigma^2(g) + \sigma^2(u_\delta))$. Specifically we have:

$$\varepsilon_\delta = \sigma_\delta \times \left(\sqrt{h^2_\delta} \times g + \sqrt{\left(1 - h^2_\delta\right)} \times u_\delta\right) \qquad (6)$$

When computed this way the standard deviation of ε_δ is equal to σ_δ, and the heritability of growth vigor is equal to the chosen value of h^2_δ (RIPLEY, 1987).

The standard deviation of growth vigor (σ_δ) was, in turn, a function of the overall population density index (*RDI*). Using the RDI of SMITH and HANN (1986) we get:

$$\sigma_\delta = 0.062 - 0.024 \times RDI \qquad (7)$$

$$\text{with } RDI = \bar{v} \times exp(-10.077 + 1.47 \times \log N)$$

where N is the number trees per hectare (alive at the particular year), and $\bar{v}$ is the average stem volume in the population ($0 \le RDI \le 1$).

To estimate the expected vigor of an individual tree and to complete the growth model we need to quantify time trends in the 2-sided competition parameter a, and the 1-sided equivalent b (see equation 4):

$$a = a^0 + \varepsilon_a(t) + \varepsilon_a(population) \qquad (8)$$

$$b = b^0 + \varepsilon_b(t) + \varepsilon_b(population)$$

where a^0 and b^0 are the expected population parameters, and the ε's are stochastic deviations from the expected population average due to annual environmental effects that impacts on the entire population ($\varepsilon(t)$), and those that are associated with the particular site and population ($\varepsilon(population)$). The latter is assumed constant throughout a simulated rotation. Appendix I provides computational details on the interrelationships and temporal correlations of the stochastic deviations (ε) in (9).

Estimates of a^0 and b^0 came from (MAGNUSSEN and BRAND, 1989):

$$a^0 = 0.5 \times \left(1 - exp\left(-\frac{1}{RDI}\right)\right) \qquad (9)$$

$$b^0 = 10 \times exp\left(-\frac{4}{RDI}\right)$$

Time trends in the 2 competition parameters are given in *figures 8* and *9*. When a decreases there is a decline in the vigor of all trees (see 4), and vice versa. A decline in b lowers the vigor of a tree and vice versa; a trend more pronounced in relatively smaller trees than in relatively bigger trees (see 5).

Trees with low vigor were more likely to die than trees with high vigor. Mortality in the growth simulations was triggered by a stochastic function of vigor. The probability (p_{mort}) that a tree would die in a given year was modelled as a linear function of the *logarithm* of its vigor (δ):

$$p_{mort} = \begin{cases} -0.003505 - 0.011569 \times log(\delta) \text{ for } \delta \le 0.74 \\ 0 \text{ for } \delta > 0.74 \end{cases} \qquad (10)$$

Death of individual trees was then triggered when p_{mort} was greater than a 'trigger' probability p_{random} drawn at random from a uniform distribution on the interval [0,1]. Any tree that was given a zero or negative relative growth rate in any given year was also eliminated (this happened in less than 0.01% of the assigned growth rates). Eliminating trees this way ensured that the population density, as measured by RDI, stayed below a ceiling value not exceeded in actual plantations (*ibid.*). *Figures 3* and *6* illustrate the outcome of this self-thinning process.

To start the growth simulations, a set of initial (age 3) stem volumes (v_o) were generated via the algorithm:

$$v_o = \bar{v}_o + \sigma_{v_o} \times \left(\sqrt{\left(1 - h^2_\delta\right)} \times u_{v_o} + \sqrt{h^2_\delta} \times g\right) \qquad (11)$$

where $\bar{v}_o$ is the mean initial volume of the population (here 0.0013 m^3) and σ_{v_o} is the initial phenotypic population standard deviation of stem volume (here 0.0005 m^3). h^2_δ is the heritability of vigor (δ). u_{v_o} and g are random normal distributed parameters with a mean of zero and a variance of one representing the stochastic variation in initial stem volume due to environmental (u) and additive genetic effects (g), respectively. g-values assigned to individual trees were kept constant throughout a rotation. From (11) one obtains that all the variation is due to the environment if and only if h^2_δ is zero or, conversely, due entirely to genetic effects if and only if $h^2_\delta = 1$. Trees that by chance were assigned a negative initial volume (less than 0.5%) were regarded as initial losses and counted as dead in the population tallies.

Estimating apparent and 'true' heritability of tree volume size

Direct genetic input to the growth simulator was limited to the relative growth rate via its role in determining vigor (δ). Genetic influence on volume growth was governed by the relationship between relative growth rate and volume growth. Stochastic events and dynamic feedback mask this relationship. To see how a known genetic control over relative growth rates (via h^2_δ) is carried forward to the actual volume accruals the heritability of volume growth was estimated. By tracking and comparing the volume growths of individual trees with and without a genetic input, the 'true' heritability estimate of stem volume at any given age could be estimated as the following ratio of variances:

$$h^2_{true}\left(vol_t\right) = var\sum_{t_0}^{t}\left(v_{t|g=g} - v_{t|g=0}\right) / var\left(v_{t|g=g}\right) \qquad (12)$$

The true heritability is purely of academic interest because it cannot be estimated in practice. Only an apparent heritability estimate ($h^2_{app.}\ (vol)$) of stem volume can be obtained from, say, a clonal test. The apparent heritability is derived from the covariance between the volume of a tree with genetic value g and the volume the tree would have had with a genetic value of 0 divided by the phenotypic variance of stem volume (at any given year). In the absence of dynamic feedback in the volume growth simulator (i.e., lag effects) which generates apparent 'genotype x environment' effects, this type of estimator ($h^2_{app.}\ (vol)$) would be identical to $1\text{-}h^2_{true}\ (vol)$ (see 12). Given the dynamic feedback in the growth model it is of interest to gauge how much a heritability estimate of stem volume growth can be inflated by genotype x environment effects (includes interaction between genetic value and direct and indirect competition effects, as well as lagged environmental and genetic effects). A comparison of h^2_{true} with h^2_{app} will gauge the importance of genotype x environment inflation of apparent heritability. Apparent ($h^2_{app.}\ (vol)$) heritability of stem volume was defined as:

$$h^2_{app}\left(vol_t\right) \equiv 1 - \frac{cov\left[v_{t|g=g}, v_{t|g=0\ \forall\ t' \le t}\right]}{var\left[v_{t|g=g}\right]} \qquad (13)$$

where $v_{t|g=g}$ is the volume of a tree at time t given its genotypic value g (i.e., the variance of $v_{t|g=g}$ is equal to the total phenotypic variance of volume at time t). $v_{t|g=0\ \forall\ t \le t}$ is the volume the tree would have had at time t if its genetic value g had been zero throughout (computed as the sum of annual increments and initial stem volume with $g=0$ at each step). In absence of any genotype x environment interaction, the numerator in (13) would estimate the genetic variance associated with volume and thus (13) would estimate the apparent heritability of stem volume.

Results

General growth and yield predictions

To better appreciate and understand the results with genetic implications, an overview is provided in *figures 3* to *12* of the most pertinent growth and yield results at the stand level. All results are the averages of nine simulations of growth and yield of 400 trees throughout two rotations. MAGNUSSEN and BRAND (1989) have published further details.

Figure 3 depicts the time trends in the number of live stems per hectare. All declines are due to mortality (self-thinning). Self-thinning was, as expected (WEINER and THOMAS, 1985; WELLER, 1987), strongest in the 1 m x 1 m spacing and weakest in the 3 m x 3 m spacing. Self-thinning was especially strong during the first 80 years in the 1 m x 1 m spacing. Both relative and absolute differences between spacings in the number of live trees per hectare diminished with age.

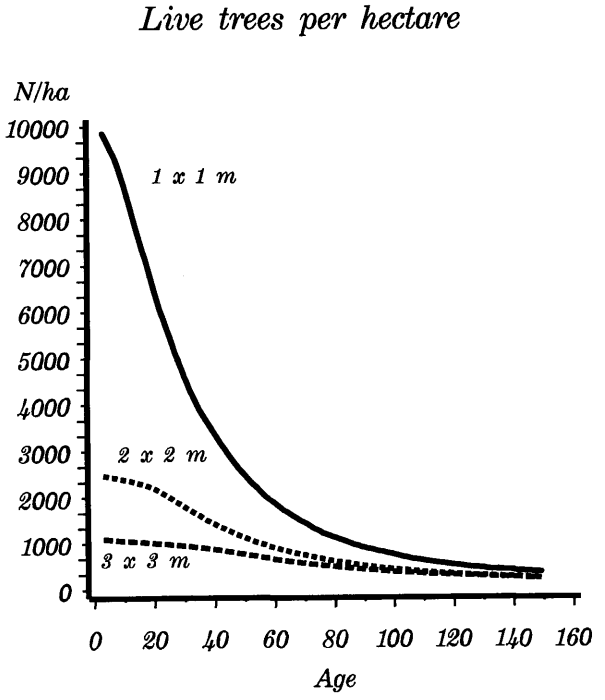

Figure 3. – Number of live trees per hectare (N/ha) at various ages and 3 levels of initial spacing (1 m x 1 m, 2 m x 2 m, and 3 m x 3 m).

Wider spacings resulted in markedly greater mean stem volumes (*Figure 4*), in agreement with other spacing trials with forest tree species (JØRGENSEN, 1967). At age 100 the mean stem volume at spacings 1 m x 1 m, 2.0 m x 2.0 m, and 3 m x 3 m was 0.8 m^3, 1.3 m^3, and 2.0 m^3, respectively. Density-induced mean volume differences were, by and large, established and maintained from age 80 years and onwards. Wider spacings reduced, on a relative scale, the within-stand variation of stem volume (*Figure 5*). In the widest spacing the coefficient of variation decreased from 37% at age 3 to about 15% at age 150 while an initial (age 3 to 60) increase followed by a

decrease was found in the intermediate and the narrow spacings. In absolute terms, the standard deviation of stem volume increased with spacings (BRAND and MAGNUSSEN, 1988; MAGNUSSEN and BRAND, 1989).

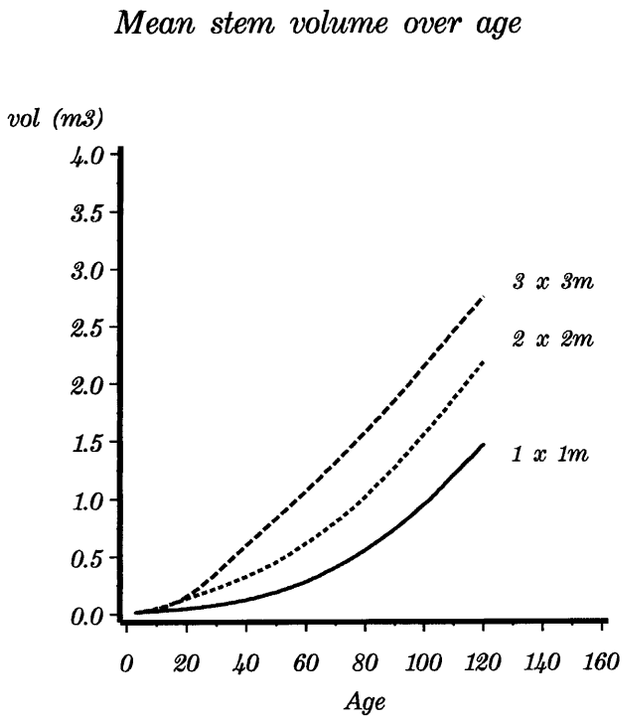

Figure 4. – Mean stem volume at various ages and 3 levels of initial spacing (1 m x 1 m, 2 m x 2 m, and 3 m x 3 m).

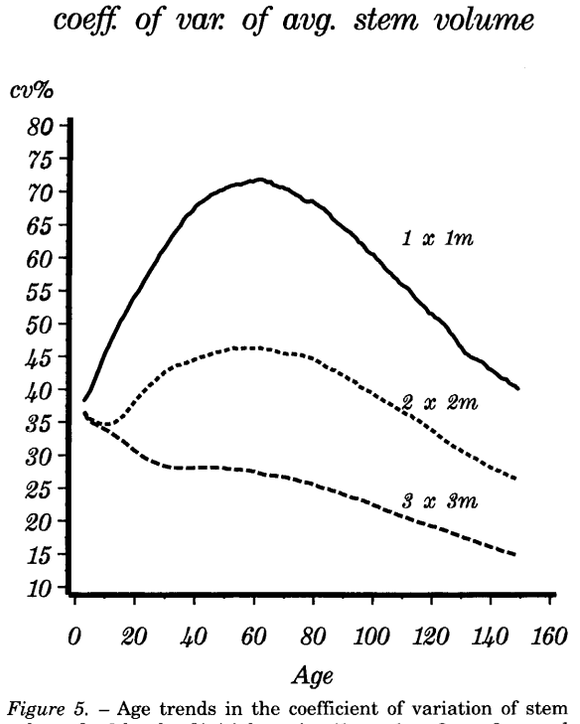

Figure 5. – Age trends in the coefficient of variation of stem volume for 3 levels of initial spacing (1 m x 1 m, 2 m x 2 m, and 3 m x 3 m). CV% = standard deviation/mean • 100.

Further insight into the dynamics of the growth model is gained from *figure 6* which illustrates the relationship between the number of live stems per hectare and the mean stem volume in a log-log scaled diagram. In coniferous stands with density induced self-thinning one would expect this relationship to form a single linear 'ceiling' with a slope of about –0.67 (for example, WELLER, 1987 and ZEIDE, 1987). Prior to the density induced self-thinning the mean stem volume expands without a concomitant decline in the number of live trees (horizontal parts of trend lines in spacings 2.0 m and 3 m). Once self-thinning commences in the various spacings (later with increased spacing) the log-stems to log-volume relationships converge to a single straight line with a slope of –0.74, not far from the aforementioned 'global' expectation. Note, that the model predictions arrive at this self-thinning line based on an elimination process based on vigor, and not an implicit adherence to the '3/2 power law of self-thinning' (*ibid.*) as seen in other models (for example, SMITH and HANN, 1986). Overall, the model predictions of stem numbers, mean volume, and volume variation is deemed realistic for conifers in the temperate zone (ASSMANN, 1970).

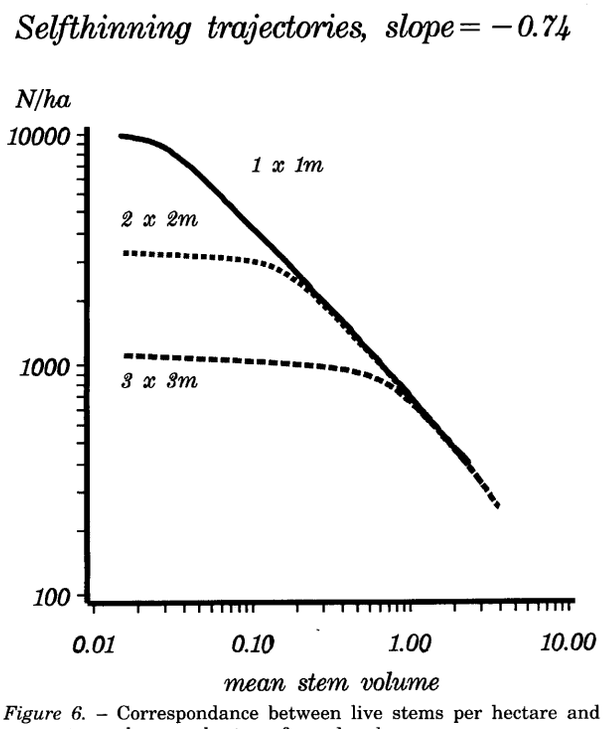

Figure 6. – Correspondance between live stems per hectare and mean stem volume on *log* transformed scales.

Stand density as quantified by the relative density index (RDI, SMITH and HANN, 1986) is central to the growth and yield predictions via its influence on the 2 growth modifying parameters '*a*' (2-sided competition effects) and '*b*' (1-sided competition effects). *Figures 7, 8,* and *9* illustrate time trends of RDI in the 3 spacings. There was a rapid juvenile increase in RDI followed by a relatively slow decline after the onset of self-thinning. Trends in the '*a*' and '*b*' parameters (*Figures 8* and *9*) follow directly from the functional dependence on RDI. Again, we see that the biggest drop in these parameters, and hence growth vigor, occurs during the initial phase of density competition. After the peak in density competition (approximately at age 20 in the 1 m x 1 m spacing, and ages 40 and 60 in the 2.0 m and 3 m spacings, respectively) both '*a*' and '*b*' recovers somewhat during the remainder of the rotation.

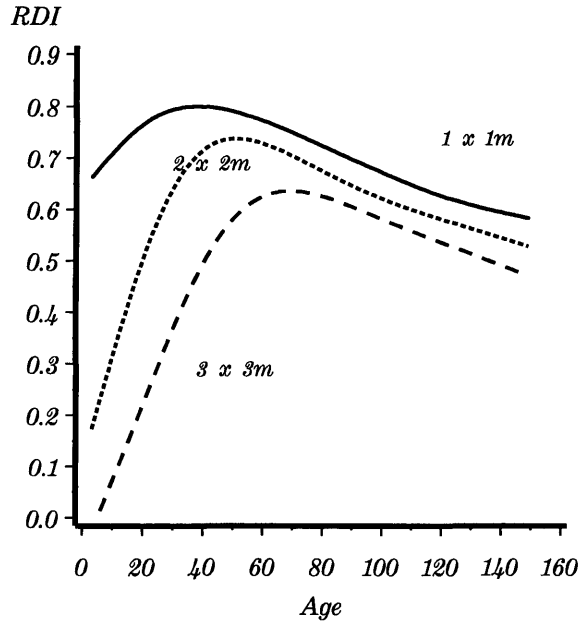

Figure 7. – Relative density index (RDI) at various ages (see text for further explanation of this index) and 3 levels of initial spacing (1 m x 1 m, 2 m x 2 m, and 3 m x 3 m).

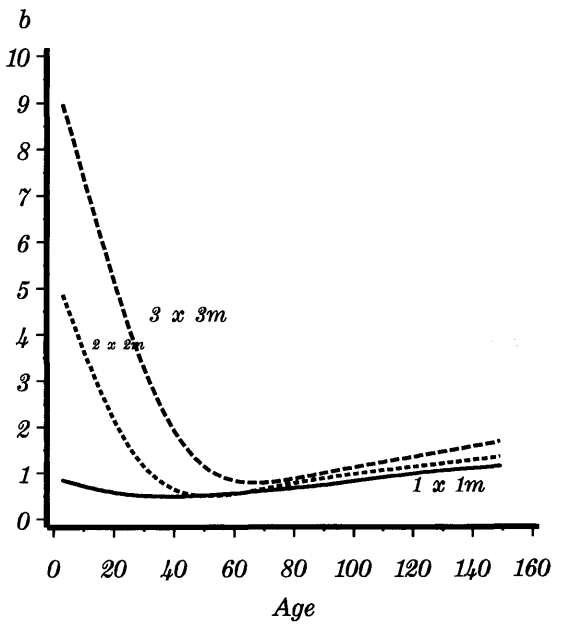

Figure 9. – Age trends in the density coefficient '*b*' of 2-sided competition (see text for further explanation of this coefficient) for 3 levels of initial spacing (1 m x 1 m, 2 m x 2 m, and 3 m x 3 m).

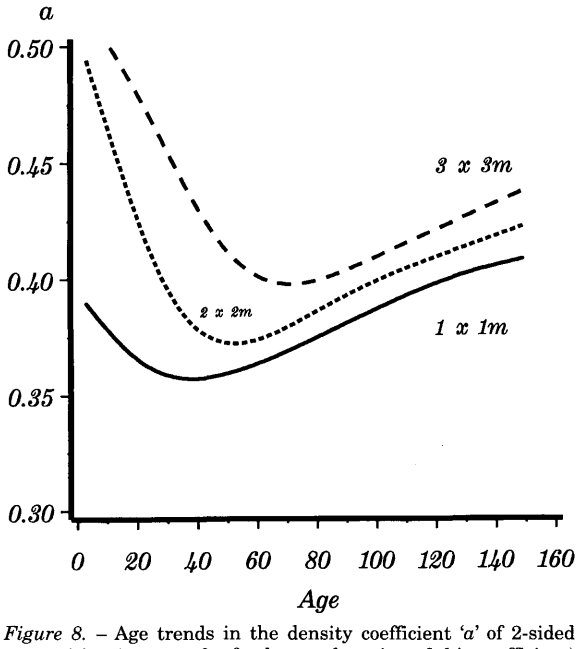

Figure 8. – Age trends in the density coefficient '*a*' of 2-sided competition (see text for further explanation of this coefficient) for 3 levels of initial spacing (1 m x 1 m, 2 m x 2 m, and 3 m x 3 m).

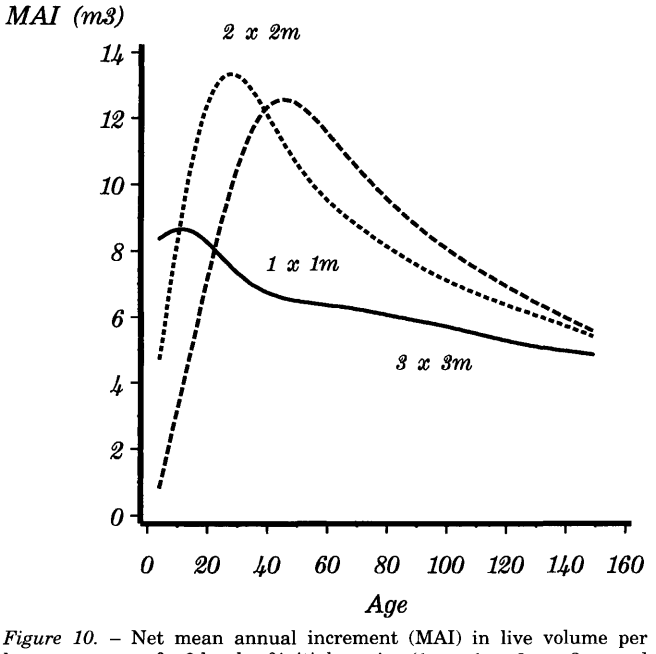

Figure 10. – Net mean annual increment (MAI) in live volume per hectare over age for 3 levels of initial spacing (1 m x 1 m, 2 m x 2 m, and 3 m x 3 m).

Mean annual increment (MAI) of survivors (net growth) followed the typical growth pattern of conifers in even-aged plantations (ASSMANN, 1970) characterized by a steep juvenile increase followed by a steady decline (*Figure 10*). For the entire rotation MAI was around 6 m^3 per hectare and per year in the 2 widest spacings compared to 5 m^3 in the narrow spacing. Density losses and suppressed growth are the main causes for this deficit. Peak values of MAI were reached around age 15 in the 1 m x 1 m spacing (maximum MAI of 9.9 m^3/ha and per year), around age 25 in the 2.0 m x 2.0 m spacing (maximum MAI of 14.3 m^3/ha and per year), and around age 50 in the 3 m x 3 m spacing (maximum MAI of 12.7 m^3/ ha and per year). Results of similar nature are commonly reported from various spacing trials with conifers in even aged plantations (JØRGENSEN, 1967).

Figures 11 and *12* complete the overview of general growth and yield results by showing time trends in the relative growth rate (RGR), and the average relationship between RGR and mean stem volume, respectively. Note the reversal in the ranking of RGR in the three spacings that occurs between ages

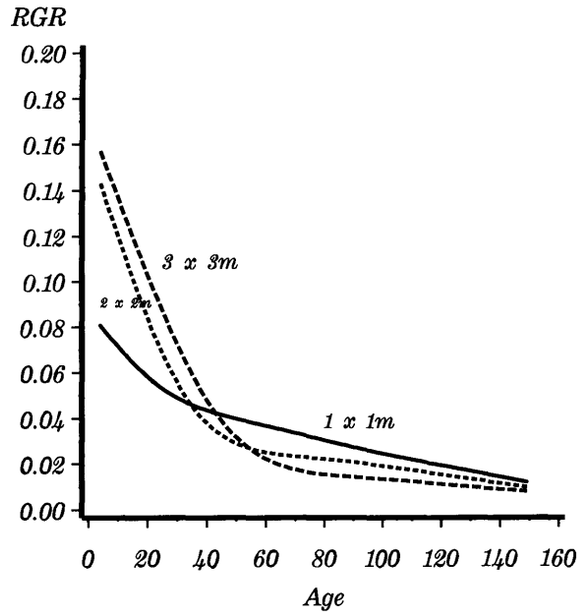

Figure 11. – Relative growth rate (RGR = volume increment per unit volume) over age for 3 levels of initial spacing (1 m x 1 m, 2 m x 2 m, and 3 m x 3 m).

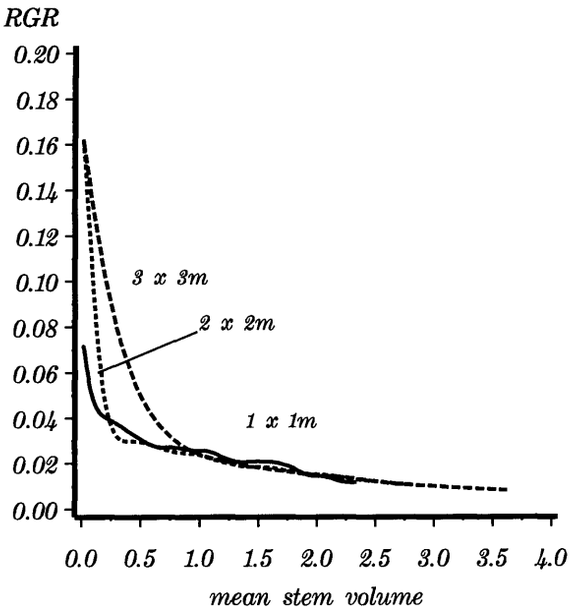

Figure 12. – Relative growth rate (RGR = volume increment per unit volume) versus mean stem volume for 3 levels of initial spacing (1 m x 1 m, 2 m x 2 m, and 3 m x 3 m).

20 and 50. In the juvenile stage RGR increases as the initial spacing goes up in accordance with the levels of experienced competition; later, however, the increasing differences in mean stem size and the negative correlation between tree size and RGR increasingly dominates the relationship which ultimately leads to a reversal in ranking. RGR versus mean stem volume is plotted in *figure 12* where a common trend line is apparent once the spacings follow the same self-thinning trajectory, which they do once the mean stem volume is approximately 1 m^3. Before that, competition pressures lowered the RGR, for a given stem size most in the 1 m x 1 m spacing, and least in the 3 m x 3 m spacing.

Genetic effects in growth and yield predictions

As outlined above, introducing genetic effects into the calculations of relative growth rates in the model did not change the stand level growth and yield results. The only change was a unique linkage between a tree's phenotype and its 'genotype', which makes it possible to account for the significance of a 'genotypic' value g on volume growth. As well, the linkage enables the calculation of a suite of quantitative genetic parameters.

Trees remaining at the end of the first rotation had a higher average genetic value (g) of vigor than the initial population average (= 0) at age 3. A measure of how efficient the simulated mortality process (a mix of density dependent and random mortality) was in selecting for trees with a high growth potential was obtained by dividing the mean genetic vigor of trees with the implied selection intensity. Because the variance of genetic vigor was held constant at 1.0 throughout, the measure of selection efficiency is, in effect, an estimate of the realized heritability of growth vigor (FALCONER, 1981). *Figure 13* illustrates how this retrospective selection efficiency for growth vigor ($\hat{h}^2_{vigor}$) changes with age, spacing, and the imposed (simulation input) heritability of vigor (h^2_δ). Apart from a brief initial juvenile phase where mortality is relatively independent of genetic worth (g) the selection for vigor became increasingly efficient with age. A narrow spacing promoted this efficiency to a larger extent than a wider spacing. As a rule, an increase in spacing of about 1 m decreased the selection efficiency by about 0.1 in stands older than 10 years. Self-thinning is consequently an efficient way of screening trees for genetic vigor.

The mean genetic value of vigor (δ) of trees remaining at the end of a simulated rotation was used as initial mean population genetic average in the subsequent rotation of growth simulations. Relative differences between results obtained in the second generation and those of the first generation are considered to be genetic gains arising from a selection against

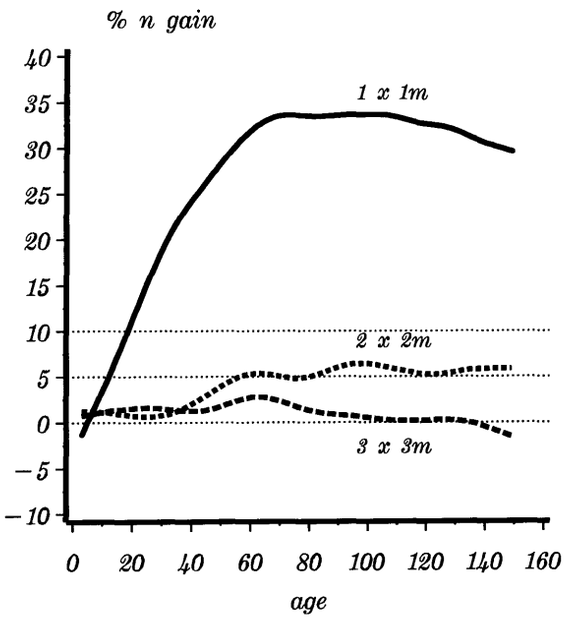

Figure 13. – Predicted relative genetic gain (gain%) in number of live stems per hectare at 3 levels of spacing after 1 generation of selection and breeding. $h^2_\delta = 0.5$.

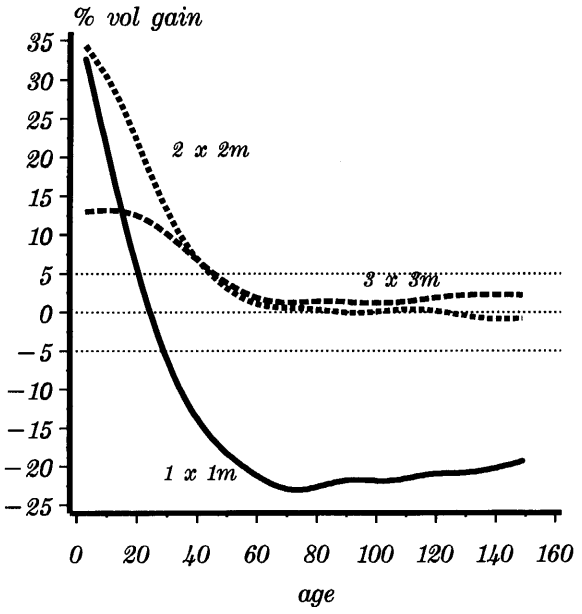

Figure 14. – Predicted relative genetic gain (gain%) in mean stem volume at 3 levels of spacing after 1 generation of selection and breeding. $h^2_\delta = 0.5$.

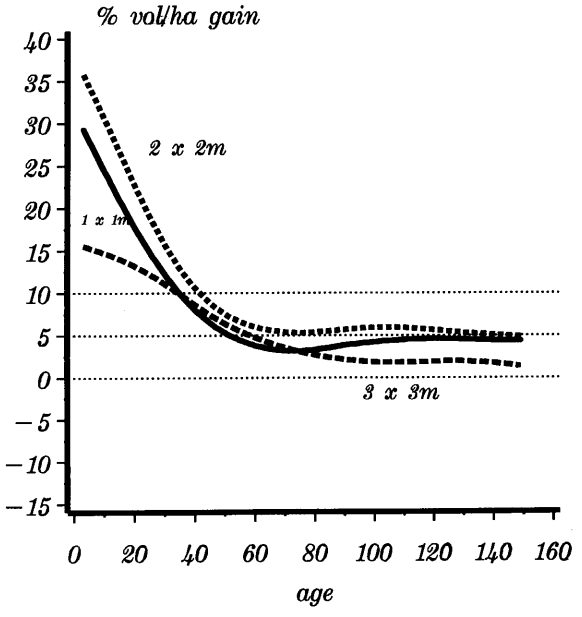

Figure 15. – Predicted relative genetic gain (gain%) in total stem volume per hectare at 3 levels of spacing after 1 generation of selection and breeding. $h^2_\delta = 0.5$.

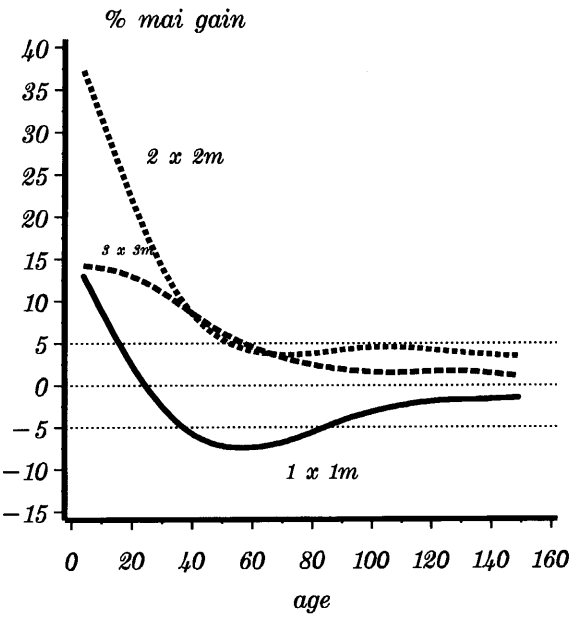

Figure 16. – Predicted relative genetic gain (gain%) in mean annual increment (MAI) at 3 levels of spacing after 1 generation of selection and breeding. $h^2_\delta = 0.5$.

weak growth rates. The magnitude of the gain depends on the intensity of the selection (FALCONER, 1981). For the 3 spacings the intensity of the selection for high growth rates was 2.3 in the 1 m spacing, 2.0 in the 2.0 m spacing, and 1.4 in the 3 m spacing. Genetic gains (in % of first generation results) in stem numbers, mean volume, volume per unit area, and mean annual increment are plotted over age in *figures 14* to *17* for the case of $h^2_\delta = 0.5$.

Because trees at the end of a rotation had, on average, a positive genetic value of vigor, trees in the second generation will, for a given tree size, everything else being equal, have a higher vigor (and thus RGR) than trees in the first generation. The higher vigor in the second generation lowered mortality compared to the first generation. Growth in the second generation, on the other hand, could be lower or higher than the corresponding growth figure of the first generation depending on the densities at the age in question.

A higher vigor in the second generation leads to a significantly higher number of live trees in the 1 m spacing, whereas the number remained more or less the same (± 5%) across generations in the 2.0 m and 3 m spacings (*Figure 14*). In the 1 m x 1 m spacing the second generation gain in numbers of live trees climbed to about 33% during the first 60 years whereafter it hovered around this level (± 7%). More trees per hectare translates into a higher density and thus an increase in density-stress and subsequent growth depression, as outlined next.

Mean stem volumes (*Figure 15*) during the first 20 years of the second generation were from 5% to 35% higher than in the first generation but, as competition intensified, this gain vanished or turned into a loss. In later years (age > 20) the mean stem volume in the 1 m spacing was actually predicted to be about 25% less than in the first generation, a direct consequence of the higher number of live trees. Mean stem volume in stands 40 years or older and planted at 2.0 m or 3.0 m square spacings was about the same (± 5%) as in the first generation simulations. Total volume per hectare (*Figure 16*) was at first (age < 30 years) much improved in the second generation (10% to 38%) but, during the second half of the rotation (age > 75), the gain dropped to about 5%. Second generation superiority in volume per hectare was most marked during the juvenile phase in the narrow and intermediate spacings, as would be expected from the aforementioned trends in gains of survival and average stem volume.

Gains in mean annual increments of survivors (*Figure 17*) were considerable (> 10%) during the first 5 to 30 years of the second generation. Later, however, these gains not only disappeared in the 1 m spacing but they turned into a net decrease at older ages due to the increased density stress created by the gain in live stems per hectare. In the 2.0 m and 3 m spacings the net improvement in MAI stabilized around 5% for stands 60 years or older.

Of foremost interest in this study is the estimation of apparent ($h^2_{app.}$ *(vol.))* and 'true' heritability (h^2_{true} *(vol.))* of stem volume size. Apparent heritability would be the only estimate available in practice. By comparing the two estimates, it can be learned how reliable the apparent heritability is for estimating the potential for additive genetic gains in volume growth.

Stem volume at any given age is the integration of past growth. As such, it is expected to be the result of direct and indirect (past) effects of the genotype, the environment, and interactions thereof. The apparent heritability includes genotype x environment interactions and autocorrelation in the growth process (current growth is influenced by previous growth). True heritability, on the other hand, isolates the main effect of the additive genetic value. Any genotype x environment and autocorrelation effect will make the apparent heritability higher than the true heritability and thereby inflate gain expectations based on apparent heritability (FALCONER, 1981).

Time trends in the apparent heritability of stem volume are shown in *figure 18* for the 3 levels of spacing and the 3 levels of imposed heritability of vigor (δ). At age 3, when the trees are given their initial size, the apparent heritability equals the imposed (true) heritability, but at any later age, the apparent heritabilities becomes inflated due to the recursive dynamics of growth that generates genotype x environment interaction. The inflation was substantial and significant ($P < 0.01$, t-test) already after five years of growth. In the 1 m x 1 m spacing the apparent heritability peaked around age 80 years, when it was about 0.4 above the nominal level of the imposed vigor heritability. The positive 'bias' of the heritability is cogent given the dynamics of tree growth and the positive feedback of competi-

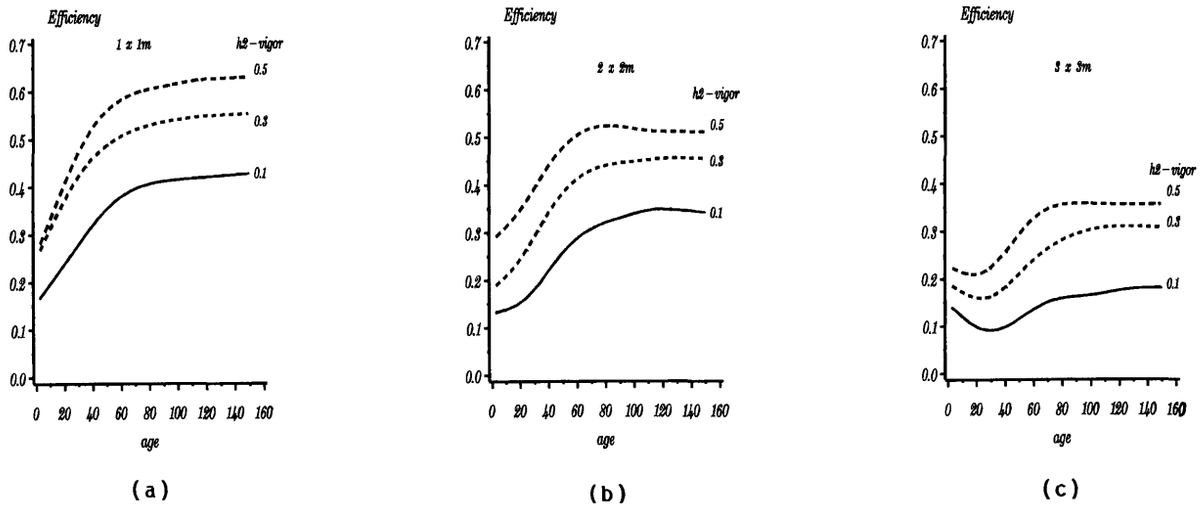

Figure 17. – Predicted selection efficiency of genetic vigor ($\hat{h}^2_{vigor}$) plotted against age for 3 levels of spacing and 3 levels of imposed heritability of vigor (h^2_δ). a) spacing = 1 m x 1 m, b) spacing = 2 m x 2 m, c) spacing = 3 m x 3 m.

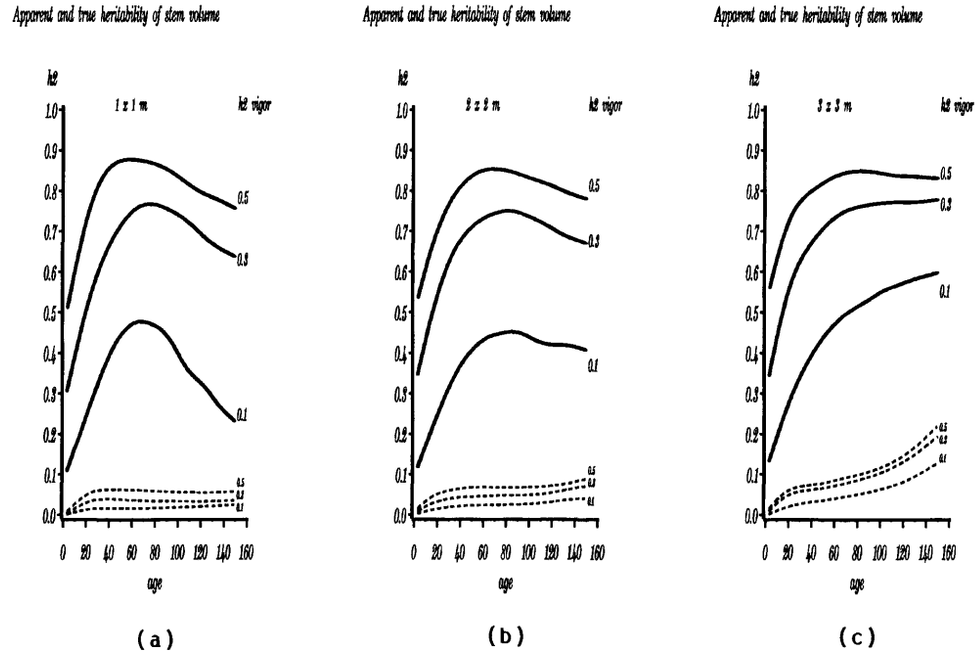

Figure 18. – Age trends in estimated heritability of stem volume for 3 levels of imposed heritability on vigor (h^2_δ). Full lines: apparent heritability $h^2_{app.}$*(vol);* dashed lines: true heritability h^2_{true}*(vol)).*
(a) spacing = 1 m x 1 m, b) spacing = 2 m x 2 m, c) spacing = 3 m x 3 m.

tive advantages (big trees 'usurp' smaller trees). At rotation age the apparent heritabilities were inflated by approximately 0.1 for h^2_δ=0.1, 0.3 for h^2_δ=0.3, and 0.2 for h^2_δ=0.5. The onset of a decline in the apparent heritability, seen in the data from the 1 m x 1 m spacing around age 50, is probably due to: i) a moderation of the density stress (cf. trends in RDI, '*a*', and '*b*') which allows smaller trees to enjoy a higher relative growth rate (size effect) and thus, in a relative sense, 'catch up' with the bigger trees in the stand (cf. trend in CV% in *Figure 5*), and ii) the increasingly random character of tree mortality. In the 2.0 m x 2.0 m and the 3 m x 3 m spacings the positive bias in the apparent heritability of stem volume either increased with age or reached a plateau around age 50. Hence, smaller trees in these wider spacing were not 'catching up' in a relative sense with their larger counterparts; rather, big trees maintained or increased their lead during the entire rotation.

The 'true' heritability ($h^2_{true}(vol.)$) of stem volume was computed as the variance of the stem volume that could be attributed to additive genetic effects on tree vigor divided by the total phenotypic variance. True heritability estimates were always much smaller than the estimates of the apparent heritability. Differences between the two heritability estimates increased with decreasing spacing. Starting close to zero at age 3, when almost all the variance of stem volume is due to size (cf. the exponential character of the RGR versus stem volume relationship), the actual heritability continued to increase at a slow rate throughout the entire rotation (*Figure 18*). In the narrow spacing the true heritabilities reached levels of 0.02 for h^2_δ=0.1, 0.04 for h^2_δ=0.3, and 0.06 for h^2_δ=0.5. The true volume heritabilities in the 2.0 m spacing reached levels at the end of the rotation that were approximately 0.04 higher than in the tightest spacing. Final heritabilities in the 3 m spacing were 0.13 for h^2_δ=0.1, 0.20 for h^2_δ=0.3, and 0.22 for h^2_δ=0.5. Consequently, apparent heritabilities were inflated by a factor 10 to 15 in the 1 m x 1 m spacing, a factor of 8 to 10 in the 2.0 m spacing, and a factor of 3 to 5 in the 3 m x 3 m spacing. Comparable inflation rates of stem volume have been reported in a juvenile jack pine (*Pinus banksiana* LAMB.) progeny test with a narrow (0.5 m x 0.5 m) spacing (MAGNUSSEN, 1989b).

Discussion and Conclusions

Any mathematically formulated growth model is only a symbolic representation of a much more complex reality (HUNT, 1982). A simple recursive growth model, whose parameters change in a dynamic fashion in response to the population density, and the predicted growth vigor of individual trees, could, when combined with a stochastic mortality function, 'mimic' the quantitative development of a forest stand in a realistic fashion (MAGNUSSEN and BRAND, 1989). Predictions, made with the model presented here, are believed to reflect tree growth and competition (for light) in many coniferous species quite well; at least for even aged plantations (ASSMANN, 1970). Additions of stochastic events not only enhanced the realism of the model (RIPLEY, 1987), but also made the introduction of random additive genetic effects at the individual level possible and straightforward. This facility makes the model better suited for demonstrating how imputed individual genetic effects on growth rate (vigor) translates into population effects on tree size (volume) than models driven by, say, the development of population means and postulated net genetic effects (BUFORD and BURKHART, 1987; KNOWE and FOSTER, 1989; REHFELDT et al., 1991; SPRINTZ, 1987). These types of models do not fully account for the effect of density competition, and the interactions with genetic parameters.

Alternative formulations of the 'driving' growth process and the main competition process make it possible to integrate genetic effects in various forms to suit analytical objectives. HAMILTON and REHFELDT (1994) have used a model where height growth and diameter increment were the driving parameters to explore the effect of genetic contributions to overall yields. Other models (for example, NANCE et al., 1986; and MITCHELL, 1975) would allow to explore the impact of genetic differences in competitive ability and crown developments on growth and yield. Competition for water and nutrients may lead to different competition processes, and thus different expressions of genetic effects, than those exemplified here. It is beyond the scope of this paper to deal with these situations although they may be important in certain regions.

Only an integration of all the genetic contributions to all growth components will furnish the overall significance of genetics on yield.

Genetic control of tree growth has been well documented, but the exact nature of the genetic effects can be very hard to diagnose due to the interrelationships and correlations of most quantitative growth related characters (NAMKOONG et al., 1988). For economically important traits like tree height, stem diameter, stem volume, and wood density, reported heritabilities vary greatly not only among and within species, but also between sites and years (BALOCCHI et al., 1993; FOSTER, 1986; ZOBEL and TALBERT, 1984). There is, however, growing evidence that much of this variation is caused by genotype-environment interactions in competition sensitive traits amplified by effects such as autocorrelation of growth, microsite effects, and competition (GALLAIS, 1976; MAGNUSSEN, 1993; STROUP and MULITZE, 1991; WRIGHT, 1982). When it is known that various growth processes are strongly dependent on the quality, quantity, and structure of the plant tissue (JARVIS and MCNAUGHTON, 1986; LAMBERS and POORTER, 1992; ZIMMERMANN and BROWN, 1977) then, clearly, these effects must be factored out before the additive genetic contributions and unbiased gain estimates can be ascertained. Otherwise the estimated genetic parameters will be confounded by the impact of non-genetic effects.

Further complicating the task of separating genetic and non-genetic effects are the temporal correlations in tree growth (for references see, for example, MAGNUSSEN, 1989b). Growth is dependent on current conditions which were, in part, generated in the past. The current tree size is the outcome of an exceedingly complex web of direct and indirect genetic and non-genetic effects (BONAN, 1988; LAMBERS and POORTER, 1992). Changes in the external environment (spatial and temporal) adds to this complexity. Non-additive genetic effects are likely to be more prone to the aforementioned distortions than additive effects (GALAIS, 1976; STROUP and MULITZE, 1991; WRIGHT, 1982). All said, caution is a prerequisite when interpreting quantitative tree genetic parameters of growth. Genetic comparisons of growth curve parameters (MAGNUSSEN and KREMER, 1993; MAGNUSSEN and PARK, 1991) call for special attention because the shape of a growth curve may be 'distorted' by non-genetic factors.

In the modelling approach, it was assumed that genetic effects operated exclusively on the relative growth rate (vigor), and that they remained constant for an individual on a standardized scale. This assumption is the simplest possible. Non-trivial and frequently non-linear temporal trends in genetic parameters in tree genetic trials have been explained by genotype x age interactions and growth phases affected by different genes (FRANKLIN, 1979; NAMKOONG et al., 1972). While this is plausible, it remains conjecture until we are able to peel all non-genetic effects away from these estimates. Claims of genetic effects in the competitive ability of trees have surfaced from time to time, but the effects have been either scale effects,

confounded by size, or unstable (ADAMS et al., 1973; BENTZER et al., 1988; CAMPBELL and WILSON, 1978; HÜHN, 1974; NANCE et al., 1983; TAUER, 1975; WILLIAMS et al., 1983; WUHLISCH et al., 1990). Theoretical considerations indicate that the interpretation and modelling of size independent, genetically mediated competitive abilities tend to become intractable (AARSSEN, 1992; BONAN, 1988). The entertained model is, therefore, the most parsimonious and suitable first approximation towards an understanding of how the growth dynamic interacts with the genetic components of growth in a population of trees. Extensions of the model to include localized competition effects should be straightforward.

Tree size and competition were confirmed as salient covariates that must be considered when trying to estimate heritability of stem volume for trees competing for light. Otherwise inflated heritability estimates of stem volume may be obtained (GALLAIS, 1976; MAGNUSSEN, 1989a, 1993; WRIGHT, 1982). Tree size (itself a measure of competitive status) has an effect that is an order of magnitude greater than what can realistically be expected from additive genetic effects on the underlying growth processes. True heritabilities of tree volume will, therefore, be low (< 0.20) in agreement with the majority of published figures (ZOBEL and TALBERT, 1984). Findings of higher heritabilities are in most cases raw estimates without any correction for size effects. Other things being equal, only genetic effects that lead to more growth will be exploitable and show up as improved yield per unit area. From this study and others it appears that growth and stand dynamic generate apparent heritability estimates that are upwardly biased (MAGNUSSEN, 1993; STROUP and MOULITZE, 1991).

Density management evidently plays an important role in capturing genetic gains (SPRINTZ, 1987). Juvenile genetic gains may inadvertently be lost if competition is allowed to take full effect. A change in management of improved stands is, therefore, necessary in order to capture the full potential of tree breeding. Narrow spacing appears to be effective for screening genotypes with a high genetic potential for rapid growth as purported by others (FRANKLIN, 1979; ST. CLAIR et al., 1991). Reducing the impact of competition is clearly important to allow genetic differences to manifest themselves in a trait like stem volume. It appears that the amount of genetic variation in stem volume will be rather limited (20% or less) despite a subsumed strong genetic control of the underlying growth process (vigor). This is a result that is reflected in many reported figures of heritability of stem volume (ZOBEL and TALBERT, 1984; BALOCCHI et al., 1993). The overwhelming effect of current tree size and competition on current growth simply dominates the growth process and leaves little 'room' for exploitable genetic influences.

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Helpful suggestions and ideas were provided by Dr. A. D. YANCHUK and an anonymous reviewer.

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Appendix I

Computing the stochastic deviations from a^0, and b^0 (i.e., $\varepsilon_a(t)+\varepsilon_a$(population), and $\varepsilon_b(t)+\varepsilon_b$ (population)) in equation (8) was based on an error budget derived from regression models relating a^0 and b^0 to the relative density (RDI) (MAGNUSSEN and BRAND, 1989). Repeated use of the TAYLOR theorem (for example, GALLANT, 1987) made it possible to obtain estimates of the standard deviation of the various stochastic deviations. The terms ε (population) are assumed to reflect 'global' fluctuations of a^0 and b^0 from one stand (population) to another. The standard deviations of these fluctuations were:

$$\sigma_a(pop.) = \sqrt{0.0064 \cdot \left(1 - exp\left(-\frac{1}{RDI}\right)\right)^2 + \frac{0.018225}{exp\left(\frac{2}{RDI}\right) \cdot RDI^2} - \frac{0.0054 \cdot \left(1 - exp\left(\frac{1}{RDI}\right)\right)}{exp\left(\frac{1}{RDI}\right) \cdot RDI}} \qquad (14)$$

and

$$\sigma_b(pop.) = \sqrt{\frac{3.6864}{exp(8 \cdot RDI)} + \frac{79.21 \cdot RDI^2}{exp(8 \cdot RDI)} - 14.4 \cdot RDI\,(exp(-(4 \cdot RDI)))^2} \qquad (15)$$

Equipped with these standard deviations the population specific stochastic effects were

$$\varepsilon_a(pop.) = u_a((pop.) \cdot \sigma_a(pop.)) \qquad (16)$$

$$\varepsilon_b(pop.) = (0.8 \cdot u_a(pop.) + 0.6 \cdot u_b(pop.)) \cdot \sigma_b$$

where u_a and u_b are random draws from a standardized normal variate (mean zero and variance one). This formulation assumes a 'built-in' correlation of 0.8 between the stochastic deviations from the expected competition parameters a^0 and b^0 (MAGNUSSEN and BRAND, 1989).

The temporal stochastic components $\varepsilon_a(t)$ and $\varepsilon_b(t)$ in (8) were derived from

$$\varepsilon_a(t) = u_a(t) \cdot \sigma_a(t) \qquad (17)$$

$$\varepsilon_b(t) = u_b(t) \cdot \sigma_b(t)$$

where u is a random normal distributed number with mean zero and a variance of one and $\sigma(t)$ is the standard deviation of $\varepsilon(t)$. The stochastic components $\varepsilon(t)$ of a and b were correlated (MAGNUSSEN and BRAND, 1989) as outlined below:

$$corr(\varepsilon_a(t), \varepsilon_a(t-1)) = 0.8 \qquad (18)$$

$$corr(\varepsilon_b(t), \varepsilon_b(t-1)) = 0.7$$

$$corr(\varepsilon_a(t), \varepsilon_b(t-1)) = corr(\varepsilon_b(t), \varepsilon_a(t-1)) = 0.8$$

Random normally distributed numbers $u_a(t)$ and $u_b(t)$ that satisfy the correlations in (18) were generated (RIPLEY, 1987) via the algorithms in (19)

$$u_a(t) = 0.7z_a(t-1) + 0.07z_b(t-1) + 0.07z_a(t) \qquad (19)$$

$$u_b(t) = 0.6z_a(t-1) + 0.7z_b(t-1) + 0.5z_b(t)$$

where z stands for a random standardized normally distributed variable. Finally, $\sigma_a(t)$ and $\sigma_b(t)$ were generated via

$$\sigma_a(t) = 0.0965 \cdot \sqrt{\left(a_t^0\right)^2 \cdot \left(1.54682 - 1.6456a_t^0 + \left(a_t^0\right)^2\right)} \qquad (20)$$

$$\sigma_b(t) = 0.0004 \cdot \sqrt{\left(b_t^0\right)^2 \cdot \left(171225 - 659.5b_t^0 + \left(b_t^0\right)^2\right)}$$