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Accounting for competition in genetic analysis, with particular emphasis on forest genetic trials

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Abstract Available experimental evidence suggests that there are genetic differences in the abilities of trees to compete for resources, in addition to non-genetic differences due to micro-site variation. The use of indirect genetic effects within the framework of linear mixed model methodology has been proposed for estimating genetic parameters and responses to selection in the presence of genetic competition. In this context, an individual's total breeding value reflects the effects of its direct breeding value on its own phenotype and its competitive breeding value on the phenotype of its neighbours. The present study used simulated data to investigate the relevance of accounting for competitive effects at the genetic and non-genetic levels in terms of the estimation of (co)variance components and selection response. Different experimental designs that resulted in different genetic relatedness levels within a neighbourhood and survival were other key issues examined. Variances estimated for additive genetic and residual effects tended to be biased under models that ignored genetic competition. Models that fitted competition at the genetic level only also resulted in biased (co)variance estimates for direct additive, competitive additive and residual effects. The ability to detect the correct model was reduced when relatedness within a neighbourhood was very low and survival decreased. Selection responses changed considerably between selecting on breeding value estimates from a model ignoring genetic competition and total breeding estimates using the correct model. Our results suggest that considering a genetic basis to competitive ability will be important to optimise selection programmes for genetic improvement of tree species.

Keywords Competition · Direct and indirect genetic effects · Linear mixed model · Genetic (co)variance components · BLUP · Response to selection

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Introduction

The ability to capture and use efficiently available resources is essential for the growth or reproductive successes of individual trees in forests (Binkley et al. 2004). Therefore, considering the long-lived nature and long rotation periods of forest trees, competition will be an important factor in the development of forest stands. Plant species with high competitive ability may evolve under conditions of low stress with low disturbance (Grime 1977). Consequently, accounting for competition is particularly important for forest tree breeding, as most species are recently domesticated and founder genotypes obtained from low stress, natural environments are likely to be competitive. In forest species, there have been several studies showing significant effects of inter-genotypic competition on stand (plot) productivity

(von Euler et al. 1992; Foster et al. 1998), but little is known about the quantitative genetics of tree competition. Several methods have been proposed to adjust for the effects of competition in forest genetic trials: examples include distance-dependent competition indices (Tuskan and McKinley 1984), use of angles of height difference (Magnussen 1989) and iterative nearest neighbour procedures (Magnussen 1994). However, these modelling approaches consider that competition operates at the phenotypic level but do not attempt to separate (and consequently confound) contributions from genetic and non-genetic (i.e. residual) competitive effects at the individual tree level.

Evolutionary theory and recent developments in quantitative genetics indicate that competition may substantially affect responses to selection, both in nature and in artificial selection programmes (Muir 2005; Bijma et al. 2007a; Bijma 2010a; Muir et al. 2010). Denison et al. (2003) suggested that most of the improvement in yield potential for the world's major crops has arisen by reducing individual plant competitiveness in favour of increasing plant community performance. Griffing (1967, 1977) introduced the concept of associative or indirect genetic effects (IGE) in order to more formally examine the dynamics of individual- versus community-level performance. In this sense, the genetic influences on an individual's phenotype are partitioned into the direct effects of its genes and the indirect effects of genes belonging to conspecifics with which the individual interacts (see also Moore et al. 1997; Wolf et al. 1998). Muir (2005) implemented IGE within a linear mixed model framework and, using forest tree breeding as an example, indicated that individuals should be selected on the basis of an index that combines genetic values for direct and indirect (i.e. competitive) effects. Though not verified with actual forestry data, the models, when tested using data from Japanese quail, indicated that in some circumstances a negative selection response might result if selecting only on direct genetic effects. Thus, artificial selection schemes in forest trees are probably sub-optimal, as current selection and breeding strategies for trait improvement most likely do not consider a genetic basis to competitive ability.

Bijma and colleagues (Bijma et al. 2007a, b; Ellen et al. 2007; Bergsma et al. 2008; Bijma 2010b, c, 2011) examined the use of IGE to model the contributions of an animal's associates. The IGE in this context are called social effects and indicate a genetic basis to either cooperative or aggressive behaviour to a subject animal. This research dealt with the quantitative genetic theory accounting for interactions among individuals and multi-level selection, explored the statistical methodology for genetic parameter estimation and examined the interrelationships between social effects, animal pen size and the relatedness of animals within pens. In the context of competition in agronomic crop species, Stringer (2006) and Stringer et al. (2011) refer to IGE fitted within a linear mixed model framework as the "treatment interference model", considering variety effects as random. Of particular interest was their modelling of competition at the residual level, either in conjunction with the use of IGE, when competition at the genetic level is present, or in absence of it. In this sense, autoregressive models were proposed and tested to account for competition as the dominant source of variation or to jointly model both competition and environmental trend. These analytical approaches for modelling competition at the residual level offer a guiding hand to plant and forest tree geneticists, more so than approaches used in animal genetics. This is because agricultural and forest field trials represent a continuum of focal plant and neighbour relationships, whereas animals are reared in discrete units such as pens or cages. Resende et al. (2005) and Cappa and Cantet (2008) were the first to verify the IGE model with actual forestry data from field trials. However, Resende et al. (2005) did not account for missing neighbours due to mortality and for the variable distance between a focal tree and its neighbours in the different spatial directions, features that were considered in the work of Cappa and Cantet (2008) by incorporating intensity of competition factors in the quantitative genetic model. Nevertheless, Cappa and Cantet (2008) did not consider a model for the residual covariance structure.

The present study brings together several aspects of modelling competition from the studies cited above. However, previous forest genetic studies have not examined the effects of different levels of relatedness and survival when modelling competition at the genetic level, nor evaluated the implications of trait genetic architecture on response to selection when inter-tree competition is present. These important issues can be effectively approached by using simulated data. In this sense, we have used simulated data to investigate the incorporation of IGE into a quantitative genetic model to account for competition, with a particular focus on forest genetic trials, and aiming to:

- Examine the effect on (co)variance component estimates of accounting for competition at the genetic and/or non-genetic levels.
- Explore the effect of different levels of genetic relatedness within the neighbourhood of a focal tree and overall survival on the ability to detect and estimate competition effects at the genetic level.
- Determine the importance of accounting for competition in genetic evaluation, by assessing the impact on the ranking of selection candidates and on expected responses to selection, and considering different magnitudes of competition (co)variance components, as well as different levels of genetic relatedness within a neighbourhood.

Materials and methods

Quantitative genetic model and definition of neighbourhoods

Because our context is competition in forest genetic trials and only additive effects of genes are considered, IGE will henceforth be denoted as competitive additive effects. The starting model we consider, which was first introduced by Griffing (1967), is the decomposition of an individual tree's phenotype into its direct breeding value, the sum of the competitive breeding values of its neighbours and a random, non-systematic, environmental (residual) effect. Thus, the model may be written as:

$$P_i = DBV_i + \sum_{j \neq i}^{n_i} CBV_j + E_i \quad (1)$$

where i denotes the focal individual, j one of its competitors (with $j=1\ldots n_i$), P_i is the phenotype of i, DBV_i is the direct breeding value of i, CBV_j is the competitive breeding value of j, and E_i denotes the residual component of the ith phenotype which may also be partitioned into direct and competitive residual terms. In forest genetic trials, the competitors are the trees planted adjacent to the focal tree, and thus it is assumed that competitive additive effects of more distant neighbours do not affect the phenotype of the focal tree. The number of first-order competitors, n_i, is conditional on the focal tree's positioning within the planting grid, as well as factors such as mortality and the presence or absence of buffer rows. The grid is usually a regular shape indexed by row and column numbers. Figure 1 shows two examples of a focal tree, denoted by F, located within subsets of the regular grid, at row and column numbers equal to R and C, respectively. The focal tree in neighbourhood 1 has the maximum possible number of eight neighbours. The focal tree in neighbourhood 2 is an example of an edge tree located on a boundary of the grid, with one of its neighbours being dead; depending on whether a buffer row is present or not, this focal tree has either seven or four neighbours.

Intensity of competition factors

Intensity of competition factors should be used to account for the differential intensity of competitive effects that neighbours exert over the phenotype of the ith focal individual, as suggested by Cappa and Cantet (2008) and Cantet and Cappa (2008). This differential intensity of competition applies within a neighbourhood and across neighbourhoods. Consider the case of neighbourhood 1 in Fig. 1: if inter-column spacing is smaller than inter-row spacing, then the intensity of competition exerted by N_1 and N_2 is greater than that exerted by N_3 and N_4. Next, consider two different neighbourhoods in the same trial with no buffer rows: one focal tree is an edge tree with only five neighbours and the other is a non-edge tree with a full complement of eight neighbours. In both of these cases, the expectation is that competitors of the ith focal tree are able to exert their competitive additive effect with greater intensity in a neighbourhood with fewer co-neighbours.

Equation (1) incorporating intensity of competition factors is represented as:

$$P_i = DBV_i + \sum_{j \neq i}^{n_i} f_{ij} CBV_j + E_i \quad (2)$$

where f_{ij} is the intensity factor exerted by neighbour j over the ith focal individual. As shown by Cappa and Cantet (2008), scaling the variance of competitive genetic effects within the phenotypic variance of P_i, while accounting

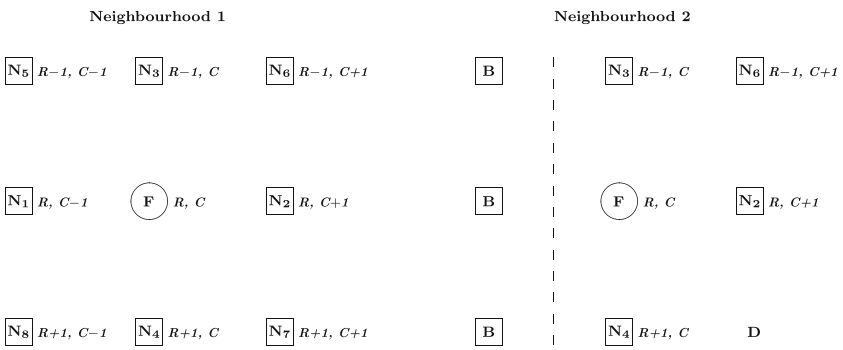

Fig. 1 Examples of neighbourhoods: neighbourhood 1 has no trees which are edge trees, and all neighbours have an alive status; neighbourhood 2 is flush on a boundary of the trial (represented by the *dashed line*), with buffer trees, denoted by the letter "B", existing beyond the boundary. A dead tree, represented by the letter "D", is found in neighbourhood 2. A focal tree, denoted by the letter "F", is situated at row = R and column = C. Under our coding system, a focal tree has a maximum of eight neighbours. The coding we have adopted is as follows: same row neighbours are denoted as N_1 and N_2; same column neighbours are denoted as N_3 and N_4; and diagonal neighbours are denoted as N_5, N_6, N_7 and N_8. The distance between N_1 and F (or between F and N_2) is referred to as inter-column spacing, while the distance between N_3 and F (or between F and N_4) is inter-row spacing

for different neighbourhood sizes, can be achieved by using intensity of competition factors under the following restriction:

$$\sum_{j=1}^{n_i} f_{ij}^2 = n_{\mathrm{R}_k} f_{\mathrm{R}_k}^2 + n_{\mathrm{C}_k} f_{\mathrm{C}_k}^2 + n_{\mathrm{D}_k} f_{\mathrm{D}_k}^2 = 1 \quad (3)$$

where n_{R_k}, n_{C_k} and n_{D_k} represent the number of row, column and diagonal neighbours, respectively, and f_{R_k}, f_{C_k} and f_{D_k} are the intensity of competition factors for row, column and diagonal neighbours, computed for the kth neighbourhood. Thus, Eq. (3) considers that neighbours positioned on a given orientation relative to the focal individual have the same f_{ij}. Cappa and Cantet (2008) demonstrated how f_{ij} can be computed for equal inter-row and inter-column spacing, assuming that the intensity of competition is related to the inverse of the distance between i and j. We have extended this approach to cases where inter-row spacing is not equal to inter-column spacing, by introducing a parameter p, which is the ratio of inter-row to inter-column spacing (for details on the derivation of formulae, see Online Resource 1, Electronic supplementary material).

Description of the data simulation

Data for two traits were simulated. One trait, which we nominally call growth and has a moderate to low heritability (i.e. in forest tree species, narrow-sense heritabilities for stem growth traits range from 0.10 to 0.30, White et al. 2007), was subject to direct and competitive effects. Since it was an objective to explore the effect of mortality within the framework of modelling competition at the genetic and non-genetic levels in forestry trials, it was necessary to consider survival in the data simulation. Hence, the second trait simulated was a binary survival trait. In this case, phenotypes were assumed to have a direct genetic component only, with moderate magnitudes for both narrow-sense heritability and positive additive correlation with the first trait (e.g. Chambers et al. 1996). The 80 and 100 % levels of survival were considered in the simulated data.

The simulation started by generating genetic values for a founder generation. The growth trait was assumed to have a normal distribution with a direct additive variance (σ_{d}^2) of 20. The nine combinations of values shown in Table 1 for competitive additive variance (σ_{c}^2) and correlation between direct and competitive additive effects (r_{dc}) were simulated to represent cases with high, moderate and low ratios of σ_{c}^2 to σ_{d}^2, and with small negative, moderate negative and large negative magnitudes for r_{dc}. The covariance between direct and competitive additive effects (σ_{dc}) is expected to be negative when competition is present at the genetic level (e.g. Muir 2005), and thus the simulated r_{dc} values were also negative. The values simulated for the additive (co)variance parameters approximate the range of estimates we have found for the ratio $\sigma_{\mathrm{c}}^2/\sigma_{\mathrm{d}}^2$ and r_{dc} in preliminary analyses (unpublished data) of diameter growth from five field trials of different forest species (*Eucalyptus globulus*, *Pinus radiata* and *Picea abies*) at several ages from planting (ranging from 4 to 18 years).

Table 1 Combinations of values used for genetic competition parameters in the simulated data

Genetic competition parameters	Simulated values								
σ_{c}^2	10	10	10	5	5	5	2	2	2
r_{dc}	−0.3	−0.6	−0.9	−0.3	−0.6	−0.9	−0.3	−0.6	−0.9

σ_{c}^2=genetic variance for competitive additive effects; r_{dc}=genetic correlation between direct and competitive additive effects

Under Eqs. (2) and (3), the scaling of competitive effects enables the σ_{c}^2 values to correspond directly to the total (i.e. summed over all neighbours) contribution of competitive additive effects to the phenotypic variance, assuming that the neighbours of a focal individual are genetically unrelated. However, under Eq. (1), the total contribution of the competitive additive variance to the phenotypic variance would equal $n_i \sigma_{\mathrm{c}}^2$, assuming again unrelated neighbours. Hence, the scaling of competitive effects using the f_{ij} factors strongly affects the interpretation of the magnitude of competitive effects. This is important when comparing studies using f_{ij} factors to those not using f_{ij} factors.

The survival trait, which on an observed scale had a phenotypic value of either 0 (dead) or 1 (alive), was assumed to have an underlying normal distribution with an additive variance of 0.5. Chambers et al. (1996) reported an average genetic correlation of 0.5 between growth and survival across a number of trials, but the authors also indicated that the magnitude of this correlation may be lower for sites under severe drought conditions. Thus, we have assumed a value of 0.3 for the genetic correlation between direct additive effects for growth and survival, as an attempt to accommodate a broad range of environmental stress conditions (such as competition, frost and drought) impacting tree survival. For each of the combinations of the (co)variance parameters shown in Table 1, direct and competitive additive values for growth, and direct additive values for survival, were sampled from a multivariate normal distribution using a mean vector (denoted by **m**) containing zeros and a (3×3) variance–covariance matrix (denoted by **C**).

The simulation proceeded by mating founders, using either cross-pollinated (CP) or open-pollinated (OP) mating designs, to produce a progeny generation. For the CP

design, 36 founders were generated and each founder was crossed four times following a partial diallel mating design, which resulted in 72 families, each containing 36 full-sibs. Breeding values for the CP progeny were sampled from a multivariate normal distribution with a mean vector:

$$\mathbf{m} = \begin{pmatrix} 0.5d_{\mathrm{G,fp}} + 0.5d_{\mathrm{G,mp}} \\ 0.5c_{\mathrm{G,fp}} + 0.5c_{\mathrm{G,mp}} \\ 0.5d_{\mathrm{S,fp}} + 0.5d_{\mathrm{S,mp}} \end{pmatrix}$$

and a variance–covariance matrix $\mathbf{C}^* = 0.5 \times \mathbf{C}$; in $\mathbf{m}$, d denotes direct breeding value and c denotes competitive breeding value, with d and c being subscripted with G to denote growth or S to denote survival, and the abbreviations fp and mp referring to female and male parents, respectively. For the OP design, 72 founders were generated and each founder was assumed to produce 36 progeny. Genetic values for the OP progeny were sampled from a multivariate normal distribution with a mean vector:

$$\mathbf{m} = \begin{pmatrix} 0.5d_{\mathrm{G,fp}} \\ 0.5c_{\mathrm{G,fp}} \\ 0.5d_{\mathrm{S,fp}} \end{pmatrix}$$

and a variance–covariance matrix $\mathbf{C}^* = 0.75 \times \mathbf{C}$. Non-additive genetic effects were assumed to be unimportant in our populations, and thus were not simulated under the assumption that an additive genetic model is adequate for the traits considered. In both designs, 2,592 progeny were generated and positioned on a regular grid composed by 72 rows and 36 columns, with equal inter-row and inter-column spacing.

Three levels of additive genetic relatedness within a neighbourhood were imposed as:

medium relatedness—achieved under the CP mating design by creating four-tree-line plots, in which case a focal tree has one or two full sibs as neighbours in a non-edge neighbourhood;
low relatedness—achieved under the CP mating design by creating single-tree plots;
very low relatedness—achieved under the OP mating design by creating single-tree plots.

The plots were placed at random on the grid, except for a restriction on randomization that ensured that there was no tendency for pairs of plots from the same two families to be located together. For a neighbourhood comprising a focal tree and its eight competitors, the resulting mean values for the additive relationship coefficients among all pairs of individuals were 0.11, 0.03 and 0.003 for medium, low and very low genetic relatedness levels, respectively. The mating designs and plot configurations described above are regularly used in the context of field testing in forest genetics (White et al. 2007). Thus, under comparable experimental settings, it is expected that the three simulated levels of relatedness will represent magnitudes of mean genetic relatedness that may be commonly found within tree neighbourhoods in actual field trials.

Growth phenotypes for progeny were simulated as described in Eq. (2). When survival was 100 %, the f_{ij} were needed to reflect the different strengths of competition exerted by row and column neighbours relative to diagonal neighbours. In addition, for 80 % survival, the f_{ij} were needed to account for the fact that n_i differed among non-edge focal individuals. In the generation of residual effects for growth, we have partitioned the random term E_i in Eq. (2) into competitive and direct residual components. We have further assumed that the competitive component follows a spatially correlated process, and the direct component is an uncorrelated residual distributed independently of the competitive term. In this context, the general form for the variance–covariance matrix of the residuals was defined as (after Gilmour et al. 1997; Stringer 2006; Stringer et al. 2011)

$$\mathrm{Var}[\boldsymbol{\xi}] + \mathrm{Var}[\boldsymbol{\eta}] = \sigma^2_{\mathrm{ce}}\,(\boldsymbol{\Sigma} \mid \boldsymbol{\alpha}) + \sigma^2_{\mathrm{ie}}\mathbf{I} \tag{4}$$

where $\boldsymbol{\xi}$ is a vector whose elements follow a spatially correlated process, $\boldsymbol{\eta}$ is a vector whose elements are pairwise independent, $\boldsymbol{\Sigma}$ is the correlation matrix for the spatially dependent process (conditional on the parameters in vector $\boldsymbol{\alpha}$) with the associated variance given by σ^2_{ce}, σ^2_{ie} is the variance of the independent residuals and $\mathbf{I}$ is an identity matrix. In addition, we have assumed separable spatially dependent processes in the row and column directions, and thus $\boldsymbol{\Sigma}$ was defined as (for data ordered as columns within rows):

$$\boldsymbol{\Sigma}|\boldsymbol{\alpha} = \boldsymbol{\Sigma}_{\mathrm{row}}|\boldsymbol{\alpha}_{\mathrm{row}} \otimes \boldsymbol{\Sigma}_{\mathrm{col}}|\boldsymbol{\alpha}_{\mathrm{col}} \tag{5}$$

where $\boldsymbol{\alpha}_{\mathrm{row}}$ and $\boldsymbol{\alpha}_{\mathrm{col}}$ are vectors for row and column autocorrelation parameters, respectively, and $\otimes$ denotes the Kronecker product operation. A first-order autoregressive (AR1) process can be used in $\boldsymbol{\Sigma}$ to model either residual competitive effects or local environmental trend but may not be effective to model both (Stringer 2006; Stringer et al. 2011). In the present study, we aim to explore the effects of competition *per se*, and thus we have assumed in our simulated data that environmental trend was unimportant at both global and local levels (e.g. a field trial located in a homogeneous site, with small environmental variability). Therefore, we have used the AR1 process in $\boldsymbol{\Sigma}$, considering competition to be the dominant source of autocorrelation at the residual level. In this sense, situations where competition is dominant over environmental trend have also been observed in preliminary analyses that we have pursued for actual diameter growth data from forest genetic trials of *Pinus radiata* (with ages from field planting ranging from 11 to 13 years) and *Picea abies* (with ages from field planting ranging from 16 to 18 years) (unpublished results). In the AR1 model, each of the vectors $\boldsymbol{\alpha}_{\mathrm{row}}$ and $\boldsymbol{\alpha}_{\mathrm{col}}$ comprises a

single autocorrelation parameter, which is typically negative when competition is present at the residual level and is dominant over environmental trend (e.g. Stringer 2006). Correlated residuals across the grid of R rows by C columns were generated from a multivariate normal distribution with a mean vector containing zeros and a covariance structure of the form described in Eq. (5), assuming σ^2_{ce} equal to 20 and the parameters in $\boldsymbol{\alpha}_{row}$ and $\boldsymbol{\alpha}_{col}$ equal to −0.5. The independent residuals were generated from a normal distribution with a zero mean and σ^2_{ie} equal to 80.

An underlying, continuous phenotype for survival, $P^*_{S,i}$, was simulated using:

$$P^*_{S,i} = BV_{S,i} + E_{S,i} \tag{6}$$

where $BV_{S,i}$ and $E_{S,i}$ denote the breeding value and the residual term, respectively, for the ith individual. The $E_{S,i}$ values were generated from a normal distribution with a zero mean and a variance equal to 1. The $P^*_{S,i}$ values were subsequently transformed to a binary scale using a threshold X computed from the cumulative standard normal distribution, where the integral from minus infinity to X had either the value 0 for 100 % survival or 0.2 for 80 % survival. That is, if $P^*_{S,i} < X$ then $P_{S,i}$=0 (dead), otherwise $P_{S,i}$=1 (alive). Then, for a focal individual i, its number of first-order neighbours (n_i) was given by:

$$n_i = \sum_{j=1}^{8} P_{S,j} \tag{7}$$

Buffer rows were assumed not to exist, and thus were not simulated; hence, when the focal individual was an edge tree, $P_{S,j}$ values were assumed to be zero for j corresponding to a position outside the grid. Thus, for both 80 and 100 % survival levels, the intensity of competition factors were also needed to reflect the different strengths of competition exerted over edge and non-edge focal individuals.

For each combination of values simulated for genetic competition parameters (Table 1), three levels of genetic relatedness and two levels of survival were tested. In total, 54 different scenarios were tested for a defined neighbourhood, and 100 replicates were generated for each scenario. Each replicate was analysed under the four statistical models described below. Also 100 replicates were used for each of the scenarios exploring the impact on selection (see below).

Statistical models

Table 2 presents an overview of the statistical models that were fitted in the present study to simulated data. The base model (B) does not fit competition at either the genetic or residual levels. The additive variance estimated under this model is not assumed to explicitly represent direct additive effects, and is denoted more generally as σ^2_a. A breeding value is not qualified as being either direct or competitive, and is denoted simply as BV. Residual effects are assumed independently and identically distributed, and the residual variance is denoted simply as σ^2_e. The B model is expressed as:

$$\mathbf{y} = \mathbf{1}\mu + \mathbf{Za} + \mathbf{e} \tag{8}$$

where $\mathbf{y}$ is the vector of simulated phenotypes, $\mathbf{1}$ is a vector of ones linking phenotypes to the mean μ, $\mathbf{Z}$ is a matrix linking phenotypes of individuals to their BV contained in the vector $\mathbf{a}$, and $\mathbf{e}$ is a vector of residuals. The variance matrix of the additive effects in vector $\mathbf{a}$ is:

$$\mathrm{Var}[\mathbf{a}] = \mathbf{A}\sigma^2_a \tag{9}$$

where $\mathbf{A}$ is a matrix of additive relationship coefficients among individuals in the population, and the

Table 2 Overview of statistical models fitted to simulated data

Abbreviation	Name	Random terms fitted	Tested against[a]	df[b]
B	Base model	σ^2_a, σ^2_e		
AR	Autoregressive model for the residuals	$\sigma^2_a, \sigma^2_{ie}, \sigma^2_{ce}, \alpha_{1_{row}}, \alpha_{1_{col}}$	B	3
GC	Genetic competition model	$\sigma^2_d,\ \sigma^2_c,\ r_{dc},\ \sigma^2_e$	B	2
GC-AR	Combined genetic competition and autoregressive model	$\sigma^2_d,\ \sigma^2_c,\ r_{dc},\ \sigma^2_{ie},\ \sigma^2_{ce}, \alpha_{1_{row}},\ \alpha_{1_{col}}$	AR	2

σ^2_a=additive genetic variance under the B and AR models, assuming no competition at the genetic level; σ^2_d=genetic variance for direct additive effects under the GC and GC-AR models; σ^2_c=genetic variance for competitive additive effects under the GC and GC-AR models; r_{dc}=genetic correlation between direct and competitive additive effects under the GC and GC-AR models; σ^2_e=residual variance under the B and GC models, assuming that the residuals are uncorrelated; σ^2_{ie}=independent residual variance under the AR and GC-AR models; σ^2_{ce}=correlated residual variance under the AR and GC-AR models; $\alpha_{1_{row}}$ and $\alpha_{1_{col}}$=first-order autocorrelation parameters for the row and column directions under the AR and GC-AR models

[a] The improvement of the model shown in the first column over the model shown in this column is tested via a likelihood ratio (LR) test. In this sense, the test statistic was calculated by twice the difference in REML log-likelihood between the two models

[b] Difference between the compared models in the number of parameters fitted, which is used to specify the degrees of freedom (df) of the chi-squared distribution to perform a LR test

variance matrix of the residual effects in vector **e** is defined as:

$$\mathrm{Var}[e] = \mathbf{I}\sigma_e^2 \tag{10}$$

The second model (AR) in Table 2 does not account for competition at the genetic level, but fits competition at the residual level using a separable AR1 process. Thus, the variance matrix of the additive effects in vector **a** was defined as in Eq. (9), and the vector of random residuals was partitioned as $\mathbf{e}=\boldsymbol{\xi}+\boldsymbol{\eta}$ and being fitted according to the variance–covariance matrix described in Eqs. (4) and (5).

The third model is the genetic competition model (GC), which explicitly separates additive effects into direct and competitive additive effects. The genetic covariance between direct and competitive additive effects is also fitted. The GC model is expressed as:

$$\mathbf{y} = \mathbf{1}\mu + \mathbf{Z}_d\mathbf{d} + \mathbf{Z}_c\mathbf{c} + \mathbf{e} \tag{11}$$

where $\mathbf{Z}_d$ and $\mathbf{Z}_c$ are matrices linking phenotypes of individuals to their direct breeding values and competitive breeding values contained in the vectors **d** and **c**, respectively. The f_{ij} factors used in the statistical analysis were identical to those used in the generation of the data, with the *i*th row in $\mathbf{Z}_c$ containing the intensities of competition factors computed for the neighbours *j* of the *i*th focal individual. The variance–covariance matrix of the additive effects in Eq. (11) is given by:

$$\mathrm{Var}\begin{bmatrix}\mathbf{d}\\ \mathbf{c}\end{bmatrix} = \begin{bmatrix}\sigma_d^2 & \sigma_{dc}\\ \sigma_{dc} & \sigma_c^2\end{bmatrix} \otimes \mathbf{A} \tag{12}$$

where σ_d^2, σ_c^2 and σ_{dc} are defined as above. The use of the matrix **A** allows us to link direct and competitive additive effects through individual relationships within and across neighbourhoods. The variance matrix of the effects in **e** is defined as in Eq. (10), as competition at the residual level is not fitted under the GC model.

The fourth model in Table 2 is the combined genetic competition and autoregressive model (GC-AR), which accounts for competition at both the genetic and residual levels. Likelihood ratio (LR) tests were performed for comparing nested models, as shown in Table 2. The LR test is implicitly two-tailed but it should be adjusted when a hypothesis test involves testing variances (as in our case), which are restricted to be greater than or equal to zero (Stram and Lee 1994). However, rather than determining the correct theoretical asymptotic distribution of the LR test statistic for each model comparison, two-tailed tests were always used with the stated degrees of freedom. Hence, all the LR tests we have performed are conservative. All models were fitted with the ASReml software (Gilmour et al. 2009), using restricted maximum likelihood for the estimation of (co)variance parameters.

Impact on selection

In the framework of a quantitative genetic model accounting for IGE, Bijma (2011) demonstrated the distinction between the heritable component of phenotypic variance and the heritable effects that an individual passes on to the next generation. The latter term is completely a genetic property of the focal individual. It represents the impact of the individual's genes on the genetic mean value of a population ($\overline{P}_{\mathrm{genetic}}$) and is referred to as the total breeding value (*TBV*). Following Eq. (2), the genetic mean of the population may be defined as:

$$\overline{P}_{\mathrm{genetic}} = \overline{DBV} + \left(\overline{n_R}\,\overline{f_R} + \overline{n_C}\,\overline{f_C} + \overline{n_D}\,\overline{f_D}\right)\overline{CBV} \tag{13}$$

and thus, for the *i*th focal individual, the total breeding value (TBV_i) will be defined as:

$$TBV_i = DBV_i + \left(\overline{n_R}\,\overline{f_R} + \overline{n_C}\,\overline{f_C} + \overline{n_D}\,\overline{f_D}\right)CBV_i \tag{14}$$

where $\overline{n_R}\,\overline{f_R}$, $\overline{n_C}\,\overline{f_C}$ and $\overline{n_D}\,\overline{f_D}$ denote products of the means taken across all focal individuals in the population for the number of their neighbours and intensity of interaction factors in the row, column or diagonal directions. For 100 % survival, the quantity $\overline{n_R}\,\overline{f_R} + \overline{n_C}\,\overline{f_C} + \overline{n_D}\,\overline{f_D}$ was 2.7 in every replicate of the simulated scenarios.

It is important to examine the impact on selection decisions (in addition to the impact on variance component estimation) from not accounting for competitive effects in genetic evaluation when they are actually present. In this context, three methods of selection were compared:

(i) selection based on estimated breeding values using the B model ($\widehat{BV}$);
(ii) selection based on estimated direct breeding values using the GC-AR model ($D\widehat{BV}$);
(iii) selection based on estimated total breeding values using the GC-AR model ($T\widehat{BV}$);

with estimated breeding values being obtained by best linear unbiased prediction (BLUP).

Progeny were ranked using the three methods. As an indication of discrepancy in ranking, Spearman rank correlations were computed for: comparing selections based on $\widehat{BV}$ and $D\widehat{BV}$; and comparing selections based on $\widehat{BV}$ and $T\widehat{BV}$. Expected genetic responses were computed for individual (offspring) selection, following selection of around the 5 % topmost ranked individuals. For simulated data, true genetic values are available, and the expected response to selection may be calculated from the average of the simulated genetic values for the individuals selected under a given method. By using the simulated (true) breeding values (denoted as *DBV*, *CBV* and *TBV* for direct, competitive and total genetic effects, respectively), the expected genetic

responses from selections based on the B or GC-AR models are expressed on the same scale, hence reflecting the differences between models in the individuals selected. Thus, the strategy was to compare the average *TBV* of the top individuals ranked under method (i) with the average *TBV* of the top individuals ranked under method (iii). Average values were also obtained for the two terms in Eq. (14), in order to evaluate the contributions of direct and competitive genetic effects to the total response from selection based on either method.

Results

We have used the median to describe the results of the studied parameters over the 100 replicates in the simulation. When comparing estimators to summarise data, Hozo et al. (2005) indicated that the median provides a consistent measure of central tendency for either normal or skewed distributions when sample sizes are larger than 25.

Parameter estimates from fitting an incomplete model

The true values of the (co)variance parameters used to simulate the data sets were best approximated by the corresponding estimates obtained under the most complete GC-AR model (Fig. 2). For the remaining models, the following main patterns in (co)variance parameter estimates were observed for σ_c^2 = 10 and 100 % survival.

For the B model (Fig. 3), $\widehat{\sigma}_a^2$ appeared to be unbiased under very low relatedness but tended to become biased downwards for r_{dc} values beyond −0.3 under the other relatedness levels. The residual variance in the B model exceeded always its expected value (i.e. $\sigma_{ie}^2 + \sigma_{ce}^2 = 100$), with the amount of bias being close to the true value of σ_c^2, and thus indicating that it was absorbing additive effects due to competition. In addition, the residual variance also seemed to absorb direct additive variance under the medium relatedness level, as suggested by the opposite trends in the estimates of these two variance components as r_{dc} became more negative (Fig. 3).

Under the AR model (Fig. 4), $\widehat{\sigma}_a^2$ appeared to be increasingly biased downwards for all relatedness levels as the values of r_{dc} became more negative, with the bias being greater for medium relatedness relative to the other relatedness levels. The $\widehat{\sigma}_{ie}^2$ was inflated for all relatedness levels under r_{dc}=−0.3, but then it decreased as r_{dc} became more negative, reaching a downward bias for the very low relatedness level under r_{dc}=−0.9. Conversely, $\widehat{\sigma}_{ce}^2$ appeared to be increasingly biased upwards for more negative values of r_{dc}. The opposite trends observed for $\widehat{\sigma}_{ce}^2$ and $\widehat{\sigma}_a^2$ (Fig. 4) suggest that $\widehat{\sigma}_{ce}^2$ may have an increasing contribution in absorbing $\widehat{\sigma}_a^2$ as r_{dc} becomes more negative. In addition, the observed biases in the residual terms indicate that $\widehat{\sigma}_{ce}^2$ may assimilate part of $\widehat{\sigma}_{ie}^2$ under more negative values of r_{dc}, with the effect being particularly strong for a very low relatedness level. Nevertheless, in general, the results indicate that residual terms in the AR model may be absorbing direct and/or indirect additive (co)variance components.

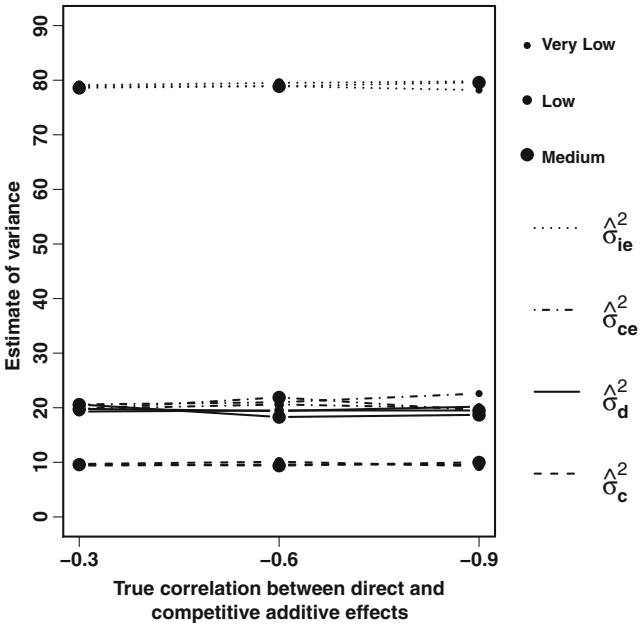

Fig. 2 Estimates of direct additive ($\widehat{\sigma}_d^2$), competitive additive ($\widehat{\sigma}_c^2$), independent residual ($\widehat{\sigma}_{ie}^2$) and correlated residual ($\widehat{\sigma}_{ce}^2$) variances from data simulated for a trait representing tree growth and analysed under the GC-AR model. The variance estimates are plotted against three simulated values (−0.3, −0.6 and −0.9) of the genetic correlation between direct and competitive additive effects, considering three levels (medium, low and very low) of genetic relatedness within a neighbourhood and 100 % survival. Variances for the underlying true effects in the simulated data were $\sigma_d^2 = 20$, $\sigma_c^2 = 10$, $\sigma_{ie}^2 = 80$ and $\sigma_{ce}^2 = 20$ for direct additive, competitive additive, independent residual and correlated residual effects, respectively

For the GC model (Figs. 5 and 6), there were in general upward biases in $\widehat{\sigma}_d^2$ and $\widehat{\sigma}_c^2$, and a $\widehat{r}_{dc}$ more negative than its true value. The biases in $\widehat{\sigma}_d^2$ and $\widehat{\sigma}_c^2$ increased as r_{dc} became more negative and tended to be smaller under very low relatedness (Fig. 5). The biases in $\widehat{r}_{dc}$ were substantial when the true values of the parameter were equal to −0.3 and −0.6 (Fig. 6). The residual variance was biased downwards relatively to its expectation (i.e. 100), with the bias being more accentuated as the r_{dc} values became more negative (Fig. 5), and thus suggesting that additive (co)variance components may be absorbing residual terms (a situation which contrasts with the tendencies indicated under the B and AR models).

Results obtained for 80 % survival and σ_c^2 = 10, as well as for both levels of survival and σ_c^2 = 5 or σ_c^2 = 2, are not presented as the patterns observed for the estimated (co)variance parameters were in general similar to those described above for 100 % survival and σ_c^2 = 10.

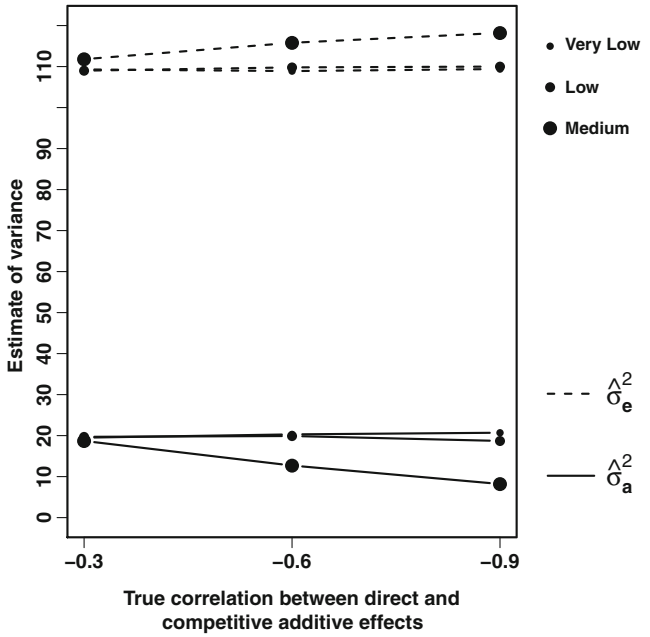

Fig. 3 Estimates of additive ($\widehat{\sigma}^2_a$) and residual ($\widehat{\sigma}^2_e$) variances from data simulated for a trait representing tree growth and analysed under the B model. The variance estimates are plotted against three simulated values (−0.3, −0.6 and −0.9) of the genetic correlation between direct and competitive additive effects, considering three levels (medium, low and very low) of genetic relatedness within a neighbourhood and 100 % survival. Variances for the underlying true effects in the simulated data were $\sigma^2_d = 20$, $\sigma^2_c = 10$, $\sigma^2_{ie} = 80$ and $\sigma^2_{ce} = 20$ for direct additive, competitive additive, independent residual and correlated residual effects, respectively

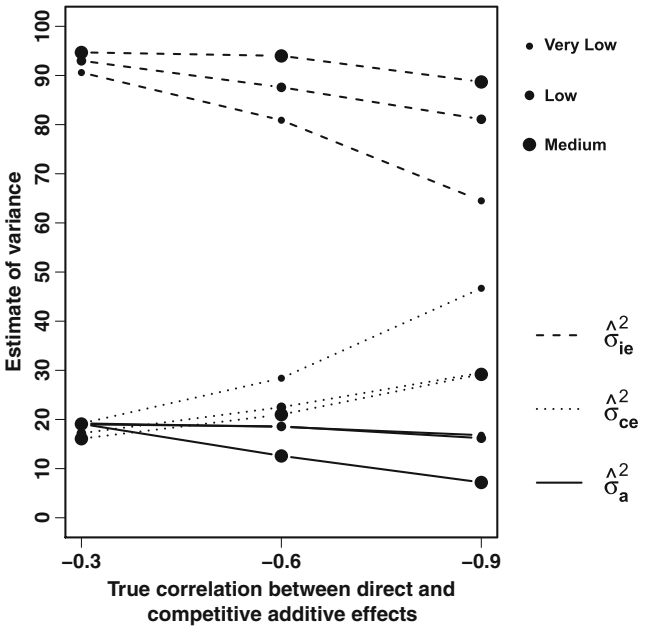

Fig. 4 Estimates of additive ($\widehat{\sigma}^2_a$), independent residual ($\widehat{\sigma}^2_{ie}$) and correlated residual ($\widehat{\sigma}^2_{ce}$) variances from data simulated for a trait representing tree growth and analysed under the AR model. The variance estimates are plotted against three simulated values (−0.3, −0.6 and −0.9) of the genetic correlation between direct and competitive additive effects, considering three levels (medium, low and very low) of genetic relatedness within a neighbourhood and 100 % survival. Variances for the underlying true effects in the simulated data were $\sigma^2_d = 20$, $\sigma^2_c = 10$, $\sigma^2_{ie} = 80$ and $\sigma^2_{ce} = 20$ for direct additive, competitive additive, independent residual and correlated residual effects, respectively

Ability to detect the most appropriate model

Following LR tests, the AR model proved to be always highly significantly ($P \leq 0.001$) better than the B model, whereas the improvement of the GC model over the B model was dependent on the magnitudes of σ^2_c, r_{dc} and relatedness for a given level of survival (not shown). These factors also appeared to determine the ability to detect the most appropriate model, as indicated by the significance probability from the LR test comparing the GC-AR model with the AR model. Based on the individual probability values obtained for each of the 100 replicated comparisons of the GC-AR and AR models, average estimates of the significance probability are plotted in Fig. 7 against the simulated values of r_{dc} , considering the different scenarios involving σ^2_c, relatedness and survival. From a total of 100 comparisons between the GC-AR and AR models, the number of LR tests that were not statistically significant at the 5 % level is presented in Online Resource 2 (Electronic supplementary material) for each of the simulated scenarios.

The ability to detect the correct GC-AR model tended to diminish when r_{dc} became less negative, the σ^2_c decreased for a given magnitude of r_{dc} and the level of relatedness decreased for given magnitudes of r_{dc} and σ^2_c. In addition, decreasing survival from 100 to 80 % reduced in general the ability to detect the most appropriate model for a given set of parameters and genetic relatedness (Fig. 7 and Online Resource 2). Yet, detecting the most appropriate model under 80 % survival was reasonably successful for σ^2_c = 10; the same applies for all scenarios under σ^2_c = 5, except for very low relatedness when r_{dc}=−0.3.

Impact on selection

Selection outcomes under contrasting magnitudes of σ^2_c and r_{dc}

The effect of contrasting magnitudes of σ^2_c and r_{dc} on selection outcomes is presented in Table 3 assuming 100 % survival and using simulated data with a mean additive genetic relatedness within a neighbourhood equal to 0.03 (i.e. corresponding to an intermediate level between the medium and very low relatedness scenarios).

The Spearman rank correlations between breeding value estimates from the B model ($\widehat{BV}$) and direct breeding value estimates from the GC-AR model ($D\widehat{BV}$) were very high (i.e. ≥0.97). Poorer agreement in ranking was found between selections based on $\widehat{BV}$ from the B model and total

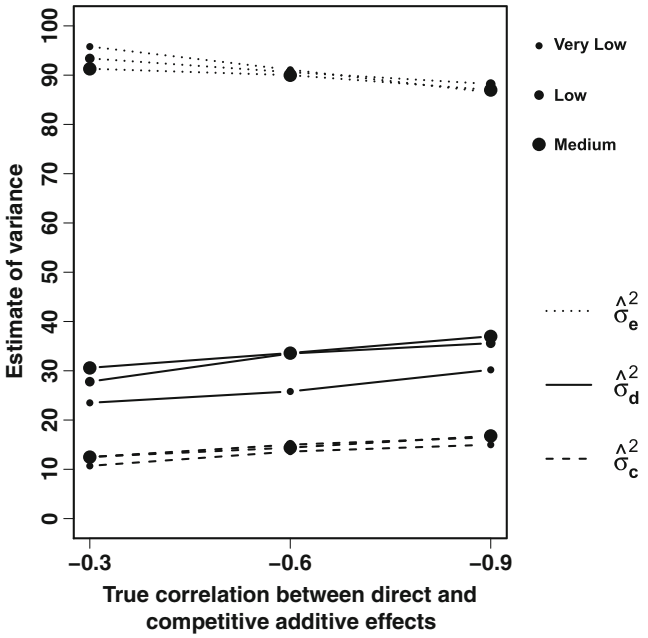

Fig. 5 Estimates of direct additive ($\widehat{\sigma}^2_d$), competitive additive ($\widehat{\sigma}^2_c$) and residual ($\widehat{\sigma}^2_e$) variances from data simulated for a trait representing tree growth and analysed under the GC model. The variance estimates are plotted against three simulated values (−0.3, −0.6 and −0.9) of the genetic correlation between direct and competitive additive effects, considering three levels (medium, low and very low) of genetic relatedness within a neighbourhood and 100 % survival. Variances for the underlying true effects in the simulated data were $\sigma^2_d = 20$, $\sigma^2_c = 10$, $\sigma^2_{ie} = 80$ and $\sigma^2_{ce} = 20$ for direct additive, competitive additive, independent residual and correlated residual effects, respectively

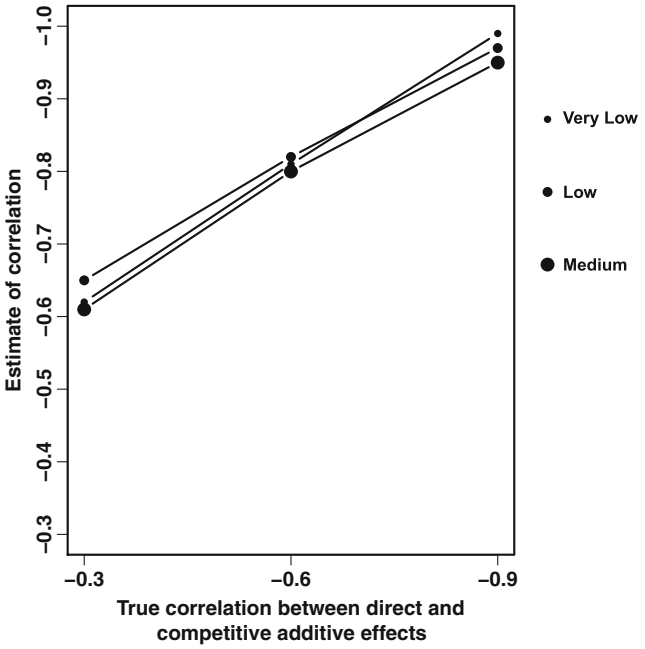

Fig. 6 Estimates of the correlation between direct and competitive additive effects from data simulated for a trait representing tree growth and analysed under the GC model. The correlation estimates are plotted against three simulated values (−0.3, −0.6 and −0.9) of the genetic correlation between direct and competitive additive effects, considering three levels (medium, low and very low) of genetic relatedness within a neighbourhood and 100 % survival. Variances for the underlying true effects in the simulated data were $\sigma^2_d = 20$, $\sigma^2_c = 10$, $\sigma^2_{ie} = 80$ and $\sigma^2_{ce} = 20$ for direct additive, competitive additive, independent residual and correlated residual effects, respectively

breeding value estimates from the GC-AR model ($T\widehat{BV}$). In this sense, the agreement in ranking based on these two models became worse when r_{dc} was more negative and/or σ^2_c increased. The Spearman rank correlations between $\widehat{BV}$ from the B and AR models were always very high (i.e. ≥0.98; not shown), indicating that similar selection outcomes are expected from comparisons between the GC-AR model and either the B or the AR model.

For r_{dc}=−0.3, the selections based on the B model resulted in a response from direct effects which was able to compensate for the negative contribution due to competitive effects, leading always to a positive total response. For r_{dc}=−0.9, the total response from selection based on the B model was negative under σ^2_c = 10, reflecting the larger contribution (in absolute value) to *TBV* of negative competitive effects relative to direct effects.

Total responses from selection based on the GC-AR model were considerably better than those based on the B model for σ^2_c = 10; in particular, for selections from the GC-AR model, the contribution of positive competitive effects to *TBV* has largely compensated the corresponding negative direct effects observed under r_{dc}=−0.9. However, the advantage of the GC-AR model over the B model became less apparent for σ^2_c = 2, as the corresponding total responses to selection tended to converge, reflecting the smaller differences between models in contributions to *TBV* due to direct or competitive effects. Yet, despite these approximations in total response, the maximum number of common selections from the two models under σ^2_c = 2 was 63 only (for a total of 130 individuals selected).

Results obtained by assuming 80 % survival (not shown) indicated similar patterns as those described above, but the advantage of the GC-AR model over the B model in total response to selection was in general slightly diminished relative to comparable scenarios for 100 % survival. This reflected a reduction in the contribution of competitive effects to *TBV*, which may be due to the decrease in the number of competitors around the focal individual under 80 % survival.

Selection outcomes under contrasting levels of relatedness within a neighbourhood

The effect on selection outcomes from contrasting levels of additive genetic relatedness within a neighbourhood (i.e. mean levels of 0.11 and 0.003, corresponding to the simulated medium and very low relatedness scenarios, respectively) is presented in Table 4, assuming 100 % survival, an

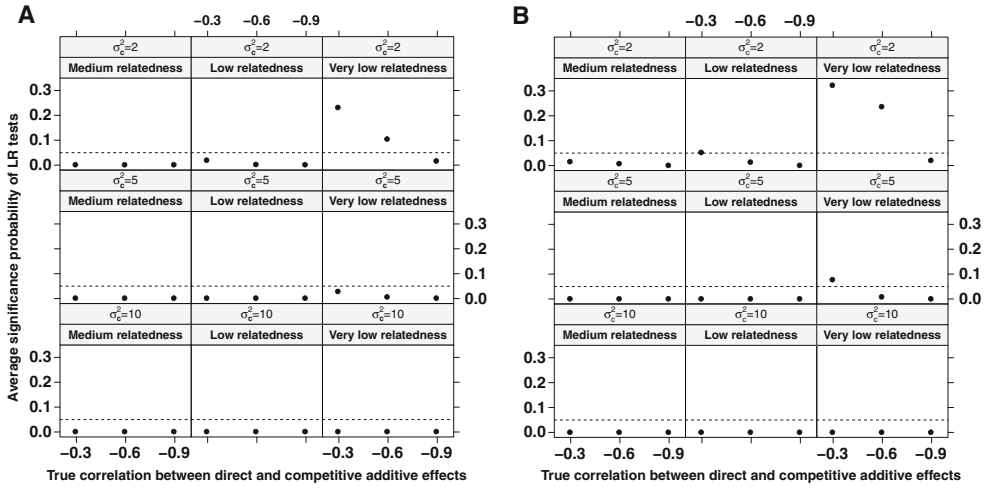

Fig. 7 Average significance probabilities from likelihood ratio (LR) tests comparing the GC-AR model and the AR model. For each of the simulated scenarios, the individual significance probabilities from LR tests performed for each of the 100 replicated comparisons of the GC-AR and AR models were averaged and plotted against simulated values of the genetic correlation between direct and competitive additive effects. Simulated scenarios involved three values of the genetic correlation between direct and competitive additive effects (−0.3, −0.6 and −0.9), three values of the genetic variance for competitive additive effects (σ_c^2 = 2, 5 and 10), three levels of genetic relatedness within a neighbourhood (medium, low and very low) and two levels of survival (100 and 80 %, pertaining to plots in A and B, respectively). The 5 % significance level is represented by a *dashed line*

intermediate value of σ_c^2 (i.e. 5) and intermediate and high values of r_{dc} (i.e. −0.6 and −0.9).

The rankings based on $D\widehat{B}V$ and $T\widehat{B}V$ from the GC-AR model were, respectively, highly and poorly correlated with the rankings based on $\widehat{B}V$ from the B model, consistent with the selection outcomes described above. Nevertheless, for a given value of r_{dc}, the Spearman rank correlations between $T\widehat{B}V$ and $\widehat{B}V$ indicated a better agreement in ranking when relatedness increased.

In general, total response from selection based on either B or GC-AR model tended to decline when the level of relatedness decreased, but the relative changes in total response with decreasing relatedness depended on the value of r_{dc}. In this sense, under medium relatedness, the advantage of the GC-AR model over the B model in total response was greater than under very low relatedness, but the difference between models was diminished when r_{dc} changed from −0.6 to −0.9. This result reflects the fact that, under selection based on the GC-AR model, lowering the relatedness level decreased total response by reducing the favourable contribution of the competitive component relatively to the part attributed to direct effects, with the difference between relatedness levels for r_{dc}=−0.6 being larger than that for r_{dc}=−0.9. Results obtained by assuming 80 % survival (not shown) indicated similar

Table 3 Expected outcomes from individual selection for contrasting magnitudes of the genetic variance for competitive additive effects (i.e. σ_c^2 = 10 and 2) and of the genetic correlation between direct and competitive additive effects (i.e. r_{dc}=−0.3 and −0.9), assuming 100 % survival and a mean level of genetic relatedness within a neighbourhood equal to 0.03 (i.e. corresponding to an intermediate level between the medium and very low relatedness scenarios). The results were obtained from data simulated for a trait representing tree growth

True values of parameters	Spearman rank correlations[a]		Genetic response to selection[b]						Common selected individuals[c]
			Direct		Competitive		Total		
r_{dc}=−0.3, σ_c^2 = 10	0.988	0.210	6.2	1.4	−3.7	8.8	2.5	10.2	11/130
r_{dc}=−0.3, σ_c^2 = 2	0.994	0.793	6.1	4.7	−1.5	0.8	4.6	5.5	63/130
r_{dc}=−0.9, σ_c^2 = 10	0.967	−0.765	6.0	−5.1	−10.4	10.4	−4.4	5.3	0/130
r_{dc}=−0.9, σ_c^2 = 2	0.990	0.793	6.1	3.2	−5.0	−2.4	1.1	0.8	51/130

[a] Spearman rank correlations were calculated between breeding value estimates from the B model and direct (left column) or total (right column) breeding value estimates from the GC-AR model

[b] Direct, competitive and total genetic responses from selection based on the B (left columns) and GC-AR (right columns) models were calculated following selection of around 5 % top individuals

[c] The number of common individuals selected from both the B and GC-AR models is given (numerator) for a total number of individuals selected by either model (denominator)

Table 4 Expected outcomes from individual selection for contrasting levels of genetic relatedness within a neighbourhood (i.e. mean levels of 0.11 and 0.003 corresponding to the simulated medium and very low relatedness scenarios, respectively), assuming 100 % survival, an intermediate value of the genetic variance for competitive additive effects (i.e. σ_c^2 = 5) and intermediate and high values of the genetic correlation between direct and competitive additive effects (i.e. r_{dc}= −0.6 and −0.9). The results were obtained from data simulated for a trait representing tree growth

True values of parameters	Spearman rank correlations[a]		Genetic response to selection[b]						Common selected individuals[c]
			Direct		Competitive		Total		
r_{dc}=−0.6, medium	0.946	0.415	5.7	0.7	−3.7	5.0	2.0	5.7	15/130
r_{dc}=−0.6, very low	0.971	0.204	4.7	0.3	−4.0	1.9	0.7	2.2	7/130
r_{dc}=−0.9, medium	0.945	−0.296	5.7	−2.4	−6.7	4.7	−1.0	2.3	0/130
r_{dc}=−0.9, very low	0.956	−0.681	4.7	−2.7	−5.7	3.5	−1.0	0.8	0/130

[a] Spearman rank correlations were calculated between breeding value estimates from the B model and direct (left column) or total (right column) breeding value estimates from the GC-AR model

[b] Direct, competitive and total genetic responses from selection based on the B (left columns) and GC-AR (right columns) models were calculated following selection of around 5 % top individuals

[c] The number of common individuals selected from both the B and GC-AR models is given (numerator) for a total number of individuals selected by either model (denominator)

patterns to those observed for 100 % survival in the effects of contrasting levels of relatedness on selection outcomes.

Discussion

Parameter estimates from fitting an incomplete model

In general, our results indicated that ignoring competitive effects at either genetic or non-genetic levels will lead to biases in (co)variance estimates of other components. Following Cappa and Cantet (2008), the expected covariance between the phenotypes of x and y can be expressed at the genetic level as:

$$A_{xy}\sigma_d^2 + \left[\sum_{i=1}^{n_x} f_{xw_i}A_{yw_i} + \sum_{j=1}^{n_y} f_{yz_j}A_{xz_j}\right]\sigma_{dc} + \left[\sum_{i=1}^{n_x}\sum_{j=1}^{n_y} f_{xw_i}f_{yz_j}A_{w_iz_j}\right]\sigma_c^2 \quad (15)$$

where focal tree x has n_x neighbours denoted by w_i, focal tree y has n_y neighbours denoted by z_j, A_{xy} is the additive relationship between x and y, A_{yw_i} (A_{xz_j}) is the additive relationship between focal tree y (x) and a neighbour of x (y), $A_{w_iz_j}$ is the additive relationship between a neighbour of x and a neighbour of y, and $f_{yz_j}(f_{xw_i})$ is the intensity of competition exerted by tree z_j (w_i) over y (x). In forest genetic tests, there are a finite number of families and multiple progeny per family. Consequently, under any planting design, it is highly probable that there will be some proportion of pairings of relatives x and y, where some neighbours of x are related to neighbours of y, or to y itself, and some neighbours of y are related to x. Thus, the coefficients of σ_{dc} and σ_c^2 are likely to be non-zero and, in models that do not take into account competition, competitive variance presents as increased residual variance.

Our results from the base model unequivocally demonstrate that the residual variance has absorbed the (co)variances arising from genetic and/or non-genetic competitive effects, and this absorption occurred irrespective of whether competition was primarily between or within families. In this sense, in conjunction with a single-tree plot design (low and very low relatedness levels), our base model produced an unbiased additive variance estimate, but a residual variance estimate that was biased upwards. However, in conjunction with a multiple-tree-line plot design (medium relatedness level), the base model yielded a downward bias in the estimate of the additive variance, and a residual variance estimate that was further biased upwards, relatively to the single-tree plot design, by an amount that matched the bias in the additive variance estimate. In preliminary testing of our models, we included a case scenario where competition was acting at the non-genetic level only: there was never an instance where residual competition variance was misconstrued as additive variance under a base model. Thus, competition at the residual level still presents a case of environmental covariance, but *per se* (i.e. in the absence of actual competitive additive effects) it will not have an influence on the genetic covariance among relatives (and consequently on the additive variance).

Why the pattern of bias for the additive variance estimated under the base model was only evident when there was a strong negative correlation (i.e. beyond −0.3) between direct and competitive additive effects is best understood by examining the nature of the genetic covariance between two relatives, as described in Eq. (15). To investigate the cause of the biases observed when r_{dc}=−0.9, we computed the average coefficients for σ_{dc} and σ_c^2 in Eq. (15) for all half-sib pairs and for the CP planting designs we have used. In the case of four-tree-

line plots, the values were 0.42 and 0.29, respectively. In the case of single-tree plots, the values were 0.15 and 0.21. Therefore, the value of the coefficient for σ_{dc} is higher than the coefficient for σ_c^2 under the four-tree-line plot design. The same tendency was observed when investigating the values of the coefficients for full-sib pairings. In addition, when r_{dc}=−0.9, the covariance between direct and competitive additive effects has a higher absolute value than the competitive additive variance (i.e. σ_{dc}=−12.7 versus σ_c^2 = 10.0). Consequently, the sum of the second and third terms in Eq. (15) has a negative value, and thus genetic covariances between half and full sibs are reduced to less than what they would be under no competitive effects. The net effect is a reduced additive variance. The "missing" additive variance, or the amount the additive variance has been reduced from the true value, is absorbed into the residual variance. The same occurs when r_{dc}=−0.6 but to a lesser extent. Cappa and Cantet (2008) have observed a similar phenomenon occurring when comparing models (with and without fitting genetic competition effects) applied to actual loblolly pine trial data, although average values of coefficients for the terms in Eq. (15) were not reported. In summary, for competition at the genetic level, it appears that the estimated additive variance will only be decreased under a base model when planting designs have the effect of emphasising the σ_{dc} component in the genetic covariance between any two relatives (as described in Eq. (15)). Such designs would emphasise intra-family competition over inter-family competition, examples of which are when families are planted in multiple-tree-line or block plots.

In simulating and analysing the data, competition at the residual level was modelled according to an autoregressive process, which may provide a residual covariance structure that is closer to the actual correlation structure in the data from field trials (Stringer 2006; Stringer et al. 2011). Autoregressive models reflect the existence of correlated errors between a focal individual and more distant neighbours, in addition to the adjacent neighbours described in Fig. 1 (Gilmour et al. 1997). Furthermore, autoregressive models provide a framework for fitting both environmental trend and residual competitive effects (Stringer 2006; Stringer et al. 2011). Stringer (2006) has explored an equal-roots second-order autoregressive (EAR2) process as an alternative to the AR1 for trials where, at the residual level, competition is dominant over local environmental trend. In preliminary analyses of diameter growth from some actual forest genetic trials, where competition at the residual level seemed to be dominant over local environmental trend (as suggested by negative estimates of the coefficients in $\boldsymbol{\alpha}$), the EAR2 did not appear to fit better the data than the AR1. A similar tendency has been generally found by Stringer (2006) in the analyses of 22 sugar cane data sets. Nevertheless, the results in the present study suggest that residual effects under the AR model may be absorbing indirect additive (co)variance components. In addition, when considerable genetic competition (e.g. as given in the simulated data by σ_c^2 = 10 and r_{dc} more negative than −0.3) is present but not modelled, accounting only for residual competition through a AR1 process may lead to a downward bias in additive variance estimates. Thus, it is recommended that the model used for data analysis always attempts to fit competition at both genetic and non-genetic levels.

Another incomplete model tested in our sequence fitted competition at the genetic, but not at the residual level. Under this situation, there is a chance for the environmental covariance to be misinterpreted as genetic covariance among relatives, and the estimates of all three additive (co)variance components (i.e. σ_d^2, σ_{dc} and σ_c^2) may become inflated as a consequence. Our results indicated that there is a stronger chance for this misinterpretation to occur when the level of relatedness within the neighbourhood of a focal individual increases.

As a final remark, it should be reminded that the results discussed above were obtained under scenarios assuming that environmental trend was negligible, and thus considered competition to be the most important source of residual autocorrelation. Consequently, our results may not strictly apply to situations where environmental trend is strong and dominant over competition at the residual level.

Ability to detect the most appropriate model

The correct model for data analysis is the GC-AR model under the scenarios defined in our simulation. Considerable genetic competition, as defined in the simulation by σ_c^2 = 10, could always be detected under the GC-AR model. The ability of this model to detect moderate to weak genetic competition (as defined by σ_c^2 = 5 and 2) was dependent on the magnitude of r_{dc}, and the levels of relatedness and mortality. Decreasing the relatedness level appeared to be an important factor in reducing the ability to detect genetic competition, with this reduction being magnified for decreased percentage of survival and for a small negative correlation between direct and competitive additive effects.

In a study by Bijma (2010b), it was found that, for increasing the precision of estimating IGE, schemes with animals grouped into two families were superior to schemes where animals were grouped at random. In our medium relatedness scheme, the majority of neighbourhoods were composed of three families. Under low and very low relatedness levels, neighbourhoods constituted a maximum of nine families. Though the parallels between animal breeding experiments and forest genetic trials are imperfect, our results support the finding in Bijma (2010b). In summary, our results indicated that, although the GC-AR model provided on average the best approximation to the simulated (co)variance parameters, it appeared to have a limited ability to detect weak levels of genetic competition (as defined by

$\sigma_c^2 = 2$ and $r_{dc}=-0.3$) under a level of very low relatedness within a neighbourhood. Thus, under these experimental conditions, trial and/or family sizes substantially greater than those used in our simulation may be needed for increasing the power to detect weak levels of genetic competition.

Impact on selection

The results of the present study indicated that ranking genotypes on the basis of estimated breeding values using models that ignore genetic competition does not correlate well with a ranking based on estimated total breeding values, which are a function of both direct and competitive additive effects. Cannell (1978) has postulated that selection of non-competitive genotypes leads to the greatest yields per unit area, and the terminology "crop ideotype" has been applied to such genotypes (Donald 1968). If selection response is measured in terms of forest productivity per unit area, then ranking and selection on the basis of total breeding values may lead to greater response, and thus it will be important to have a framework that allows the breeder to distinguish direct breeding values from competitive breeding values. Mixed model methodology and the ability to model IGE within this framework has now provided us with a suitable approach for identifying family differences in terms of conforming to a particular ideotype.

For a given level of relatedness, our results indicated that the total responses to selection (i.e. based on *TBV*) under the GC-AR model were in general greater than under the B model. This was achieved via selection of genotypes with favourable values for *CBV*, rather than genotypes with higher values for *DBV*. In fact, this implies selecting for genotypes that are cooperative rather than competitive, in which case a genotype with a positive *CBV* will benefit the performance of its neighbours. However, the potential of a population to respond to selection will be determined by the magnitude of the heritable variation (Bijma 2011) which, given the definition of *TBV* in Eq. (14) for the *i*th focal individual, may be described as:

$$\mathrm{Var}(TBV) = \sigma_d^2 + 2\left(\overline{n_R}\,\overline{f_R} + \overline{n_C}\,\overline{f_C} + \overline{n_D}\,\overline{f_D}\right)\sigma_{dc} + \left(\overline{n_R}\,\overline{f_R} + \overline{n_C}\,\overline{f_C} + \overline{n_D}\,\overline{f_D}\right)^2\sigma_c^2 \qquad (16)$$

indicating that the magnitude of heritable variance determining the change in the population mean trait value may be reduced when strong negative σ_{dc} and small σ_c^2 are occurring simultaneously. This is supported by our results as, for a given level of relatedness, the total responses to selection based on the B and GC-AR models became similar under $r_{dc}=-0.9$ and $\sigma_c^2 = 2$. Nevertheless, a breeder may also choose to maximise gain for *DBV*, subject to maintaining the mean *CBV* at a zero value.

As indicated by Bijma et al. (2007a), the degree of additive genetic relatedness among neighbourhood members is a key factor in determining the response to selection under the presence of competitive interactions among individuals. Increasing relatedness among interacting individuals will contribute to convert the covariance between phenotypic trait values and *TBV* of individuals into total heritable variation, and thus will increase the potential of the trait to respond to selection (Bijma et al. 2007a). Consequently, relatedness within a neighbourhood may help to lessen the adverse impact of the competitive (co)variance components on response to selection, hence contributing to reduce the negative consequences of competition (Bijma et al. 2007a; Ellen et al. 2007). This is sustained by our results in that total responses to selection under the GC-AR model increased with higher levels of relatedness within a neighbourhood. In this sense, the improvement in total response to selection with higher relatedness levels reflected an increase in the beneficial contribution from competitive effects to *TBV* relatively to the component attributed to direct effects. Our results also suggest that, although selection accuracy may have been improved by using BLUP to obtain estimates of *TBV*, relatedness among neighbourhood members remained an important factor to optimise selection response under sizeable negative r_{dc} (see also Muir et al. 2010). Yet, the impact of relatedness in determining the extent to which selection utilises total heritable variance may be reduced when competitive effects have a moderate to small additive variance and are strongly negatively correlated with direct additive effects (Bijma 2011). This is supported by our results under $\sigma_c^2 = 5$ as, when r_{dc} changed from −0.6 to −0.9, the advantage of the GC-AR model over the B model in total response to selection was diminished for medium relatedness relatively to very low relatedness.

Further issues

Number of neighbours with a significant competitive effect on a focal individual

An issue that we have not properly explored in the present study is the number of neighbours that have a significant competitive effect on a focal individual. In the simulated data, it was assumed that all surviving neighbours out of the eight possible first-order neighbours impart their competitive breeding values to the determination of the focal individual's phenotype. This may not always be the case. Cannell et al. (1984) have found competition to be asymmetrical, meaning that small trees suffer more from suppression, rather than large trees benefit from their dominance. This finding led these authors to suggest that light is the main environmental resource competed for. If this would be the case, and taking the geographic context in the southern hemisphere as an example, then it would not be unexpected

that only northern neighbours could be the significant competitors. It is even conceivable that second-order neighbours may be significant competitors. Related to this issue is the concept of dilution, which Bijma et al. (2007a) and Bijma (2010b, c) have defined as the decrease in the magnitude of IGE with group size, although there is no clear analogy with forest genetic trials. In essence, the whole field trial is the group over which there is a finite amount of resources distributed, perhaps non-uniformly, but within which there is a continuum of neighbourhoods. Thus, tree density per unit area may be more of an important factor in determining dilution than neighbourhood size. Yet, it is important to gauge the correct number of significant competitors because: (a) if this number is overestimated, there is a risk that genetic competition will not be detected, especially if the signal is already inherently weak; and (b) it has implications for the prediction of selection response, assuming that response is based on total breeding value. Consequently, an important step in the analysis of genetic competition will be to pursue a pre-analysis to evaluate the number of neighbours with a significant competitive effect on a focal individual, and this will comprise a topic for future research.

Ability to separate direct and competitive additive (co)variance components

Bijma et al. (2007b) derived an expression for the expected covariance between two focal individuals in different neighbourhoods (akin to Eq. (15)), and demonstrated that σ_c^2 could be separated from σ_d^2 only when the combined relatedness between the neighbourhood members of two given focal individuals differed from the relatedness between these two individuals themselves. Thus, the capacity of the information in the data to separate σ_d^2, σ_{dc} and σ_c^2 may be impaired if there is a large number of instances where the coefficients for σ_{dc} and/or σ_c^2 in Eq. (15) equal A_{xy}, which is the coefficient for σ_d^2.

Previous analyses from four-neighbour simulations, where competitive effects were assumed to originate mainly from row and column neighbours (i.e. assuming non-significant competitive effects for diagonal neighbours) revealed that, under the medium relatedness level, the ability to detect the correct GC-AR model was reduced relatively to the low relatedness level. This is the reverse of what occurred when all the eight first-order neighbours were considered as being significant competitors, although the poorest ability to detect the most appropriate model was observed under the very low relatedness level for both four- and eight-neighbour simulations. In order to explain the observed difference in the four- and eight-neighbour results, we have calculated the number of instances that the coefficients for σ_{dc} and σ_c^2 in Eq. (15) equalled 0.5 (when x and y are full sibs) or 0.25 (when x and y are half sibs), as a percentage of the total number of full- and half-sib pairings (Online Resource 3, Electronic supplementary material). Under the medium relatedness scenario, the relative number of instances in which the coefficients for σ_{dc} and σ_c^2 in Eq. (15) equalled 0.5 or 0.25 was larger for the four-neighbour simulations when compared with the eight-neighbour simulations. However, this tendency was only found for half-sib pairings under the low relatedness scenario (Online Resource 3). Therefore, these differences between relatedness scenarios in equivalence relationships may have led to differences in the capacity to separate σ_d^2, σ_{dc} and σ_c^2 and thus diminish the ability to identify the correct GC-AR model under the medium relatedness level for the four-neighbour simulations.

As the medium relatedness scenario in the four-neighbour simulations had a greater mean level than that in the eight-neighbour simulations (i.e. 0.21 versus 0.11), our results also suggest that an adequate separation of direct and competitive additive effects may require an optimum level of additive relatedness within a neighbourhood. Thus, although increasing relatedness among interacting individuals will improve the utilisation of heritable variance for response to selection, greater relatedness within a neighbourhood may also make it more difficult to estimate indirect genetic (co)variances. Indeed, previous studies have indicated that non-identifiable (Bijma et al. 2007b) or biased (Cheng et al. 2009) indirect (co)variances will appear when all neighbourhoods are composed of single families of half or full sibs. Nevertheless, the equivalence relationships mentioned above may not occur in a field trial when inter-row distance is different to inter-column distance ($p \neq 1$), or when a combination of diagonal and row or column neighbours comprises the significant competitors (see Online Resource 3). Given that the number and location of significant competitors to a focal tree is an empirical issue, and can only be determined once a pre-analysis step is undertaken, a precautionary measure to take when designing field trials is to have unequal inter-row and inter-column distances. This will ensure that the intensity of competition factors in the row and column directions will be different (assuming insignificant diagonal neighbours), which may improve the identifiability of (co)variance components in models fitting genetic competition (Cantet and Cappa 2008). In addition, the distance differential should not be so great as to cause dilution of competitive effects from trees in a different row or column.

Intensity of competition factors

Uniformity of scaling of competitive effects enabled by the restriction $\sum_{j=1}^{n_i} f_{ij}^2 = 1$ will account for differences in tree density per unit of area, both among neighbourhoods within a trial (e.g. due to differences in tree mortality) and across trials (e.g. due to differences in inter-tree spacing).

Competitive additive (co)variances are trial specific, as they may depend on the given population of genotypes tested at a given age. This may limit direct comparisons among trials. Nevertheless, differences among trials in competitive additive (co)variances, arising from different numbers of significant competitors affecting on average a focal tree, may be attenuated through the incorporation of the intensity of competition factors in models used for genetic evaluation.

Conclusion

In general, our results indicated that estimates of (co)variance components and predictions of responses to selection may be inaccurate when competition exists at the genetic level but is not accounted for by statistical models used in genetic evaluation. The underlying quantitative genetic model should allow the genetic variance of competitive effects and the genetic covariance between direct and competitive effects to become part of the heritable variance, which determines the full potential for a population to respond to selection. Considering their implications for tree improvement programmes (which typically have not been considering genetic competition effects), key conclusions to be drawn from the current work can be summarised as follows:

- If genetic competition is present but not modelled, variance estimates obtained for additive genetic and residual effects may be biased. In this context, we found a downward bias in the additive variance estimate when the actual genetic correlation between direct and competitive effects became more negative (i.e. beyond −0.3), with a propensity for the bias to occur under increasing levels of genetic relatedness within a neighbourhood. In addition, there was a general tendency for inflated variance estimates in residual terms. Under models that fitted competition at the genetic level only, we also observed biased (co)variance estimates for direct additive, competitive additive and residual effects. Thus, the model used for data analysis should always attempt to fit competition at both genetic and non-genetic levels.
- The ability to detect low to moderate magnitudes of genetic competition effects (as given in our study by ratios of competitive additive variance to direct additive variance of 0.1 and 0.25) may be reduced as levels of genetic relatedness within a neighbourhood decrease, with the reduction being magnified with lower survival and less negative values of the actual genetic correlation between direct and competitive effects.
- When competition is present at both genetic and non-genetic levels, accounting only for non-genetic competition may lead to selection outcomes that are similar to those obtained by ignoring competition at both levels, as suggested in our study by the high agreement in individual rankings. However, allowing for a genetic basis to competitive ability will be important to optimise artificial selection schemes, as total response to selection based on breeding values estimated from a model ignoring genetic competition may actually be lower than when based on total breeding value estimates from an appropriate model incorporating competition at both genetic and non-genetic levels. We observed that the disparity in terms of total responses to selection was greater for higher ratios of competitive additive variance to direct additive variance and for increasing levels of genetic relatedness within a neighbourhood. Nevertheless, our results also suggested that the heritable variance will decrease when a strong negative genetic correlation between direct and competitive effects occurs simultaneously with moderate to low ratios of competitive additive variance to direct additive variance, hence constraining the change in the population mean trait value. This will reduce the difference in total response to selection between a model ignoring genetic competition and the appropriate model.

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