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Predicted rates of inbreeding with additive maternal effects

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Summary

Maternal effects play an important role in fitness and other aspects of individual performance in many species, particularly mammalian, yet their impact on genetic variation within species and its rate of loss during selection has been neglected. In this paper we extend the theory of expected long-term genetic contributions to include maternal effects, and tested the accuracy of predicted rates of inbreeding for populations under mass selection by comparison with simulations. The model includes selective advantages of direct and maternal additive genetic effects, and also the selective advantage of a common maternal environmental effect. The population structures investigated had a fixed number of dams per sire and fixed family size. Most prediction errors of the rate of inbreeding (ΔF) were less than 8% of the simulated means and were lower in magnitude than the prediction errors of genetic gain (ΔG). The predictions of ΔG from contributions equalled previously published predictions. A variation in maternal genetic effects resulted in a much larger ΔF than for an equally sized variation in common maternal environmental effects. For a fixed genetic gain, ΔF increased as the maternal heritability increased. The influence of family size, mating ratio and age structure on ΔF was greater with maternal effects than with only direct genetic effects included. In conclusion, maternal effects can be a very important aspect to consider when predicting ΔF in populations under selection, and the developed methodology gives good predictions.

1. Introduction

Maternal effects play an important role in fitness and other aspects of individual performance in many species, particularly mammalian, and their impact on selection response has been investigated extensively. However, their impact on genetic variation and its rate of loss during selection has received little attention. This rate of loss is measured by the rate of inbreeding (ΔF) and a better understanding of how maternal effects influence ΔF will inform and improve the design of animal breeding schemes and genetic conservation programmes of wild populations subjected to natural selection.

In phenotypic models including maternal effects, the dam affects her offspring's phenotype in two ways. Firstly, her genetic contribution to the offspring's genes influences the phenotype of the offspring. Secondly, her ability to contribute to the development of the offspring's phenotype is modelled as a phenotypic component attributable to the dam. This latter part of the model is the maternal effect, and several different models of this phenomenon have been developed; these have been reviewed by Kirkpatrick & Lande (1989). One of the main differentiating factors among these models is whether the trait measured in the offspring is regarded as the same trait (e.g. Falconer, 1965) or a different trait (Willham, 1963, 1972) from that describing the dam's ability to contribute to the offspring's development. Intermediate models have also been developed where the environmental component of the mother's ability is correlated with the non-maternal environmental component of the phenotype measured in the offspring (Mueller & James, 1985; Riska *et al.*, 1985). The studies reviewed by Kirkpatrick & Lande (1989) focused on the selection

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Table 1. *Notation of frequently used parameters*

ΔF, ΔF_L	Annual rates of inbreeding and rates of inbreeding per generation
ΔG_{dir}, ΔG_{mat}	Direct and maternal genetic gain per year
$\Delta \mathbf{G}$, ΔG	Vector of annual genetic gain and total genetic gain ($\Delta G_{dir} + \Delta G_{mat}$)
P_i, A_i, E_i	Phenotype, additive direct genetic effect and environmental effect
M_d, C_d	Additive maternal genetic effect and environmental maternal effect of the dam
$\mathbf{s}_{i(q)}$	Vector of selective advantages for individual i in category q equal to $(A_i\ M_i\ C_i)^T$; mean over all selected in category q is $\bar{\mathbf{s}}_q$
$r_{i(q)}$, $u_{i(q)}$, $\mu_{i(q)}$	Long-term genetic contribution, expected long-term genetic contribution and linear predictor of long-term genetic contributions
$\boldsymbol{\alpha}_q$, $\boldsymbol{\beta}_q$	Vectors of the coefficients for $\mu_{i(q)}$
$\boldsymbol{\lambda}_{pq}$	Regression coefficients of proportion selected in category p on $\mathbf{s}_{i(q)}$ for parents in category q
$\boldsymbol{\pi}_{pq}$	Regression coefficients of $\mathbf{s}_{j(p)}$ on $\mathbf{s}_{i(q)}$ for parents in category q
a_i, m_i, $\mathbf{g}_{i(p)}$	Direct and maternal Mendelian sampling terms of individual i, and vector of Mendelian sampling terms equal to $(a_i\ m_i\ 0)^T$
h^2_A, h^2_M, h^2_W	Direct, maternal and Willham heritabilities
c^2, ρ, σ^2_I	Common environmental variance, direct-maternal genetic correlation, and phenotypic variance
N_m, N_f, d	Number of male parents, number of female parents, and dams per sire (N_f/N_m)
n_o, T	Total number of offspring per dam, and number of candidates available for selection in each sex
$\mathbf{N}$	Vector of number of individuals in each category
i_p, k_p, i	Intensity of selection and variance reduction term in sex p, and mean selection intensity
Subscripts	
p, q, $i(p)$	Indicators of categories, $i(p)$ denotes individual i in category p
m, f	Indicators of male and female
i, d, s	Indicators of individual, dam and sire

response in populations, and did not investigate the influence of maternal effects on ΔF.

The partition of the covariance between offspring and dam among the direct additive effects and maternal effects, both inherited and environmental, has been shown to influence genetic gain (Willham, 1963), but this partition will also be critical in determining ΔF. This is because a mother that is more able to take good care of her offspring will tend to have more offspring selected under mass selection, and so increase the co-selection of related individuals. Thus the maternal effect is a selective advantage for the dam. Wray *et al.* (1994) considered the impact of maternal effects on predicted ΔF with directional selection, but assumed that the maternal selective advantage was not inherited, i.e. a daughter's maternal effect was independent of its dam's maternal effect. However, ΔF has been shown to increase when selective advantages are inherited (Robertson, 1961; Wray & Thompson, 1990).

Woolliams & Bijma (2000) showed that ΔF could be predicted from the expected genetic contributions of individuals conditional upon their selective advantages. These expected contributions can be calculated for mass and index selection with direct additive effects using general methods developed by Woolliams *et al.* (1999), and the generality of their approach suggested that inherited maternal effects could also be incorporated into their model. This incorporation would remove a major limitation of the methods of Wray *et al.* (1994).

The development of accurate predictions of genetic gain and rates of inbreeding is desirable since the alternative is to use stochastic simulations for all predictions, which is time-consuming and specific to situations simulated, and restricts extrapolation, interpretation and insight. Therefore our aim is to extend the theory of expected long-term genetic contributions to include maternal effects and, thereby, investigate the influence of maternal effects on predicted ΔF for populations under mass selection, and to test the accuracy of these predictions by comparison with simulations. The development is initially based upon the phenotypic model of maternal effects developed by Willham (1963, 1972), since this is relatively simple and is the one most commonly used for estimation of genetic (co)variances and breeding values.

2. Methods

The notation for frequently used parameters of the model is given in Table 1.

(i) *Population models and parameters*

We studied a model for maternal effects where the phenotype of individual i is composed of an individual component, $P_{i,\,\text{self}}$, and a component determined by the dam, $P_{i,\,\text{maternal}}$ (Willham, 1963):

$$P_i = P_{i,\,\text{self}} + P_{i,\,\text{maternal}}.$$

Each of the genetic subcomponents of each phenotypic component is assumed to have Mendelian inheritance determined by an infinite number of loci,

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each having an infinitesimal effect (the infinitesimal model; Fisher, 1918), with:

$$P_{i,\,\text{self}} = A_i + E_i$$
$$P_{i,\,\text{maternal}} = M_d + C_d,$$

where A_i and M_d are the additive direct genetic effect and additive maternal genetic effects, respectively, and where subscripts i, s, d denote belonging to individual i, sire s and dam d. C_d will be referred to as the *common environmental effect* and is assumed to represent environmental effects related to the dam's own attributes or other influences on its offspring that are shared by the maternal sibs alone. Thus effects common only to specific litters have been ignored here. C_d was the sole maternal component modelled by Wray *et al.* (1994). The terms A_i, E_i, M_d and C_d are assumed to be mutually independent with the exception of A_i and M_d. Furthermore, for a female i, E_i (the environmental component specific to her own performance) is independent of her maternal contribution to her offspring. This latter assumption distinguishes the model above from that of Falconer (1965); see Discussion.

For each genetic component, inheritance is modelled by:

$$A_i = \tfrac{1}{2}A_s + \tfrac{1}{2}A_d + a_i$$

where $V(a_i) = \tfrac{1}{2}h_A^2$, and

$$M_i = \tfrac{1}{2}M_s + \tfrac{1}{2}M_d + m_i$$

where $V(m_i) = \tfrac{1}{2}h_M^2$.

An unrelated and randomly selected base population is assumed in which $V(P_i)$ = the total phenotypic variance = 1, h_A^2 = total direct additive genetic variance, and h_M^2 = total maternal additive genetic variance. Within the base population $Cov(A_i, M_i) = \rho h_A h_M$, where ρ is the direct-maternal genetic correlation, and in all subsequent generations (neglecting inbreeding) $Cov(a_i, m_i) = \tfrac{1}{2}\rho h_A h_M$. Using these assumptions and denoting $V(C_d)$ by c^2, gives

$$V(E) = 1 - (h_A^2 + h_M^2 + \rho h_A h_M + c^2).$$

Using this model, Willham (1972) showed that for mass selection in the base population, the ratio of the response to the selection differential applied is given by $h_W^2 = h_A^2 + \tfrac{3}{2}\rho h_A h_M + \tfrac{1}{2}h_M^2$. We refer to this fraction as the *Willham heritability*.

(ii) *Population structures*

Each sire was mated at random to a fixed number of d dams, and each dam produced n_o full-sibs (hierarchical mating) with equal numbers of males and females. For discrete generations the numbers of male and female parents were N_m and N_f, respectively, with the mating ratio $d = N_f/N_m$. The phenotype used for selection was assumed measurable in both sexes, with the same genetic and environmental parameters. Parents were selected by ranking the phenotypes within each sex, and selecting the required number of individuals with the highest rank. For overlapping generations, the individuals were ranked within age classes and were selected each year. There was no reordering of ranking between ages.

(iii) *Expected genetic contributions, gain and rate of inbreeding*

In this section predictions of genetic gain and rates of inbreeding are derived for discrete generations using the concept of long-term genetic contributions. The basic approach follows Woolliams & Bijma (2000) and Bijma *et al.* (2000), and uses similar notation. The extension to overlapping generations is given in the Appendix. All predictions were derived for a population after several generations of selection (Woolliams *et al.*, 1999) where equilibrium genetic (co)variances had been attained (Bulmer, 1971).

The long-term genetic contribution, $r_{i(q)}$, of individual i in category q born at t_1 is defined as the proportion of genes present in individuals in cohort t_2 deriving by descent from i, where $(t_2 - t_1) \to \infty$ (Woolliams *et al.*, 1993).

The long-term genetic contribution of an individual i depends on the category that the individual belongs to, where a category (in the present paper) is defined by the individual's sex and age. Furthermore, in a selected population superior parents are likely to have more offspring selected than average individuals. The superiority is defined by selective advantages (Woolliams *et al.*, 1999) and the model assumes that the selective advantages of an individual i are given by A_i, M_i and C_i, since these three components, and the corresponding terms for its mate(s), influence the selection of future descendants of individual i. For females, all of these will influence selection of offspring and A_i and M_i will influence selection of later descendants. For males, A_i will influence selection of their offspring and later descendants, M_i does not influence the selection of their offspring but will influence selection through their selected female descendants, whereas C_i does not influence the selection of their offspring or descendants and need not be defined for a male.

Let $\mathbf{s}_{i(q)}$ be a vector of selective advantages for individual i in category q so that $\mathbf{s}_{i(q)} = (A_{i(q)}\ M_{i(q)}\ C_{i(q)})^T$, where superscript T denotes the transpose of matrices. The expected long-term genetic contribution $u_{i(q)}$ is then defined as $u_{i(q)} = \mathrm{E}(r_{i(q)} | \mathbf{s}_{i(q)} - \bar{\mathbf{s}}_q)$, where $\bar{\mathbf{s}}_q$ is the average of selected individuals in

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category q. The linear predictor of $u_{i(q)}$ is:

$$\mu_{i(q)} = \alpha_{i(q)} + \boldsymbol{\beta}_q^T(\mathbf{s}_{i(q)} - \bar{\mathbf{s}}_q) \quad (1)$$

where $\alpha_m = 1/(2N_m)$ and $\alpha_f = 1/(2N_f)$. For discrete generations, solutions for the coefficients in $\boldsymbol{\beta}_q^T$ are obtained from Woolliams *et al.* (1999):

$$\begin{pmatrix} N_m\boldsymbol{\beta}_m \\ N_f\boldsymbol{\beta}_f \end{pmatrix} = \frac{1}{4}\left(\mathbf{I} - \frac{1}{2}\begin{pmatrix} \boldsymbol{\pi}_{mm} & \boldsymbol{\pi}_{fm} \\ \boldsymbol{\pi}_{mf} & \boldsymbol{\pi}_{ff} \end{pmatrix}\right)^{-1}\begin{pmatrix} \boldsymbol{\lambda}_{mm} + \boldsymbol{\lambda}_{fm} \\ \boldsymbol{\lambda}_{mf} + \boldsymbol{\lambda}_{ff} \end{pmatrix} \quad (2)$$

where $\mathbf{I}$ is the 6×6 identity matrix, $\boldsymbol{\pi}_{pq}$ are 3×3 submatrices containing regression coefficients of selective advantages of selected progeny in sex p on selective advantages of parents in sex q, $\boldsymbol{\lambda}_{pq}$ are 3×1 submatrices containing regression coefficients of proportion selected in sex p on selective advantages of parents in sex q, and subscripts m and f denote males and females, respectively. Following equations (A5) and (A6) in the Appendix for Willham's phenotypic model, the estimates of $\boldsymbol{\lambda}_{pq}$ and $\boldsymbol{\pi}_{pq}$ are given by:

$$\boldsymbol{\lambda}_{p,m} = \frac{i_p}{2\sigma_I}(1 \quad 0 \quad 0)^T$$

$$\boldsymbol{\lambda}_{p,f} = \frac{i_p}{2\sigma_I}(1 \quad 2 \quad 2)^T$$

$$\boldsymbol{\pi}_{p,m} = \frac{1}{2}\left[\begin{pmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 0 \end{pmatrix} - \frac{k_p}{\sigma_I^2}\begin{pmatrix} x & 0 & 0 \\ y & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}\right]$$

$$\boldsymbol{\pi}_{p,f} = \frac{1}{2}\left[\begin{pmatrix} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 0 \end{pmatrix} - \frac{k_p}{\sigma_I^2}\begin{pmatrix} x & 2x & 0 \\ y & 2y & 0 \\ 0 & 0 & 0 \end{pmatrix}\right]$$

where $x = Cov(P_i, A_i)$, $y = Cov(P_i, M_i)$, i_p is the selection intensity in sex p, k_p is the variance reduction term in sex p, and σ_I is the phenotypic standard deviation.

The annual genetic gain (ΔG) was predicted by

$$\Delta\mathbf{G} = N_m\mathrm{E}[r_{i(m)}\mathbf{g}_{i(m)}] + N_f\mathrm{E}[r_{i(f)}\mathbf{g}_{i(f)}] \quad (3)$$

where $\mathbf{g}_{i(p)}$ is the vector of Mendelian sampling terms corresponding to the selective advantages in $\mathbf{s}_{i(p)}$, i.e. $\mathbf{g}_{i(p)} = (a_{i(p)}, m_{i(p)}, 0)^T$ (Woolliams *et al.*, 1999). The expectations of $r_i\mathbf{g}_i$ are given in equation (A9) in the Appendix.

The rate of inbreeding per year, ΔF, was predicted using the results of Woolliams & Bijma (2000):

$$\mathrm{E}[\Delta F] = \frac{1}{2}\sum_{q=m,f} N_q\mathrm{E}(u_{i(q)}^2) - \frac{1}{8T} \quad (4)$$

where the last term is a correction factor for fixed family size, with T equal to the total number of progeny of each sex before selection, and $u_{i(q)}$ includes the selective advantages of the mates so that:

$$\mathrm{E}(u_{i(m)}^2) = \alpha_m^2 + (1 - 1/N_m)\boldsymbol{\beta}_m\mathbf{V}_{mm}^*\boldsymbol{\beta}_m^T + d(1 - 1/N_f)\boldsymbol{\beta}_f\mathbf{V}_{ff}^*\boldsymbol{\beta}_f^T \quad (5a)$$

$$\mathrm{E}(u_{i(f)}^2) = \alpha_f^2 + (1 - 1/N_f)\boldsymbol{\beta}_f\mathbf{V}_{ff}^*\boldsymbol{\beta}_f^T + \frac{1}{d^2}(1 - 1/N_m)\boldsymbol{\beta}_m\mathbf{V}_{mm}^*\boldsymbol{\beta}_m^T \quad (5b)$$

where $\mathbf{V}_{qq}^*$ is the (co)variance matrix of selective advantages *after* selection in sex q as defined in the Appendix.

The method implies that ΔF is an equilibrium value. The rates of inbreeding for the alleles defined in an arbitrary base will be reached after a small number of generations (Woolliams & Bijma, 2000), if equilibrium genetic covariances are assumed.

(iv) *Stochastic simulations*

The stochastic simulation programme described by Bijma & Woolliams (1999) was developed to include the maternal effects model described above for a population undergoing mass selection with fixed family size.

Since the randomly selected and unrelated base population was assumed to have phenotypic variance = 1, the genetic covariance matrix for the direct and maternal effects in the base population was

$$\mathbf{V_0} = \begin{pmatrix} h_A^2 & \rho h_A h_M \\ \rho h_A h_M & h_M^2 \end{pmatrix},$$

for which the lower matrix of the Cholesky factorization is

$$\mathbf{L} = \begin{pmatrix} h_A & 0 \\ \rho h_M & \sqrt{h_M^2(1-\rho^2)} \end{pmatrix}.$$

Additive direct and maternal genetic effects of the sires and dams of the base population were simulated as $(A_i, M_i)^T = \mathbf{Lx}$, where $\mathbf{x}$ is a vector of two independent random N(0, 1) numbers. The phenotypic value of individual i was then calculated as $P_i = A_i + M_d + C_d + E_i$, where C_d was sampled from N(0, c^2) and E_i was sampled from N(0, $V(E)$).

In subsequent generations the procedure was similar, but additive effects were calculated as $A_i = \frac{1}{2}A_s + \frac{1}{2}A_d + a_i$ and $M_i = \frac{1}{2}M_s + \frac{1}{2}M_d + m_i$, with the covariance between Mendelian sampling terms $Cov(a_i, m_i) = \frac{1}{2}\rho h_A h_M$. Inbreeding was neglected in the calculation of Mendelian sampling terms. Thus sampling was

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conducted using

$(a_i, m_i)^T = \sqrt{0{\cdot}5}\mathbf{Lx}$.

For the calculation of genetic contributions, the ancestor cohort t_1 was set to 10 and the descendent cohort t_2 was 20. The long-term genetic contribution r_i of an ancestor in cohort t_1 to individuals in cohort t_2 was obtained by summing contributions via all pedigree paths leading from i to individuals in t_2.

For each replicate observed, genetic contributions were analysed using the linear model: $r_i = \alpha + \beta_1(A_i - \overline{A}) + \beta_2(M_i - \overline{M}) + \beta_3(C_i - \overline{C}) + e_i$, and β_i was then estimated from multiple regression of r_i on the selective advantages. Asymptotic rates of annual direct and maternal genetic gain were calculated as $\Delta G_{dir} = (\overline{A}_{t_2} - \overline{A}_{t_1})/(t_2 - t_1)$ and $\Delta G_{mat} = (\overline{M}_{t_2} - \overline{M}_{t_1})/(t_2 - t_1)$. Inbreeding coefficients of individuals in cohorts t_1 and t_2 were calculated from the simulated pedigree, using the algorithm of Meuwissen & Luo (1992). Rates of inbreeding per year were calculated as $\Delta F = 1 - \left(\frac{1-\overline{F}_{t_2}}{1-\overline{F}_{t_1}}\right)^{(t_2 - t_1)^{-1}}$. Results were averaged over 500 replicates to give ΔF_{sim}.

(v) *Structure of population parameters investigated*

To explore both the properties of the genetic model and the validity of the predictions we considered five sets of studies with specific objectives. This helped to overcome the problems associated with the large number of parameters that could be varied simultaneously, e.g. h_A^2, h_M^2, ρ, c^2, N_m, N_f, n_o, age structure.

Case I. The origin of maternal effects. Parameters h_M^2, c^2 and n_o were varied to exemplify differences between inherited and non-inherited maternal effects in determining ΔF. In this case $h_A^2 = 0$, $N_m = 25$, and $d = 1$.
Case II. Partitioning a constant ($h_A^2 + h_M^2$). The impact of h_A^2 and h_M^2 on ΔF was examined for fixed $h_A^2 + h_M^2$ ($= 0{\cdot}4$) and with $\rho = 0$.
Case III. Partitioning a constant h_W^2. The differential impact of h_A^2, h_M^2 and ρ on ΔF was examined when these parameters were constrained to give a fixed $h_W^2 = 0{\cdot}2$. Thus, h_M^2 was varied from 0 to 0·4 as in case II but for fixed ΔG. The population structures were varied so that with random selection and mating and Poisson family size ΔF was expected to be 0·01.
Case IV. Varying h_W^2. The impact of varying h_W^2 on ΔF was examined. The values of h_A^2, h_M^2 and ρ were constrained by using the results from the review of genetic parameters in beef cattle by Mohiuddin (1993): $h_M^2/h_A^2 = 0{\cdot}6$, $\rho = -0{\cdot}15$. In addition, it was assumed that $c^2 = 0{\cdot}07$ (Mohiuddin, 1993).
Case V. Overlapping generations. The influence of age structure on rates of inbreeding per generation (ΔF_L) was investigated in a population with two age classes, following the structure described in table 2 of Bijma *et al*. (2000). Differences between direct and maternal effects were compared for $N_m = 20$ and $N_f = 20$, and the age distribution was varied for one sex at a time. The proportion of individuals in age class 2 of each sex was varied from $p_2 = 0$ to 1; so $\mathbf{N} = [20,\ 0,\ 20(1-p_2),\ 20p_2]^T$ when the dam distribution was varied, and $\mathbf{N} = [20(1-p_2),\ 20p_2,\ 20,\ 0]^T$ when the sire distribution was varied.

Table 2. *Algebraic expressions for male and female* β *for each separate selective advantage when the variances of the other two selective advantages are zero* (*derived from eq. [2]*)

	Male β	Female β
A_i [a]	$\frac{1}{\Delta_1} \times \frac{i}{2N_m\sigma_I}$	$\frac{1}{\Delta_1} \times \frac{i}{2N_f\sigma_I}$
M_i [b]	$\frac{1}{2\Delta_2} \times \frac{i}{2N_m\sigma_I}$	$\frac{3}{2\Delta_2} \times \frac{i}{2N_f\sigma_I}$
C_i	0	$\frac{i}{2N_f\sigma_I}$

[a] $\Delta_1 = 1 + \frac{V(A_i)}{2\sigma_I^2}(k_f + k_m)$.

[b] $\Delta_2 = 1 + \frac{V(M_d)}{8\sigma_I^2}(3k_f + k_m)$.

For all cases the results of the simulations were compared with predictions of ΔG and ΔF given above. In the case of ΔG the predictions were also compared with those from 'conventional' selection theory (see e.g. Van Vleck, 1993):

$$\Delta G_{dir} = \left[V(A_i) + \tfrac{1}{2}Cov(A_d,\ M_d)\right] i/\sigma_I \qquad (6a)$$

$$\Delta G_{mat} = \left[Cov(A_i,\ M_i) + \tfrac{1}{2}V(M_d)\right] i/\sigma_I \qquad (6b)$$

where i is the mean intensity of selection, and subscripts i and d denote the individual and its dam, respectively. No substitution has been made for h_A^2, h_M^2 and ρ, since the expressions given above will hold for both the base and the equilibrium genetic parameters (Bulmer, 1971). The term 'genetic gain' refers to $\Delta G = \Delta G_{dir} + \Delta G_{mat}$, unless stated otherwise.

3. Results

(i) *Case I. The origin of maternal effects*

This case was explored quantitatively by deriving the algebraic expressions of β (Table 2). For females the ratio between genetic and environmental β is within the range 1·0 to 1·5. Hence, a genetic maternal effect has greater impact on the long-term genetic contributions than an environmental maternal effect. This is in accordance with the predicted ΔF in Table 3, where maternal effects that were inherited had greater

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Table 3. *Predicted rates of inbreeding compared with simulations for populations with $N_f=25$, $d=1$, varying number of offspring per dam, n_o, and varying values of the variance of common maternal environment, c^2 and maternal heritability, h^2_M (and $h^2_A=0$). Standard errors of simulated rates of inbreeding were less than 3% of ΔF_{sim}*

		$n_o=4$		$n_o=8$		$n_o=50$	
c^2	h^2_M	ΔF_{pred} [a]	Error %[d]	ΔF_{pred} [b]	Error %	ΔF_{pred} [c]	Error %
0·1	0	0·0081	+3	0·0103	+1	0·0142	−2
0	0·1	0·0089	+3	0·0122	+3	0·0197	+4
0·2	0	0·0087	+2	0·0119	+3	0·0189	−7
0	0·2	0·0101	+6	0·0151	+3	0·0277	0
0·4	0	0·0099	+4	0·0150	+1	0·0276	−23
0	0·4	0·0121	+10	0·0198	0	0·0401	−8
0·6	0	0·0112	+5	0·0181	−2	0·0365	−36
0	0·6	0·0137	+8	0·0234	−7	0·0496	−19
1	0	0·0163	−2	0·0243	−40	0·0543	+184
0	1	0·0163	−2	0·0291	−13	0·0640	+235

[a] $\Delta F_{pred}=0{\cdot}0075$ for random selection ($h^2_W=0$).
[b] $\Delta F_{pred}=0{\cdot}0088$ for random selection.
[c] $\Delta F_{pred}=0{\cdot}0098$ for random selection.
[d] $100\% \times (\Delta F_{pred}-\Delta F_{sim})/\Delta F_{sim}$.

effect on rates of inbreeding than those of equal size but environmental origin. An additional component to this increase in ΔF was that $\beta>0$ for males, unlike the environmental case. Table 2 shows the regression on M_i was $3/d$ times greater in females than in males, i.e. was equal in magnitude when $d=3$ and greater when $d>3$.

For low to moderate litter sizes and maternal effects, the prediction accuracy of ΔF was approximately the same as the prediction errors given by Bijma *et al.* (2000) for additive direct effects only. However, ΔF could not be predicted for large maternal variances and large litter sizes. In the model for predicting ΔF it was assumed that the expected long-term contributions were linearly related to the selective advantages (eq. 1). However, in the extreme case of Table 3 ($n_o=50$, $c^2=1$ or $h^2_M=1$) the dam with the highest maternal effect would have all its offspring selected and all other dams would have no offspring selected. Consequently, the assumption of linearity was severely violated and predictions were poor.

For random selection and Poisson variance of family size, the numbers of sires and dams in the present case ($N_m=N_f=25$) correspond to $\Delta F=0{\cdot}01$, which has been suggested as a maximum acceptable level (see Franklin, 1980; Meuwissen & Woolliams, 1994). However, for mass selection and moderate variances of maternal effects, the values of ΔF exceeded 0·01, even though the family size was fixed (Table 3). Furthermore, ΔF was increased by more than 20% for moderate h^2_M compared with c^2 of equal size.

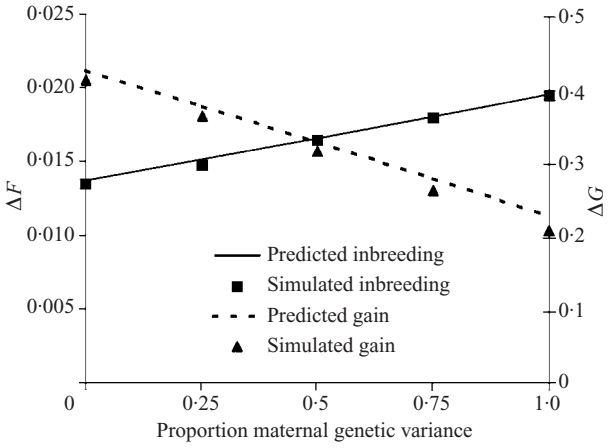

Fig. 1. Rates of inbreeding and total genetic gain for a fixed sum of direct and maternal genetic variance equal to 0·4 (i.e. $h^2_A+h^2_M=0{\cdot}4$, $\rho=0$). The proportion of maternal genetic variance is equal to $h^2_M/(h^2_A+h^2_M)$. Total genetic gain is the sum of direct and maternal gain. $N_f=25$, $n_o=8$, and $d=1$.

(ii) *Case II. Partitioning a constant* ($h^2_A+h^2_M$)

ΔF increased and ΔG decreased when the proportion of the total additive genetic variance attributed to maternal effects increased with $\rho=0$, as shown in Fig. 1. The prediction errors of ΔF (<3%) were smaller than the prediction errors of ΔG (<9%), where the predictions of ΔG equalled conventional ones (difference <0·7% of ΔG). The maximum prediction error was obtained for $h^2_M=0{\cdot}4$, and ΔG was over-predicted as expected due to finite family size and the intra-family correlation (see Hill, 1976; Meuwissen, 1991).

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Table 4. *Predicted rates of inbreeding for different combinations of h^2_A, h^2_M and ρ, in the base population with $h^2_W = 0{\cdot}2$ ($c^2 = 0$). Standard errors of simulated rates of inbreeding were less than 2% of ΔF_{sim}*

			$N_m = 25$, $d = 1$, $n_o = 4$		$N_m = 25$, $d = 1$, $n_o = 8$		$N_m = 15$, $d = 4$, $n_o = 8$	
h^2_A	h^2_M	ρ	ΔF_{pred} [a]	%Error[d]	ΔF_{pred} [b]	%Error	ΔF_{pred} [c]	%Error
0·2	0	0	0·0091	+3	0·0125	+3	0·0141	+4
0·1	0·1	0·33	0·0100	+5	0·0147	+4	0·0154	+1
0·2	0·2	−0·33	0·0103	+6	0·0154	+4	0·0160	+1
0·1	0·2	0	0·0106	+8	0·0162	+3	0·0164	+1
0	0·4	0	0·0121	+10	0·0198	0	0·0185	−6

[a] The predictions of ΔG varied within 0·144–0·146, $\Delta F_{pred} = 0{\cdot}0075$ for random selection ($h^2_W = 0$).
[b] The predictions of ΔG varied within 0·226–0·229, $\Delta F_{pred} = 0{\cdot}0088$ for random selection.
[c] The predictions of ΔG varied within 0·287–0·290, $\Delta F_{pred} = 0{\cdot}0095$ for random selection.
[d] $100\% \times (\Delta F_{pred} - \Delta F_{sim})/\Delta F_{sim}$.

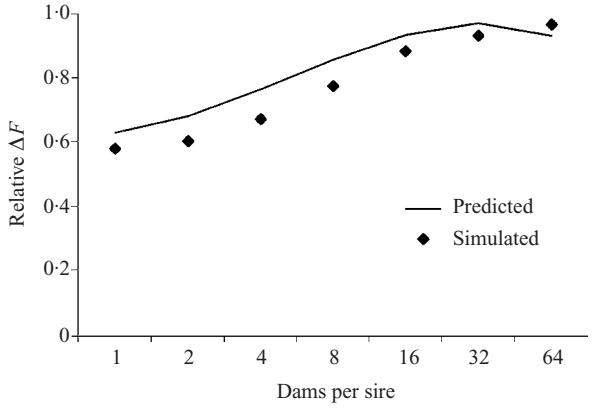

Fig. 2. Dams per sire related to relative ΔF. Relative ΔF is the ratio of the rates of inbreeding when h^2_W is made up of direct effects ($h^2_A = 0{\cdot}2$, $\rho = 0$) compared with when it only includes maternal effects ($h^2_M = 0{\cdot}4$, $\rho = 0$). $N_f = 64$, $n_o = 8$, and N_m varied from 64 to 1.

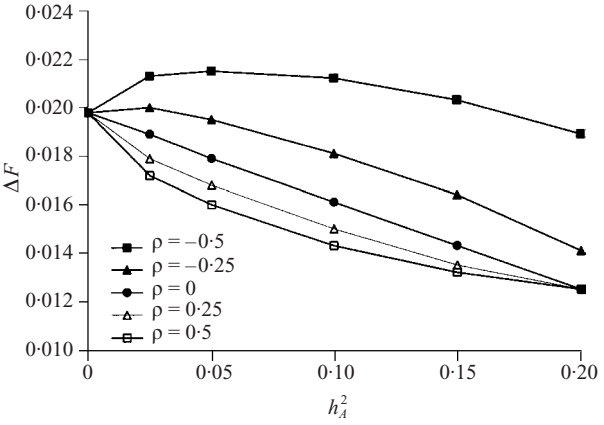

Fig. 3. Relationship between h^2_A and predicted ΔF for fixed $h^2_W = 0{\cdot}2$ ($c^2 = 0$) and different values of ρ. $N_f = 25$, $n_o = 8$, and $d = 1$.

Table 2 also shows the magnitude of β when comprised of either pure direct effects or maternal effects. For similar population structure and the same equilibrium heritability (either direct or maternal), the ratio β_M/β_A was approximately 0·5 for males and 1·5 for females. Thus, the long-term contributions are more evenly spread among males for maternal effects than for direct additive effects, whereas the distribution of long-term contributions among female parents is more varied for maternal effects than for direct effects.

(iii) *Case III. Partitioning a constant h^2_W*

In this case ΔG was constant within each population structure by studying a constant h^2_W. The rates of inbreeding increased when the component of h^2_W due to maternal genetic effects increased (Table 4), because there is an increase in the co-selection of full-sib families when maternal effects increase. Accordingly, the increment of ΔF with h^2_M was smaller when the number of offspring per dam was reduced. Furthermore, the increase in ΔF was also smaller when the number of dams per sire was increased, because for a high mating ratio ($d = N_f/N_m$) the influence of the maternal effects diminishes and the male part of ΔF dominates. In the case where there are only maternal genetic effects $\beta_f/\beta_m = 3/d$ (Table 2), and consequently the selective advantages of each individual female have less impact on ΔF as d becomes higher. This is in accordance with the results in Fig. 2. The ratio between the rates of inbreeding when h^2_W is made up of only direct effects compared with only maternal effects, increased for low mating ratios and was close to 1 for large mating ratios.

Fig. 3 shows that ρ has a considerable effect on ΔF when h^2_A is high. Because these comparisons are made at equal rates of gain, making the correlation negative increases the magnitude of h^2_M required to maintain the same ΔG, and as noted in the earlier cases I and II, this has a potent effect on ΔF. When ρ is positive the magnitude of h^2_M required to achieve equal ΔG is decreased. The trends observed for increasing h^2_A are

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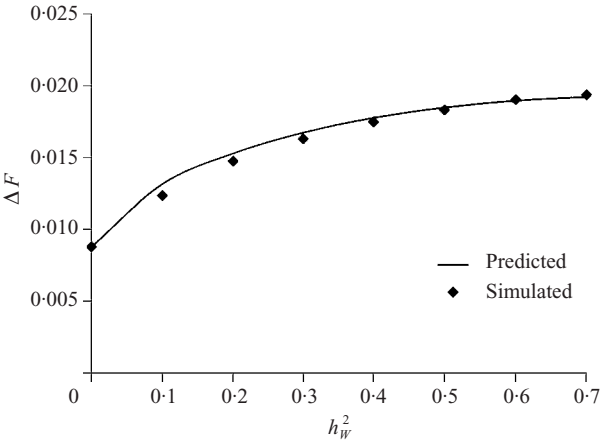

Fig. 4. Relation between the Willham heritability and ΔF under the constraints of $h^2_M/h^2_A = 0{\cdot}6$, $\rho = -0{\cdot}15$, $c^2 = 0{\cdot}07$ and $V(E) > 0$. $N_f = 25$, $n_o = 8$, and $d = 1$.

consistent with case II, where it was observed that a partition in favour of more h^2_A and less h^2_M produces greater ΔG and lower ΔF.

The predictions of direct and maternal genetic gain in Table 4 equalled those from conventional predictions using equilibrium genetic (co)variances (difference $< 0{\cdot}5\%$ of ΔG). Further, even though we used fixed h^2_W ($= 0{\cdot}2$), the predicted equilibrium genetic gains (ΔG) varied slightly between predictions within the three population structures in Table 4. These small variations ($< 1\%$ of ΔG) were caused by the differential influence of the Bulmer effect on the genetic (co)variances.

(iv) *Case IV. Varying* h^2_W

For typical estimates of direct and maternal (co)variances ($h^2_M/h^2_A = 0{\cdot}6$, $\rho = -0{\cdot}15$, $c^2 = 0{\cdot}07$, from Mohiuddin, 1993), the rates of inbreeding increased when the Willham heritability increased (Fig. 4). Note that with these assumptions $h^2_W < 0{\cdot}7$ since otherwise $V(E) < 0$ in the base population. The prediction errors were small ($< 7\%$).

(v) *Case V. Overlapping generations*

Fig. 5 shows the relationship between the rate of inbreeding per generation, ΔF_L, and the distribution of parents over two age classes. For pure maternal genetic effects ($h^2_M = 0{\cdot}5$, $h^2_A = 0$, $c^2 = 0$) the variation in age distribution of dams, with all sires being 1-year-olds, had a greater affect on ΔF_L than did a variation in age distribution of sires, with all dams being 1-year-olds. Further, for direct additive effects ($h^2_A = 0{\cdot}5$, $h^2_M = 0$, $c^2 = 0$), a variation in dam or sire distribution influenced ΔF_L equally. Searches close to $p_2 = 0{\cdot}5$ were performed and we found that for both direct effects and maternal effects the maximum of ΔF_L was

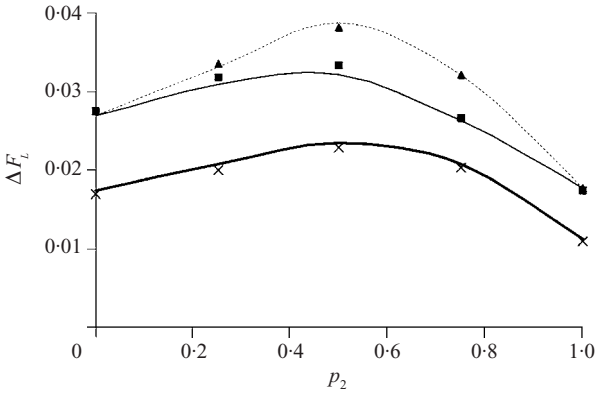

Fig. 5. Relation of the proportion of parents from the second age class (p_2) with predicted (lines) and simulated (symbols) rates of inbreeding per generation (ΔF_L). There were 20 sires and 20 dams ($n_o = 8$), and the age distribution was varied for one sex at a time. Predictions of ΔF_L are given for $h^2_A = 0{\cdot}5$ ($h^2_M = 0$, $c^2 = 0$) as a continuous thick line. Predictions of ΔF_L are given separately for variation in male and female age structures, for $h^2_M = 0{\cdot}5$ ($h^2_A = 0$, $c^2 = 0$), as a continuous thin line and a dashed line, respectively. Corresponding simulations are shown as crosses, boxes and triangles, respectively.

reached when p_2 was 0·5, i.e. when the number of parents entering the population per generation was minimized. Fig. 5 also shows that the effect of dam age distribution on ΔF_L was less pronounced for direct genetic effects than for maternal genetic effects. For maternal effects the rate of inbreeding was equal for $p_2 = 1$ whichever sex was varied (i.e. ΔF_L was 0·018 for both $\mathbf{N} = [20, 0, 0, 20]$ and $\mathbf{N} = [0, 20, 20, 0]$), because the lifetime contributions of females and of males are the same in both cases.

4. Discussion

This study has developed methods for predicting expected long-term genetic contributions to predict ΔF and ΔG, with good precision, for a phenotypic model including maternal effects of both genetic and environmental origin, as well as direct genetic effects. This extends the models of Woolliams *et al.* (1999) and Bijma *et al.* (2000) by incorporating the inheritance of maternal effects. The extension allowed us to quantify the impact of maternal effects on ΔF, and the results showed the impact was much greater when maternal effects were genetic in origin rather than environmental (ΔF was increased by more than 20% in a small population; Table 3). Furthermore, selection for traits with maternal effects that have equal h^2_W, i.e. with equal expected ΔG, may result in considerably different ΔF, indicating the inadequacy of h^2_W as the single summary parameter for determining selection outcomes when maternal effects are present. When compared at the same ΔG, lower ΔF would be expected as the partitioning of the variance favours direct additive genetic rather than maternal genetic

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effects, and for these to be positively rather than negatively correlated. More widely these results accentuate the importance of considering inheritance of selective advantages in predictions of ΔF, and not merely selective advantages in one generation, as pointed out by Bijma *et al*. (2000).

A general conclusion from the study was that the predictive precision of ΔF was good, and that, except in extreme cases, the overall prediction errors for ΔF were similar to those of ΔG (whether using long-term contribution methods or conventional methods). In the specific setup of case III the prediction errors of ΔF were even smaller than those of ΔG. This quality of prediction extended to overlapping generations where (as shown in Fig. 5) the complex relationships between ΔF per generation and the inheritance models were modelled very closely. The major errors arose when the litter sizes were large compared with the numbers selected and when the variation in maternal effects was large, as previously noted by Wray *et al*. (1994). As h^2_M tends to 1, unlike the case of $h^2_A = 1$, all sibs have similar phenotypes and selection becomes 'family selection'. The reason for this discrepancy is that the expected contributions were assumed to be linearly related to the selective advantages – often a reasonable assumption (Wray & Thompson, 1990), but not with very high selection intensities. More importantly, ΔF was predicted satisfactorily for litter sizes and maternal effects corresponding to practical situations in animal breeding. If acceptable predictions are to be made for the more extreme situations, then our model has to be developed for non-linear predictions of genetic contributions, which may also be the case in BLUP selection (Bijma & Woolliams, 2000).

The predictions of ΔG made using genetic contributions (eq. 3) and using the conventional approach (eqs. 6a, 6b) were very close, differing only in the third significant digit. One of the findings of Woolliams *et al*. (1999) was to show the equivalence of the conventional and contribution approaches in predicting ΔG for direct genetic effects; however, in this extended model, the proof of equivalence is more challenging and requires the algebraic inversion of a 4×4 matrix. This has not been done and so the question of the consistency of the extension with the existing tenets of quantitative genetics remains open.

The study has shown that the impact of maternal genetic variance on ΔF can be dramatic, more so than when the maternal effects are purely environmental in origin, and that the relationship between ΔG and ΔF depends critically on the partitioning of the genetic variance. An immediate consequence of this is to make clear that consideration of selection schemes with maternal variance using h^2_W alone is inadequate for describing the properties of the scheme. Furthermore, h^2_W is often referred to as the *total heritability* (e.g. Meyer, 1992; Koch *et al*., 1994; Mohiuddin, 1993). We recommend that this all-embracing term, *total heritability*, should *not* be used, because h^2_W does not completely describe the genetic properties of the population and its unconsidered use may seriously mislead the design of breeding programmes.

The influence of population structure on ΔF with maternal effects may best be viewed through consideration of the regressions of the long-term contributions on the selective advantages. Some general principles concerning litter size and mating ratio are predictable from consideration of the action of the selective advantages and the gene-flow equations. Firstly, there is the potentiating effect of family size (i.e. n_o) since, as described above, maternal effects work directly through the co-selection of maternal half-sib families, and the larger the family size, the more intense the selection and the stronger the relationship between long-term contribution and M_i. Secondly, increasing d reduces the relative influence of $V(M_i)$ relative to $V(A_i)$ in determining ΔF. This is due to an asymmetry between the sexes and the selective advantages in that long-term contributions are more strongly influenced by maternal effects in female ancestors than male ancestors (since, unlike a female, M_i does not influence selection of its offspring; see Table 2). When d is increased the important individual contributions to ΔF come primarily from the male ancestors, thereby reducing the impact of $V(M_i)$.

The results suggest that the impact of operational tools for maximizing genetic gain for a predefined rate of inbreeding by controlling the population structure and pedigree development (Meuwissen, 1997; Grundy *et al*., 1998) may be even greater with maternal effects than for only direct effects. Evidence for this conclusion comes from two observations: (i) ΔF was more sensitive to the population structure (n_o, d and age distribution in overlapping generations) with maternal effects compared with when there were only direct effects (Bijma *et al*., 2000); and (ii) ΔF was higher when maternal effects contributed to the Willham heritability, suggesting that selection decisions to satisfy pre-determined policies on ΔF are more demanding when $V(M) > 0$. A further implication of these observations is that not only may such tools be more valuable in breeding schemes where maternal effects are part of the evaluation models used, but they may also be more needed.

An important, and straightforward, extension of the method would be to encompass more general models of maternal effects (see Kirkpatrick & Lande, 1989). This study has followed Willham's model, where the maternal effect satisfies the property that $Cov(P_i, E_d) = 0$, i.e. the dam's environmental component of its own performance is independent of its offspring's performance. However, other models do

not make this assumption (e.g. Falconer, 1965), and in such models the selection effects upon the environmental means of the dams have an impact upon the mean phenotypic value in the progeny generation. Consequently, the environmental part of the maternal effect is inherited, e.g. as a socially inherited trait. Thus, the influence of the maternal effect on ΔF may be even greater for populations where Falconer's model applies.

We conclude that, even for a moderately low variation in maternal effects, it is important to consider maternal effects when predicting ΔF in a population under mass selection, especially if the maternal effects are inherited. The method of expected long-term genetic contributions gives good predictions of ΔF and ΔG in populations with maternal effects under mass selection, for both discrete and overlapping generations. The extension to BLUP selection (Bijma & Woolliams, 2000) with maternal effects may require further research. However, the method is easily extended to index and multi-trait selection (using the Appendix) in the same manner as for direct effects (Bijma & Woolliams, 1999) and can be developed for models of maternal effects other than Willham's.

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Appendix. Expected long-term genetic contributions with overlapping generations and multiple selective advantages

This appendix summarizes the theory of expected contributions with multiple selective advantages and overlapping generations as developed by Woolliams *et al.* (1999), Bijma & Woolliams (1999), Woolliams & Bijma (2000) and Bijma *et al.* (2000). A more detailed description of the method is found at http://journals.cambridge.org.

The linear predictor of the expected long-term genetic contribution $u_{i(q)}$ of individual i in category q is given by:

$$\mu_{i(q)} = \alpha_{i(q)} + \boldsymbol{\beta}_q^T(\mathbf{s}_{i(q)} - \bar{\mathbf{s}}_q) \tag{A1}$$

Let n_s be the number of defined selective advantages in $\mathbf{s}_{i(q)}$ and n_c be the number of categories. For simplicity of notation, the equations to calculate $\alpha_{i(q)}$ and $\boldsymbol{\beta}_q^T$ were slightly changed by defining the age structure by a vector $\mathbf{N}$ of length n_c (instead of a diagonal matrix $\mathbf{N}$ as in equations 7b and 9 of Woolliams *et al.*, 1999):

$$(\mathbf{N}\otimes\boldsymbol{\alpha}) = [\mathbf{G}^T + (\mathbf{G}^T\otimes\mathbf{D}^T)(\mathbf{I}-\mathbf{G}^T\otimes\boldsymbol{\Pi}^T)^{-1} \times (\mathbf{G}^T\otimes\boldsymbol{\Lambda}^T)](\mathbf{N}\otimes\boldsymbol{\alpha}) \tag{A2}$$

$$(\mathbf{N}\otimes\boldsymbol{\beta}) = (\mathbf{I}-\mathbf{G}^T\otimes\boldsymbol{\Pi}^T)^{-1}(\mathbf{G}^T\otimes\boldsymbol{\Lambda}^T)(\mathbf{N}\otimes\boldsymbol{\alpha}) \tag{A3}$$

where $\otimes$ denotes element-by-sub-matrix multiplication of matrices, $\mathbf{I}$ is the $n_cn_s \times n_cn_s$ identity matrix, $\mathbf{N}$ is a vector with elements N_k equal to the numbers of parents selected from each category, $\boldsymbol{\Pi}$ is a $n_cn_s \times n_cn_s$ matrix containing sub-matrices $\boldsymbol{\pi}_{pq}$ $(n_s \times n_s)$ of regression coefficients of selective advantages of selected progeny in category p on selective advantages of parents in category q, $\boldsymbol{\Lambda}$ is a $n_c \times n_cn_s$ matrix containing sub-matrices $\boldsymbol{\lambda}_{pq}$ $(1 \times n_s)$ of regression coefficients of proportion selected in category p on selective advantages of parents in category q, $\mathbf{G}$ is a $n_c \times n_c$ modified gene flow matrix connecting selected offspring to parental categories, $\mathbf{D}$ is a $n_cn_s \times n_c$ matrix of deviations of selective advantages from the mean of the selected category, α is a vector (length n_c) of elements α_q, and $\boldsymbol{\beta}$ is a vector of length n_cn_s containing the sub-vectors $\boldsymbol{\beta}_q$.

Let $(\mathbf{s}_{i(q)}\ \mathbf{s}_{j(p)}\ I_{j(p)})^T$ have the partitioned covariance matrix:

$$\mathbf{V} = \begin{pmatrix} \mathbf{V}_{qq} & \mathbf{V}_{pq} & \mathbf{v}_p \\ \mathbf{V}_{pq}^T & \mathbf{V}_{pp} & \mathbf{v}_q \\ \mathbf{v}_p^T & \mathbf{v}_q^T & \sigma_I^2 \end{pmatrix} \tag{A4}$$

where p and q are progeny and parent categories, respectively, and $I_{j(p)}$ is the index upon which the selection of individual $j(p)$ will be determined. $\boldsymbol{\Pi}$ and $\boldsymbol{\Lambda}$ are then obtained from (see appendix B in Woolliams *et al.*, 1999):

$$\boldsymbol{\pi}_{pq} = \mathbf{V}_{pq}^*\mathbf{V}_{qq}^{-1} \tag{A5}$$

$$\boldsymbol{\lambda}_{pq} = i_p\sigma_I^{-1}\mathbf{v}_q\mathbf{V}_{qq}^{-1} \tag{A6}$$

where $\mathbf{V}_{pq}^*$ is the genetic (co)variance matrix *after* selection

$$\mathbf{V}_{pq}^* = (\mathbf{V}_{pq} - k_p\sigma_I^{-2}\mathbf{v}_p\mathbf{v}_q^T) \tag{A7}$$

and k_p is the variance reduction term in category p.

Define $\mathbf{g}_{j(p)}$ as a vector of Mendelian sampling terms corresponding to the selective advantages in $\mathbf{s}_{i(p)}$ (in our paper $\mathbf{g}_{j(p)} = (a_{j(p)}, m_{j(p)}, 0)^T$). The annual genetic gain is then:

$$\Delta\mathbf{G} = \sum_{q=1}^{n_c} N_q\mathrm{E}[r_{i(q)}\mathbf{g}_{i(q)}] \tag{A8}$$

and

$$\mathrm{E}[r_{i(q)}\mathbf{g}_{i(q)}] = \alpha_q i_q\sigma_I^{-1}\mathbf{v}_g + \boldsymbol{\beta}_q^T(\mathbf{V}_{pg} - k_q\sigma_I^{-1}\mathbf{v}_p\mathbf{v}_g^T) \tag{A9}$$

follows by extension of appendix B in Bijma & Woolliams (1999) to multiple selective advantages, where the matrices $\mathbf{V}_{pg}$, $\mathbf{v}_g$ and $\mathbf{v}_p$ are covariance

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matrices of $(\mathbf{s}_{j(p)}\ \mathbf{g}_{j(p)})^T$, $(\mathbf{g}_{j(p)}\ I_{j(p)})^T$ and $(\mathbf{s}_{j(p)}\ I_{j(p)})^T$, respectively.

Rates of inbreeding per year, ΔF, are predicted as (equation 29 in Woolliams & Bijma, 2000):

$$\mathrm{E}[\Delta F] = \frac{1}{2}\sum_{s_{males}} n_s \mathrm{E}(u_{i,s}^2) + \frac{1}{2}\sum_{s_{females}} n_s \mathrm{E}(u_{i,s}^2) + \frac{1}{8}\sum_{s} n_s \delta_s \qquad \text{(A10)}$$

where n_s is the number of individuals with a certain life history of reproduction defined by the categories that an individual was selected in. The third term is the correction for non-Poisson distribution of family size (Bijma *et al*., 2000).

The generation interval, L, is defined as the time in which the long-term contributions sum to unity (Woolliams *et al*., 1999): $L = 1/\sum_{k=1}^{n_c} n_k \alpha_k$. The predicted rate of inbreeding per generation, ΔF_L, may then be calculated as $\Delta F_L = \mathrm{E}(\Delta F) \times L$.

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