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, and their impact on geneticvariation 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 at the offspring (Mueller & James, 1985; Riska et al., 1985). The studies reviewed by Kirkpatrick & Lande (1989) focused on the selection...
response in populations, and did not investigate the influence of maternal effects on $\Delta F$.

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 $\Delta F$. 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 $\Delta F$ 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, $\Delta F$ has been shown to increase when selective advantages are inherited (Robertson, 1961; Wray & Thompson, 1990).

Woolliams & Bijma (2000) showed that $\Delta F$ 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 $\Delta F$ 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, $P_i$, self, and a component determined by the dam, $P_i$, 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.

Table 1. Notation of frequently used parameters

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Delta F, \Delta F_L$</td>
<td>Annual rates of inbreeding and rates of inbreeding per generation</td>
</tr>
<tr>
<td>$\Delta G_{dir}, \Delta G_{mat}$</td>
<td>Direct and maternal genetic gain per year</td>
</tr>
<tr>
<td>$\Delta G, \Delta G_L$</td>
<td>Vector of annual genetic gain and total genetic gain ($\Delta G_{dir} + \Delta G_{mat}$)</td>
</tr>
<tr>
<td>$P_i, A_i, E_i$</td>
<td>Phenoype, additive direct genetic effect and environmental effect</td>
</tr>
<tr>
<td>$M_i, C_i$</td>
<td>Additive maternal genetic effect and environmental maternal effect of the dam</td>
</tr>
<tr>
<td>$s_{iq}$</td>
<td>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 $s_q$</td>
</tr>
<tr>
<td>$r_{iq}, h_{iq}, \mu_{iq}$</td>
<td>Long-term genetic contribution, expected long-term genetic contribution and linear predictor of long-term genetic contributions</td>
</tr>
<tr>
<td>$a_p, b_q$</td>
<td>Vectors of the coefficients for $\mu_{iq}$</td>
</tr>
<tr>
<td>$\lambda_{pq}$</td>
<td>Regression coefficients of proportion selected in category $p$ on $s_{iq}$ for parents in category $q$</td>
</tr>
<tr>
<td>$\pi_{pq}$</td>
<td>Regression coefficients of $s_{iq}$ on $s_{iq}$ for parents in category $q$</td>
</tr>
<tr>
<td>$a_i, m_i, g_{(p)}$</td>
<td>Direct and maternal Mendelian sampling terms of individual $i$, and vector of Mendelian sampling terms equal to $(a_i, m_i, 0)^T$</td>
</tr>
<tr>
<td>$h^2_A, h^2_M, h^2_F$</td>
<td>Direct, maternal and Willham heritabilities</td>
</tr>
<tr>
<td>$\sigma^2, \rho, \sigma^2_i$</td>
<td>Common environmental variance, direct-maternal genetic correlation, and phenotypic variance</td>
</tr>
<tr>
<td>$N_m, N_f, d$</td>
<td>Number of male parents, number of female parents, and dams per sire ($N_f/N_m$)</td>
</tr>
<tr>
<td>$n_i, T$</td>
<td>Total number of offspring per dam, and number of candidates available for selection in each sex</td>
</tr>
<tr>
<td>$N_i$</td>
<td>Vector of number of individuals in each category</td>
</tr>
<tr>
<td>$l_p, k_p, i$</td>
<td>Intensity of selection and variance reduction term in sex $p$, and mean selection intensity</td>
</tr>
<tr>
<td>$p, q, i(p)$</td>
<td>Indicators of categories, $i(p)$ denotes individual $i$ in category $p$</td>
</tr>
<tr>
<td>$m, f$</td>
<td>Indicators of male and female</td>
</tr>
<tr>
<td>$i, d, s$</td>
<td>Indicators of individual, dam and sire</td>
</tr>
</tbody>
</table>
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 \textit{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 \textit{et al.} (1994). The terms \( A_i, E_i, M_d \) and \( C_d \) are assumed to be mutually independent with the exception of \( A_i, M_d \). 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:

\[ A_i = \frac{1}{s} A_s + \frac{1}{d} A_d + a_i \]

where \( V(a_i) = \frac{1}{s} h_s^2 \), and

\[ M_i = \frac{1}{s} M_s + \frac{1}{d} M_d + m_i \]

where \( V(m_i) = \frac{1}{s} h_M^2 \).

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

\[ V(E) = 1 - (h_s^2 + h_M^2 + \rho h_s h_M + c^2). \]

Using this model, Wilham (1972) showed that for mass selection in the base population, the ratio of the response to the selection differential applied is given by \( h_s^2 = h_s^2 + \frac{1}{s} \rho h_s h_M + \frac{1}{s} h_M^2 \). We refer to this fraction as the \textit{Willham heritability}.

\section*{(ii) Population structures}

Each sire was mated at random to a fixed number of \( d \) dams, and each dam produced \( n_s \) 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.

\section*{(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 \textit{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 \textit{et al.}, 1999) where equilibrium genetic (co)variances had been attained (Bulmer, 1971).

The long-term genetic contribution, \( r_{(g)q} \), of individual \( i \) in category \( q \) born at \( t_q \) is defined as the proportion of genes present in individuals in cohort \( t_q \) deriving by descent from \( i \), where \( (t_q - t_i) \rightarrow \infty \) (Woolliams \textit{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 \textit{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 \( s_{(g)q} \) be a vector of selective advantages for individual \( i \) in category \( q \) so that \( s_{(g)q} = (A_{(g)q}, M_{(g)q}, C_{(g)q})^T \), where superscript \( T \) denotes the transpose of matrices. The expected long-term genetic contribution \( u_{(g)q} \) is then defined as \( u_{(g)q} = E(r_{(g)q} | s_{(g)q} - \bar{s}_{q}) \), where \( \bar{s}_q \) is the average of selected individuals in
category \( q \). The linear predictor of \( u_{i(q)} \) is:
\[
\mu_{i(q)} = \alpha_{i(q)} + \beta^*_q (s_{i(q)} - \bar{s}_q) \tag{1}
\]
where \( \alpha_i = 1/(2N_i) \) and \( \alpha_f = 1/(2N_f) \). For discrete generations, solutions for the coefficients in \( \beta^*_q \) are obtained from Woolliams et al. (1999):
\[
\left( \frac{N_m \beta_m}{N_f \beta_f} \right) = \frac{1}{4} \left( \frac{1 - \frac{1}{2} \left( \pi_{mm} - \pi_{mf} \right)}{\pi_{mf} + \pi_{ff}} \right)^{-1} \left( \frac{\pi_{mm} + \pi_{mf}}{\pi_{mf} + \pi_{ff}} \right) \tag{2}
\]
where \( I \) is the 6 \times 6 identity matrix, \( \pi_{pq} \) are 3 \times 3 sub-matrices containing regression coefficients of selective advantages of selected progeny in sex \( p \) on selective advantages of parents in sex \( q \), \( I_{pq} \) are 3 \times 1 sub-matrices 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 \( \lambda_{pq} \) and \( \pi_{pq} \) are given by:
\[
\lambda_{p,m} = \frac{i_p}{2\sigma_f} (1 \ 0 \ 0)^T
\]
\[
\lambda_{p,f} = \frac{i_p}{2\sigma_f} (1 \ 2 \ 2)^T
\]
\[
\pi_{p,m} = \frac{1}{2} \left[ \begin{array}{ccc} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 0 \end{array} \right] - \frac{k_p}{\sigma^2_f} \left[ \begin{array}{ccc} x & 0 & 0 \\ y & 0 & 0 \\ 0 & 0 & 0 \end{array} \right]
\]
\[
\pi_{p,f} = \frac{1}{2} \left[ \begin{array}{ccc} 1 & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 0 \end{array} \right] - \frac{k_p}{\sigma^2_f} \left[ \begin{array}{ccc} x & 2x & 0 \\ y & 2y & 0 \\ 0 & 0 & 0 \end{array} \right]
\]
where \( x = \text{Cov}(P_i, A_i) \), \( y = \text{Cov}(P_i, M_i) \), \( i_p \) is the selection intensity in sex \( p \), \( k_p \) is the variance reduction term in sex \( p \), and \( \sigma_f \) is the phenotypic standard deviation.

The annual genetic gain (\( \Delta G \)) was predicted by
\[
\Delta G = N_m \text{E}[f(m)g(m)] + N_f \text{E}[f(f)g(f)] \tag{3}
\]
where \( g_{i(p)} \) is the vector of Mendelian sampling terms corresponding to the selective advantages in \( s_{i(p)} \), i.e. \( g_{i(p)} = (a_{i(p)}m_{i(p)})^T \) (Woolliams et al., 1999). The expectations of \( r_{i(p)}g \) are given in equation (A9) in the Appendix.

The rate of inbreeding per year, \( \Delta F \), was predicted using the results of Woolliams & Bijma (2000):
\[
\text{E}[\Delta F] = \frac{1}{2} \sum_{q=m,f} N_q \text{E}(u_{i(q)}) - \frac{1}{8T} \tag{4}
\]
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:
\[
E(u_{i(q)}) = \alpha_{i(q)}^2 + (1 - 1/N_m)\beta_m \text{V}_{mm}/\beta_m^T + d(1 - 1/N_f)\beta_f \text{V}_{ff}/\beta_f^T \tag{5a}
\]
\[
E(u_{i(f)}) = \alpha_f^2 + (1 - 1/N_f)\beta_f \text{V}_{ff}/\beta_f^T + \frac{1}{d^2}(1 - 1/N_m)\beta_m \text{V}_{mm}/\beta_m^T \tag{5b}
\]
where \( \text{V}_{qq} \) is the (co)variance matrix of selective advantages after selection in sex \( q \) as defined in the Appendix.

The method implies that \( \Delta F \) 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
\[
\text{V}_g = \left( \begin{array}{ccc} h_A^2 & \rho h_A h_M & h_A^2 \\ \rho h_A h_M & \rho h_A h_M & h_M^2 \\ h_A^2 & \rho h_A h_M & h_M^2 \end{array} \right),
\]
for which the lower matrix of the Cholesky factorization is
\[
\text{L} = \left( \begin{array}{ccc} h_A & 0 & \sqrt{h_A^2(1 - r^2)} \\ \rho h_M & 0 & \sqrt{h_M^2(1 - r^2)} \end{array} \right).
\]
Additive direct and maternal genetic effects of the sires and dams of the base population were simulated as \( (A_i, M_i)^T = \text{Lx} \), where \( x \) is a vector of two independent random \( N(0, 1) \) numbers. The phenotypic value of individual \( i \) was then calculated as \( P_i = A_i + M_i + C_i + E_i \), where \( C_i \) was sampled from \( N(0, e^2) \) and \( E_i \) was sampled from \( N(0, V(E)) \).

In subsequent generations the procedure was similar, but additive effects were calculated as \( A_i = \frac{1}{2} A_i + \frac{1}{8} A_d + a_i \) and \( M_i = \frac{1}{2} M_i + \frac{1}{4} M_d + m_i \), with the covariance between Mendelian sampling terms \( \text{Cov}(a_i, m_i) = \frac{1}{2} \rho h_A h_M \). Inbreeding was neglected in the calculation of Mendelian sampling terms. Thus sampling was
conducted using
\[(a_i, m)^T = \sqrt{0.5} L x.\]

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:
\[ r_i = \alpha + \beta_i (A_i - \overline{A}) + \beta_i (M_i - \overline{M}) + \beta_i (C_i - \overline{C}) + e_i, \]
and \( \beta_i \) 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 \( \Delta G_{dir} = (\overline{A}_i - \overline{A})/(t_2 - t_1) \) and \( \Delta G_{mat} = (\overline{M}_i - \overline{M})/(t_2 - t_1) \). 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 \( \Delta F = 1 - \left(1 - \frac{F}{F_{sim}}\right)^{(t_2 - t_1) \nu} \). Results were averaged over 500 replicates to give \( \Delta F_{sim} \).

(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. \( h^2_D, h^2_M, \rho, c^2, N_m, N_f, n_a, n_m, \) age structure.

Case I. The origin of maternal effects. Parameters \( h_D, c^2 \) and \( n_i \) were varied to exemplify differences between inherited and non-inherited maternal effects in determining \( \Delta F \). In this case \( h^2_D = 0, N_m = 25, \) and \( d = 1. \)

Case II. Partitioning a constant \( (h^2_D + h^2_M) \). The impact of \( h^2_D \) and \( h^2_M \) on \( \Delta F \) was examined for fixed \( h^2_D + h^2_M \) (equal to 4) and with \( \rho = 0. \)

Case III. Partitioning a constant \( h^2_M \). The differential impact of \( h^2_D, h^2_M \) and \( \rho \) on \( \Delta F \) was examined when these parameters were constrained to give a fixed \( h^2_M = 2 \). Thus, \( h^2_M \) varied from 0 to 4 as in case II but for fixed \( \Delta G \). The population structures were varied so that with random selection and mating and Poisson family size \( \Delta F \) was expected to be 0-01.

Case IV. Varying \( h^2_M \). The impact of varying \( h^2_M \) on \( \Delta F \) was examined. The values of \( h^2_D, h^2_M \) and \( \rho \) were constrained by using the results from the review of genetic parameters in beef cattle by Mohiuddin (1993): \( h^2_M = 0.6, \rho = -0.15 \). In addition, it was assumed that \( c^2 = 0.07 \) (Mohiuddin, 1993).

Case V. Overlapping generations. The influence of age structure on rates of inbreeding per generation (\( \Delta F_g \)) 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 \( N = [20, 0, 20(1 - p_2), 20p_2]^T \) when the dam distribution was varied, and \( N = [20(1 - p_2), 20p_2, 20, 0]^T \) when the sire distribution was varied.

For all cases the results of the simulations were compared with predictions of \( \Delta G \) and \( \Delta F \) given above. In the case of \( \Delta G \) the predictions were also compared with those from ‘conventional’ selection theory (see e.g. Van Vleck, 1993):

\[
\Delta G_{dir} = \left[ V(A_d) + \frac{1}{2} \text{Cov}(A_d, M_d) \right] i/\sigma_f \]

(6a)

\[
\Delta G_{mat} = \left[ \text{Cov}(A_i, M_i) + \frac{1}{2} V(M_d) \right] i/\sigma_f \]

(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 \( h^2_D, h^2_M \) and \( \rho \), 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 \( \beta \) (Table 2). For females the ratio between genetic and environmental \( \beta \) 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 \( \Delta F \) in Table 3, where maternal effects that were inherited had greater

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

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Male ( \beta )</th>
<th>Female ( \beta )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( A_i )</td>
<td>( \frac{1}{\Delta_i} \times \frac{i}{2N_m o_f} )</td>
<td>( \frac{1}{\Delta_i} \times \frac{i}{2N_m o_f} )</td>
</tr>
<tr>
<td>( M_i )</td>
<td>( \frac{1}{\Delta_2} \times \frac{i}{2N_m o_f} )</td>
<td>( \frac{3}{\Delta_2} \times \frac{i}{2N_m o_f} )</td>
</tr>
<tr>
<td>( C_i )</td>
<td>0</td>
<td>( \frac{i}{2N_m o_f} )</td>
</tr>
</tbody>
</table>

*\( \Delta_1 = 1 + \frac{V(A_d)}{2\sigma_f^2} (k_f + k_m) \).*

*\( \Delta_2 = 1 + \frac{V(M_d)}{8\sigma_f^2} (3k_f + k_m) \).*

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effect on rates of inbreeding than those of equal size but environmental origin. An additional component to this increase in $\Delta F$ was that $\beta > 0$ for males, unlike the environmental case. Table 2 shows the regression on $M_f$ 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 $\Delta F$ was approximately the same as the prediction errors given by Bijma et al. (2000) for additive direct effects only. However, $\Delta F$ could not be predicted for large maternal variances and large litter sizes. In the model for predicting $\Delta F$ 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 $\Delta F = 0.01$, 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 $\Delta F$ exceeded 0.01, even though the family size was fixed (Table 3). Furthermore, $\Delta F$ was increased by more than 20% for moderate $h^2_M$ compared with $c^2$ of equal size.

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 $\Delta F_{sim}$

<table>
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<tr>
<th>$c^2$</th>
<th>$h^2_M$</th>
<th>$\Delta F_{pred}^a$</th>
<th>Error%$^d$</th>
<th>$\Delta F_{pred}^b$</th>
<th>Error%</th>
<th>$\Delta F_{pred}^c$</th>
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<td>+3</td>
<td>0.0197</td>
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<tr>
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<td>-8</td>
</tr>
<tr>
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<td>0.0291</td>
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<td>0.0640</td>
<td>+235</td>
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</table>

$a^D$ $\Delta F_{pred} = 0.0075$ for random selection ($h^2_{D_{r}} = 0$).

$b^D$ $\Delta F_{pred} = 0.0088$ for random selection.

c $\Delta F_{pred} = 0.0095$ for random selection.

d $100\% \times (\Delta F_{pred} - \Delta F_{sim})/\Delta F_{sim}$.

(ii) Case II. Partitioning a constant ($h^2_A + h^2_M$) $\Delta F$ increased and $\Delta G$ 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 $\Delta F (<3\%)$ were smaller than the prediction errors of $\Delta G (<9\%)$, where the predictions of $\Delta G$ equaled conventional ones (difference 0.7% of $\Delta G$). The maximum prediction error was obtained for $h^2_M = 0.4$, and $\Delta G$ 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 2. Predicted rates of inbreeding for different combinations of \( h_A^2, h_M^2 \) and \( \rho \), in the base population with \( h_W^2 = 0.2 (\sigma^2 = 0) \). Standard errors of simulated rates of inbreeding were less than 2% of \( \Delta F_{\text{sim}} \)

<table>
<thead>
<tr>
<th>( h_A^2 )</th>
<th>( h_M^2 )</th>
<th>( \rho )</th>
<th>( \Delta F_{\text{pred}}^a )</th>
<th>% Error$^d$</th>
<th>( \Delta F_{\text{pred}}^b )</th>
<th>% Error</th>
<th>( \Delta F_{\text{pred}}^c )</th>
<th>% Error</th>
</tr>
</thead>
<tbody>
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<td>0.0198</td>
<td>0</td>
<td>0.0185</td>
<td>-6</td>
</tr>
</tbody>
</table>

$^a$ The predictions of \( \Delta G \) varied within 0.144–0.146, \( \Delta F_{\text{pred}} = 0.0075 \) for random selection (\( h_W^2 = 0 \)).

$^b$ The predictions of \( \Delta G \) varied within 0.226–0.229, \( \Delta F_{\text{pred}} = 0.0088 \) for random selection.

$^c$ The predictions of \( \Delta G \) varied within 0.287–0.290, \( \Delta F_{\text{pred}} = 0.0095 \) for random selection.

$^d$ 100% \times (\( \Delta F_{\text{pred}} - \Delta F_{\text{sim}} \))/\( \Delta F_{\text{sim}} \).

Table 2 also shows the magnitude of \( \beta \) 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 \( \beta_M/\beta_A \) 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 \( h_W^2 \)

In this case \( \Delta G \) was constant within each population structure by studying a constant \( h_W^2 \). The rates of inbreeding increased when the component of \( h_W^2 \) 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 \( \Delta F \) with \( h_M^2 \) was smaller when the number of offspring per dam was reduced. Furthermore, the increase in \( \Delta F \) 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 \( \Delta F \) dominates. In the case where there are only maternal genetic effects \( \beta_M/\beta_A = 3/d \) (Table 2), and consequently the selective advantages of each individual female have less impact on \( \Delta F \) as \( d \) becomes higher. This is in accordance with the results in Fig. 2. The ratio between the rates of inbreeding when \( h_W^2 \) 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 \( \rho \) has a considerable effect on \( \Delta F \) when \( h_A^2 \) is high. Because these comparisons are made at equal rates of gain, making the correlation negative increases the magnitude of \( h_M^2 \) required to maintain the same \( \Delta G \), and as noted in the earlier cases I and II, this has a potent effect on \( \Delta F \). When \( \rho \) is positive the magnitude of \( h_M^2 \) required to achieve equal \( \Delta G \) is decreased. The trends observed for increasing \( h_M^2 \) are

![Fig. 2. Dams per sire related to relative \( \Delta F \). Relative \( \Delta F \) is the ratio of the rates of inbreeding when \( h_W^2 \) is made up of direct effects (\( h_A^2 = 0.2, \rho = 0 \)) compared with when it only includes maternal effects (\( h_M^2 = 0.4, \rho = 0 \)). \( N_f = 64, n_a = 8, \) and \( N_m \) varied from 64 to 1.](image1)

![Fig. 3. Relationship between \( h_A^2 \) and predicted \( \Delta F \) for fixed \( h_M^2 = 0.2 (\sigma^2 = 0) \) and different values of \( \rho \). \( N_f = 25, n_a = 8, \) and \( d = 1 \).](image2)
consistent with case II, where it was observed that a partition in favour of more $h_A$ and less $h_M$ produces greater $\Delta G$ and lower $\Delta F$.

The predictions of direct and maternal genetic gain in Table 4 equalled those from conventional predictions using equilibrium genetic (co)variances (difference $<0.5\%$ of $\Delta G$). Further, even though we used fixed $h_M^2 = 0.2$, the predicted equilibrium genetic gains ($\Delta G$) varied slightly between predictions within the three population structures in Table 4. These small variations ($<1\%$ of $\Delta G$) were caused by the differential influence of the Bulmer effect on the genetic (co)variances.

(iv) Case IV. Varying $h_W$

For typical estimates of direct and maternal (co)variances ($h_A^2/h_M^2 = 0.6, \rho = -0.15, c^2 = 0.07$, from Mohiuddin, 1993), the rates of inbreeding increased when the Willham heritability increased (Fig. 4). Note that with these assumptions $h_W^2 < 0.7$ 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, $\Delta F_L$, and the distribution of parents over two age classes. For pure maternal genetic effects ($h_M^2 = 0.5, h_A^2 = 0, c^2 = 0$) the variation in age distribution of dams, with all sires being 1-year-olds, had a greater affect on $\Delta F_L$ than did a variation in age distribution of sires, with all dams being 1-year-olds. Further, for direct additive effects ($h_A^2 = 0.5, h_M^2 = 0, c^2 = 0$), a variation in dam or sire distribution influenced $\Delta F_L$ equally. Searches close to $p_2 = 0.5$ were performed and we found that for both direct effects and maternal effects the maximum of $\Delta F_L$ was 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 $\Delta F_L$ was less pronounced for direct genetic effects than for maternal genetic effects. For maternal effects the rate of inbreeding was equal for $p_2 = 1$ whenever sex was varied (i.e. $\Delta F_L$ was $0.018$ for both $N = [20, 0, 0, 20]$ and $N = [0, 20, 20, 0]$), 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 $\Delta F$ and $\Delta G$, 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 $\Delta F$, and the results showed the impact was much greater when maternal effects were genetic in origin rather than environmental ($\Delta F$ was increased by more than 20% in a small population; Table 3). Furthermore, selection for traits with maternal effects that have equal $h_M^2$, i.e. with equal expected $\Delta G$, may result in considerably different $\Delta F$, indicating the inadequacy of $h_M^2$ as the single summary parameter for determining selection outcomes when maternal effects are present. When compared at the same $\Delta G$, lower $\Delta F$ would be expected as the partitioning of the variance favours direct additive genetic rather than maternal genetic
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The predictions of $\Delta G$ 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 the approach (eqs. 6a, 6b) were very close, differing only in the third significant digit. More widely these results accentuate the importance of considering inheritance of selective advantages in predictions of $\Delta F$, 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 $\Delta F$ was good, and that, except in extreme cases, the overall prediction errors for $\Delta F$ were similar to those of $\Delta G$ (whether using long-term contribution methods or conventional methods). In the specific setup of case III the prediction errors of $\Delta F$ were even smaller than those of $\Delta G$. This quality of prediction extended to overlapping generations where (as shown in Fig. 5) the complex relationships between $\Delta F$ 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 $h^2_F$ tends to 1, unlike the case of $h^2_V = 1$, 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, $\Delta F$ 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 $\Delta G$ can be dramatic, more so than when the maternal effects are purely environmental in origin, and that the relationship between $\Delta G$ and $\Delta F$ 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 $h^2_V$ alone is inadequate for describing the properties of the scheme. Furthermore, $h^2_V$ 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 $h^2_V$ 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 $\Delta F$ 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. $n_d$) 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 $\Delta F$. 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 $\Delta F$ 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) $\Delta F$ was more sensitive to the population structure ($n_d$, $d$ and age distribution in overlapping generations) with maternal effects compared with when there were only direct effects (Bijma et al., 2000); and (ii) $\Delta F$ was higher when maternal effects contributed to the Willham heritability, suggesting that selection decisions to satisfy pre-determined policies on $\Delta F$ 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 $\text{Cov}(P_d, E_d) = 0$, 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.

L. R. thanks Prof. Öje Danell for arranging a visit to the Roslin Institute and for enabling the co-operation with J.A.W. J.A.W. is grateful to the Department for the Environment, Food and Rural Affairs (UK) for funding. We thank Erling Strandberg for valuable comments on an earlier version of the manuscript.

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:

\[
\mu_{i(q)} = \alpha_{i(q)} + \beta^T_q (s_{i(q)} - \bar{s}_q)
\]  

(A1)

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

\[
(N \otimes \alpha) = (G^T + (G^T \otimes D^T)(I - G^T \otimes \Pi^T)^{-1} \times (G^T \otimes A^T))(N \otimes \alpha)
\]  

(A2)

where \( \otimes \) denotes element-by-sub-matrix multiplication of matrices, \( I \) is the \( n_c \times n_c \) identity matrix, \( N \) is a vector with elements \( N_k \) equal to the numbers of parents selected from each category, \( \Pi \) is a \( n_c \times n_c \) matrix containing sub-matrices \( \pi_{pq} \) (\( n_c \times n_c \) of regression coefficients of selective advantages of selected progeny in category \( p \) on selective advantages of parents in category \( q \), \( A \) is a \( n_c \times n_c \) matrix containing sub-matrices \( \lambda_{pq} \) (\( 1 \times n_c \) of regression coefficients of proportion selected in category \( p \) on selective advantages of parents in category \( q \), \( G \) is a \( n_c \times n_c \) modified gene flow matrix connecting selected offspring to parental categories, \( D \) is a \( n_c \times n_c \) matrix of deviations of selective advantages from the mean of the selected category, \( \alpha \) is a vector (length \( n_c \) of elements \( \alpha_{pq} \), and \( \beta \) is a vector (length \( n_c \times n_c \) containing the sub-vectors \( \beta_{pq} \).

Let \( (s_{i(q)} \ s_{j(p)})^T \) have the partitioned covariance matrix:

\[
V = \begin{pmatrix}
V_{qq} & V_{pq} & V_p \\
V_{pq} & V_{pp} & V_q \\
V_p^T & V_q^T & \sigma^2
\end{pmatrix}
\]

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

\[
\pi_{pq} = V_{pq}^{-1}V_{qq}^{-1}
\]

(A5)

\[
\lambda_{pq} = p_i \sigma^{-1}_q V_{q}^{-1}
\]

(A6)

where \( V_{pq}^* \) is the genetic (co)variance matrix after selection

\[
V_{pq}^* = (V_{pq} - k_p \sigma_{q}^{-2}V_q V_q^T)
\]

(A7)

and \( k_p \) is the variance reduction term in category \( p \). Define \( g_{p(q)} \) as a vector of Mendelian sampling terms corresponding to the selective advantages in \( s_{p(q)} \) (in our paper \( g_{p(q)} = (a_{p(q)}, m_{p(q)}, 0)^T \)). The annual genetic gain is then:

\[
\Delta G = \sum_{q=1}^{n_c} N_q E[r_{i(q)}g_{i(q)}]
\]

(A8)

and

\[
E[r_{i(q)}g_{i(q)}] = \alpha_q i_q \sigma_q^{-1}V_q + \beta^T_q (V_{pq} - k_q \sigma_q^{-2}V_q V_q^T)
\]

(A9)

follows by extension of appendix B in Bijma & Woolliams (1999) to multiple selective advantages, where the matrices \( V_{pq}, \ V_q \) and \( V_p \) are covariance matrices.
matrices of $(s_{f(p)} g_{f(p)})^T$, $(g_{f(p)} I_{f(p)})^T$ and $(s_{f(p)} I_{f(p)})^T$, respectively.

Rates of inbreeding per year, $\Delta F$, are predicted as (equation 29 in Woolliams & Bijma, 2000):

$$E[\Delta F] = \frac{1}{2} \sum_{f=0}^{\infty} n_e E(\alpha_f^2) + \frac{1}{2} \sum_{f=0}^{\infty} n_e E(\alpha_f^2)$$

$$+ \frac{1}{8} \sum_{j} n_j \delta_j$$  \hspace{1cm} (A10)

where $n_i$ 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): $L = 1/\sum_{k=1}^{\infty} n_k \alpha_k$. The predicted rate of inbreeding per generation, $\Delta F_L$, may then be calculated as $\Delta F_L = E(\Delta F) \times L$.

References


