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THE EVOLUTION OF MATERNAL CHARACTERS

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Abstract. — We develop quantitative-genetic models for the evolution of multiple traits under maternal inheritance, in which traits are transmitted through non-Mendelian as well as Mendelian mechanisms, and maternal selection, in which the fitness of offspring depends on their mother's phenotype as well as their own. Maternal inheritance results in time lags in the evolutionary response to selection. These cause a population to evolve for an indefinite number of generations after selection ceases and make the rate and direction of evolution change even when the strength of selection and parameters of inheritance remain constant. The rate and direction of evolution depend on the inheritance of traits that are not under selection, unlike under classical Mendelian inheritance. The models confirm earlier findings that the response to selection can be larger or smaller than what is possible with simple Mendelian inheritance, and even in a direction opposite to what selection favors. Maternal selection, in which a mother's phenotype influences her offspring's fitness, is frequency-dependent and can cause a population to evolve maladaptively away from a fitness peak, regardless of whether traits are transmitted by Mendelian or maternal inheritance. Maternal selection differs from other forms of selection in that its force depends not only on the fitness function but also on the phenotypic resemblance of parents and offspring.

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Parents have two kinds of influence on their offspring. The first of these is heredity. While biologists usually assume that the resemblance between parents and offspring is caused by Mendelian genetics, additional factors often contribute to inheritance. Non-Mendelian forms of heredity can be transmitted through the cytoplasm, parental care, and various forms of learning. The second kind of influence parents have is through direct effects on the fitness of their offspring. In many populations the fitness of an individual depends on the phenotype of its parents as well as its own phenotype, for example the amount of food the parents provide to their offspring. We refer to these two effects as maternal inheritance and maternal selection, respectively, because often mothers have the strongest influence on the offspring, but our results apply equally to cases in which non-Mendelian inheritance or direct fitness effects result from the father. Here we develop quantitative-genetic models for the evolution of multiple traits under maternal inheritance (including both Mendelian and non-Mendelian effects) and maternal selection. Our primary goal is to understand how the mean phenotypes of one or more characters evolve under the influence of selection.

Numerous mechanisms are responsible for maternal inheritance. The early development of all metazoan embryos involves RNA transcripts and other cytoplasmic fac-
tors packaged in the egg by the mother during oogenesis (Davidson, 1986); embryogenesis therefore involves characters that are maternally inherited. Mitochondria, chloroplasts, and other cytoplasmic factors that have physiological effects throughout the life of an individual are transmitted directly from one parent to the offspring, often through the mother (Whitehouse, 1973; Grun, 1976). Maternal influences during the rearing of offspring can be strong. Body size in mammals, for example, typically shows effects from the uterine environment (Atchley and Newman, 1989) and maternal lactation performance (e.g., Bradford, 1972; Koch, 1972; Legates, 1972; Robison, 1972; Van Vleck et al., 1977). Cultural inheritance is another pathway by which the maternal phenotype can influence the phenotype of the offspring (Wright, 1931; Cavalli-Sforza and Feldman, 1981; Boyd and Richerson, 1985). Maternal inheritance is known from a wide variety of taxa; in addition to mammals, it has been identified in mollusks (Boycott et al., 1930), insects (Bondari et al., 1978; Janssen et al., 1988), fish (Reznick, 1981), birds (Price and Grant, 1985), and many groups of plants (Schaal, 1984; Roach and Wulff, 1987).

Experimental studies have shown that maternal inheritance can produce qualitatively different evolutionary outcomes than does simple Mendelian inheritance. Falconer (1965) found that artificial selection on litter size in mice results in a temporarily reversed evolutionary response. In the first generation of his experiment, selection for increased litter size produced offspring whose litters were smaller, while selection for decreased litter size produced offspring whose litters were larger. Falconer inferred that this was the consequence of a maternal effect acting through the body size of the mothers. Large females give birth to large litters consisting of pups that grow to be individually smaller than those born to small females. This is caused by competition between the offspring for maternal resources, particularly the mother’s milk during the lactation period. This physiological effect produces a negative correlation between the litter size of mothers and their offspring, rather than the positive correlation between parents and offspring that is assumed to exist for most traits because of Mendelian inheritance. Another striking example of maternal inheritance is the strongly negative mother-offspring correlation for age at maturity in natural populations of the springtail Orchesella cincta (Janssen et al., 1988).

Animal breeders have long been interested in maternal inheritance because of its substantial effects on the results of artificial selection. This interest has led to models for the evolution of a single trait that maternally affects itself (Falconer, 1965), and for the evolution of two traits, one of which maternally affects the other (e.g., Dickerson, 1947; Willham, 1963; Hanrahan, 1976; Van Vleck et al., 1977; Cheverud, 1984; Mueller and James, 1985; Lynch, 1987). The two-character model has been extended to cases in which the maternally affected trait is sexually dimorphic (Hanrahan and Eisen, 1973) and in which the maternal character affects its own expression as well as that of the offspring character (Willham, 1972; Riska et al., 1985). Many of these studies were primarily oriented towards analyzing sources of phenotypic variation.

In this paper, we analyze a model for the evolution of multiple, maternally inherited traits that includes the previous one- and two-character models as special cases. The results show that earlier studies did not obtain the correct general predictions for the response to selection because they did not account for evolutionary time lags that result from maternal inheritance. Furthermore, we find that the evolution of a single trait under selection depends on all the characters that maternally affect it, even when those other characters are not under selection. Predicting the response to selection in natural or artificial populations thus may require consideration of many characters.

Maternal selection can occur whether or not maternal inheritance is present. An example of maternal selection comes from the classic data of Karn and Penrose (1951). They studied the survival of human infants to 28 days of age as a function of their birth weight and the length of the gestation period. Data from 13,730 births show that stabilizing selection acts on both characters, favoring a birth weight of 7.8 pounds (3.5 kg) and a gestation period of 283 days. Furthermore, they found a pattern of correla-
tional selection between these characters: infants that were heavier than average had higher survival if their mothers had longer than average gestation periods. Conversely, infants that were lighter than average had higher survival if their mothers had shorter than average gestation periods. If one assumes that birth weight is an attribute of the offspring (although possibly influenced by maternal inheritance; e.g., Mi et al., 1986) and that gestation period is an attribute of the mother, these data illustrate how an interaction between maternal and offspring phenotypes can influence offspring fitness.

Maternal inheritance and maternal selection operate simultaneously on some characters. A potential example is hatching size in birds. The size of a hatching is a trait of the offspring. Its phenotypic value, however, is largely determined by the mother through the size of egg she makes, and so this trait is maternally inherited. In addition, hatching size would be under maternal selection if the probability that a chick survives depends on its mother’s phenotype (for example, the amount of care she provides) as well as on the size of the chick.

**Maternal Inheritance**

The size of a hatching is said to be inherited maternally because the phenotype of the offspring is influenced by the mother above and beyond her contribution to the nuclear genes of the offspring. We will begin by analyzing the simplest possible case, a single character maternally affecting itself. This case, although not biologically compelling, gives insight into the evolutionary consequences of maternal inheritance that distinguish it from Mendelian inheritance. These results are then generalized to more realistic situations involving multiple traits.

**One Character Maternally Affecting Itself.**—We begin by analyzing a model that was introduced by Falconer (1965) for a single maternally inherited trait. The phenotypic value for a trait in an individual is determined by the sum of three components: an additive genetic component, an uncorrelated component caused by environmental and nonadditive genetic effects, and the maternal effects contributed by the phenotype of the individual’s mother. The first two of these components form the basis for standard quantitative genetics (Falconer, 1981 p. 100). In principle, maternal effects may influence a trait in complex, nonlinear ways. We follow earlier workers (Dickerson, 1947; Willham, 1963; Falconer, 1965) by assuming that the maternal effect on an offspring is simply a linear function of the mother’s phenotype.

In the case of a single maternally inherited trait, the phenotypic value for an individual in generation $t + 1$ is

$$z(t + 1) = a(t + 1) + e(t + 1) + mz*(t)$$

where $a(t + 1)$ is the additive genetic component of the trait, $e(t + 1)$ is the component from the environment and from genetic interaction (dominance and epistasis), $m$ is the maternal-effect coefficient, and $z*(t)$ is the phenotypic value of the mother (a female that survived selection in the previous generation). The additive genetic component is assumed to be normally distributed with variance $G$. Following standard quantitative genetics, the “environmental” effect is defined so that it is independent of the additive genetic effect in zygotes and is assumed to be normally distributed with mean 0 and variance $E$. The overall phenotypic variance for the trait is denoted $P$. We will assume for simplicity that the variances $G$, $E$, and $P$ remain constant in time, a reasonable approximation when the character is determined by many loci and selection is weak (Bulmer, 1985). A submodel that accounts for the evolution of the variances and covariances, however, could be incorporated into this framework. Throughout this paper, we assume that mating is random and that the genes are inherited autosomally.

The coefficient $m$ measures the strength of the effect that the mother’s phenotype has on determining the phenotype of her offspring via maternal inheritance. It is defined as the partial regression of the offspring’s phenotype on its mother’s phenotype, holding genetic sources of variation (i.e., $a$) constant. This coefficient can be either positive or negative. A negative value indicates that females that are larger than average for the trait tend to produce offspring that are smaller than average when the genetic component of the trait is held constant. Several
cases of negative maternal effects are known, including litter size in mice (for which Falconer [1965] estimated \( m = -0.13 \)), and age at maturity in springtails (for which Janssen et al. [1988] estimated \( m \) to be between \(-0.58 \) and \(-0.40 \)). In the special case of \( m = 0 \), Equation (1) reduces to the classical equation for the determination of a quantitative character under Mendelian inheritance. Appendix 1 (section ii) shows that the absolute value of \( m \) must be less than one for the phenotypic variance to remain finite. Equation (1) can be extended to cases in which fathers (or other individuals in the population) also contribute non-Mendelian hereditary influence by adding terms analogous to the \( mz^*(t) \) term to describe the paternal influence.

We will assume that generations do not overlap beyond the period of parental expenditure. By taking the expectation of Equation (1), we find the mean value of the trait among newborns in generation \( t + 1 \) is

\[
\bar{z}(t + 1) = \bar{a}(t + 1) + mz^*(t) \\
= \bar{a}^*(t) + mz^*(t) \\
= \bar{a}(t) + C_{az}\hat{\beta}(t) \\
+ m[\bar{z}(t) + P\hat{\beta}(t)]
\]

where \( \bar{a}^* \) is the mean additive genetic component after selection. \( \hat{\beta}(t) \) is the selection gradient acting on the trait, which is a convenient measure of the force of directional selection. It is defined as the regression of relative fitness onto the phenotypic value for the trait and is related to the widely used phenotypic selection differential \( s(t) \) by the equation \( \hat{\beta}(t) = s(t)/P \). The selection gradient can be calculated for natural or domesticated populations using simple statistical methods (Lande and Arnold, 1983). \( C_{az} \) is the covariance between \( a(t) \) and \( z(t) \). In the absence of maternal effects, \( C_{az} \) is equal to the additive genetic variance. [Then \( C_{az}\hat{\beta}(t) \) reduces to the usual selection response \( h^2s(t) \), where \( h^2 \) is the heritability of the trait (Falconer, 1981 p. 172).] Appendix 1 (section ii) evaluates the covariance \( (C_{az}) \) and the phenotypic variance \( (P) \) in terms of the basic parameters \( G, E, \) and \( m \) and shows that maternal inheritance can cause \( C_{az} \) to be either larger or smaller than the additive genetic variance \( G \).

The evolutionary change in the mean of the character across one generation is

\[
\Delta\bar{z}(t) = \bar{z}(t + 1) - \bar{z}(t) \\
= [C_{az} + mP]\hat{\beta}(t) + m\Delta\bar{z}(t - 1) \\
- mP\hat{\beta}(t - 1).
\]

The three components of Equation (3) have straightforward interpretations. The first term represents the change in the character mean caused by selection in the current generation, \( \beta(t) \), and is comprised of two parts: \( C_{az} \) accounts for the change in the genetic component of the trait, while \( mP \) accounts for the change in the average maternal contribution to the next generation caused by the phenotypic effects of selection in the current generation. The second component of Equation (3), \( m\Delta\bar{z}(t - 1) \), is the change in the average maternal contribution caused by evolution of the trait in the previous generation. The third component, \(-mP\hat{\beta}(t - 1) \), accounts for the loss of the purely phenotypic effects of selection on the maternal contribution to the previous generation. Equation (3) assumes that selection acts on the maternal character in the same way in both sexes, as might be the case for a trait like body size in a species that is not sexually dimorphic. If the character is expressed only in females, as with litter size, then \( \hat{\beta}(t) \) refers to the selection gradient on females only, and the term \( C_{az} \) in Equation (3) is multiplied by \( 1/2 \).

Equation (3) reveals an evolutionary property of maternally influenced traits that distinguishes them from characters under Mendelian inheritance. In the absence of maternal effects, \( m = 0 \) and \( C_{az} = G \). Equation (3) then reverts to the conventional equation for the evolution of traits under Mendelian inheritance, \( \Delta\bar{z}(t) = G\hat{\beta}(t) = h^2s(t) \), which does not involve the previous history of selection on the population. Maternally influenced characters, in contrast, evolve in response to previous selective forces, as indicated by the factors \( \Delta\bar{z}(t - 1) \) and \( \beta(t - 1) \) in Equation (3), as well as current selective forces, contained in \( \hat{\beta}(t) \). The direct phenotypic transmission of characters from mother to offspring through maternal inheritance evidently introduces time lags into the evolutionary response to selection.
This has two interesting consequences. First, a population can exhibit a reversed response to selection. That is, selection favoring an increase of the character can actually cause the mean of the trait to evolve to smaller values, at least temporarily. Reversed response to selection will be observed when a population that is initially at equilibrium is subject to selection if \( C_{az} + mP < 0 \). If selection of constant intensity persists, however, the response to selection will change sign in the second generation. Eventually the oscillations will damp, and the population will evolve thereafter in the direction favored by selection. This evolutionary pattern was actually observed by Falconer (1965) in replicated selection experiments on litter size in mice. The possibility of reversed response to selection has been emphasized in several previous theoretical studies of maternal inheritance (e.g., Hanrahan, 1976; Cheverud, 1984; Lynch, 1987).

A second consequence of the time lags introduced by maternal inheritance is that, even under a constant intensity of selection, the ultimate evolutionary rate of change is only reached asymptotically. With Mendelian inheritance, in contrast, a population with nonoverlapping generations immediately attains its ultimate evolutionary rate. When selection on a maternally inherited trait ceases, the character does not immediately stop evolving. Rather, the mean of the trait will evolve at a geometrically decreasing rate, with \( \Delta z(t) = m\Delta z(t - 1) \). Using Equation (3) and results from Appendix 1 [Equations (A13) and (A14)], we find that, following the cessation of selection, the mean will evolve in an oscillatory manner when maternal effects are negative \( (m < 0) \), while the phenotypic mean can evolve in either the same direction or the opposite direction to that of the earlier selection when maternal effects are positive \( (m > 0) \), depending on the parameters. Maternal inheritance thus creates a form of evolutionary momentum in which the effects of selection in a particular generation persist and cause evolutionary change in subsequent generations. Simple Mendelian inheritance (i.e., involving autosomal genes with no epistasis), on the other hand, produces no such momentum effects: the mean phenotype stops evolving as soon as selection ceases. These properties of maternal inheritance are illustrated in Figures 1 and 2, which show numerical examples of evolutionary trajectories computed from Equation (3).

The evolutionary dynamics described by Equation (3) are general to any form of natural or artificial selection. We now examine the evolution of a population's mean phenotype for a single maternally inherited character under specific assumptions about the fitness function acting on the trait. In particular, we will ask how the trait evolves in the vicinity of a fitness peak.

A fitness function with a single peak can be approximated by a Gaussian function
with an optimum at \( z = \theta \) and a “variance” \( W \). The analysis presented in Appendix 2 (section i) shows that if selection is weak (\( W \gg P \)), the equilibrium at \( z = \theta \) will always be stable, which implies that the mean of the population will evolve toward the fitness peak. Under certain conditions, however, the initial generations of the approach to equilibrium involve oscillations in which the phenotypic mean may repeatedly overshoot the optimum in successive generations. Following this period, the mean approaches the optimum smoothly at an exponential rate. Further details are given in Appendix 2 (section i).

**Multiple Maternally Inherited Characters.**—Maternal effects generally involve more than a single character. We therefore wish to extend the approach of the previous section to situations involving multiple characters. If there are \( n \) characters, the phenotypic value for character \( i \) in an individual at generation \( t + 1 \) is

\[
z_i(t + 1) = a_i(t + 1) + e_i(t + 1) + \sum_{j=1}^{n} M_{ij} z_j^*(t)
\]

by analogy with Equation (1).

The component of character \( i \) that is caused by maternal effects is represented by the last term on the right-hand side of Equation (4). The coefficient \( M_{ij} \) measures the strength of the maternal effect of character \( j \) in the mother on character \( i \) in the offspring. It is defined as the partial regression of the phenotypic value of character \( i \) in the offspring on the phenotypic value of character \( j \) in its mother, holding the additive genetic value for that character \( (a_i) \) and the phenotypic values for all other characters constant. The additive genetic effects \( a_i \) are assumed to have a multivariate normal distribution with a covariance matrix \( G \) in zygotes. The “environmental” effects \( e_i \) are normally distributed with mean 0 and a covariance matrix \( E \) and are defined to be independent of the additive genetic effects in zygotes. The covariance matrix for the overall phenotypes is denoted as \( P \).

The model of Equation (4) allows for any network of Mendelian and maternal pathways of inheritance between the characters. This is illustrated by three special cases that have been considered by earlier workers and which are shown in Figure 3. The first (Fig. 3a) is that of a single character maternally affecting itself, as described by Falconer (1965) and analyzed in the previous section. The second case (Fig. 3b) involves a single maternal character with Mendelian inheritance, such as milk production or litter size, which affects a single offspring trait, such as body size (e.g., Dickerson, 1947; Willham, 1963; Cheverud, 1984). Denoting the maternal trait as character 1 and the offspring trait as character 2, the effect of the maternal trait on the offspring trait is measured in our model by the coefficient \( M_{12} \). If neither of the two traits maternally affects itself, then \( M_{11} = M_{22} = M_{12} = 0 \). In the third special case considered previously (Willham, 1972; Riska et al., 1985), the maternal trait influences itself as well as the offspring trait in a non-Mendelian way (Fig. 3c). In our notation, this effect is reflected in a coefficient \( M_{11} \) that is different from 0. The model of Equation (4) thus unifies the previous models into a single framework. It furthermore allows them to be generalized to more characters and to alternative pathways of maternal effects.

Proceeding as in the derivation of Equation (3), we find that the evolutionary change in \( \bar{z}(t) \), the vector of phenotypic means in generation \( t \), is

\[
\Delta \bar{z}(t) = \left[ C_{a2} + MP \right] \beta(t) + M \Delta \bar{z}(t - 1) - MP \beta(t - 1)
\]

where \( C_{a2} = \text{Cov}(a(t), z(t)^T) \) (T denotes matrix transposition), \( a(t) \) is the vector of additive genetic effects, \( \beta(t) \) is the vector of selection gradients, and \( M \) is an \( n \times n \) matrix of maternal-effect coefficients. The elements of the vector of selection gradients \( \beta \) are measures of the force of selection acting directly on each trait, independently of selection acting on the other traits. These gradients are equal to the partial regression coefficients in the multiple regression of individual relative fitness on the phenotypic value of the characters (Lande and Arnold, 1983). For characters expressed only in females, such as litter size and lactation performance, the gradients refer to selection on females only, and the matrix \( C_{a2} \) in Equation (5) should be multiplied by \( \frac{1}{2} \). The matrices \( C_{a2} \) and \( P \) are evaluated in Appendix
1 (section i). Eisen (1967) describes methods for estimating some of the parameters of Equation (5) for some special cases.

As in the case of a single trait maternally affecting itself, the evolutionary change in the means of multiple characters depends on the evolutionary change and selection gradients in the previous as well as the present generation. The interpretations of the terms of Equation (5) are identical to those for the single-character case in Equation (3).

Evolutionary momentum, the continued evolution of trait means after selection has ceased, will appear with multiple traits whenever there are pathways of maternal effects that lead back to the character where they originated after one or more generations—when one of the traits maternally affects itself, for example, or when one trait maternally affects a second trait which in turn maternally affects the first. (Technically, the condition is that there be cycles [Harary, 1969] in the maternal-effects matrix M.) The persistent effects will ultimately decline at a geometric rate governed by the eigenvalues of M. Multiple characters that are maternally inherited can also show reversed responses to selection in which one or more of the traits evokes against the direction favored by selection.

The resemblance between parents and offspring, specifically the regression of offspring phenotype on parental phenotype, is widely used to predict the evolutionary response to selection of characters under Mendelian inheritance (Falconer, 1981 pp. 136–140, 152–154; Bulmer, 1985 pp. 77–81). Although earlier workers have assumed that parent-offspring (or offspring-grandparent) regression can also be used with maternal inheritance (Dickerson, 1947; Williams, 1963; Hanrahan, 1976; Van Vleck, 1976; Cheverud, 1984; Riska et al., 1985; Lynch, 1987), our analysis shows that this is not generally valid. Under Mendelian inheritance, evolution of the character means in generation t is predicted from the equation \( \Delta \bar{z}(t) = b_{op}s(t) \), where \( b_{op} \) is the matrix of regression coefficients of offspring on their mid-parents and \( s(t) \) is the vector of phenotypic selection differentials within generation t (Lande, 1979; Falconer, 1981). (In the case of a single trait with Mendelian inheritance, the regression \( b_{op} \) equals \( h^2 \), the narrow-sense heritability, while in the multivariate case \( b_{op} = GP^{-1} \)). With maternal inheritance, however, this procedure only works for a single generation of selection applied to a population initially at equilibrium in the absence of selection. Using the value for \( b_{op} \) obtained in Appendix 1 [Equation (A12)] and the identity \( s = P\beta \) (Lande and Arnold, 1983), we see from Equation (5) that the response to one generation of selection is

\[
\Delta \bar{z}(1) = (C_{az} + MP)\beta(1) = b_{op}s(1). \tag{6}
\]

Appendix 1 [Equation (A12)] shows that, in the case of a single character, maternal inheritance can cause the regression \( b_{op} \) to be less than 0 or greater than 1, both of which outcomes are impossible with strictly Mendelian inheritance.

While Equation (6) agrees with the earlier theoretical predictions of the response to selection for maternally inherited characters, this result breaks down after the first generation of selection. As with a single maternally inherited trait, the ultimate rate of evolution for multiple traits generally will differ from the initial rate. Even when the force of selection is constant and the parameters of inheritance (\( G, E, \) and \( M \)) remain constant, the evolutionary rate will fluctuate and then settle down to an asymptotic rate of evolution different than the initial rate.
Equations (5) and (A2) show that the asymptotic rate of evolution will be
\[
\Delta \bar{x}(\infty) = (I - M)^{-1}C_{az}\beta \\
\approx (I - M)^{-1}G(I - \frac{1}{2}M^T)^{-1}\beta. \quad (7)
\]
The asymptotic rate can be either larger or smaller than the initial rate, depending on the parameters of inheritance. More generally, when the strength or direction of selection is not constant, the time lags introduced by maternal inheritance ensure that it is usually not possible to predict the evolutionary change using only information about the force of selection acting in the current generation. Thus, the method of basing predictions about a population’s response to selection on the phenotypic regression of offspring on parents is not generally valid when maternal effects are present.

Evolutionary momentum causes fluctuations in response to the onset of selection and continued evolution after selection ceases, as we have described. The net cumulative change in the means of the characters after these transient effects pass can be determined by summing across generations in Equation (5). If a population experiences \(k\) generations of selection and then is allowed to reach equilibrium in the absence of further selection, the total evolutionary change in the phenotypic means will be
\[
\Delta \bar{x}_T = \bar{x}(\infty) - \bar{x}(0) \\
= (I - M)^{-1}C_{az}\sum_{t=1}^{k} \beta(t) \quad (8a) \\
= (C_{az} + MC_{az} + M^2C_{az} + \ldots) \sum_{t=1}^{k} \beta(t). \quad (8b)
\]
The first term on the right-hand side of (8b), \(C_{az}\), stems from the changes in the additive genetic components of the traits. The following terms \((MC_{az}, M^2C_{az}, \text{ etc.})\) represent changes in the average maternal contributions to the traits, with each successive term arising from an additional loop of feedback through the pathways of maternal effects. While Mueller and James (1985) and Van der Steen (1985) recognized that changes in the average maternal effects through this feedback contribute to the total evolutionary change, earlier calculations that consider only the genetic component (e.g., Dickerson, 1947; Hanrahan, 1976) do not give the correct predictions.

An important conclusion from these analyses is that the evolution of the means of characters experiencing selection can depend on the parameters of inheritance of traits that are not themselves under selection. Even if a single maternally influenced trait such as body size is the only trait under selection in a population, its evolution depends on the details of the genetic and maternal interactions \((C_{az} \text{ and } M)\) between all other traits tied to it through pathways of maternal inheritance (e.g., qualities of the placenta, lactation rate, litter size). This differs from Mendelian inheritance, in which only information on the inheritance of the traits actually experiencing selection is needed to predict their evolution. Furthermore, the lumping of multiple characters that have maternal influences into a single “maternal performance” trait, as suggested by some workers (Willham, 1972; Cheverud, 1984; Riska et al., 1985; Lynch, 1987), will produce inaccurate predictions of the evolutionary trajectory. Equations (5)–(8) cannot in general be reduced to a two-character model, even when only a single trait is under selection, if that trait is connected (directly or indirectly) to more than one other character by pathways of maternal inheritance. One reason for this is that a set of \(n\) characters can produce lags in evolutionary response of up to \(n\) generations. It is possible to construct examples in which one generation of selection on one of \(n\) traits produces no immediate evolutionary change in that trait but does cause the trait to evolve \(n\) generations later.

Some of the evolutionary consequences of maternal inheritance for multiple characters can be illustrated by a special case involving only two characters. Consider a situation in which a trait in offspring is maternally influenced by another character that is itself inherited by simple Mendelian mechanisms (studied previously by Dickerson [1947], Willham [1963], Van Vleck [1976], Cheverud [1984], and Riska et al. [1985]). Appendix I (section iii) shows that the evolutionary dynamics of the two traits are given by
\[ \Delta z_m(t) = \left( G_{m0} + \frac{m}{2} G_{mm} \right) \beta_o(t) + G_{mm} \beta_m(t) \]

and

\[ \Delta \bar{z}_o(t) = \left( G_{oo} + \frac{3m}{2} G_{mo} + \frac{m^2}{2} G_{mm} \right) \cdot \beta_o(t) + (G_{mo} + mG_{mm}) \beta_m(t) + mE_{mo} [\beta_o(t) - \beta_o(t - 1)] + mE_{mm} [\beta_m(t) - \beta_m(t - 1)] \]

where the subscript 0 indicates the offspring character and the subscript m the maternal character (e.g., \( E_{mo} \) is the environmental covariance between the maternal and offspring characters). The strength of the maternal effect is denoted \( m = M_{on} \). In the event that the maternal character is limited to females, the maternal selection-gradient \( \beta_m(t) \) again refers to the females only. In this case, the terms \( G_{mm} \beta_m(t) \) in Equation (9) and \( G_{mo} \beta_m(t) \) in Equation (10) are multiplied by \( \frac{1}{2} \).

Equation (10) agrees with the results derived by previous authors, who implicitly assumed \( \beta_m = 0, m = 1 \), and \( E_{mo} = 0 \) (Dickerson, 1947; Willham 1963; Hanrahan, 1976; Cheverud, 1984; Riska et al., 1985). Under these conditions, there is no contribution to the change in the mean of the offspring trait from the selection gradient in generation \( t - 1 \), so there are no evolutionary lags, although evolutionary momentum will be seen when these conditions do not hold. Among those who have considered this case previously, Mueller and James (1985), Van Vleck et al. (1977), and Van der Steen (1985) mention that with a constant intensity of selection the evolutionary response in the second generation will differ from that in the first. Falconer (1965) made the same observation in the case of a single, maternally inherited character.

Examination of these equations shows that a system of multiple, maternally inherited traits, unlike the case of a single character, can cause evolution to proceed in a direction opposite to what selection favors for an indefinitely long time. This outcome, which has been discussed by Cheverud (1984), is favored by a strong negative relationship between the traits on the genetic or environmental levels \( mG_{mo} < 0 \) or \( mE_{mo} < 0 \). Even when such antagonistic effects between the maternal and offspring characters are not strong enough to cause a reversed response to selection, a negative genetic covariance between the offspring and maternal characters \( G_{mo} \) will impede the evolutionary response to selection, since selection to increase the offspring character causes maternal performance to decrease as a correlated response (Dickerson, 1947; Willham, 1963).

Some maternal characters, such as lactation performance in mammals, have maternal effects on themselves as well as on the offspring character. The evolutionary dynamics for this more general case are derived in Appendix 1 (section iii).

**Maternal Selection**

Maternal selection on an offspring character occurs when the fitness of the offspring is determined jointly by the value of its character and by the value of some trait in the mother. For example, hatching size in chicks is under maternal selection when the probability of chick survival depends on both the size of the chick and the amount of care the mother provides. The presence or absence of maternal selection does not depend on whether the traits are influenced by maternal inheritance. Hatching size in birds is likely to be under both maternal selection and maternal inheritance. Maternal selection differs from what population geneticists refer to as “fertility selection” (Bodmer, 1965) because it affects the survival of zygotes rather than the number of gametes produced by a female.

A complexity introduced by maternal selection is that it changes the distribution among offspring of traits that they have not yet expressed. Maternal selection acting on chicks, for example, can cause selection on the body size that the chicks will reach as adults. Because we are unable to measure changes in characters that have not yet been expressed, we will determine the statistical impact of maternal selection acting on unexpressed traits using data from observable characters. Once the intensity of selection has been determined, models of either Mendelian inheritance or maternal inheri-
tance, as appropriate, can be used to predict the evolution of the population.

We begin by considering the simplest case of maternal selection acting on a single character that is expressed in offspring and in mothers with no ontogenetic change. Having illustrated the concepts, we then treat more realistic situations involving multiple traits that may be expressed differently in offspring and adults.

One Trait under Maternal Selection. — To determine the impact of maternal selection on the offspring, we first consider the effects of the mother’s phenotype and the offspring’s phenotype separately. The effect of the phenotypes of offspring on their own fitness in generation \( t \) is measured by the offspring selection component, \( b_o(t) \), while the effect of maternal phenotypes on the fitness of offspring in generation \( t \) is measured by the maternal selection component, \( b_m(t) \). The selection components \( b_o(t) \) and \( b_m(t) \) are defined, respectively, as the partial regression coefficients from the multiple regression of relative offspring fitness in generation \( t \) onto offspring phenotypes and maternal phenotypes. The selection component \( b_o \) measures the effect of the offspring phenotype on offspring fitness, holding the maternal phenotype constant, while the selection component \( b_m \) measures the effect of the maternal phenotype on offspring fitness, holding the offspring phenotype constant. Given data on the phenotypes of mothers and their offspring and the fitnesses of the offspring, \( b_o \) and \( b_m \) can be determined using standard statistical methods (see Lande and Arnold, 1983).

The impact of maternal selection on the offspring phenotypes can now be calculated in terms of the selection differential in generation \( t \), the change in the mean phenotype of offspring of generation \( t \) caused by maternal selection. This selection differential is the sum of the separate contributions from the two selection components:

\[
s(t) = Pb_o(t) + C_m b_m(t) \tag{11}
\]

where \( P \) is the phenotypic variance in offspring and \( C_m \) is the covariance between the phenotypic values of the trait in mothers and in offspring, both measured before selection acts. The value of \( C_m \) depends on the form of inheritance. If the resemblance of parents and offspring is caused only by Mendelian inheritance, \( C_m \) is equal to one half of the additive genetic variance (Falconer, 1981 p. 136), while under maternal inheritance it is given by Equation (A15) from Appendix 1. If common-environmental effects contribute to the phenotypic covariance between mothers and offspring, the value of \( C_m \) should be increased by the appropriate amount. Equation (11) can be extended to include simultaneous paternal and maternal selection by adding the term \( C^P b_o(t) \) to the right-hand side, where \( b_o(t) \) is the paternal selection component and \( C^P \) is the phenotypic covariance between fathers and offspring.

The two terms on the right-hand side of Equation (11) have simple interpretations. The first is the usual change in the mean phenotype within a generation caused by selection acting directly on offspring. The second is the effect of the maternal phenotype on offspring fitness, which generates selection in the offspring through the phenotypic covariance between mothers and offspring. This component depends in part on the resemblance between parents and offspring, represented by \( C_m \). Maternal selection differs in this way from other forms of selection, where the phenotypic effects of selection can be measured independently of the parameters of inheritance.

The selection differential can be used to determine the evolution of the mean of the trait using an appropriate model of inheritance. Under Mendelian inheritance, the evolutionary change in the phenotypic means is

\[
\Delta z(t) = G s(t) = G \left[ b_o(t) + \frac{C_m}{P} b_m(t) \right]. \tag{12}
\]

If the trait is maternally inherited, the evolution of its mean is described by Equation (3). For use in that equation we calculate the net selection gradients \( \beta(t) = s(t)/P \) and \( \beta(t - 1) = s(t - 1)/P \), where the selection differentials \( s(t) \) and \( s(t - 1) \) are again determined using Equation (11).

We now consider the evolutionary consequences of weak stabilizing maternal selection acting on a single trait under Men-
delian inheritance. As suggested by the data of Karn and Penrose (1951), we imagine that offspring survival is proportional to a bivariate Gaussian function of the phenotype of the offspring and its mother. For simplicity, the optimal value for the trait is assumed to be the same in mothers and offspring. The strength of selection favoring the optimum is determined by the "covariance" matrix,

\[
W = \begin{bmatrix} w_{mm} & w_{mo} \\ w_{mo} & w_{oo} \end{bmatrix}.
\]

The coefficients \(w_{mm}\) and \(w_{oo}\) are inversely proportional to the strength of stabilizing selection acting on the trait in mothers and offspring, respectively, while the coefficient \(w_{mo}\) measures the strength of selection favoring a correlation between the phenotypic values of mothers and their offspring.

An analysis of the evolution of the population mean is presented in Appendix 2 (section ii). The evolutionary equilibrium for the population is at the point where the mean phenotypes of both mothers and offspring lie at the optimum value for the trait. The equilibrium is unstable if

\[
G(w_{oo} - w_{mo}) + 2P(w_{mm} - w_{mo}) < 0. \tag{13}
\]

This indicates that instability is favored by stabilizing selection acting more strongly on mothers than offspring (\(w_{oo} \gg w_{mm}\)), strong correlational selection (\(w_{mo} > w_{mm}\)), and low heritability (\(G \ll P\)). Under these conditions, the trait will continue to evolve maladaptively, away from the fitness peak, until the parameters of selection or inheritance change enough that Condition (13) is no longer met. An example of such a trajectory is shown in Figure 4. Condition (13) may be sufficient extreme that it is satisfied only very rarely in nature, but the relevant parameters have not been measured.

If the equilibrium is stable, the population will evolve towards it. The approach can involve oscillations, including possible overshoot of the optimum, if \(2Pw_{mo} < Gw_{oo}\). These oscillations will ultimately damp out, and the population will then evolve smoothly toward the optimum.

**Multiple Characters under Maternal Selection.** — We now generalize our results for the evolutionary dynamics to situations in which there are multiple characters. Traits that change during ontogeny can also be included in this framework by treating each trait that changes as two separate characters, one expressed only in offspring and the other only in mothers.

For \(n\) characters under maternal selection there are \(2n\) selection components, as each trait can potentially be expressed in both offspring and mothers. These components can be written as the vectors \(\mathbf{b}_m(t)\), with elements that are the maternal selection components for characters 1 through \(n\), and \(\mathbf{b}_o(t)\), with elements that are the corresponding offspring selection components. Selection components for characters that are not expressed at one of the two life stages take on values of 0. Maternal selection produces a vector of \(n\) selection differentials for the characters in the offspring. The selection differentials are given by the multivariate generalization of Equation (11):

\[
\mathbf{s}(t) = \mathbf{Pb}_o(t) + \mathbf{C}^m \mathbf{b}_m(t) \tag{14}
\]

where \(\mathbf{C}^m_{ij}\) is again the phenotypic covariance between trait \(i\) in the offspring and trait \(j\) in the mother before selection acts. With Mendelian inheritance, the elements are equal to one half of the corresponding additive genetic covariance, while under maternal inheritance these covariances are given by Equation (A8) of Appendix 1.

Under Mendelian inheritance, the evolutionary dynamics of the vector of means of the characters are determined by the standard equation.
\[ \Delta \mathbf{z} = \mathbf{G} \mathbf{P}^{-1} \mathbf{s}(t) \]  

(Magee, 1965; Dickerson et al., 1974; Yamada, 1977; Lande, 1979), where \( \mathbf{s}(t) \) is given by Equation (14). If the traits are maternally inherited, their evolution is described by Equation (5), using the vectors of net selection gradients \( \mathbf{\beta}(t) = \mathbf{P}^{-1} \mathbf{s}(t) \) and \( \mathbf{\beta}(t - 1) = \mathbf{P}^{-1} \mathbf{s}(t - 1) \), where the vectors \( \mathbf{s}(t) \) and \( \mathbf{s}(t - 1) \) are again given by Equation (14).

Biologists are often interested in maternal selection involving two characters, the first of which is expressed only in adult females and the second only in young. An example is Karn and Penrose’s (1951) study of how gestation time and birth weight affect human infant survival. Denote gestation time as trait 1 and birth weight as trait 2. Two of the four possible selection components, \( b_{21}(t) \) and \( b_{22}(t) \), take on values of 0, because the gestation time is not expressed in young, while birth weight is not expressed in adults. By writing out Equation (14) in full, we find that the selection differentials for the two characters are

\[ s_1(t) = C^{m}_{11} b_{m1}(t) + P_{12} b_{02}(t) \]
\[ s_2(t) = P_{22} b_{02}(t) + C^{m}_{21} b_{m1}(t). \]

Each selection differential is the sum of two terms. In Equation (16), the first term is the change in the mean gestation time the offspring will express as adults that is caused by the effects of the mothers’ gestation time on offspring fitness. The second term is the change in that mean caused by selection acting on offspring birth weight. In Equation (17), the first term is the change in the mean birth weight of offspring caused by selection acting directly on that trait, while the second term is the change in that mean caused by effects of the mother’s gestation time.

This case illustrates the point that maternal selection generates selection differentials in offspring for characters that they have not yet expressed. For example, the selection differential for the gestation time that the offspring will show when they become mothers, \( s_1(t) \), can be nonzero, even though those individuals do not express that trait until they mature. This selection differential has evolutionary effects, even though the differential cannot be observed directly. The evolution of the phenotypic means for the two characters is determined for either Mendelian or maternal inheritance as described above [Equation (15) and following].

**DISCUSSION**

Maternal characters evolve in qualitatively different ways than do most traits studied by evolutionary biologists. The differences arise from two sources: maternal inheritance and maternal selection.

Our analysis of maternal inheritance shows that it produces time lags in the response of a population to selection. Conventional forms of Mendelian inheritance produce evolutionary dynamics in which the response of a population to selection depends on the force of selection in the current generation. Under maternal inheritance, however, the response to selection depends on the force of selection and the evolutionary response in the previous generation, as well as the force of selection in the current generation. The time lags lead to unusual effects. The rate of evolution generally will change even when the strength of selection and the parameters of inheritance remain constant. The rate after two or more generations of selection can be either greater or less than the rate after one generation of selection. The population will ultimately achieve an asymptotic evolutionary rate if the parameters remain constant. The asymptotic rate of evolution may not be reached in real populations, however, since the parameters may change. Even should the strength of selection and the genetic variables stay constant, the maternal-effect coefficients may change as the means of the traits evolve (Arnold, 1988).

Another novelty introduced by the time lags is that the effects of selection persist after selection ceases, causing the trait means to continue evolving. We refer to this as evolutionary momentum. The total amount of evolutionary change that can occur after selection has ended is limited, however, since the momentum ultimately declines in a geometric manner. A hypothetical example gives a better intuitive understanding of the cause of the momentum effect. Consider a strain of mice in which there is no genetic variation. Imagine that body size is
inherited with a negative maternal effect \((-1 < m < 0)\), perhaps because large mothers produce poor quality milk for physiological reasons. The population, which is initially at equilibrium, is subjected to a single generation of selection for increased body size and thereafter experiences no selection. The offspring of the selected generation will then be smaller than their parents because of the negative maternal effect. These individuals, in turn, will produce offspring that are on average larger than themselves. Successive generations will oscillate in mean body size from the effects of evolutionary momentum. The oscillations are damped out as the impact of the maternal effect is diluted by random environmental variation which is introduced in each generation. If instead the maternal effect is positive \((0 < m < 1)\), there will also be evolutionary momentum, but the approach to equilibrium will be monotonic rather than oscillatory. In these two examples, the mean body size ultimately returns to its original value, because none of the variation is genetic. When a genetic component is present, maternal inheritance will produce the same kind of momentum effects as just described, but there will be a permanent change in the mean [see Eq. (8)].

The evolutionary momentum produced by maternal inheritance is reminiscent of Pearson's (1898) formulation of Galton's "Law of Ancestral Heredity," in which all previous ancestors of an individual contribute to its phenotype. Pearson's model, however, produces an infinite series of time lags that involve all previous generations, while in our model of maternal inheritance the evolution of the phenotypic mean depends only on the means of the current and immediately preceding generations. The physiological mechanisms considered by more recent workers (e.g., Van Vleck, 1976; Reznick, 1981; Riska et al., 1985) also involve non-Mendelian effects that extend only across a single generation and so are compatible with (and described by) the models developed here. In contrast, some characters, such as cultural traits, are influenced by non-Mendelian effects contributed from more distant relatives (Cavalli-Sforza and Feldman, 1981; Boyd and Richerson, 1985), and our models would have to be extended to include such effects.

Predicting the evolution of maternally inherited traits requires knowledge not only of the inheritance of the traits actually experiencing selection, but also of all the maternal characters affecting them. This is fundamentally different from Mendelian inheritance, in which characters not under selection have no effect on the evolution of the selected traits. Empirical workers are thus confronted with a formidable challenge. First, the full set of characters under selection must be identified, as is the case with Mendelian inheritance (Lande and Arnold, 1983). Second, the set of all maternal traits affecting them must be identified. Third, the parameters of maternal and genetic inheritance for these traits must be estimated. In many cases, merely identifying the traits involved may be difficult. Furthermore, methods for estimating \(G\) and \(M\) in cases involving more than two traits are still being developed (R. Lande and T. Price, unpubl.). Some workers (e.g., Dickerson, 1947; Willham, 1972; Riska et al., 1985) have suggested grouping sets of traits with maternal effects on a character of interest into a single "maternal performance" meta-character. While this approach may be useful for describing the sources of phenotypic variation in the target character, it generally gives inaccurate predictions for the evolution of traits influenced by multiple maternal effects.

Maternal inheritance can augment or retard evolution relative to what is possible with Mendelian inheritance. With a single character that influences itself through a large, positive maternal effect, the response to selection, \(\Delta z(t)\), can actually exceed the selection differential, \(s(t)\). A negative maternal effect, on the other hand, can cause the population to evolve initially in the direction opposite to that favored by selection. While evolution will ultimately reverse and proceed in the direction favored by selection in the case of a single character, this is not necessarily true for systems comprised of two or more traits. If a trait is negatively genetically correlated with another character that influences it maternally, selection to increase the trait will reduce maternal performance as a correlated response (William, 1963). This situation is found in mammals, where selection for increased body size
often adversely affects lactational performance and other components of maternal care (e.g., Legates, 1972; Hanrahan and Eisen, 1973; Van Vleck et al., 1977; see also Riska et al. [1985]).

Maternal inheritance allows two pathways by which a trait can respond to selection. The trait can be altered by changes in its additive genetic component or its maternal component. If selection favors the evolution of a particular mean value of a maternally inherited trait, this can be achieved by evolution of the additive genetic component, evolution of the maternal component, or a combination of the two.

Maternal characters have effects on selection as well as on inheritance. Maternal selection causes the fitness of an individual to depend not just on its own phenotype, but on that of its mother as well. Since the probability of having a certain type of mother depends on the composition of the population, maternal selection is necessarily frequency-dependent. This means that there is no guarantee that selection will lead to an increase in the population’s mean fitness, as is the case with frequency-independent selection and Mendelian inheritance (Wright, 1969; Lande, 1979). Our finding that weak stabilizing maternal selection can cause a population to evolve away from a fitness peak shows that this kind of selection can in fact lead to maladaptive evolution.

This outcome can be intuitively understood in the case of a single trait under maternal selection and Mendelian inheritance. Whenever there is a correlational component in the fitness function \( w_{mo} \neq 0 \), the value of the trait in mothers that maximizes their offspring’s fitness depends on the phenotype of their offspring. The pattern of selection can be such that an increase in offspring phenotype favors a more than proportional increase in the mother’s phenotype. If the increase in the maternal phenotype that is favored is sufficiently large, it can override direct selection favoring a smaller optimal value of the trait in offspring. The mean value of the trait will then increase between generations, which in turn favors even larger values of the trait in mothers, thus creating positive feedback and an unstable evolutionary process. Under these conditions, the population will evolve deterministically away from a fitness peak, leading to continually lower population mean fitness.

A peculiar feature of maternal selection is that its strength depends not only on extrinsic ecological factors, but also on the phenotypic correlation of parents and offspring. This is because the impact of a mother’s phenotype on the fitness of her offspring is translated into selection on the offspring through this correlation. The strength of simple viability selection, in contrast, is independent of this correlation.

Maternal selection can therefore lead to a confusion between selection and heredity, since the strength of selection is influenced by heredity through its effect on mother-offspring resemblance. To clarify the concepts, consider the problem of predicting the evolution of hatchling size in a population of birds when mothers influence both the size of the hatchlings they produce through maternal inheritance and the probability that their offspring successfully fledge through maternal selection. The parameters of inheritance for egg size, including the additive genetic effects and the maternal effects, can be calculated from phenotypic measurements of hatchling size and other traits in various relatives (see Willham, 1963; Eisen, 1967; Hanrahan and Eisen, 1973; Riska et al., 1985; R. Lande and T. Price, unpubl.). A study of hatchling size would typically show that it is strongly maternally inherited (Price and Grant, 1985). Maternal selection on size at hatching can be quantified by first determining the selection components acting on the offspring trait (hatchling size) and the maternal characters (such as the amount of parental care mothers provide). The multiple-regression coefficients of relative offspring fitness on the maternal and offspring traits are used to estimate these two selection components (see Lande and Arnold, 1983; Arnold and Wade, 1984; Price et al., 1984). Given these data on inheritance and selection, the evolutionary impact of maternal selection and maternal inheritance can be determined from the dynamic equations presented above.

We have considered here only maternal influences on heredity and selection. Many traits, however, are influenced in both regards by relatives other than the mother.
For example, many cultural traits show more complex forms of inheritance (Wright, 1931; Cavalli-Sforza and Feldman, 1981; Boyd and Richerson, 1985). Biparental care and other social interactions can be expected to cause selection that involves additional kinds of between-generation interactions. As indicated above, the models described here can be extended to include these effects. We expect that they will produce evolutionary dynamics even more complex than those described here.

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APPENDIX 1

This appendix develops results concerning characters influenced by maternal inheritance. We first derive the variances and covariances for traits at equilibrium in the absence of selection. These results are then used to determine the dynamics of populations under weak selection for special cases of interest involving one and two characters.

1) Equilibrium covariances in the absence of selection. —In the general case of multiple characters, the formulas for the evolution of the phenotypic means involves the covariance matrices C_m and P. By taking the covariances of a and z, from Equation (4) we find that

$$C_{az}(t) = G + C_{a}*(t, 1)M^T + C_{a}*(t, 2)M^T + \ldots, \quad (A1)$$

where $C_{a}*(t, r) = Cov[a(t), a^{*}(t-r)].$ This expression is difficult to evaluate in general. The problem is considerably simplified if selection is absent and the population has attained equilibrium. In this case, $C_{az}(t, r) = (\langle a \rangle)C_{a}(t),$ and we find that $C_{a}(t)$ equilibrates to the value

$$C_{a} = G \sum_{r=0}^{\infty} \left( \frac{1}{2} M^T \right)^r = G(1 - \frac{1}{2} M^T)^{-1}, \quad (A2)$$

where I is the identity matrix.

By taking the variance of Equation (4), we find that the phenotypic covariance matrix P(t) reaches an equilibrium determined implicitly by the matrix equation

$$P = G + E + M M^T + \frac{1}{2} M C_{a} M^T + \frac{1}{2} C_{a} M^T.$$

The general solution of Equation (A3) is obtained by defining a column vector p with n^2 elements, formed by stacking the rows of P:

$$p = [P_1, P_2, \ldots, P_n]^T, \quad (A4)$$

in which P_i is the i^th row of P. Similarly, let $Y = G + E + \frac{1}{2} M C_{a} M^T + \frac{1}{2} C_{a} M^T$ and

$$y = [Y_{1}, Y_{2}, \ldots, Y_{n}]^T, \quad (A5)$$

in which Y_i is the i^th row of Y. The system of equations in (A3) then can be written as

$$p = (M \otimes M)p + y, \quad (A6)$$

where the $n^2 \times n^2$ matrix in parentheses is the Kronecker product of M with itself (Bellman, 1960). The i^th block of n \times n elements of $M \otimes M$ is the matrix M multiplied by the element $M_{i.j}.$ The solution for p, which is the vector of equilibrium phenotypic variances and covariances, is

$$p = (I - (M \otimes M))^{-1} y. \quad (A7)$$

To evaluate this expression in the particular cases discussed below, we make use of identities from Bellman (1960 p. 228) which show that

$$[I - (M \otimes M)]^{-1} = \sum_{i=0}^{\infty} (M \otimes M)^{i} = \sum_{i=0}^{\infty} (M \otimes M)^{i}.$$

This last expression implies that the eigenvalues of M must all have modulus less than 1 for the variances to
remain finite (Bellman, 1960). In the case of a single maternally inherited trait, this implies that $|m| < 1$.

The phenotypic covariances between traits in parents and their offspring can also be evaluated at this equilibrium. Defining $C_m$, as the covariance between trait $i$ in an offspring and trait $j$ in its mother, taking covariances from text Equation (4) in the absence of selection shows that

$$C_m = \frac{1}{2}C_{ai} + MP = \frac{1}{2}G(I - \frac{1}{2}M^T)^{-1} + MP \tag{8}$$

Denoting the covariance between trait $i$ in an offspring and trait $j$ in its father as $C_{ij}$, the equilibrium values are given by the relation

$$C_i = \frac{1}{2}C_{ai} = \frac{1}{2}G(I - \frac{1}{2}M^T)^{-1} \tag{9}$$

From these results, we find that the matrix of partial regression coefficients of offspring on their mothers is

$$b_{om} = C = P^{-1} = \frac{1}{2}C_{ai}P^{-1} + M \tag{10}$$

the regression of offspring on fathers is

$$b_{op} = C'P^{-1} = \frac{1}{2}C_{ai}P^{-1} \tag{11}$$

and the regression of offspring on the mean of their parents is

$$b_{op} = (C + C')P^{-1} = C_{ai}P^{-1} + M \tag{12}$$

In the case of a single character, this last measure reduces to $h^2$ (the narrow-sense heritability) when the maternal effect $m$ equals 0. Maternal inheritance, in contrast to Mendelian inheritance, can cause the regression of offspring on midparent to be less than 0 or greater than 1.

The results above can be applied to predict the response to selection, under the assumption that selection is sufficiently weak that it does not appreciably alter the variances and covariances. We will now use this assumption to examine two special cases.

ii) A single maternally inherited trait. —This case was considered previously by Falconer (1965) and is illustrated schematically in Figure 3a. At an equilibrium in the absence of selection, the variances and covariances for a single maternally inherited trait can be calculated from Equations (A2) and (A7–A9) to be

$$C_{ai} = \frac{2G}{2 - m} \tag{13}$$

$$P = \frac{(2 + m)G + (2 - m)E}{(2 - m)(1 - m^2)} \tag{14}$$

$$C_m = \frac{G}{2 - m} + mP \tag{15}$$

$$C_i = \frac{1}{2}C_{ai} = \frac{G}{2 - m} \tag{16}$$

These results were obtained previously by Falconer (1965).

The dynamics of the population mean is determined by substituting Equations (A13) and (A14) into Equation (3). The response of a population that is initially at equilibrium to one generation of selection is

$$\Delta\theta(1) \approx \left[ \frac{2G}{2 - m} + mP \right] \beta(1). \tag{17}$$

The ultimate rate of evolution under constant directional selection is found from Equation (7) to be

$$\Delta\theta(\infty) \approx \frac{2G}{(2 - m)(1 - m)} \beta. \tag{18}$$

This rate of change in the character mean is only reached asymptotically because of the effects of the maternal contribution to the phenotype. The initial response to selection will be greater than the asymptotic rate if the maternal effect is positive and smaller if the maternal effect is negative. Equation (A18) shows that increasingly large values of the maternal effect coefficient $m$ will always have the effect of increasing the ultimate rate of evolution.

iii) One maternal and one offspring trait. —The second special case involves two characters, a maternal character $z_m$ and an offspring character $z_o$. We will study the case in which the maternal character maternally affects the offspring character and itself. This situation, considered previously by Willham (1972) and Riska et al. (1985), is a generalization of the case in which the maternal character itself is determined by purely Mendelian inheritance, which has been discussed by many workers (e.g., Dickerson, 1947; Willham, 1963; Hanrahan, 1976; Van Vleck, 1976; Cheverud, 1984). The paths of inheritance are shown schematically in Figure 3b and c.

Writing the vector of characters as $z = (z_m, z_o)^T$, the maternal effects matrix is

$$M = \begin{bmatrix} p & 0 \\ m & 0 \end{bmatrix}.$$ 

For situations in which the maternal character is under pure Mendelian inheritance, $p = 0$. From (A2) we have

$$C_{ai} = \begin{bmatrix} 2G_{nm} & G_{mo} + mG_{mn} \\ \frac{2}{2 - p} & \frac{2}{2 - p} \end{bmatrix} \tag{19}$$

Since in this case $M^2 = pM$, it follows that

$$[I - (M \otimes M)]^{-1} = I + M \otimes M \sum_{r=0}^{\infty} p^r = I + (1/(1 - p))M \otimes M.$$ 

Hence,

$$\begin{bmatrix} p_{mm} \\ p_{mo} \\ p_{cm} \\ p_{co} \end{bmatrix} = \begin{bmatrix} 1/(1 - p^2) & 0 & 0 & 0 \\ mp/(1 - p^2) & 1 & 0 & 0 \\ mp/(1 - p^2) & 0 & 1 & 0 \\ m^2/(1 - p^2) & 0 & 0 & 1 \end{bmatrix} \begin{bmatrix} \frac{2G_{nm}}{2 - p} & \frac{2G_{mo} + mG_{mn}}{2 - p} \\ \frac{1}{2 - p}G_{nm} + E_{nm} & \frac{1}{2 - p}G_{mo} + mE_{mn} + m(2/p)p_{mm} \\ \frac{2}{2 - p}G_{nm} + E_{nm} + m(2/p)p_{mm} & \frac{2}{2 - p}G_{mo} + mE_{mn} + m(2/p)p_{mm} \end{bmatrix}.$$  

(A20)
Multiplying this expression out, we find

\[ P_{mn} = \frac{(2 + p)G_{mm} + (2 - p)E_{mm}}{(1 - p^2)(2 - p)} \]  

(A21)

\[ P_{oo} = G_{oo} + E_{oo} + \frac{2m}{2 - p} G_{mo} \]

\[ + \frac{m^2(2 + p)}{(2 - p)(1 - p^2)} G_{mm} \]

\[ + \frac{m^2}{(1 - p^2)} E_{mm}. \]  

(A22)

\[ P_{mo} = P_{om} \]

\[ = \frac{m(1 + 2p)}{(1 - p^2)(2 - p)} G_{mm} + \frac{2}{2 - p} G_{mo} \]

\[ + E_{mo} + \frac{mp}{1 - p^2} E_{mm}. \]  

(A23)

The only restriction on the parameters is that \(|p| < 1\). Our results for the equilibrium variances differ from those obtained by Riska et al. (1985 p. 296).

The equilibrium phenotypic covariances between mothers and offspring are

\[ C^m = \frac{1}{2} C_{ar} + \begin{bmatrix} pP_{mm} & pP_{mo} \\ mp_{mm} & mp_{mo} \end{bmatrix} \]  

where the values for \(P_{mm}\) and \(P_{mo}\) are given above by Equations (A21) and (A23). The phenotypic covariances between fathers and offspring are

\[ C^f = \frac{1}{2} C_{ar}, \]  

(A25)

where \(C_{ar}\) is given by (A19).

The equation for the evolution of the mean phenotypes is, from Equation (5), that given by Equation (A26) below. This again assumes that the characters are not sexually dimorphic. If the maternal character is limited to females, the selection gradient \(r_{fa}(t)\) is multiplied by \(\frac{1}{2}\) where it appears for the first time on the right side of Equation (A26). When the maternal trait itself is under simple Mendelian inheritance (see Fig. 3b), \(p = 0\) and Equation (A26) reduces to Equations (9) and (10).

The response of a population at equilibrium to one generation of selection on the offspring trait is

\[ \Delta z^o(\infty) \approx \begin{bmatrix} \Delta z_{oo}^o(t) & \Delta z_{om}^o(t) \\ \Delta z_{mo}^o(t) & \Delta z_{mm}^o(t) \end{bmatrix} = \frac{m^2}{(1 - p^2)(2 - p)} \]  

(A28)

\[ \begin{bmatrix} G_{oo} & \frac{m^2}{2 - p} G_{mm} \\ \frac{(2 - p)(1 - p)}{2 - p} G_{mo} \end{bmatrix} \beta_o. \]  

**APPENDIX 2**

Here, we present stability analyses for two special cases discussed in the text: a single trait with maternal inheritance under weak stabilizing viability selection and a single trait with Mendelian inheritance under weak stabilizing maternal selection.

1) **Stabilizing selection on a maternally inherited trait.**—For a population with phenotypes that are normally distributed, a fitness function that is proportional to a Gaussian function with optimum \(\theta\) and variance \(W\) results in the selection gradient

\[ \beta(t) = \frac{\theta - z(t)}{W + P}. \]  

(A29)

The equation for the per-generation change in the population mean is obtained by substituting this expression into Equation (3), which gives:

\[ \Delta z(t) = \frac{C_{ar}}{W + P} [z(t) - \theta] \]

\[ + \frac{mW}{W + P} \Delta z(t - 1). \]  

(A30)

The first term on the right-hand side of (A30) represents the force of selection in the current generation, while the second involves the evolutionary response in the previous generation.

The evolution of the mean phenotype can be analyzed by forming a linear system from Equation (A30). The dynamics can be represented in matrix form as

\[ \begin{bmatrix} z(t + 1) - \theta \\ z(t) - \theta \end{bmatrix} = \begin{bmatrix} A_1 & A_2 \\ 1 & 0 \end{bmatrix} \begin{bmatrix} z(t - \theta) \\ \delta(t - \theta) \end{bmatrix}, \]  

(A31)

where \(A_1 = 1 - A_2 - C_{ar}(W + P)\) and \(A_2 = -mW/(W + P)\).

The evolution of the mean phenotype with respect to the optimum \(\theta\) is determined by the eigenvalues of the matrix in Equation (A31). These can be evaluated under the assumptions that selection is sufficiently weak that \(W \gg P\) and that \(C_{ar}\) and \(P\) are approximately constant and equal to their equilibrium values in the absence of selection ([A13] and [A14]). The eigenvalues of the matrix in (A31) are then

\[ \begin{bmatrix} \Delta z_{mm}(t) \\ \Delta z_{mm}(t) \end{bmatrix} = C_{ar} \begin{bmatrix} \beta_m(t) \\ \beta_m(t) \end{bmatrix} + \begin{bmatrix} pP_{mm} & pP_{mo} \\ mp_{mm} & mp_{mo} \end{bmatrix} \begin{bmatrix} \beta_m(t) \\ \beta_o(t) \end{bmatrix} \]

\[ + \frac{m\Delta z_{mo}(t - 1)}{m\Delta z_{mm}(t - 1)} - \begin{bmatrix} pP_{mm} & pP_{mo} \\ mp_{mm} & mp_{mo} \end{bmatrix} \begin{bmatrix} \beta_o(t - 1) \\ \beta_o(t - 1) \end{bmatrix}. \]  

(A26)
λ_+ ≈ 1 - \frac{2G}{(1 - m)(2 - m)W} \quad (A32a)

λ_- ≈ m \left[ 1 - \frac{(2 + m)G + (2 - m)E}{(1 - m^2)(2 - m)W} \right. \\
\quad \left. + \frac{2G}{(1 - m)(2 - m)W} \right]. \quad (A32b)

Inspection of (A32a) shows that if selection is weak, λ_+ lies between 0 and 1. This implies that the equilibrium at z = θ is stable. After about |1/λ_-| generations, during which damped oscillations will occur if m is negative, there will be a smooth and approximately exponential approach towards the optimum. After t generations, the deviation of the population from the optimum will be proportional to \exp \{ -2G/(1 - m)(2 - m)W \}.

In the case of multiple, maternally inherited characters experiencing stabilizing selection, stability of the equilibrium at the fitness peak is determined by the eigenvalues of a matrix that is the multivariate extension of that appearing on the right side of (A31).

**ii) Stabilizing maternal selection.** Here we analyze the evolutionary stability of a single trait with Mendelian inheritance evolving under the influence of weak stabilizing maternal selection. The optimal value for the trait in both mothers and offspring is z = θ. The strength of selection favoring the optimum is determined by the covariance matrix described in the text [following Equation (12)].

In the case of weak Gaussian selection, the maternal selection component b_m and the offspring selection component b_o are given by the matrix equation

\[
\begin{bmatrix}
  b_m(t) \\
  b_o(t)
\end{bmatrix} = -(W + Π)^{-1} \begin{bmatrix} z_0(t - 1) - θ \\
  z_0(t) - θ
\end{bmatrix}
\]

\[
= -(W + Π)^{-1} \begin{bmatrix} z(t - 1) - θ \\
  z(t) - θ
\end{bmatrix} + \begin{bmatrix} P \\
  \frac{1}{2} G
\end{bmatrix} \beta(t - 1), \quad (A33)
\]

where P is the phenotypic variance, G is the additive variance, and s(t - 1) is the selection differential. β(t - 1) is the net selection gradient in generation t - 1, which is defined as β(t - 1) = s(t - 1)/P. The matrix Π is the phenotypic covariance matrix of the trait as expressed in mothers and their offspring:

\[
Π ≈ \begin{bmatrix} P & \frac{1}{2} G \\
  \frac{1}{2} G & P
\end{bmatrix}.
\]

The evolution of the character is described by the matrix equation

\[
\begin{bmatrix}
  z(t + 1) - θ \\
  β(t + 1)
\end{bmatrix} ≈ \begin{bmatrix} 1 & G \\
  B_1 & B_2
\end{bmatrix} \begin{bmatrix} z(t) - θ \\
  β(t)
\end{bmatrix} \quad (A34)
\]

where \( B_1 = D[1, 1]^T \), \( B_2 = D[P, G]^T \), and \( D = -[G/2P, 1] (W + Π)^{-1} \). Equation (A34) reveals that maternal selection, like maternal inheritance, increases the dimensionality of the evolutionary dynamics. While the description of the evolution of a character under simple viability selection requires only one first-order equation, two such equations are needed for a single character under maternal selection.

The evolution of the trait with respect to the optimum is determined by the two eigenvalues of the matrix on the right-hand side of Equation (A34). Solving for these reveals that the largest eigenvalue can exceed 1 in magnitude, which means that the equilibrium at z = θ can be unstable. The condition for instability is given by Equation (13). Even if the equilibrium is stable, the approach towards the equilibrium will involve oscillations if the smaller eigenvalue is negative, as discussed in the text.