EVOLUTION

INTERNATIONAL JOURNAL OF ORGANIC EVOLUTION

PUBLISHED BY

THE SOCIETY FOR THE STUDY OF EVOLUTION

Vol. 52 **April 1998** **No. 2**

Evolution, 52(2), 1998, pp. 299–308

THE COADAPTATION OF PARENTAL AND OFFSPRING CHARACTERS

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Abstract.—Parents often have important influences on their offspring's traits and/or fitness (i.e., maternal or paternal effects). When offspring fitness is determined by the joint influences of offspring and parental traits, selection may favor particular combinations that generate high offspring fitness. We show that this epistasis for fitness between the parental and offspring genotypes can result in the evolution of their joint distribution, generating genetic correlations between the parental and offspring characters. This phenomenon can be viewed as a coadaptive process in which offspring genotypes evolve to function with the parentally provided environment and, in turn, the genes for this environment become associated with specific offspring genes adapted to it. To illustrate this point, we present two scenarios in which selection on offspring alone alters the correlation between a maternal and an offspring character. We use a quantitative genetic maternal effect model combined with a simple quadratic model of fitness to examine changes in the linkage disequilibrium between the maternal and offspring genotypes. In the first scenario, stabilizing selection on a maternally affected offspring character results in a genetic correlation that is opposite in sign to the maternal effect. In the second scenario, directional selection on an offspring trait that shows a nonadditive maternal effect can result in selection for positive covariances between the traits. This form of selection also results in increased genetic variation in maternal and offspring characters, and may, in the extreme case, promote host-race formation or speciation. This model provides a possible evolutionary explanation for the ubiquity of large genetic correlations between maternal and offspring traits, and suggests that this pattern of coinheritance may reflect functional relationships between these characters (i.e., functional integration).

Key words.—Correlational selection, epistasis, genetic correlation, genetic integration, indirect genetic effects, kin effects, maternal effects.

Received September 9, 1997. Accepted January 28, 1998.

While individual traits can directly affect an individual's fitness, multiple traits often have epistatic effects on fitness (Whitlock et al. 1995). The result of epistasis for fitness is that selection favors particular combinations of traits expressed within individuals. This phenomenon is known as correlational selection and results from functional integration, wherein traits are expected to act in concert to determine fitness (Olsen and Miller 1958; Cheverud 1982, 1984, 1996; Brodie 1992). This functional relationship among characters can generate genetic integration, or coadaptation of traits, by promoting linkage disequilibrium and/or favoring pleiotropic mutations. Individuals are consequently more likely to inherit the particular combinations of traits favored by correlational selection (Lande 1980, 1984; Brodie 1992; Cheverud 1996).

This process is traditionally thought to be important when two traits directly work together to affect the fitness of an individual. However, in many organisms, traits expressed in one individual can also be influenced by, and function together with, traits that are expressed in different individuals in the previous generation. These so-called maternal effects (or more generally, indirect genetic effects) are present whenever the environment provided by parents (i.e., the parents' phenotype) influences the expression of characters in offspring (reviewed by Cheverud and Moore 1994; Rossiter 1996; Wolf et al. 1998). In such a case, the fitness of offspring is likely to be affected by the combination of their own genes and the specific environmental trait provided by parents (e.g., nutrients, gestation environment, oviposition sites, seed characteristics) (Kirkpatrick and Lande 1989; Wade 1998). Often certain combinations of offspring genotypes and environmental traits provided by parents result in high offspring fitness (e.g., offspring adapted to develop in the oviposition environment provided by the mother). Although these two traits are not expressed simultaneously by the same individual, a genetic correlation between the parental and offspring traits results in predictable combinations of offspring genotypes and parental environmental effects because of relat-

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edness. Such genetic integration of parental and offspring traits could be adaptive, but it is unclear how selection acting in one generation could generate genetic integration of traits that are expressed in different generations.

The relationship between functional integration and genetic correlations has been investigated from both a theoretical and empirical perspective (Cheverud 1982, 1984, 1996; Lande 1984; Tallis and Leppard 1988; Phillips and Arnold 1989; Tallis 1989; Brodie 1992; Dudley 1996), but few attempts have been made to understand how selection might result in the genetic integration of parental and offspring traits (but see Wade 1998). Existing treatments of functional integration assume that fitness is determined by the combination of traits expressed in the same individual. Because parental and offspring traits are not expressed simultaneously by a single individual (or sometimes ever, as in a maternal trait that is not expressed by sons), and because fitness is determined by a combination of traits expressed in different individuals (e.g., the mother and her offspring), these previous investigations do not address the problem of parental and offspring trait coadaptation.

To examine conditions under which selection results in the genetic integration of offspring and parental characters, we adopt a quantitative genetic perspective of maternal inheritance. Using a quadratic fitness model (cf. Lande and Arnold 1983), we show how simple stabilizing or directional selection on maternally affected offspring traits results in functional integration of these traits, favoring particular combinations of maternal and offspring genetic effects. A Gaussian infinitesimal genetic model (cf. Bulmer 1985) of the maternal and offspring traits is employed to investigate how this selection will affect linkage disequilibrium between these traits. We present two general scenarios that lead to epistasis for fitness between the maternal and offspring genotypes. In these examples, maternal effects on the offspring phenotype lead to the evolution of genetic correlations between maternal and offspring traits when selection acts only on the maternally affected offspring trait. The resulting genetic correlations are expected to produce combinations of traits in parents and offspring that lead to high offspring fitness. This model provides a possible evolutionary explanation for the ubiquity of large genetic correlations between the genes for maternal and offspring traits (reviewed by Cheverud and Moore 1994; Roff 1997). Further, we suggest that the pattern of coinheritance of maternal and offspring characters in many systems may reflect functional relationships between these characters. While we focus on maternal effects, this model can also be directly applied to paternal effects and can easily be extended to include any relationship between individuals where traits in one individual affect the fitness of relatives (see Lynch 1987).

The Model

Additive Maternal Inheritance

Consider a single offspring trait (z_o) that is maternally affected by a single maternal trait (z_m). We assume a Gaussian infinitesimal model (GIM) for the genetics of the traits with purely additive effects and autosomal inheritance (cf. Fisher 1918; Bulmer 1985). The GIM assumes that genetic variation in a quantitative trait is produced by an infinite number of unlinked loci, each with an infinitesimally small effect on the phenotype (Fisher 1918; Bulmer 1985). We assume that the two traits have equilibrium genetic variances maintained at mutation-selection balance with no initial disequilibrium (see Bulmer 1971, 1985; Lande 1976). Following the model developed by Kirkpatrick and Lande (1989) for maternal inheritance, the offspring trait can be decomposed into three parts: (1) additive genetic effects (a_o); (2) random environmental effects (e_o); and (3) maternal environmental effects, such that

$$z_o = a_o + e_o + mz^*_{m(t-1)} \quad (1)$$

(eq. 1 in Kirkpatrick and Lande 1989).

The maternal trait value of the mother is $z^*_{m(t-1)}$, where $t - 1$ indicates a trait expressed in the previous generation (all traits and components not designated with a $t - 1$ occur in the present generation, [t], and the asterisk denotes the fact that the value of the mother is given after selection in the previous generation). The maternal effect coefficient is denoted m and determines the degree to which the phenotype of the offspring is determined by the phenotype of the mother (Kirkpatrick and Lande 1989). The maternal effect coefficient is defined as the partial regression of the offspring's phenotype on the mother's phenotype holding all other sources of variation constant (Kirkpatrick and Lande 1989). We assume that the maternal phenotype is determined solely by direct genetic and environmental effects (i.e., experiences no maternal effect itself) and therefore the maternal phenotype is partitioned into just two terms, the additive genetic value ($a^*_{m(t-1)}$) and the random environmental component ($e^*_{m(t-1)}$). We will refer to the additive genetic value of the offspring trait (a_o) as the direct genetic effect (Fisher 1918) and the contribution of the maternal phenotype ($mz^*_{m(t-1)}$) as the maternal effect (Cheverud and Moore 1994). Substituting $(a^*_{m(t-1)}) + (e^*_{m(t-1)})$ for the maternal phenotype ($mz^*_{m(t-1)}$) in equation (1) yields

$$z_o = a_o + e_o + ma^*_{m(t-1)} + me^*_{m(t-1)}. \quad (2)$$

We will refer to $ma^*_{m(t-1)}$ as the maternal genetic effect and $me^*_{m(t-1)}$ as the maternal environmental effect to indicate the causal origin of these indirect effects on the offspring phenotype (Rossiter 1996). The offspring phenotype is thus determined jointly by the phenotype of the mother and its own genotype. Because the maternal trait is heritable, offspring with a particular trait value have a predictable genetic value of the maternal effect (even if they do not express the maternal trait themselves, as for sons).

Selection on Traits with Additive Maternal Effects

To illustrate how selection acts to alter the joint distribution of maternal and offspring characters, we start with the common scenario in which the offspring trait is under stabilizing selection. Early body size in mammals is a classic example of a trait that is influenced by maternal effects and that also experiences stabilizing selection. In mammals, larger offspring generally have higher postpartum survival but are more likely to experience problems during birth, while small individuals experience fewer birthing problems but may have

lower postpartum survival (e.g., Karn and Penrose 1951; Brown et al. 1993). Data from humans demonstrate clear stabilizing selection for birth weight (Karn and Penrose 1951; see also Schluter and Nychka 1994) and the influence of maternal effects on birth weight (maternal genotype accounts for 25% of the variance in human birth weight; Robson 1978; see also Mi et al. 1986). Early body size in mammals shows a strong component attributable to the uterine environment (Atchley and Newman 1989; Cowley 1991; Gregory and Maurer 1991) and is affected postpartum by lactation performance (for examples, see Kirkpatrick and Lande 1989; Roff 1997).

We explore how stabilizing selection on offspring alone can generate genetic integration through epistatic effects of traits on fitness. In all examples presented here, we do not consider selection acting on the maternal phenotype because this selection is not expected to lead to epistasis for fitness between the maternal and offspring genotypes under most conditions (Wolf and Brodie, unpubl. data). Selection on maternal phenotypes will, however, alter the distribution of maternal environments (see Kirkpatrick and Lande 1989) and, when there is an existing genetic correlation between the maternal and offspring genotypes, will alter the distribution of offspring genotypes (i.e., a correlated response to selection; Lande and Arnold 1983).

Stabilizing selection on the offspring trait acts to reduce the phenotypic variance of the trait (Lande and Arnold 1983). This form of selection can be modeled using a simple quadratic fitness equation

$$w(z_o) = \alpha + \beta_o z_o + \frac{1}{2}\gamma_o z_o^2, \quad (3)$$

where $w(z_o)$ is the relative fitness of an individual with phenotype z_o, β_o is the directional selection gradient, γ_o (when negative) is the stabilizing selection gradient (also referred to as a nonlinear selection gradient, or quadratic selection gradient; Phillips and Arnold 1989; Brodie et al. 1995), and α is baseline fitness (Lande and Arnold 1983). In this example, we assume that the mean of the offspring trait is at its optimum, making β_o zero. Directional selection is not explored here because it does not generate epistasis for fitness between the maternal and offspring genotypes. Directional selection on the offspring trait alters the means of both the direct and maternal effects separately, but does not directly alter the genetic covariance between them (for details, see Appendix). For simplicity we assume that the values of the traits and trait components (i.e., additive genetic values and environmental values) are standardized to a mean of zero and variance of one.

To calculate how stabilizing selection acts on individuals that have particular combinations of direct genetic effects, and maternal effects we use the definition of z_o from equation (1) and substitute into equation (3) for z_o to solve for the fitness of individuals with particular combinations of direct genetic effect (a_o) and maternal environment (i.e., their mothers' phenotype, $z^*_{m(t-1)}$):

$$w[a_o, z^*_{m(t-1)}] = \alpha + \frac{1}{2}\gamma_o a_o^2 + \frac{1}{2}\gamma_o[mz^*_{m(t-1)}]^2 + \gamma_o a_o[mz^*_{m(t-1)}]. \quad (4)$$

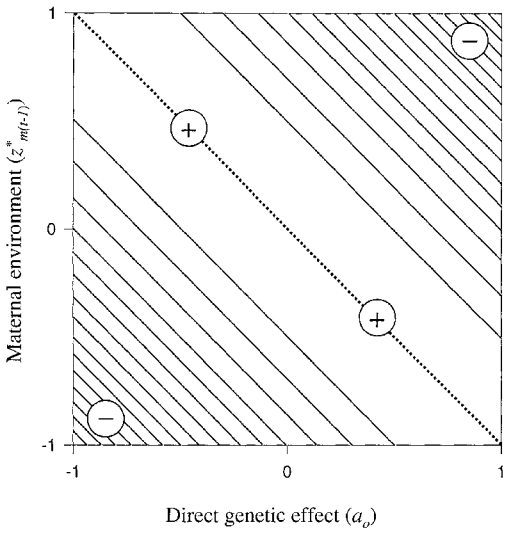

FIG. 1. The surface describing the fitness of offspring with combinations of maternal environments ($z^*_{m(t-1)}$) and additive genetic values (a_o) assuming additive maternal effects and only stabilizing selection on the offspring trait. Fitness is assigned based on the phenotypic value of the offspring (z_o). The case illustrated is for $m = 1$, $\gamma_o = -0.8$. The lines represent fitness isoclines for offspring with particular combinations of maternal and direct genetic effects.

We have removed all of the environmental components (e_o) because the random environmental components do not contribute to evolutionary changes. This equation produces a fitness surface that is a ridge corresponding to combinations of maternal phenotypes and offspring genotypes that result in the optimal offspring phenotype (Fig. 1). This view of fitness treats the maternal effect ($z^*_{m(t-1)}$) as a trait of the offspring, which allows us to describe the fitness of offspring that have unique combinations of the maternal effect and direct genetic effect. The selection gradient (γ_o) acting on combinations of these two effects ($a_o[mz^*_{m(t-1)}]$) is a sort of "correlational" selection gradient (Lande and Arnold 1983; Phillips and Arnold 1989; Brodie 1992), in that particular combinations of direct and maternal effects have high fitness. This correlational selection gradient can be used to predict changes in the joint distribution of the maternal and offspring genotypes by considering the relationship between the maternal phenotype and the offspring genes for the maternal phenotype (i.e., the relationship between $z^*_{m(t-1)}$ and a_m). That is, because the maternal trait is heritable, there is a predictable relationship between the maternal phenotype ($z^*_{m(t-1)}$) that produced the maternal effect and the genes that the offspring possesses for that maternal trait (a_m).

Because the mother and offspring are related by a factor of one-half, an offspring that experiences a particular maternal environment (i.e., has a particular maternal effect contribution to its phenotype) has a somewhat predictable additive genetic value for the maternal trait. Under the GIM we expect the offspring's additive genetic value to be one-half the maternal additive value plus one-half the paternal additive value (Falconer and Mackay 1996). Because it is only the mother's phenotype that affects the expression of

the offspring trait in this scenario, there is no predictable relationship between offspring fitness and the paternal component of the offspring's additive genetic value of the maternal trait (assuming random mating). However, due to the predictable relationship between the genes that an individual possesses for the maternal trait and its value for the offspring trait, selection acting on the offspring trait "exposes" the genes for the maternal trait to selection. To derive an equation describing the fitness of an individual that has a particular combination of additive genetic values for the offspring and maternal traits we substitute 1/2 a_m for $z^*_{m(t-1)}$. The one-half reflects the fact that only one-half of the offspring additive genetic value for the maternal trait is expected to covary with offspring fitness. Other relationships between interacting individuals can easily be included in this analysis by substituting in the relatedness of the interacting individuals for the one-half in the maternal-offspring equations presented here (see Lynch 1987). Because the offspring in the current generation are derived from the mothers that existed after selection in the previous generation, the asterisk notation is dropped (i.e., offspring genotypes this generation reflect the post-selection distribution in the previous generation). The resulting fitness equation is:

$$w[a_o, a_m] = \alpha + \frac{1}{2}\gamma_o a_o^2 + \frac{1}{2}\gamma_o\left(\frac{m}{2}a_m\right)^2 + \frac{m}{2}\gamma_o a_o a_m. \quad (5)$$

The coefficient ($m/2\ \gamma_o$) associated with the product of the additive genetic values for the maternal and offspring traits (the cross product, $a_o a_m$) is interpretable as a correlational selection gradient that describes selection on the combination of maternal and offspring additive genetic values. This gradient results from maternal-offspring genotype-by-genotype epistasis for fitness and represents an epistatic interaction between genomes (Wade 1998). Note that this is a function of the gradient that describes stabilizing selection on the offspring phenotype because the offspring phenotype is produced by a combination of maternal and offspring characters.

Generation of Linkage Disequilibrium

Selection affecting the variances and covariances can cause cross-generational changes in linkage disequilibrium (Lande 1984; Phillips and Arnold 1989; Brodie 1992; Wade 1998). Under the GIM within-generation changes in the genetic variance-covariance matrix (**G**) can be predicted from changes in the phenotypic variance-covariance matrix (**P**), or from the nonlinear selection gradients (γ; Lande and Arnold 1983; Tallis and Leppard 1988). The within-generation changes in **G** owing to changes in linkage disequilibrium ($\Delta\mathbf{G}$) are described by the multivariate equation

$$\Delta\mathbf{G} = \mathbf{G}(\boldsymbol{\gamma} - \boldsymbol{\beta}\boldsymbol{\beta}^{\mathrm{T}})\mathbf{G}, \quad (6)$$

where $\boldsymbol{\gamma}$ is the matrix of nonlinear selection coefficients and $\boldsymbol{\beta}$ is the vector of directional selection gradients and the superscript T denotes matrix transposition (eq. 1 of Phillips and Arnold 1989; after Lande 1980; Lande and Arnold 1983). The diagonal elements of the $\boldsymbol{\gamma}$-matrix contain selection gradients affecting the variances of the maternal and offspring traits and the off-diagonal elements contain selection gradients affecting the covariance between the traits. Because we have assumed that the directional selection gradients are zero in this example, equation (6) simplifies to:

$$\Delta\mathbf{G} = \mathbf{G}\boldsymbol{\gamma}\mathbf{G}. \quad (7)$$

In a standard model of fitness, the entries in the $\boldsymbol{\gamma}$-matrix contain the partial regression of individual fitness on the pairwise products of character deviations from the mean (see Lande and Arnold 1983; Phillips and Arnold 1989). These partial regressions describe selection that alters the second moment of the bivariate distribution (i.e., the variances and covariances). In the usual quadratic fitness equation the partial regression of fitness on the squared deviations is an estimate of half the nonlinear gradient (see Lande and Arnold 1983; Phillips and Arnold 1989; and Brodie et al. 1995). Therefore, the estimates of the nonlinear selection gradients associated with the squared deviations are twice the partial regression coefficient (Lande and Arnold 1983). The coefficients associated with the partial regression of fitness on the cross-product are estimates of the full value, and thus are not doubled (Lande and Arnold 1983).

It is usually assumed that the partial regression of fitness on the phenotype is equivalent to the partial regression of fitness on the deviations of additive genetic values from the mean, and in the case of nonlinear selection, on the pairwise products of deviations of additive genetic values from the mean (Lande and Arnold 1983; Phillips and Arnold 1989; Brodie 1992; Walsh and Lynch, unpubl.). However, in the case of maternal and offspring traits, this relationship does not hold. In the case of maternally affected traits the elements in the $\boldsymbol{\gamma}$-matrix are more complex because of the relationship between the genotype (whose distributions in which we are predicting changes) and the offspring phenotype (which experiences selection). To describe selection acting to alter the distribution of additive genetic values for the maternal and offspring traits we need to extract the complete term from equation (5) describing the partial regression of fitness onto the pairwise products of deviations of additive genetic values from the mean. By doubling the regression coefficients associated with the squared deviations, we get:

$$\boldsymbol{\gamma} = \begin{bmatrix} \gamma_o & \frac{m}{2}\gamma_o \\ \frac{m}{2}\gamma_o & \frac{m^2}{4}\gamma_o \end{bmatrix}, \quad (8)$$

where the top-left element describes selection affecting the genetic variance of the direct genetic effect (a_o) and the lower-right element describes selection affecting the genetic variance of the maternal character (a_m). The top-right and lower-left elements describe selection affecting the genetic covariance between the maternal and offspring traits.

Expanding equation (7) using equation (8) for our two-trait example yields an expression for the change in the genetic covariance within a generation due to linkage disequilibrium between the maternal and direct genetic effects:

$$\Delta G_{om} = \gamma_o G_{oo} G_{om} + \frac{m}{2}\gamma_o G_{om}^2 + \frac{m}{2}\gamma_o G_{oo} G_{mm} + \frac{m^2}{4}\gamma_o G_{om} G_{mm}. \quad (9)$$

G_{om} is the genetic covariance between the maternal and offspring traits (i.e., cov(a_o, a_m)) and G_{mm} is the additive genetic variance of the maternal trait (i.e., cov(a_m, a_m)) and G_{oo} is the additive genetic variance for the offspring trait (i.e., cov(a_o, a_o)). Changes in the additive genetic variances of the two traits are given by:

$$\Delta G_{oo} = \gamma_o G_{oo}^2 + \frac{m}{2}\gamma_o G_{om} G_{oo} + \frac{m}{2}\gamma_o G_{oo} G_{om} + \frac{m^2}{4}\gamma_o G_{om}^2 \tag{10}$$

and

$$\Delta G_{mm} = \gamma_o G_{om}^2 + \frac{m}{2}\gamma_o G_{om} G_{mm} + \frac{m}{2}\gamma_o G_{mm} G_{om} + \frac{m^2}{4}\gamma_o G_{mm}^2. \tag{11}$$

These within-generation changes can then be translated into cross-generational changes in linkage disequilibrium under GIM (i.e., Tallis and Leppard 1988; Tallis 1989). Assuming random mating, the change in linkage disequilibrium is (cf. eq. 6 in Tallis and Leppard 1987; eq. 3 in Tallis and Leppard 1988):

$$\Delta \mathbf{D}_t = \frac{\Delta \mathbf{G}}{2} - \frac{\mathbf{D}_t}{2}, \tag{12}$$

where $\mathbf{D}_t$ is the matrix of linkage disequilibrium among traits. The disequilibrium in the next generation ($t + 1$) is simply $\mathbf{D}_t + \Delta\mathbf{D}_t$. Under the Gaussian infinitesimal model the **G**-matrix in the next generation will be

$$\mathbf{G}_t = \mathbf{G}_o + \mathbf{D}_t, \tag{13}$$

where $\mathbf{G}_0$ is the starting value of **G** (where covariances are due to pleiotropy) and $\mathbf{D}_t$ is the linkage disequilibrium in generation t (Tallis and Leppard 1988; Tallis 1989). Equation (12) points out two important effects of recombination on the disequilibrium: (1) only half of the disequilibrium generated by selection is passed on each generation; and (2) only half of the disequilibrium from the previous generation is passed onto the next generation (Tallis and Leppard 1988; Walsh and Lynch, unpubl.). Because of the complex asymptotic nature of the changes in disequilibrium, the equilibrium values for linkage disequilibrium between the traits ($\hat{\mathbf{D}}$) were solved for iteratively using equations (9)–(13) (Walsh and Lynch, unpubl.; cf. Tallis and Leppard 1987, 1988). Assuming a lack of pleiotropy, (i.e., $\mathbf{G}_0 = 0$) the equilibrium values for linkage disequilibrium are equivalent to the equilibrium genetic variances and covariances ($\hat{\mathbf{G}}$). To show the equilibrium relationship between the traits we present the equilibrium genetic correlations, which are defined as

$$\hat{\rho}_{ij} = \hat{G}_{ij}/\sqrt{\hat{G}_{ii}\hat{G}_{jj}}.$$

The outcome of the iteration shows that whenever the maternal effect is negative, the genetic correlation that develops is positive and vice versa (Fig. 2). This occurs because selection on offspring phenotypes favors opposing effects of direct genetic and maternal effects. Thus, the offspring and maternal genotypes become integrated in such a way that together they produce offspring with trait values that have high fitness. This apparent coadaptation results from simple stabilizing selection on a single offspring trait. Integration

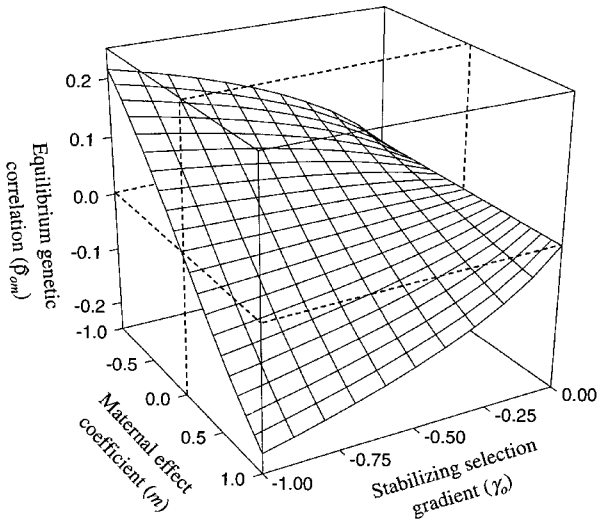

FIG. 2. Equilibrium values for the direct-maternal genetic correlation resulting from stabilizing selection on the offspring character and no pleiotropy. Disequilibrium is plotted as a function of the maternal effect coefficient (m) and the strength of stabilizing selection on the offspring character (γ_o).

of maternal and offspring traits is expected to be very common, and may explain the ubiquity of negative direct-maternal genetic correlations (Cheverud and Moore 1994; Roff 1997). Further the pattern of genetic covariation of maternal and offspring traits may reveal functional integration of the characters.

Nonadditive Maternal Inheritance

Although maternal effects are most commonly modeled as additive effects (as in the first example), they also can be nonadditive (Wade 1998). Nonadditive maternal effects are equivalent to a genotype-by-environment interaction, where the environment is that provided by the mother and the genotype is the direct genetic effect in the offspring. This type of genotype-by-environment interaction will occur whenever the effect of the maternal phenotype (the environment) on the offspring phenotype is dependent on the genotype of the offspring. Interactions of this type may arise prenatally in mammals as a result of the complex interactive development of the placenta (Redman et al. 1993). Embryo transfer experiments have shown a highly significant interaction between the offspring genotype and the maternal uterine environment (see examples in mice: Cowley et al. 1989; in cattle: Gregory and Maurer 1991) and there is some evidence of a similar effect in studies of crossbred cattle (e.g., Núñez-Dominguez et al. 1993). In fact, the variance component for the uterine environment-by-progeny genotype interaction in mice accounts for a larger percent of the total variance in body weight at many ages than does either the progeny genotype or maternal genotype alone (Cowley et al. 1989; Cowley 1991). Interactions between genetically based maternal effects and direct genetic effects can be viewed as genotype-genotype epistasis and have also been called maternal-off-

spring "intermixing" ability (Wade 1998). This form of epistasis has been recognized as a potentially important component of the evolution of maternal-offspring interactions (Wade 1998).

Empirical evaluations of nonadditive maternal effects are lacking, primarily due to the logistical difficulties inherent in their estimation including: embryo transfers, cross fostering, or a breeding design such as reciprocal crosses of inbred strains that allows one to detect an interaction between the offspring genotype and maternal environment (Roff 1997). In addition, detection of these interactions is statistically difficult, even when the magnitude of their effect is considerable (see Wade 1992). The statistical bias in the detection of main effects (i.e., additive effects) and the relative inability to detect interaction effects has probably lead to the belief that these interaction effects are less important (see Wahlsten 1990; Wade 1992).

Nonadditive effects can be modeled with an equation analogous to (1) with the addition of a maternal-offspring interaction term. For simplicity, we assume that there is no direct genetic or maternal effect independent of the interactive effect:

$$z_o = na_o z^*_{m(t\ \ 1)} + e_o. \tag{14}$$

This simple case will be used to model selection under these conditions and can be combined with the additive model above to look at cases where there are both additive and nonadditive maternal effects. The coefficient n is a nonadditive maternal effect coefficient that is defined as the partial regression of the offspring phenotype on the product of the maternal phenotype and offspring genotype, holding all other sources of variation (i.e., maternal phenotype and offspring genotype) constant. This type of interaction is an additive-by-additive interaction. When the maternal trait is heritable, this represents additive-by-additive genotype-by-genotype epistasis for fitness (Wade 1998). Other forms of epistatic interaction (e.g., additive-by-dominance) are presented and discussed elsewhere (e.g., Cheverud and Routman 1996) and can easily be incorporated into this framework. In this scenario we assume a purely nonadditive maternal effect, such that the maternal phenotype and offspring genotype have no independent contribution to the offspring phenotype. We have made this simplifying assumption so that the consequences of each system of inheritance can be considered independently. The results shown for the additive case above (and the results shown in the Appendix) can be combined to consider other models of inheritance (i.e., combinations of these pure forms).

Selection on Traits with Nonadditive Maternal Effects

Nonadditive maternal effects can easily be incorporated into the additive model presented above. However, nonadditive effects, due to the nature of the maternal-offspring interaction, result in very different evolutionary outcomes. To explore the evolution of the genetic variance and covariance in the case of nonadditive maternal inheritance, we can again use the model of selection defined in equation (3). In this scenario we do not explore stabilizing selection, as it can be shown that stabilizing selection on a trait with nonadditive

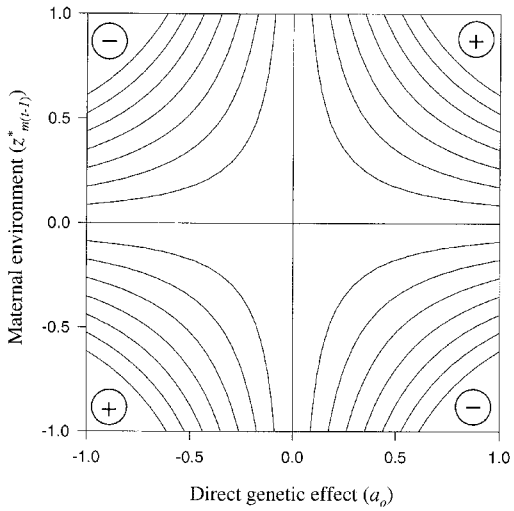

FIG. 3. The fitness surface describing the fitness of offspring with combinations of maternal environments ($z^*_{m(t\ -\ 1)}$) and additive genetic values (a_o) resulting from nonadditive maternal effects and directional selection (see eq. 15). Fitness is assigned based on the phenotypic value of the offspring (z_o). The case illustrated is for $n = 1$, $\beta_o = 0.8$. The lines represent fitness isoclines for offspring with particular combinations of maternal and direct genetic effects.

maternal effects alters only the fourth moment of the bivariate distribution (i.e., does not directly affect covariances; see Appendix).

By substituting the definition of the phenotype from equation (14) into equation (3) and focusing only on the directional selection gradient, we get the fitness equation:

$$w[a_o z^*_{m(t\ -\ 1)}] = \alpha + \beta_o[na_o z^*_{m(t\ \ 1)}]. \tag{15}$$

Again we have not included the random environmental component because it does not contribute to evolutionary change. Solving for the fitness of individuals with combinations of maternal and offspring genotypes as before, we get

$$w[a_o, a_m] = \alpha + \frac{n}{2}\beta_o a_o a_m. \tag{16}$$

This fitness equation corresponds to a saddle-shaped bivariate fitness surface (Fig. 3) on which combinations of maternal and direct genetic effects with similar influences are favored because of directional selection on offspring phenotypes.

Because the directional gradient (β_n) acts upon the combination of the direct and maternal genetic effects, it results in epistasis for fitness between the maternal and offspring genotypes. In addition, this form of selection has a disruptive effect on both the maternal and the offspring genetic variances. Thus, the matrix describing selection acting to alter the variances and covariances contains directional selection gradients (βs) because these gradients describe how selection alters the variances of, and covariances between, the direct and maternal effects. Using the coefficients from equation (16) to create a matrix of nonlinear selection terms (i.e., a γ-matrix), we can use equation (7) to derive equations describing changes in the genetic variances and covariances. In

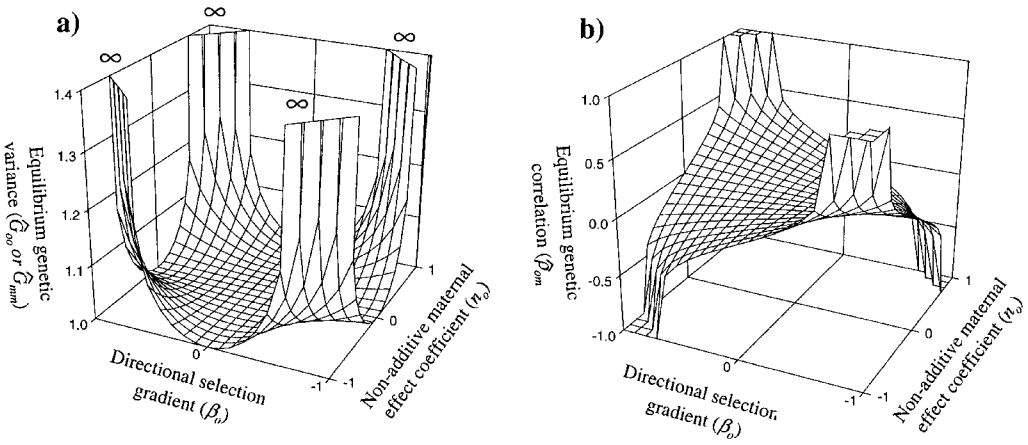

FIG. 4. Genetic consequences of directional selection on an offspring trait that experiences nonadditive maternal effects. (a) Equilibrium additive genetic variance for the maternal trait ($\hat{G}_{mm}$) and the offspring trait ($\hat{G}_{oo}$); in this example the two show equivalent equilibrium variances and therefore are shown in a single graph. The regions labeled with ∞ indicate portions of the surface that have been truncated because they approach infinity. (b) Equilibrium genetic correlation ($\hat{\rho}_{om}$) due solely to linkage disequilibrium between the maternal and offspring traits.

this case the changes in genetic variances and covariances are predicted by

$$\Delta G_{oo} = \frac{n}{2}\beta_o G_{oo} G_{om}, \tag{17a}$$

$$\Delta G_{mm} = \frac{n}{2}\beta_o G_{mm} G_{om}, \tag{17b}$$

and

$$\Delta G_{om} = \frac{n}{2}\beta_o G_{om}^2 + \frac{n}{2}\beta_o G_{oo} G_{mm}. \tag{17c}$$

When the sign of n and β_o are the same, selection will act to increase the additive genetic variance of the maternal and offspring traits (i.e., selection has a "disruptive" effect because ΔG_{oo} and ΔG_{mm} are > 0). Thus, even though there is linear directional selection on the offspring phenotype, there can be a disruptive effect on the direct genetic component of that phenotype due to the nonadditive maternal effect. Likewise there is a disruptive effect on the genetic component of the maternal trait.

Solving iteratively for the equilibrium variances and covariances, using equations (12), (13), and (17a–c), we find that (Fig. 4): (1) for most of the parameter space the variances remain close to their starting values and the correlation remains small; and (2) with extreme values for the maternal effect coefficient combined with strong directional selection the variances become unbounded and approach infinity while the correlation (ρ_{om}) approaches one (due to the covariance also approaching infinity). We see that, while the genetic variances approach infinity at the extremes, the correlation approaches one (Fig. 4). This is because the genetic covariance also approaches infinity in those same regions, and thus the correlation approaches its upper boundary of one. The unbounded increase in variance and covariances occurs because the increase in disequilibrium caused by the disruptive effects of selection always remains greater than the loss of disequilibrium to recombination, and therefore the changes in genetic variance and covariance continue to accumulate each generation (Bulmer 1985).

DISCUSSION

Parental phenotypes, in conjunction with offspring genes, determine a large portion of the variance of characters expressed early in an offspring's life (Cowley et al. 1989; Gregory and Maurer 1991; Cheverud and Moore 1994). Because genes expressed in parents and offspring often interact to determine offspring phenotypes (e.g., Cowley et al. 1989; Gregory and Maurer 1991), they can have nonindependent (i.e., epistatic) effects on fitness. Selection thereby favors the coinheritance of combinations of parental and offspring genes, so that offspring genotypes will experience the parental environment in which they have high fitness. In other words, because offspring inherit genes for the parental environment (i.e., the parental trait), epistasis for fitness between the offspring and parental genotypes results in combinations of environments and offspring genotypes that function together to produce fit offspring. The resulting genetic integration (sensu Cheverud 1996) of the offspring genotype with the parental environment allows the two to become coadapted while the environment itself is evolving (Wade 1998; Wolf et al. 1998). Because the variance in fitness early in life is so great, the opportunity exists for this form of selection to be extremely intense.

To illustrate how selection on an offspring trait alone can result in the functional integration of parental and offspring characters, we have presented two scenarios that result in epistasis for fitness between the direct and indirect genetic effects (i.e., results in maternal-offspring genotype-by-genotype epistasis for fitness). First, we have shown that stabilizing selection on a trait experiencing an additive maternal effect favors a genetic correlation between the direct and maternal genetic effect that is opposite in sign to the maternal

effect coefficient. This genetic correlation results in offspring genotypes that effectively compensate for the influence of maternal effects, generating intermediate offspring phenotypic values. Because stabilizing selection favors individuals with phenotypes corresponding to an intermediate fitness optimum, opposing values of the direct and maternal effect result in high offspring fitness. Thus, the genetic correlation between the direct and maternal effect that results from selection is "adaptive" because it results in combinations of maternal and direct genetic effects that together produce fit offspring.

In the second scenario we have shown that directional selection on a trait with nonadditive maternal inheritance generates a genetic correlation between the direct and maternal genetic effects that is the same sign as the selection gradient. In this case, directional selection on the offspring traits acts to increases the genetic variance of both the maternal and offspring characters. This is true because with nonadditive maternal inheritance it is the product, rather than the sum, of the direct and maternal effects that determines the offspring's phenotype. Thus, direct and maternal effects of the same sign result in positive values of the offspring phenotype, while opposing signs result in negative phenotypic values (negative values indicating values lying below the mean, as traits are measured as deviations from the mean). Since positive directional selection favors large positive phenotypic values, it also generates a positive direct-maternal genetic correlation (and vice versa for negative directional selection).

With nonadditive maternal inheritance, selection affecting the variance and covariance of traits could favor the divergence of the population into distinct groups (i.e., polymorphism; Wade 1998) or may fix the population for a single coadapted set of maternal and offspring characters. For example, if maternal oviposition site choice is based on host-plant chemistry and the offspring phenotype (e.g., growth rate) is some measure of offspring performance on the plant, then maternal oviposition behavior should coevolve with and be genetically correlated with offspring performance on that plant. Selection in this case should favor combinations of maternal phenotypes and offspring genotypes that result in good offspring performance on the host plant (Futuyma 1983). When selection is strong enough, a disruptive process may occur in which the population diverges into two or more groups with coadapted traits (as in Fig. 4). This divergence occurs because the increase in linkage disequilibrium due to selection every generation continues to be greater than the loss due to recombination. As the population diverges into two distinct groups, the genetic variance approaches infinity and the genetic correlation between the traits approaches one. This process might result in "host-race" formation and, coupled with other population processes such as assortative mating, in speciation (Futuyma 1983; Wade, in press).

Although we have focused here on the genetic correlations that result from linkage disequilibrium, genetic correlations can also be due to pleiotropy. Selection that generates linkage disequilibrium also favors pleiotropic mutations and/or physical linkage (Lande 1980; Bulmer 1985). Pleiotropy allows the equilibrium genetic correlation to be larger than the level predicted solely on the basis of linkage disequilibrium, as recombination does not result in a loss of genetic covariation due to pleiotropy (Bulmer 1971, 1985). Pleiotropic effects may explain why observed genetic correlations are usually much larger in magnitude than those predicted to result solely from linkage disequilibrium generated by selection (Cheverud and Moore 1994; Robinson 1996; Roff 1997). Because they are not eroded by recombination, correlations due to pleiotropy are also more stable than those due to linkage disequilibrium. Therefore pleiotropy is more likely to play a role in long-term evolutionary changes (Lande 1980). The equilibrium genetic correlations owing to linkage disequilibrium can be considered lower bounds on the genetic correlations that will result from selection for genetic integration.

In general, the pattern of coinheritance due to pleiotropy is expected to evolve to match the pattern of nonlinear selection, assuming that the appropriate pleiotropic variation is available to achieve such a pattern (Lande 1980; Cheverud 1984, 1996). Thus, in the case of maternal and offspring characters, the pattern of coinheritance is expected to reflect the strength of selection on combinations of maternal and offspring characters. However, in the case of maternal and offspring characters, the size of the expected correlation is diminished by the fact that selection on offspring traits exposes only half of the maternal genetic value to selection (e.g., eq. 8). Of course, genetic correlations may also be due to factors other than selection, and the equilibrium genetic covariance structure observed will also reflect patterns of pleiotropic mutation, the mating system, and recombination (i.e., physical linkage patterns) (Lande 1980).

Although we have made some simplifying assumptions regarding inheritance to keep this model computationally simple, the qualitative results of our model are expected to apply even in systems that do not adhere to the strict criteria set forth here. For example, we assume that the maternal trait shows pure Mendelian inheritance (i.e., is not maternally affected). Violations of this assumption may affect the short-term response to selection shown by the covariance, but is expected to have little impact on the equilibrium genetic covariance under most circumstances. As with the evolutionary response to selection shown by trait means (e.g., Kirkpatrick and Lande 1989), a maternal effect on the maternal trait may introduce time lags into the response to selection shown by the genetic covariance. We also assume a GIM for inheritance of the maternal and offspring traits. While the GIM gives results that are simple, it may be an inappropriate model of inheritance in many cases. Despite the deviation of most genetic systems from the strict GIM, the qualitative results (e.g., the direction and magnitude of the genetic correlations) obtained assuming a GIM are expected be general (i.e., we expect these systems to show coadaptation). Lastly, while we examine only "pure" forms of maternal inheritance (i.e., purely additive maternal effects or purely nonadditive effects), these pure forms can be combined to consider other more complex systems of inheritance (i.e., cases where both additive and nonadditive effects occur).

Despite the ubiquity of genetic correlations among direct genetic effects and maternal effects (i.e., the "direct-maternal genetic correlation"; see Cheverud and Moore 1994; Roff 1997), there have been few explanations for the origin and nature of these correlations (but see Wade 1998). The direct-maternal genetic correlation is of interest because it plays a

major role in determining the rate and direction of phenotypic evolution of maternal and offspring characters (Kirkpatrick and Lande 1989; Cheverud and Moore 1994; Roff 1997; Wolf et al. 1998). Similarly, in agricultural genetics the direct-maternal genetic correlation has been investigated because of its role in determining the response to selection for economically important animal traits, such as lactation performance and body size (Baker 1980). Empirical estimates of the direct-maternal genetic correlation are usually large in magnitude and mostly negative (reviewed by Cheverud and Moore 1994; Robinson 1996; Roff 1997). This pattern of coinheritance could be explained simply by stabilizing selection on offspring traits when maternal and direct genetic effects are additive, as shown herein.

All interactions between traits have the potential to generate coadaptation through epistatic effects on fitness. The relative ubiquity and uniformity of direct-maternal genetic correlations (Cheverud and Moore 1994; Robinson 1996; Roff 1997; Mousseau and Fox 1998) suggests selection has played a role in shaping the genetic architecture of these traits. We have shown that a relatively simple combination of selection on offspring and either additive or nonadditive maternal effects can cause the pattern of genetic correlations to reflect functional relationships. Further explorations of the nature and degree of interaction between maternal and offspring genotypes are necessary before we can fully evaluate the pervasiveness of coadaptation between parent and offspring traits.

Acknowledgments

We thank A. J. Moore, S. Welter, A. Sih, J. B. Walsh, J. M. Cheverud, and M. J. Wade for helpful input and/or stimulating discussions that helped improve this paper. We thank A. J. Moore, A. Agrawal, and A. Storfer for thoughtful comments on the manuscript. This research was supported by a National Science Foundation Graduate Research Training fellowship to JBW and IBN-9600775 to EDB III.

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Corresponding Editor: L. Leamy

APPENDIX

In the text we present the two combinations of inheritance and selection in which selection favors the coadaptation of the maternal and offspring traits. Here we show that the other two possible combinations of inheritance and selection do not result in the coadaptation of the parental and offspring traits.

Directional Selection with Additive Maternal Effects.—The combination of an additive maternal effect and directional selection does not result in epistasis between the maternal and offspring genotypes, because in this case the maternal and offspring genotypes act independently to determine offspring fitness. Using the model of inheritance (additive maternal effect) shown in equations (1) and (2) and the model for fitness in equation (3), and ignoring the stabilizing selection handled in the text, we solve for the fitness of individuals with particular combinations of direct genetic effect (a_o) and maternal environment (i.e., their mothers' phenotypes, $z^*_{m(t-1)}$):

$$w[a_o, z^*_{m(t-1)}] = \alpha + \beta_o(a_o + mz^*_{m(t-1)}). \quad \text{(A1)}$$

Solving for the fitness of individuals with combinations of maternal and offspring genotypes as before, we find

$$w[a_o, a_m] = \alpha + \beta_o\left(a_o + \frac{m}{2}a_m\right) = \alpha + \beta_o a_o + \beta_o\frac{m}{2}a_m. \quad \text{(A2)}$$

Equation (A2) clearly shows that directional selection does not produce epistasis for fitness between the maternal and offspring genotypes (i.e., there is no selection targeting combinations of both maternal and offspring genetoypes). We see that directional selection on the offspring trait with an additive maternal effect simply results in directional selection on both the maternal and offspring traits.

Stabilizing Selection with Nonadditive Maternal Effects.—In the case of the combination of stabilizing selection with a nonadditive maternal effect, selection does not directly affect the second moments (i.e., the variance or covariance) of the bivariate distribution. Using the model of inheritance (nonadditive maternal effect) shown in equation (14) and the model for fitness shown in equation (3), and ignoring the directional selection handled in the text, we solve for the fitness of individuals with particular combinations of direct genetic effect and maternal environment:

$$w[a_o, z^*_{m(t-1)}] = \alpha + \gamma_o[na_o z^*_{m(t-1)}]^2. \quad \text{(A3)}$$

Solving for the fitness of individuals with combinations of maternal and offspring genotypes as before, we get

$$w[a_o, a_m] = \alpha + \gamma_o\left[\frac{n}{2}a_o a_m\right]^2. \quad \text{(A4)}$$

Equation (A4) shows that selection in this case acts only on the squared cross-product of genetic effects (i.e., square of the product of the deviations from the bivariate mean, recalling that traits are measured as deviations from the mean) and does not directly affect the second moment, as would be required for coadaptation to occur. Instead stabilizing selection affects a fourth moment of the distribution (e.g., kurtosis), and is not expected to alter the covariance of parental and offspring characters.