Vol. 150, No. 5 The American Naturalist November 1997

THE EVOLUTION OF INDICATOR TRAITS FOR PARENTAL QUALITY: THE ROLE OF MATERNAL AND PATERNAL EFFECTS

JASON B. WOLF,[1,*,†] ALLEN J. MOORE,[2] AND EDMUND D. BRODIE III[1,†]

[1]Center for Ecology, Evolution and Behavior, T. H. Morgan School of Biological Sciences, 101 T. H. Morgan Building, University of Kentucky, Lexington, Kentucky 40506-0225;
[2]Department of Entomology, S-225 Agricultural Science Center North, University of Kentucky, Lexington, Kentucky 40546-0091

Submitted January 16, 1997; Revised April 18, 1997; Accepted May 5, 1997

Abstract.—In systems where individuals provide material resources to their mates or offspring, mate choice based on traits that are phenotypically correlated with the quality of resources provided is expected to be adaptive. Several models have explored the evolution of mating preference where there are direct benefits to choice, but few have addressed how a phenotypic correlation can be established between a male indicator trait and the degree of parental investment. We present a model with three quantitative traits: male and female parental investment and a potential male indicator trait. In our model, the expression of the ''indicator'' trait in offspring is affected by parental investment. These effects are referred to as maternal or paternal effects, or as ''indirect genetic effects'' when parental investment is heritable. With genetic variation for degree of parental investment, offspring harbor genes for parental investment that are unexpressed before mating but will affect the investment that they provide when expressed. Because the investment received from the parents affects the expression of the indicator trait, there will be a correlation between the genes for parental investment inherited and the degree of expression of the indicator trait in the offspring. The indicator trait is thus an ''honest'' signal for the degree of paternal investment.

A considerable theoretical debate has focused on the nature of the traits that are the subject of mate discrimination (see Andersson 1994). There is a particularly contentious debate surrounding the evolution of elaborate characters when there is no direct benefit to mate choice. Explanations for mate preference based on characters that are uncorrelated with direct benefits have fallen into two camps: preferred characters reflect the genetic ''quality'' of a mate or preferred characters are arbitrary with respect to viability fitness (Lande 1981; Arnold 1985; Kirkpatrick 1987; Andersson 1994). In contrast to the evolution of mate choice where no direct benefits are provided by the mate, mate choice based on traits correlated with the quality of benefits provided by the mate has been less controversial because, in this scenario, choice can directly affect fitness. Perhaps because of this relative lack of controversy, there have been fewer theoretical treatments (e.g., Trivers 1972; Kodric-Brown and Brown 1984) and explicit ge-

* To whom correspondence should be addressed; E-mail: jbwolf@ceeb.uky.edu.
† Present address: Department of Biology, Indiana University, Bloomington, Indiana 47405.

Am. Nat. 1997. Vol. 150, pp. 639–649.

netic models (e.g., Kirkpatrick 1985; Heywood 1989; Hoelzer 1989; Price et al. 1993) of mate choice based on direct benefits. There are abundant empirical studies that show that mates provide material resources that enhance the fitness of their mates or offspring (Thornhill and Alcock 1983; Clutton-Brock 1991) and mating decisions often relate to such allocations (Andersson 1994).

While the advantage of selecting a mate that provides direct benefits is obvious, it is less clear how such mate discrimination might happen or originate. One proposal is that mate choice is based on ''indicator'' traits (Michod and Hasson 1990; Grafen and Johnstone 1993) that are correlated with the quality of the resources provided by the chosen mate. Such a phenotypic correlation between a character and a benefit should clearly lead to mate choice based on that indicator trait. Left unclear by these models is exactly how such a correlation could arise. Previous genetic models either rely on an existing genetic correlation without explicitly addressing what creates this correlation (e.g., Kirkpatrick 1985; Hoelzer 1989) or rely on an underlying nongenetically determined trait, such as ''condition,'' that influences the joint expression of the indicator and the quality of the resources provided (e.g., Kodric-Brown and Brown 1984; Andersson 1986; Price et al. 1993). Here we suggest that indirect genetic effects are a potentially important yet previously unexplored cause of this correlation.

Indirect genetic effects can occur whenever individuals provide nongenetic resources (e.g., nutrition, gestation environment, protection, care) to their relatives (Cheverud and Moore 1994). These resources can vary in quality, providing a source of environmental variation to the individuals that receive these resources. Some of this variation may reflect genetic differences among individuals. Because these environmental effects have a genetic basis, they are considered indirect genetic effects (Moore et al. 1998). These effects may also be considered ''inherited environments'' because, while they are environmental effects in the offspring generation, the phenotypes in the parental generation that produce these environmental effects are heritable. Empirical studies have shown that there is heritable variation in levels of parental investment (Cheverud and Moore 1994). Indirect genetic effects have traditionally been investigated under the rubric of ''maternal effects'' models, because the most commonly investigated indirect genetic effects are associated with mammalian maternal care (Cheverud and Moore 1994). Maternal effect models can be quite general and have been used to investigate such topics as kin selection (Cheverud 1984; Lynch 1987), social selection (Moore et al. 1997, 1998), and general evolution (Cheverud 1984; Kirkpatrick and Lande 1989; Cheverud and Moore 1994; Wade 1998). In many cases, maternal effects or indirect effects result in unique evolutionary outcomes (Kirkpatrick and Lande 1989; Cheverud and Moore 1994; Moore et al. 1998).

We present a quantitative genetic model that examines the conditions under which indirect genetic effects result in a male trait that acts as an honest indicator of the quality of the resources that he provides to his offspring. Male and female care, both together and separately, are allowed to act as an environmental effect on the indicator trait. We show that when genetically variable male parental investment influences variation in the expression of an offspring trait (i.e.,

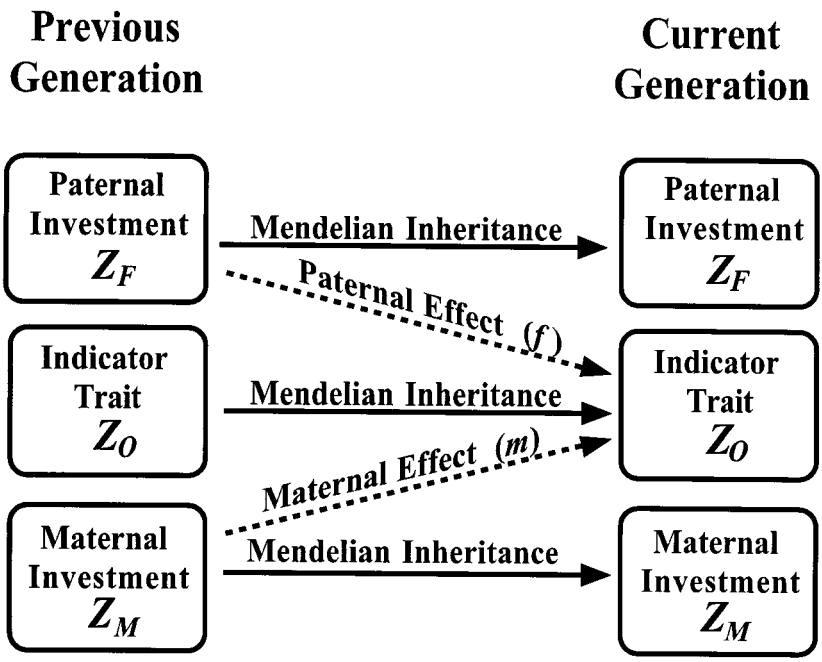

FIG. 1.—The system of inheritance for the three traits. Male and female parental investment show Mendelian inheritance (direct genetic effect). The male indicator trait has a Mendelian component as well as maternally and paternally inherited components (indirect genetic effects).

shows a paternal effect) the indicator will be an honest reflection of the quality of male parental investment. The indicator trait will also reflect mate quality whenever female investment affects its expression, as long as male and female investment are genetically correlated.

DIRECT AND INDIRECT GENETICS OF INDICATORS

Phenotypes and Inheritance

We define the genetics and phenotypes of three traits with sex-limited expression, namely, a potential male indicator trait, paternal investment, and maternal investment. Throughout, we denote parameters involving the indicator trait with the subscript ''O,'' paternal investment with subscript ''F,'' and maternal investment with subscript ''M.'' The system of inheritance for each of these traits is shown in figure 1. We assume that all traits show Mendelian inheritance. Parental investment may also show a maternal and/or paternal effect on the expression of the indicator trait (i.e., male and female investment act as an environment that affects the expression of the indicator trait; see Cheverud and Moore 1994).

All three traits are composed of an additive genetic component (a) and an en-

vironmental component (e), where the latter includes random (general) environmental and nonadditive genetic effects. These two components entirely define maternal and paternal investment. The phenotypic value of an individual (denoted by a z) for each of these traits is

$$z_F = a_F + e_F$$

and

$$z_M = a_M + e_M. \tag{1}$$

Because the male indicator trait is influenced by parental investment (i.e., maternal or paternal effect), we partition the environment into the random component (e_O), the maternal component (e_D, where ''D'' stands for dam), and the paternal component (e_S, where S stands for sire):

$$z_O = a_O + e_S + e_D + e_O. \tag{2}$$

The amount of investment received from the father is denoted $z^*_{F(t-1)}$ and from the mother as $z^*_{M(t-1)}$, where the asterisk indicates that the father and mother have survived selection in the previous generation and $t - 1$ indicates a trait expressed in the previous generation. The parental effect coefficients are denoted f (the paternal effect coefficient) and m (the maternal effect coefficient). These parental effects determine the degree to which the phenotype of the offspring is determined by the phenotype of the parent. They are defined as the partial regression of the offspring's phenotype on the parents phenotype, holding all other sources of variation constant (Kirkpatrick and Lande 1989). We can therefore rewrite the equation describing the indicator trait in generation t (eq. [2]) by substituting the definition of the paternal and maternal components [$e_S(t) = fz^*_{F(t-1)}$ and $e_D(t) = fz^*_{M(t-1)}$] as

$$z_O(t) = a_O(t) + e_O(t) + fz^*_{F(t-1)} + mz^*_{M(t-1)}. \tag{3}$$

The additive genetic components are assumed to be multivariate normally distributed with variance-covariance matrix **G**. The environmental variances are also assumed to be multivariate normally distributed and have a diagonal matrix **E**, where all environmental covariances are assumed to be zero. The phenotypic values are also assumed to be multivariate normally distributed with mean vector $\bar{\mathbf{z}}$, and variance-covariance matrix **P**. Phenotypic variances and covariances, except those involving z_O, are equal to the value from the **G** matrix plus the corresponding term from the **E** matrix. Because of the complex inheritance of z_O, the phenotypic variances and covariances P_{OF}, P_{OM}, and P_{OO} are somewhat more involved (Kirkpatrick and Lande 1989). Taking the expected covariance between traits using equations (1) and (3), the phenotypic covariance between z_O and the two parental investment traits (z_F and z_M) is

$$P_{OF} = G_{OF} + \frac{f}{2} G_{FF} + \frac{m}{2} G_{MF} \tag{4a}$$

and

$$P_{OM} = G_{OM} + \frac{m}{2} G_{MM} + \frac{f}{2} G_{MF}, \tag{4b}$$

where the first term on the right side of the equations accounts for phenotypic covariance resulting from a genetic covariance, the second term accounts for phenotypic covariance resulting from paternal inheritance (eq. [4a]) or maternal inheritance (eq. [4b]), and the last term accounts for the covariance resulting from pleiotropy or linkage disequilibrium between the maternally and paternally inherited components. In some maternal effects models, the covariance between traits after a single generation differs from that which would exist at equilibrium (Kirkpatrick and Lande 1989). This result occurs whenever a maternal or paternal trait affects the expression of that same trait in the offspring (as in Falconer 1965) or when there are cycles in the maternal- or paternal-effects matrix (Kirkpatrick and Lande 1989). When a parental trait affects the expression of a different offspring trait and there are no cycles in the parental-effect matrices, as in our example, the equilibrium covariances are equivalent to these single generation covariances.

Assuming a random association of parental investment phenotypes between a mating pair and taking the variance of z_O from equation (3), the phenotypic variance of the indicator trait, P_{OO}, equals

$$P_{OO} = G_{OO} + E_{OO} + (fG_{OF} + f^2G_{FF} + f^2E_{FF}) + (mG_{OM} + m^2G_{MM} + m^2E_{MM}). \quad (5)$$

The first term in parentheses is the variance due to paternal inheritance and the second term accounts for variance due to maternal inheritance. Nonrandom matings with respect to parental investment phenotypes would add an additional term, $2mf\ \mathrm{cov}[z^*_{M(t-1)}, z^*_{F(t-1)}]$, where the covariance accounts for the nonrandom association of parental phenotypes.

Using the variance and covariance equations above, the phenotypic correlation between the male indicator trait and paternal investment (ρ_{OF}) is:

$$\rho_{OF} = \frac{P_{OF}}{\sqrt{P_{OO}P_{FF}}}. \quad (6)$$

Implications of Indirect Genetic Effects for Honest Indicators

The ''honesty'' of the indicator trait is determined by the degree to which it is phenotypically correlated with male parental investment and is thereby a reliable predictor of the amount of investment that a male will give. Either a positive or negative correlation results in a predictable relationship between the indicator trait and paternal investment; therefore, a correlation of either sign is honest in the sense that parental investment can be predicted based on the value of the indicator trait. The magnitude of the correlation will determine the degree of predictability and therefore the degree of honesty of the indicator trait. A correlation of either sign means that when a female bases her mate choice on the indicator trait she will receive a greater amount of parental investment by the male than will a female that mates at random (assuming her choice of males is in the direction positively associated with investment). The larger the magnitude of the

correlation, the more predictable the relationship between the indicator trait and paternal investment, and the higher the potential gain of female choice based on that trait. The factors that determine the magnitude of the correlation between parental investment and the indicator trait are given in equations (4a), (5), and (6). The phenotypic covariance (eq. [4a]) between the indicator trait and paternal investment contains three components that in sum determine the degree of phenotypic association between the two traits. These three factors are described in detail below.

The first factor affecting the phenotypic covariance between the indicator trait and paternal investment (eq. [4a]) is the genetic covariance (G_{OF}) between the two traits. This source of phenotypic covariance has traditionally been considered, but the source of the genetic covariance is usually not explained. Genetic covariance between the traits is due to pleiotropy and/or linkage disequilibrium between the traits.

The second term in equation (4a), $(f/2)G_{FF}$, accounts for covariance due to paternal inheritance. Whenever the expression of a trait is sensitive to the amount of genetically determined paternal investment received, this term will be nonzero. This covariance between the paternal investment and indicator trait phenotypes exists because a male inherits genes that influence paternal investment from its father while at the same time the investment he receives from his father affects the expression of his indicator trait. Therefore, even when there is no genetic covariance, a phenotypic covariance may still exist and have a genetic basis.

The last term in equation (4a), $(m/2)G_{MF}$, accounts for the covariance due to maternal inheritance and the genetic covariance between male and female parental investment. While in some cases it may be reasonable to assume that this term is the result of linkage disequilibrium, perhaps because of males and females with high investment that have mated assortatively, the term G_{MF} may also reflect pleiotropy between the two traits (i.e., male and female parental investment are under similar genetic control). If this term is nonzero, it reflects the fact that the amount of investment received from the mother affects the expression of the indicator trait and, because of the genetic covariance between maternal and paternal investment, the paternal investment phenotype expressed by the sons will be similar to that expressed by the mother.

Because it is the phenotypic correlation, not the phenotypic covariance, that determines the degree of honesty of the indicator trait, it is important to consider what phenotypic correlation will result in systems where a phenotypic covariance is generated by this process. While the effect of G_{OF} and f on the phenotypic covariance, P_{OF}, is linear, these parameters do not have a linear effect on the phenotypic correlation (eq. [6]). These same factors affect the phenotypic variance of the indicator trait (eq. [5]), which is in the denominator of the correlation equation. While the two parameters directly affect the covariance, the correlation that results will not be as large as expected because these parameters also inflate the phenotypic variance. The correlation that results is shown in figure 2*A*.

The effect of the maternal component $(m/2)G_{MF}$ on the phenotypic covariance

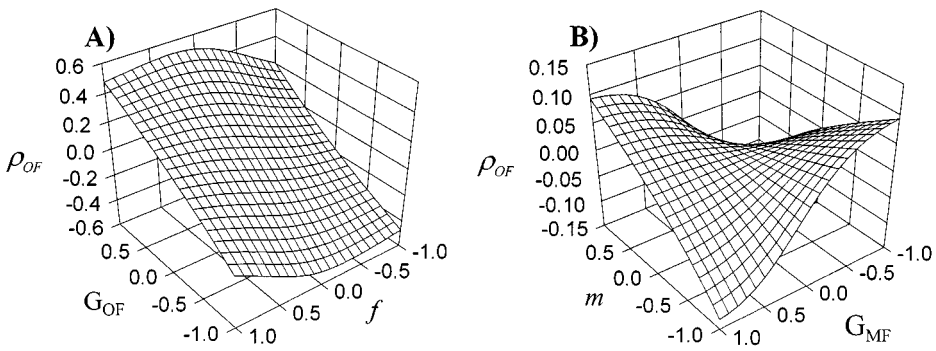

FIG. 2.—The direct and indirect genetic components of the correlation between the male indicator trait and paternal investment (ρ_{OF}). *A*, The portion due to the direct genetic covariance (G_{OF}) and the paternal effect (f). *B*, The ''maternal'' component of this correlation that is a function of the magnitude of the maternal effect (m) and the genetic covariance between maternal and paternal investment (G_{MF}).

between the indicator and paternal investment is nonlinear because of the multiplicative contribution of m and G_{MF}. Because these two parameters also interact to produce the phenotypic variance, they exaggerate the nonlinear effect on the correlation (ρ_{OF}). The resulting phenotypic correlation (fig. 2*B*) is a saddle-shaped surface where most of the parameter space is near zero. The correlation is large only when both maternal components are fairly large, and very large only when both parameters are at their extremes. Therefore, the effects of the maternal component on the phenotypic correlation will be important only when there is both a moderately large maternal effect and a high genetic covariance (i.e., pleiotropy) between male and female investment.

DISCUSSION

Whenever traits honestly signal how much parental investment a male will give, preferences for these traits will be adaptive. However, few genetic mechanisms have been proposed that can generate a correlation between a male indicator trait and male parental investment (but see Price et al. 1993). Our model proposes one particular set of conditions that could generate this relationship—indirect genetic effects. The indirect genetic effect from the father (i.e., the paternal effect) results in a phenotypic covariance because the parental investment phenotype expressed by the father affects the expression of the indicator trait by his male offspring, who also inherit half the genes that influence paternal investment from their father. Therefore, the phenotypic value of the offspring's indicator trait will, to some degree, reflect the genes that influence parental investment. The indirect genetic effect from the mother (i.e., maternal effect) contributes to the phenotypic covariance in a similar fashion. The maternal investment phenotype of the mother affects the expression of the indicator trait in her male offspring, and those male offspring may show a similar level of investment as their

mother if male and female investment are genetically correlated (i.e., pleiotropic or in linkage disequilibrium).

The existence of a genetic covariance between male and female investment will, in part, rely on the nature of investment. When the traits that determine the amount of parental investment given by males and females are similar, there may be at least some common genetic basis (perhaps due to common underlying physiological or hormonal mechanisms). For example, in ring doves male and female regurgitative feeding and other parental behaviors are regulated in part by prolactin (Horseman and Buntin 1995). Levels of this hormone in both sexes are likely to be controlled by at least some common genes, generating a pleiotropic genetic covariance between maternal and paternal investment.

Empirical results supporting the role of indicator traits for parental quality in mate choice are becoming more common. In many of these systems there is also the opportunity for indirect genetic effects to play a role. For example in the house finch, *Carpodacus mexicanus,* females preferentially mate with males that have brighter plumage, and males with brighter plumage are better fathers (Hill 1991). Hill also found a significant positive correlation between plumage brightness of fathers and sons. Hill (1991, p. 338) suggests that this correlation is due to a ''good genes'' effect but points out that ''it could also be due to maternal or paternal effects as colorful males tend to provide more food and probably pair with higher quality females than drab males.'' Mate choice in the house finch based on plumage brightness may be adaptive because of indirect genetic effects as predicted in our model and thus not rely on a good genes process. A similar scenario may exist in the collared flycatcher, *Ficedula albicollis.* Gustafsson et al. (1995) have shown that the rearing environment (altered by experimental manipulation of brood size) affects the size of the forehead patch (a sexually selected character) developed by yearling males. The authors point to the causal relationship between reduced patch size and the condition of the young in the large broods. If amount of care received from the male and/or female parent has effects similar to the experimental alteration of brood size then it seems possible that the forehead patch trait fits our model.

One set of traits that may provide an adequate test of the predictions from our model are traits that exhibit fluctuating asymmetry (FA; Van Valen 1962). The degree of FA has been linked to environmental factors such as stress and food availability and has been suggested to be involved in mate choice (see Møller 1992; Swaddler and Witter 1994; Markow 1995). Symmetry may be influenced by environmental factors, and therefore it may not be possible to genetically canalize perfect symmetry (Markow 1995). However, there is currently no empirical example of parentally provided environments affecting FA, and therefore the degree to which FA may be used as an indicator of parental quality is unknown.

Despite the lack of data, FA remains a strong candidate trait to test our model. In the scorpionfly, *Panorpa vulgaris,* males provide a salivary nuptial gift. In one study of this species, Thornhill and Sauer (1992) have shown that males that provide a larger salivary gift have more symmetrical sons. While the authors believe that these differences reflect a correlation with an underlying trait (i.e., het-

erozygosity) that would affect both FA and ability to compete for resources for the nuptial gift, they do point out that the ''possibility that the side effects . . . derive from differences in nongenetic paternal benefits to offspring'' (p. 264). Thornhill and Sauer (1992) propose the proper test of the alternative hypothesis, one where nuptial gift size is held constant and direct genetic effects are measured. They do not, however, consider the role that indirect effects might have. Rather, they lump these into ''nongenetic'' paternal benefits that create the correlation between the nuptial gift size and offspring traits. In another study, Møller (1992) has shown that, counter to the predictions of our model, females mate with males that are more symmetrical but that those males give less care than the more symmetrical males. There is no report of data on the effects of care on FA in his system.

CONCLUSIONS

Whenever the degree of expression of a trait is contingent on the amount of parental resources received or the rearing environment experienced by an individual, that trait may be a reliable indicator of mate quality. Therefore, we suggest that traits that are sensitive to the parentally provided environment may be the focus of mate choice when the parental traits are heritable. This process provides a new mechanism that can generate indicator traits for parental investment and may explain the initiation of the sexual selection process, as envisioned as the first phase of the runaway process by Fisher (1915), where females make adaptive mate choice.

While empirical evidence supports the contention that maternal effects are ubiquitous (see Mousseau and Fox 1998), adequate data to assess the assumptions and conditions of our model are lacking. For example, there are few data on the degree of genetic correlation between male and female parental traits or on the prevalence of paternal effects. Systems in which males provide material resources or alter the rearing environment are not uncommon, but few attempts have been made to quantify the effect of these paternal traits on the expression of offspring traits.

ACKNOWLEDGMENTS

We thank J. M. Cheverud, A. Sih, J. B. Walsh, and an anonymous reviewer for helpful discussions or comments on the manuscript. This research was supported by the National Science Foundation (NSF) Graduate Research Fellowship to J.B.W. Grants IBN-9514063, DEB-9521821, and IBN-9616203 from the NSF, as well as State and Federal Hatch, supported A.J.M. Grants DEB-9509295 and IBN-9600775 from the NSF supported E.D.B.

LITERATURE CITED

Andersson, M. 1986. Evolution of condition-dependent sex ornaments and mating preferences: sexual selection based on viability differences. Evolution 40:804–816.

———. 1994. Sexual selection. Princeton University Press, Princeton, N.J.
Arnold, S. J. 1985. Quantitative genetic models of sexual selection. Experientia (Basel) 41:1296–1310.
Cheverud, J. M. 1984. Evolution by kin selection: a quantitative genetic model illustrated by maternal performance in mice. Evolution 38:766–777.
Cheverud, J. M., and A. J. Moore. 1994. Quantitative genetics and the role of the environment provided by relatives in the evolution of behavior. Pages 67–100 *in* C. R. B. Boake, ed. Quantitative genetic studies of behavioral evolution. University of Chicago Press, Chicago.
Clutton-Brock, T. H. 1991. The evolution of parental care. Princeton University Press, Princeton, N.J.
Falconer, D. S. 1965. Maternal effects and selection response. Pages 763–774 *in* S. J. Geerts, ed. Genetics today: Proceedings of the XI International Congress on Genetics. Vol. 3. Pergamon, Oxford.
Fisher, R. A. 1915. The evolution of sexual preference. Eugenics Review 7:184–192.
Grafen, A., and R. A. Johnstone. 1993. Why we need ESS signalling theory. Philosophical Transactions of the Royal Society of London B, Biological Sciences 340:245–250.
Gustafsson, L., A. Qvarnström, and B. C. Sheldon. 1995 Trade-offs between life-history traits and a secondary sexual character in male collared flycatchers. Nature (London) 375:311–313.
Heywood, J. S. 1989. Sexual selection by the handicap mechanism. Evolution 43:1387–1397.
Hill, G. E. 1991. Plumage coloration is a sexually selected indicator of male quality. Nature (London) 350:337–339.
Hoelzer, G. A. 1989. The good parent process of sexual selection. Animal Behaviour 38:1067–1078.
Horseman, N. D., and J. D. Buntin 1995. Regulation of pigeon cropmilk secretion and parental behaviors by prolactin. Annual Review of Nutrition 15:213–238.
Kirkpatrick, M. 1985. Evolution of female choice and male parental investment in polygynous species: the demise of the sexy son. American Naturalist 125:788–810.
———. 1987. Sexual selection by female choice in polygynous animals. Annual Review of Ecology and Systematics 18:43–70.
Kirkpatrick, M., and R. Lande. 1989. The evolution of maternal characters. Evolution 43:485–503.
Kodric-Brown, A., and J. H. Brown. 1984. Truth in advertising: the kinds of traits favored by sexual selection. American Naturalist 124:309–323.
Lande, R. 1981. Models of speciation by sexual selection on polygenic traits. Proceedings of the National Academy of Sciences of the USA 78:3721–3725.
Lynch, M. 1987. Evolution of intrafamilial interactions. Proceedings of the National Academy of Sciences of the USA 84:8507–8511.
Markow, T. A. 1995. Evolutionary ecology and developmental instability. Annual Review of Entomology 40:105–120.
Michod, R. E., and O. Hasson. 1990. On the evolution of reliable indicators of fitness. American Naturalist 135:788–808.
Møller, A. P. 1992. Female swallow preferences for symmetrical male sexual ornaments. Nature (London) 357:238–240.
Moore, A. J., E. D. Brodie III, and J. B. Wolf. 1997. Interacting phenotypes and the evolutionary process. I. Direct and indirect genetic effects of social interactions. Evolution (in press).
Moore, A. J., J. B. Wolf, and E. D. Brodie III. 1998. The influence of direct and indirect genetic effects on the evolution of behavior: sexual and social selection meet maternal effects. *In* T. A. Mousseau and C. Fox, eds. Maternal effects as adaptations. Oxford University Press, Oxford (in press).
Mousseau, T. A., and C. Fox, eds. 1998. Maternal effects as adaptations. Oxford University Press, Oxford (in press).
Price, T., D. Schluter, and N. E. Heckman 1993. Sexual selection when the female directly benefits. Biological Journal of the Linnean Society 48:187–211.
Swaddler, J. P., and M. S. Witter. 1994. Food, feathers and fluctuating asymmetries. Proceedings of the Royal Society of London B, Biological Sciences 255:147–152.
Thornhill, R., and J. Alcock. 1983. The evolution of insect mating systems. Harvard University Press, Cambridge, Mass.

Thornhill, R., and P. Sauer. 1992. Genetic sire effects on the fighting ability of sons and daughters and mating success of sons in a scorpionfly. Animal Behaviour 43:255–264.

Trivers, R. 1972. Parental investment and sexual selection. Pages 136–179 *in* B. Campbell ed. Sexual selection and the descent of man, 1871–1971. Heinemann, London.

Van Valen, L. 1962. A study of fluctuating asymmetry. Evolution 16:125–142.

Wade, M. J. 1998. The evolutionary genetics of maternal effects. *In* T. A. Mousseau and C. Fox, eds. Maternal effects as adaptations. Oxford University Press, Oxford (in press).

Associate Editor: J. Bruce Walsh