On the assignment of fitness to parents and offspring: whose fitness is it and when does it matter?

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Abstract

There has been a long-standing conceptual debate over the legitimacy of assigning components of offspring fitness to parents for purposes of evolutionary analysis. The benefits and risks inherent in assigning fitness of offspring to parents have been given primarily as verbal arguments and no explicit theoretical analyses have examined quantitatively how the assignment of fitness can affect evolutionary inferences. Using a simple quantitative genetic model, we contrast the conclusions drawn about how selection acts on a maternal character when components of offspring fitness (such as early survival) are assigned to parents vs. when they are assigned directly to the individual offspring. We find that there are potential shortcomings of both possible assignments of fitness. In general, whenever there is a genetic correlation between the parental and direct effects on offspring fitness, assigning components of offspring fitness to parents yields incorrect dynamical equations and may even lead to incorrect conclusions about the direction of evolution. Assignment of offspring fitness to parents may also produce incorrect estimates of selection whenever environmental variation contributes to variance of the maternal trait. Whereas assignment of offspring fitness to the offspring avoids these potential problems, it introduces the possible problem of missing components of kin selection provided by the mother, which may not be detected in selection analyses. There are also certain conditions where either model can be appropriate because assignment of offspring fitness to parents may yield the same dynamical equations as assigning offspring fitness directly to offspring. We discuss these implications of the alternative assignments of fitness for modelling, selection analysis and experimentation in evolutionary biology.

Introduction

When considering the evolution of a wide variety of traits, ranging from social behaviours to parental care and germination strategies, it is common practice to consider the numbers, viability and reproductive success of offspring as components of maternal fitness (e.g. Williams, 1966, p. 184; Alexander, 1974, p. 26; Hamilton & Zuk, 1982; see also Cheverud & Moore, 1994). This approach is often also adopted uncritically in the analysis of data. For example, in experimental studies of parental investment, offspring fertility and offspring fecundity are explicitly considered components of mothers' fitness in order to evaluate theories of the evolution of maternal strategies (e.g. see chapters in Clutton-Brock, 1988a). In contrast to this practice, phenotypic selection theory explicitly cautions against the assignment of fitness in one generation (offspring) to individuals in another (parents) when analysing empirical data (e.g. Arnold, 1983; Lande & Arnold, 1983; Cheverud, 1984; see also Cheverud & Moore, 1994).

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Which, if either, of these opposing recommendations should be followed when analysing empirical data? This debate over which model is the appropriate one when analysing data has been contentious (Grafen, 1988; Cheverud & Moore, 1994).

On one hand, evolutionary genetic theorists assert that the fitness of one individual should never be assigned to another (e.g. Cheverud & Moore, 1994). Under this theoretical genetic perspective, it is recommended that, in empirical studies, fitness be counted from conception to death and not be allowed to cross the generational boundary (Arnold, 1983; Lande & Arnold, 1983; Cheverud, 1984; Cheverud & Moore, 1994). This perspective maintains that, accurate estimates of the strength and direction of evolution require this measurement of individual lifetime fitness (e.g. Cheverud, 1984; Queller, 1992a, b; Cheverud & Moore, 1994; Wolf *et al*., 1999).

The other side of this debate is best represented by behavioural ecologists using optimality theory (Grafen, 1988; Clutton-Brock, 1988b; Cheverud & Moore, 1994). In practice, when evolutionary stable strategy (ESS) theory is applied to the analysis of empirical data, a definition of fitness is used in which components of offspring fitness (e.g. survival to a given age and later reproductive success) are assigned as components of parental fitness. This allows analyses of those cases where parental behaviour influences offspring fitness. For example, two-thirds of the empirical studies (particularly those focusing on birds and mammals) compiled by Clutton-Brock (1988a) included one or more components of offspring fitness into calculations of parental fitness. Indeed, it was concluded that 'One of the most obvious generalizations arising from the analysis of fitness components in females is that individual differences in offspring survival [offspring fitness] are one of the most important components of variation in lifetime reproductive success among breeding females [parental fitness] in many birds and mammals… In particular, differences in offspring survival after fledging or weaning [offspring fitness] were the principal source of variation in reproductive success among breeding adults [parental fitness] in several species' (Clutton-Brock (1988b, p. 473). One example from these studies is an analysis of lifetime fitness in the Great Tit, *Parus major*. Its authors conclude that the largest source of variance in lifetime reproductive success of parents is 'attributable to variation in the recruitment rate of [their] offspring' (McCleery & Perrins, 1988, p. 153). Clearly, a causal connection between offspring fitness and parental behaviour is assumed by these analyses, and it is this assumption that justifies the consideration of offspring fitness traits as components of maternal fitness (Grafen, 1988).

The assignment of offspring fitness to parents is not only practiced but also recommended in some contexts. Indeed, the inability to measure offspring fitness, especially viability, and assign it to parental fitness, is considered a general limitation of invertebrate systems for testing behavioural evolution theory. Empirical studies with invertebrates have been criticized as inadequate because '…they have not yet been able to measure individual differences in offspring survival' and, hence, omit a crucial component of parental fitness (Clutton-Brock, 1988b, p. 474).

Different approaches to the assignment of fitness have been used in behavioural ecology and evolutionary genetics primarily because different sorts of evolutionary questions have been addressed (e.g. predicting optimal values, which are rarely influenced by underlying genetics except when traits show genetic multicollinearity vs. evolutionary trajectories, which are largely determined by the quantitative genetics of characters). The two approaches have been successful and have lead to evolutionary insights because they were employed in analyses that were based on implicit assumptions about the genetic basis of characters, which allowed for the exploration of certain evolutionary questions. However, these assumptions are often not explicitly defined and the impact that assumptions about the genetics of characters can have on evolutionary predictions are rarely addressed using explicit models (but see Discussion of this issue in Cheverud, 1984, 1985; Grafen, 1988; Cheverud & Moore, 1994).

In this paper, we consider the consequences of alternative assignments of fitness in empirical and theoretical analyses of evolution and determine the magnitude of the error that can accrue when either approach is misapplied in data analysis. Using a simple quantitative selection and genetic model, we consider two kinds of influences on offspring viability fitness: (1) maternal effects, wherein characteristics of the mother (phenotypic or genetic) influence the fitness of her offspring ('maternal selection' *sensu* Kirkpatrick & Lande, 1989) and (2) direct effects, wherein the viability fitness of an offspring is influenced by its own genotype and phenotype. In this way, we clarify the circumstances under which assigning offspring fitness to mother (or either parent) is permitted and when it is not in the analysis of data. We also determine the size of the error that can be introduced into data analysis if the approach is applied where it is not permitted.

The model

Trait evolution can be analysed from the perspective of quantitative genetic and phenotypic selection theory, which permits our results to be applied to characters with continuous and discrete variation in natural populations. We utilize a simple fitness model to understand the implications of the assignment of offspring fitness to mothers. We follow this section with a discussion of the genetics of the parental and offspring characters and show how maternal effects on the offspring phenotype may alter the outcome of the model. Within the general theoretical framework presented here, one can analyse

several different scenarios for the assignment of fitness in data analyses. This approach permits us to determine under what circumstances the results of empirical analyses are model dependent or model independent. In the context of the introduction, we are asking when can the assignment of fitness in data analysis safely cross the generational boundary (as recommended by some, e.g. Grafen 1988; Clutton-Brock, 1988b) and when can it not (as recommended by others, e.g. Cheverud & Moore, 1994).

Underlying fitness model

We begin by defining the underlying model for fitness of an individual, where an individual's fitness is measured from the time of conception to the time of death (and thus does not contain cross-generational assignments of fitness). This is the true model for fitness, where we look at all influences on all components of fitness of an individual within the entire length of that individual's lifetime. It is important to understand that this is a mechanistic theoretical model, which we are using to define fitness. The statistical model that one would *estimate* from a data set may or may not capture these underlying causal components of fitness. Using this mechanistic model for fitness, we compare the evolutionary inferences drawn by two experimental biologists who use the same data set but different assignments of fitness. One biologist analyses the data by assigning offspring viability fitness to the mother whereas the other analyses the same data but assigns all components of offspring viability to the offspring. We ask whether these two biologists come to the same inference regarding how selection is acting on a maternal trait, given the underlying mechanistic model. This is the ultimate test of a method of data analysis: does it provide an estimate of the underlying causal fitness components or not? In natural populations, the underlying causes are not known *a priori* as they are in our theoretical model. We must test a method of data analysis against the theory to know whether or not and under what circumstances to apply it to analyse data from nature. The conclusions we draw here also impact the construction of models because it is common for theoretical analyses to use a particular assignment of fitness without analysing the consequences of the approach chosen relative to alternative assignments.

We assume that individuals express two traits (the basic conclusions from this simple two-trait system can easily be extended to understand more complex multi-trait systems). One trait is tied to early viability and we refer to it as the 'offspring trait' and denote its phenotypic value by z_o. The second trait is an adult, maternally expressed trait with phenotypic value, z_m, i.e. by definition, only expressed in female parents. (Note: this maternal trait could be expressed by either parent but we assume it to be maternal here for simplicity.) This second trait measures maternal quality, i.e. some characteristic of the female parent that affects the viability fitness of her offspring in the next generation (Fig. 1). These are the kinds of traits that motivate the recommendations of Clutton-Brock (1988b) discussed in Introduction and that are commonly the focus of theoretical analyses in behavioural ecology. In the perspective of phenotypic selection theory, this means that an offspring's fitness is affected by the value of an adult trait expressed by one of its parents. We assume that we measure the phenotypic value of the offspring trait, z_o, in all individuals and the value of the maternal trait in all female parents (under the assumption that all female adults reproduce).

With these definitions, individual viability fitness is given by three components (using the notation of Kirkpatrick & Lande, 1989):

$$w_{(t)} = \alpha_{(t)} + b_m z_{m(t-1)} + b_d z_{m(t)} + b_o z_{o(t)} + \varepsilon_{(t)}. \quad (1)$$

Here, $\alpha_{(t)}$ is baseline fitness at generation t (which is the sum of all deterministic components of fitness that are uncorrelated with the traits being considered) and $\varepsilon_{(t)}$ is

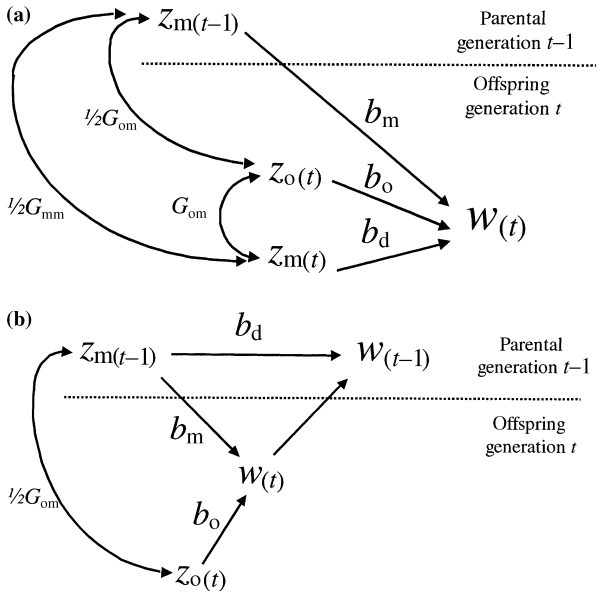

Fig. 1 Fitness components under alternative assignments of offspring fitness. (a) Pathways of effect in the underlying model for offspring fitness. Effects on fitness ($w_{(t)}$) are decomposed into the influence of traits expressed by the offspring itself ($z_{o(t)}$ and $z_{m(t)}$) as well as the influence of the phenotype of its mother ($z_{m(t-1)}$). Covariances between traits are marked with double headed arrows and are labelled with the appropriate covariance parameter. The strength of influence that the three traits have on offspring fitness are b_o, b_d, b_m for the effect of direct selection on the offspring trait, direct selection on the maternal trait and the effect of maternal selection, respectively. The generational boundary is marked with a dashed line. (b) Assignment of offspring fitness to the mother. Offspring fitness ($w_{(t)}$) appears as a component of maternal fitness ($w_{(t-1)}$).

the random (environmental) component that is uncorrelated with the phenotype of the mother or the offspring. The coefficient, b_m, defines the linear relationship between an individual's viability and the parental quality of its mother (which is denoted $z_{m(t-1)}$). The subscript, $(t-1)$, denotes that this trait is measured on the individual's mother, a member of the previous generation (i.e. generation, $t-1$). This term is analogous to the maternal selection gradient of Kirkpatrick & Lande (1989), except that our value of b_m *defines* the relationship between offspring fitness and maternal quality, whereas the maternal selection gradient is a parameter *estimated* from data in a population. The coefficient, b_d, defines the linear relationship between viability and an individual's own value of the maternal character ($z_{m(t)}$). The subscript 'd' indicates that this is the *direct* relationship between the maternal character and the individual's fitness. Lastly, b_o defines the linear relationship between viability and an individual's phenotypic value of the offspring character ($z_{o(t)}$). We assume that selection on the juvenile character ($z_{o(t)}$) is equal in both sexes and that the phenotype, (z_m), is only expressed in females. Note that, for the last two components of fitness we have used the subscript (t) to denote that these traits are measured in a different generation than the parental trait. We emphasize that traits, $z_{o(t)}$ and $z_{m(t)}$, are measured in the *same* individual but at two different times in its lifetime, whereas $z_{m(t-1)}$ is a character measured in the female parent. Because z_m is not expressed in males by definition, the term $b_d z_{m(t)}$ will be zero for males. However, because all individuals have a mother, the term $b_m z_{m(t-1)}$ will be nonzero for both males and females.

This simple model allows us to analyse a number of scenarios for fitness assignment in data analysis as we can make assumptions about what the traits represent. For example, $z_{o(t)}$ and $z_{m(t)}$ could be the same trait (such as body size) measured early in life and again later in life or they could be two separate traits, genetically correlated owing to pleiotropy or linkage disequilibrium. Although simple, this basic model allows us to understand how alternative fitness assignments affect the analysis of empirical data and the inferences drawn from them.

Assigning offspring fitness to the offspring

We now use this model of fitness and examine how the assignment of all components of fitness to the offspring as per eqn 1 affects our inference about the evolution of the maternal character. We imagine that we have measurements for each individual's phenotypic value for both its offspring and maternal traits and its mother's value of the maternal trait. We derive the net selection differential (s_m) acting on the maternal trait, z_m, by finding the total covariance of the trait value with individual fitness, $\mathrm{cov}(z_{m(t)}, w_{(t)})$, for all individuals in the population (cf. eqns 16 and 17 in Kirkpatrick & Lande, 1989). The $\mathrm{cov}(z_{m(t)}, w_{(t)})$ within generation t defines the selection differential on the maternal trait:

$$S_m = \mathrm{cov}(z_{m(t)}, w_{(t)}) \tag{2a}$$

$$S_m = b_m \mathrm{cov}(z_{m(t)}, z_{m(t-1)}) + b_o P_{om} + 1/2 b_d P_{mm}. \tag{2a}$$

The quantity, $\mathrm{cov}(z_{m(t)}, z_{m(t-1)})$, is the covariance between the phenotypic value of the maternal trait measured in the mother, $z_{m(t-1)}$, and in her offspring, $z_{m(t)}$. The phenotypic covariance, P_{om}, is the covariance measured in the offspring between their maternal trait, $z_{m(t)}$, and their offspring trait, $z_{o(t)}$. P_{mm} is the phenotypic variance of trait z_m (assumed to be the same in both mothers and daughters). Because direct selection acting on the maternal character is sex-limited (i.e. males do not contribute to the selection differential via this component), this component of direct selection has a coefficient of ½. Note that maternal selection generates selection on the maternal character in the offspring, $z_{m(t)}$ that has not yet been expressed, and, in the case of the maternal trait in male offspring, will *never* be expressed (Kirkpatrick & Lande, 1989). The covariance between maternal and offspring phenotypes (generated by the covariance of their genotypes) produces a force of selection on $z_{m(t)}$, the parental quality of the offspring *before* the offspring express the maternal quality trait. Because this form of kin-selection (*sensu* Cheverud, 1984) acts before the trait has been expressed by the offspring, it is not sex limited. As a result, one must use information from traits that can be measured (the maternal phenotype and the eventual maternal phenotypes of female progeny) to infer the impact of maternal selection on trait distributions (Kirkpatrick & Lande, 1989). Because $z_{m(t)}$ is not expressed in males, the covariance, P_{om}, cannot be measured directly in males. However, males still harbour this trait in unexpressed form and direct selection on the offspring trait, $z_{o(t)}$, in males does contribute to the value of s_m. We must measure this covariance in males by using the average of their sisters. The implications of this complication imposed by maternal selection on evolutionary inference are discussed below.

Because we have defined fitness as a linear function of $z_{m(t-1)}$, $z_{o(t)}$ and $z_{m(t)}$ (eqn 1) which are all measured for the offspring to whom we are assigning fitness in this case, eqn 2 yields the true phenotypic selection differential. That is, we know that the expression in eqn 2 is the correct estimate of selection acting on the maternal character, because we both defined fitness for the individual and have assigned fitness to those same individuals.

Selection on traits with Mendelian inheritance

Equation 2 can be re-written in a simplified form assuming Mendelian inheritance and no environmental covariances between traits or across generations. Under Mendelian inheritance with additive effects (an assumption we make only for pedagogy, cf. Wolf *et al.*, 2000), we

can partition the maternal and juvenile traits into additive genetic effects, a_o and a_m, respectively, and environmental effects, e_o and e_m, respectively (see Falconer & Mackay, 1996). Under this assumption, the genetics of the two traits are defined as:

$$z_{o(t)} = a_o + e_o \quad (3)$$

and

$$z_{m(t)} = a_m + e_m. \quad (4)$$

Assuming no genotype–environment covariances, we can now simplify the phenotypic covariance terms in eqn 2 so that selection on the maternal trait becomes,

$$S_m = 1/2b_m G_{mm} + b_o G_{om} + 1/2b_d P_{mm}. \quad (5)$$

Here, G_{mm} is the additive genetic variance of the maternal character and G_{om} is the additive genetic covariance between the maternal and offspring characters. (These are not subscripted with a t because we assume they are constant over the time scale and strength of selection that we are investigating.) In this simple case, the covariance between the juvenile and maternal traits is completely determined by the genetic covariance between these traits. The mother–offspring covariance expression is defined as one-half the total additive genetic variance for the maternal trait, because one-half represents the coefficient of relatedness between mothers and offspring.

In the Appendix, we present the selection coefficient for the case where there are maternal effects on the expression of the offspring phenotype, $z_{o(t)}$ (i.e. maternal inheritance, *sensu* Kirkpatrick & Lande, 1989) in addition to the maternal effects on offspring fitness that are contained in these equations. With maternal inheritance, a mother's phenotype determines, at least in part, the trait values of her offspring. Maternal inheritance is likely to occur whenever there is considerable maternal investment (Mousseau & Fox, 1998), which is also the condition under which maternal selection is most likely to occur. Our analysis of maternal inheritance in the Appendix shows that the general conclusions reached under the model of simple Mendelian inheritance also hold under maternal inheritance. This is not surprising because, when the maternal phenotype affects the expression of an offspring character that is itself under selection, the maternal character ultimately influences offspring fitness in a way that is analogous to the maternal effects on offspring fitness that are captured in the equations above. When maternal inheritance occurs, the only difference is that the maternal effect on fitness is indirect as it is mediated through the expression of the offspring character.

Assignment of offspring fitness to the mother

There is more than one way to assign offspring fitness to the mother. We could assume that lifetime fitness of the offspring is maternal fitness or we could assume that only early survival of offspring is a component of maternal fitness. Here, we consider the latter as it is early survival that is usually assumed to be influenced by the mother, and it is early survival that is most often assigned as a component of maternal fitness in practice. However, it is worth noting that it is not uncommon for all components of offspring fitness to be assigned as components of maternal fitness when considering offspring quality (see recommendation of Clutton-Brock, 1988b).

As an example, consider a maternal character that influences offspring survival, like feeding at the nest in birds (for simplicity, we assume no paternal care). For characters like maternal feeding, it is commonplace to assign offspring survival to the mother when considering evolutionary questions about parental investment (e.g. McCkeery & Perrins, 1988; Merilä & Sheldon, 2000). In this sort of system, the offspring trait might be something like early downy feather growth, which influences thermoregulation and thereby, early survival. We illustrate this assignment of offspring fitness to the mother with the path-diagram shown in Fig. 1.

Further, we assume that each fitness component is entered into the analysis only once so that, if offspring fitness is assigned to the mother, then the early fitness of the mother was assigned to her mother, and is not being considered a component of her fitness. Violations of this assumption would mean that a given fitness component is entered into an analysis multiple times and thus a given fitness component is assigned to more than one individual (Grafen, 1988). We avoid this problem here and defer discussion of the multiple counting problem to Grafen (1988) and Cheverud & Moore (1994).

Under the assumption that offspring fitness is part of the mother's fitness, we can take our mechanistic model (eqn 1) and divide the single equation into two separate fitness equations, one for the mother and one for her offspring (see Fig. 1). Offspring fitness is given by

$$w_{o(t)} = \alpha_{o(t)} + b_m z_{m(t-1)} + b_o z_{o(t)} + \varepsilon_{o(t)} \quad (6)$$

whereas the fitness of the mother is

$$w_{m(t-1)} = \alpha_{m(t-1)} + b_d z_{m(t-1)} + \varepsilon_{m(t-1)}. \quad (7)$$

In eqn 7, we see that maternal fitness is affected by the value of the maternal trait at the time of reproduction (i.e. by $b_d z_{m(t-1)}$). These two equations can be viewed as two bouts of selection, one occurring early in life and the other occurring later in the adult/parental phase of females but not males. These two equations clearly point out the concept introduced earlier: the early bout of selection is not sex-limited because it acts on all progeny before the maternal character is expressed.

To assign offspring fitness to the mother we assume that eqn 6 is evaluated with respect to the offspring whereas eqn 7 is evaluated with respect to the mother. To indicate this, we have labelled terms in eqn 7 as occurring in generation $t - 1$, but all values in eqn 6 are

evaluated in generation *t*, except the cross-generational maternal effect on fitness which is labelled with a $t - 1$ in eqn 6. We assign offspring fitness to the mother by summing eqns 6 and 7 and then evaluating the consequences. In this case, maternal fitness is now *defined* as the sum of female adult fitness and the viability fitness of her progeny, a practice that is common in empirical and theoretical analyses (Clutton-Brock, 1988a). Summing these quantities yields

$$w_{m(t-1)} + w_{o(t)} = \alpha_{o(t)} + b_m z_{m(t-1)} + b_o z_{o(t)} + \alpha_{m(t-1)} + b_d z_{m(t-1)} + \varepsilon_{(t)} + \varepsilon_{(t-1)} \quad (8)$$

where $\alpha_{(t)}$ in eqn 1 is simply the sum of the baseline fitness in the two equations ($\alpha_o + \alpha_m$) under the assumption that baseline fitness is constant across generations. Similarly, $\varepsilon_{(t)}$ in eqn 1 is assumed to have the same expected value as the sum of the two random components in eqn 8 ($\varepsilon_{(t)} + \varepsilon_{(t-1)}$). Note that one can keep track of whose traits are being considered as all traits measured in the mother are designated with a $t - 1$ and all those in her offspring are marked with a *t*. Also note that, although we have an equation that is analogous to eqn 1, we have a difference in the generations in which we evaluate selection on the maternal character.

Taking the covariance of the value of the maternal trait (eqn 4) with the expression in eqn 8 for the entire population (such that eqn 9 will be the average of the male and female selection equations) yields the expression for selection on the maternal trait:

$$S_m = 1/2[b_m P_{mm} + b_o \mathrm{cov}(z_{o(t)}, z_{m(t-1)}) + b_d P_{mm}] \quad (9)$$

where the factor of ½ outside of the brackets appears because, in this case, all components of selection are sex limited as we are assigning all components of fitness to mothers.

Under the assumption of Mendelian inheritance eqn 9 can be expressed as

$$S_m = 1/2[b_m P_{mm} + 1/2 b_o G_{om} + b_d P_{mm}]. \quad (10)$$

Agreement between the two approaches under Mendelian inheritance

We can compare the results of these alternative assignments of fitness by comparing eqn 5, derived under the assumption that lifetime fitness is measured and fitness is assigned to the individual, with eqn 10 in which we assigned some component of offspring fitness to the mother. From these expressions and under the assumption of Mendelian inheritance of all traits, we see that assigning offspring fitness to the mother affects our analysis of selection on the maternal trait (see Table 1). We have: (1) *over-estimated* the contribution of maternal selection, when there is environmental variance (Table 1) and (2) *under-estimated* the contribution of direct correlated selection (b_o) by a factor of ¼.

The over-estimate of the strength of maternal selection results because the component of environmental variation on the maternal trait is not translated into selection in the offspring generation. It is omitted because it does not contribute to the parent–offspring covariance; this component of maternal fitness variance is random with respect to the offspring phenotype by definition and does not contribute a component to selection. This result, that the parent–offspring covariance influences the strength of maternal selection, does not occur for natural selection because the form of inheritance is generally independent

No environmental effects (E_{mm} = 0)				With environmental effects (E_{mm} = 0.5)			
G_{om}	S_m – to offspring	S_m – to mother	Δ	G_{om}	S_m – to offspring	S_m – to mother	Δ
No phenotypic maternal effects (m = 0)							
–2.0	–1.0	0.5	–1.5	–2.0	–1.0	1.0	–2
–1.0	0.0	0.75	–0.75	–1.0	0.0	1.25	–1.25
–0.5	0.5	0.875	–0.375	–0.5	0.5	1.375	–0.875
0.0	1.0	1.0	0.0	0.0	1.0	1.5	–0.5
0.5	1.5	1.125	0.375	0.5	1.5	1.625	–0.125
1.0	2.0	1.25	0.75	1.0	2.0	1.75	0.25
2.0	3.0	1.5	1.5	2.0	3.0	2.0	1
With phenotypic maternal effects (m = 0.75)							
–2.0	–0.625	0.875	–1.5	–2.0	–0.625	1.5625	–2.1875
–1.0	0.375	1.125	–0.75	–1.0	0.375	1.8125	–1.4375
–0.5	0.875	1.25	–0.375	–0.5	0.875	1.9375	–1.0625
0.0	1.375	1.375	0.0	0.0	1.375	2.0625	–0.6875
0.5	1.875	1.5	0.375	0.5	1.875	2.1875	–0.3125
1.0	2.375	1.625	0.75	1.0	2.375	2.3125	0.0625
2.0	3.375	1.875	1.5	2.0	3.375	2.5625	0.8125

Table 1 Examples of selection differentials under the assignment of offspring fitness to either the offspring (S_m – to offspring) or to the mother (S_m – to mother). Selection values under the two assignments of fitness and the difference between these values (Δ) are given as a function of the genetic covariance between the maternal and offspring characters for cases where phenotypic maternal effects are either present or absent (m = 0 or m = 0.75) and where environmental effects on the maternal character are present or absent (E_{mm} = 0 or E_{mm} = 0.5). All values were calculated with G_{mm} = 1 and all selection parameters (b_m, b_o and b_d) equal to 1.

of the form of selection in the case of natural selection (see Kirkpatrick & Lande, 1989).

The fact that we have under-estimated the contribution of direct correlated selection is the result of two factors. First, correlated selection acting on the offspring character ($z_{o(t)}$) covaries with the expression of the mother's maternal character ($z_{m(t-1)}$) by a factor of only ½ the additive genetic covariance. Second, maternal selection affects offspring of both sexes; it is not sex limited. Thus, it should not be multiplied by the factor of ½ that was used to account for the fact that selection only occurred in females when fitness was assigned to the mother. This kind of *under-estimation* is likely to occur whenever the genes for the maternal trait are pleiotropic for some juvenile character. These sources of over and under estimation do not necessarily 'cancel out' and can lead to misidentification of the direction of selection when b_o and b_m are of opposite sign and of appropriate relative magnitudes.

It is perhaps most interesting to note the conditions under which these two approaches yield the *same* expression for selection on the maternal character. We find that when the maternal character is wholly additively genetically determined (i.e. all variance is additive genetic) and there is no genetic covariance between the maternal and offspring characters (i.e. $G_{om} = 0$), both expressions yield $s_m = ½b_m G_{mm}$ as the strength of selection on the maternal character (see example in Table 1). Thus, when there is a lack of pleiotropy (or possibly linkage disequilibrium) between the maternal character and offspring characters, and a lack of environmental variation for the maternal character, our results are similar using either approach. These conditions have been recognized previously (Grafen, 1988; Cheverud & Moore, 1994), but have not been given explicit theoretical demonstration.

Discussion

The analysis presented here shows explicitly where problems can arise in assigning offspring fitness to the mother. Our analysis also highlights the potential shortcomings of phenotypic selection analyses that may occur when lifetime fitness is assigned to the individual. The success of either approach will rest largely on the components that influence fitness, the genetics of the traits and the ability of the researcher to either separate these components empirically, or to be explicit in the assumptions underlying theoretical analyses.

We first consider assignment of lifetime fitness of an individual to the individual offspring itself. We can see from eqn 2 that, when we assign offspring fitness directly to the offspring, we get the proper dynamical equation for evolution. No correction factor is needed as the dynamical equation includes the proper doses of direct and maternal selection. This is not surprising, given that we have defined fitness for the individual to whom we assigned all components of fitness. However, the correct analysis of selection is based on an assumption that we have some *a priori* understanding of the causal effects on fitness in this case and, therefore, are able to include all of the relevant components of selection. Most noteworthy perhaps is the assumption that we have recognized the component of maternal selection that acts on males. That is, as maternal selection acts prior to the expression of the maternal character (as it acts early in life) it is not a sex-limited component of selection (Cheverud, 1984; Kirkpatrick & Lande, 1989). However, this component of selection is 'hidden' in that, we cannot detect it empirically by measuring the selection differential within a generation as the difference in the mean of a trait before and after selection. We cannot measure it because males never express the maternal phenotype, although maternal selection on males influences the way in which the maternal trait experiences selection and evolves. Because of the possibility of missing the component of 'hidden' maternal selection acting on males, it may also be easy for one to estimate selection incorrectly when assigning lifetime fitness to the individual without understanding the causal influences on fitness. Without a causal understanding of fitness effects, it is also difficult to correct for the hidden component of selection because, if one were to simply double the strength of selection acting on females, we would over-estimate the component of direct selection on females by a factor of 2. Because the components of maternal and direct selection are confounded, if one does not recognize the causal basis of the fitness effects, then there is no simple way to arrive at the correct selection equation. Thus we can see that there can be conditions where the assignment of offspring fitness to the mother may actually be beneficial because it is difficult to account for the hidden component of maternal selection acting on the distribution of the maternal character in males.

If we assign offspring fitness to the mother we find that we can grossly under-estimate the contribution of correlated selection to the net selection differential acting on the maternal character (eqn 9 and Table 1). The correlation between the maternal and offspring characters will occur when genes influencing the maternal character have pleiotropic direct effects on offspring fitness or when genes influencing these two characters are in linkage disequilibrium (Cheverud & Moore, 1994). When the maternal character being considered is genetically correlated to a direct effect on offspring fitness, then assigning offspring fitness to the mother underestimates the contribution of the direct selection component by a factor of 4. This can be corrected by multiplying *only* the direct component of selection by a factor of 4. This creates a major empirical hurdle, for, in order to multiply only the direct component of selection by 4, we need to isolate this effect from the maternal effect on fitness with which it is genetically confounded. Just as we found for the alternative assignment of offspring

fitness to offspring (see above), we need an *a priori* understanding of the causal components of offspring fitness. Without this causal understanding, we cannot produce the proper dynamical equation because we cannot dissect out the proper component to be corrected. This can be particularly problematic when the two components are of opposite sign, as one might even infer the incorrect direction of selection (see cases in Table 1 where the two selection parameters are of opposite sign). This situation, when maternal and direct effects on fitness are not independent, has been identified verbally as a potential shortcoming of assigning offspring fitness to the mother (Cheverud & Moore, 1994), but the under-estimation of net selection when this nonindependence is present has not been considered. Cheverud (1984) points out that the very presence of correlated selection on the offspring character could introduce biases into the analysis of selection and evolution of maternal characters, because this component would appear to be a component of direct selection on the maternal trait.

Problems with assigning offspring fitness to the mother also arise when the maternal trait is influenced by nonheritable factors. Comparing eqns 5 and 9 we see that the two differ whenever the maternal trait is not completely determined by additive genetic effects (i.e. when $\frac{1}{2}b_m P_{mm}$ does not equal $\frac{1}{2}b_m G_{mm}$). This difference occurs because nonheritable effects on the maternal trait are not translated into selection on the offspring character as they occur at random with respect to the value of the offspring character. Thus, when narrow-sense heritability is low, we are likely to over-estimate the contribution of maternal selection when assigning offspring fitness to the mother, but when heritability is high the two approaches will include approximately the same component of maternal selection.

To illustrate the implications of our model, consider a hypothetical example based on data from the analysis of the genetics of growth in mice by Riska *et al*. (1984). For this example, we assume that the offspring character (z_o) is growth during the last week of parental care (from 2 to 3 weeks of age) and the maternal character being considered (z_m) is growth from week 8 to 9, a time well after females have attained sexual maturity and may be likely to breed. Because Riska *et al*. (1984) present only within sex genetic correlations, we will focus on the data from females. We assume that growth during both time periods is under positive selection for increased weight gain. However, we will assume that early growth is under stronger selection than later growth (let $b_o = 2$ and $b_d = 1$) as it is assumed to be linked to early survival, which accounts for a large proportion of variation in fitness in many species (e.g. Deevy, 1947). We assume that progeny reared by faster growing females show better early survival (let $b_m = 1$), perhaps because superior overall vigour of these females that makes them better parents. Thus growth from week 8 to 9 can be viewed as a correlate of parental quality for this example, and will be treated as a surrogate of parental quality for the discussion presented here. Riska *et al*. (1984) estimate the additive genetic variance of the maternal trait (G_{mm}) as 26, the environmental variance of this trait (E_{mm}) as 247 and the additive genetic covariance between the maternal and offspring traits as –20.1 (the genetic covariance can be calculated using their estimated genetic correlation). For this example, we assume that there are no maternal phenotypic effects as there is no causal analysis presented by Riska *et al*. (1984) describing the relationship between these two traits because of maternal effects. This assumption will impact the calculated selection values, but does not alter the basic phenomena demonstrated by this example. We can now ask the question 'how does selection appear to act to change the distribution of parental quality (late growth) when we assign offspring fitness to the offspring vs. when we assign offspring fitness to the mother?'. This question about the direct of selection could be rephrased from the view of an optimality problem to ask 'does selection appear to favour increased or decreased parental quality in this system'. To answer these questions we can calculate the selection differential (s_m) acting on the maternal trait (late growth) under assignment of offspring fitness to the offspring or to the mother using eqns 5 and 10. We get a selection differential of –14.2 when assigning offspring fitness to the offspring but a much larger and positive selection differential 263 when we assign offspring fitness to the mother. Clearly, we get very different pictures of how selection acts on late growth (i.e. parental quality) under the two assignments of fitness and thus we can get very different answers to our questions. Whereas this example may be somewhat artificial, it is based on genetic data from a real system and it demonstrates the possible problems that can occur when assigning offspring fitness to parents. Also note that the data from Riska *et al*. (1984) demonstrate that genetic correlations between parental and offspring size and growth related characters in mice are the rule, not the exception. Thus, we can expect to encounter these kinds of problems in the assignment of fitness.

Perhaps as significant as the conditions under which the assignment of offspring fitness to the mother fails are the conditions under which this assignment is not problematic. Whenever there is a component of offspring fitness that is *entirely* determined by a genetic characteristic of the mother (i.e. there is no genetic correlation with direct effects), then assigning offspring fitness to the mother is not problematic with respect to understanding evolutionary dynamics (see Table 1). This confirms Grafen's (1988) contention that the critical concept is 'independence of control', i.e. causality, where one must consider only fitness components that are controlled in whole by either the mother or the offspring (see also Cheverud & Moore, 1994). This suggests that empirical and theoretical analyses must be much more explicit

about this assumption. This also lends support to the approach of optimality modelling, wherein analyses generally assume that the trait being examined is completely under genetic control and is independent of any direct effects on fitness. However, it is also important to keep in mind that this is a theoretical assumption and that, in real empirical systems, this assumption may rarely hold. Roff (1997) presents a list of systems for which genetic correlations between direct and maternal effects have been measured, and he shows that, as in mice, nonindependence is the rule, not the exception. Thus, although the assumption of no genetic correlation between direct and maternal effects may be warranted in theoretical analyses that wish to understand conditions under which selection favours particular maternal characters, it seems problematic in most natural systems. It will be especially problematic in studies involving birds and mammals, where genetically correlated maternal and direct effects appear to be common (Cheverud & Moore, 1994; Roff, 1997; see also Mousseau & Fox, 1998).

Our analysis leads to the difficult conclusion that, in an empirical system, there are potential shortcomings to both methods of fitness assignment. It is clear from eqn 2 that the ideal situation would be one in which it is possible to identify all causal components of fitness *a priori* and thus, correctly weight all components. Because each approach has shortcomings, one must decide on an assignment of fitness carefully and justify the assignment of fitness chosen. Whenever there are biologically compelling reasons to believe that components of offspring viability fitness are controlled wholly or largely by the mother, then it may be beneficial in terms of logistic considerations to assign these components to the mother. Discounting very small direct effects may not substantially alter the predictive value of an analysis. However, because there is the potential for a very large underestimation of direct selection, one must proceed with caution when assigning a component of offspring fitness to the mother. For example, one might have compelling reasons to believe that size and survival at hatching in some birds are overwhelmingly determined by maternally controlled egg size. In addition, it is plausible that there is a direct effect as an offspring's genotype may influence its ability to develop within a given sized egg, or may influence its ability to break out of an egg of a particular size. However, as long as these latter offspring characters are not genetically correlated to the maternal genetic effects on egg size, then offspring size and survival at hatching may be assigned to the mother when considering evolution of egg size. (An analogous argument could be made using the example of seed size in plants.) Thus, we suggest that individuals analysing components of selection in natural populations consider the possibility of genetic correlations between parental and offspring characters, and when absent, cautiously consider certain components of offspring fitness as parental fitness in order to simplify analysis of selection in natural populations.

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Appendix

Maternal inheritance of the offspring trait

Maternal traits (especially maternal quality) often affect the expression of offspring characters (recently reviewed in Mousseau & Fox, 1998). This results in a situation where the relationship between the juvenile character and the maternal character becomes more complex than the simple case of Mendelian inheritance used above. To analyse how maternal effects alter the consequences of our alternative partitionings of fitness, we examine a simple model for maternal inheritance (after Kirkpatrick & Lande, 1989).

We assume that the offspring trait is influenced by direct additive genetic effects, environmental effects and by the phenotype of its mother:

$$z_{o(t)} = a_{o(t)} + e_{o(t)} + mz_{m(t-1)} \quad \text{(A1)}$$

where $a_{o(t)}$ is the additive genetic value, $e_{o(t)}$ is the environmental value and the coefficient m is a measure of the degree to which the phenotype of an individual's mother affects the expression of the individual's juvenile character (see Kirkpatrick & Lande, 1989). For simplicity we assume that the maternal character is itself not influenced by the phenotype of the mother such that the phenotypic value of the maternal trait is just the sum of the additive genetic component and the environmental component. We make this assumption because the maternal character is expressed in adulthood, where the likelihood that the expression of the trait would be influenced by the individual's mother is diminished. Violations of this assumption can be analysed using the equations presented in Kirkpatrick & Lande (1989).

Under this alternative model for inheritance we can derive new expressions for the covariance in eqns 5 and 10. Under the maternal inheritance model the covariance terms used in the selection equations take on the values (see Kirkpatrick & Lande, 1989 for a derivation of these equations):

$$\mathrm{cov}(z_{\mathrm{m}(t)}, z_{\mathrm{m}(t-1)}) = 1/2G_{\mathrm{mm}} \quad \text{(A2)}$$

$$\mathrm{cov}(z_{\mathrm{o}(t)}, z_{\mathrm{m}(t-1)}) = 1/2G_{\mathrm{om}} + mP_{\mathrm{mm}} \quad \text{(A3)}$$

$$P_{\mathrm{om}} = G_{\mathrm{om}} + (m/2)G_{\mathrm{mm}} \quad \text{(A4)}$$

$$P_{\mathrm{oo}} = G_{\mathrm{oo}} + mG_{\mathrm{om}} + E_{\mathrm{oo}} + m^2P_{\mathrm{mm}} \quad \text{(A5)}$$

$$P_{\mathrm{mm}} = G_{\mathrm{mm}} + E_{\mathrm{mm}}. \quad \text{(A6)}$$

Using these altered covariances we can re-write eqn 5 that predict selection when lifetime fitness is measured for the offspring and assigned to the offspring:

$$S_{\mathrm{m}} = 1/2b_{\mathrm{m}}G_{\mathrm{mm}} + b_{\mathrm{o}}[G_{\mathrm{om}} + (m/2)G_{\mathrm{mm}}] + 1/2b_{\mathrm{d}}P_{\mathrm{mm}}. \quad \text{(A7)}$$

Re-writing eqn 10 that describes the outcome of assigning offspring fitness to the mother under the assumption of maternal inheritance:

$$S_{\mathrm{m}} = 1/2[b_{\mathrm{m}}P_{\mathrm{mm}} + b_{\mathrm{o}}(1/2G_{\mathrm{om}} + mP_{\mathrm{mm}}) + b_{\mathrm{d}}P_{\mathrm{mm}}]. \quad \text{(A8)}$$

Thus we see that the two approaches are equivalent under the same circumstances as under Mendelian inheritance. In addition, we see that, when there are direct effects, we under-estimate the contribution of direct selection by the same factor of 4.

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