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MATERNAL EFFECTS AND THE POTENTIAL FOR EVOLUTION IN A NATURAL POPULATION OF ANIMALS

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Abstract.—Maternal effects are widespread and can have dramatic influences on evolutionary dynamics, but their genetic basis has been measured rarely in natural populations. We used cross-fostering techniques and a long-term study of a natural population of red squirrels, *Tamiasciurus hudsonicus*, to estimate both direct (heritability) and indirect (maternal) influences on the potential for evolution. Juvenile growth in both body mass and size had significant amounts of genetic variation (mass $h^2 = 0.10$; size $h^2 = 0.33$), but experienced large, heritable maternal effects. Growth in body mass also had a large positive covariance between direct and maternal genetic effects. The consideration of these indirect genetic effects revealed a greater than three-fold increase in the potential for evolution of growth in body mass ($h_t^2 = 0.36$) relative to that predicted by heritability alone. Simple heritabilities, therefore, may severely underestimate or overestimate the potential for evolution in natural populations of animals.

Key words.—Direct-maternal genetic covariance, heritability, indirect genetic effects, maternal effects, nestling growth rate, *Tamiasciurus hudsonicus*.

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Evolution by natural selection pervades all aspects of biology, but an evolutionary response to selection can occur only if the trait under selection has a genetic basis. Most studies of natural populations have estimated only direct genetic effects (heritability; Weigensberg and Roff 1996; Hoffmann 2000), but recent theoretical and laboratory work suggests that heritable maternal effects can have important indirect influences on the potential for evolution (Wolf et al. 1998). Maternal effects arise when the phenotype of a mother or the environment she experiences has a phenotypic effect on her offspring (Mousseau and Fox 1998). When maternal effects exist, a response to selection depends not only on direct genetic effects, but also on indirect genetic effects (heritable maternal effects), which can result in accelerated, dampened, or even nonintuitive responses to selection (Wolf et al. 1998). Dickerson (1947) expanded the simple definition of heritability ($h^2 = V_{AO}/V_{PO}$), to include not just direct genetic effects (V_{AO}), but also indirect genetic effects (V_{AM}) and the direct-indirect genetic covariance ($Cov[A_O, A_M]$) as a proportion of the total phenotypic variation (V_{PO}) in the offspring trait (h_t^2 represents total heritability):

$$h_t^2 = [V_{AO} + 1/2V_{AM} + 3/2\ \mathrm{Cov}(A_O, A_M)]/V_{PO}. \quad (1)$$

Large maternal effects have been found in many natural populations of animals (Mousseau and Fox 1998). In collared flycatchers (*Ficedula albicollis*), over 25% of variation in clutch size was attributed to maternal effects (Schluter and Gustafsson 1993; Price 1998; but see Merilä et al. 2001), and significant maternal effects have also been reported for a range of life-history traits in red deer (*Cervus elaphus*) including total fitness (Kruuk et al. 2000). Our ability to quantify the genetic basis of maternal effects (i.e., indirect genetic effects) and the direct-indirect genetic covariance, however, has been limited to captive animals (Roff 1997; Thiede 1998; Shaw and Byers 1998). In plants, controlled breeding designs under combined greenhouse and field conditions suggest that indirect genetic effects can either accelerate or constrain the potential response to selection (Byers et al. 1997; Thiede 1998). The influence of indirect genetic effects on the potential for evolution in a natural population of animals is not known.

In this study we measured the potential for evolution of nestling growth rates in a natural population of North American red squirrels (*Tamiasciurus hudsonicus*). In this model system we had both the unique ability to cross-foster newborn squirrels and 11 previous years of data that together allowed us to estimate both direct and indirect genetic contributions to the potential for evolution in a natural environment. Studies of natural populations are particularly important because it is within these variable environments that natural selection shapes both the direct and indirect sources of genetic variation and, consequently, the potential for future evolutionary change.

MATERIALS AND METHODS

Experimental Design

A natural population of red squirrels was monitored from March through August 1999 near Kluane National Park in the southwestern Yukon, Canada (Berteaux and Boutin

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2000). Litters of squirrels were paired as closely as possible based on parturition dates. Offspring from 33 pairs of litters were reciprocally cross-fostered so that roughly half the offspring of one litter was exchanged with an equal number of offspring from the paired litter. Growth of nestling red squirrels was monitored from the time of cross-fostering to approximately 25 days of age, which was just prior to first emergence from the natal nest. Body mass was measured (± 0.1 g) using portable electronic balances and the width of the zygomatic arch (± 0.1 mm) was used as an index of overall body size (Becker 1992). Nonlinear growth trajectories were assessed using differential equations involving specific growth rates (Kaufmann 1981).

Sources of Variation

Sources of variation in growth in body mass and growth in body size were estimated using two separate ANOVAs. In each analysis, total phenotypic variation within dyads was partitioned using a two-way nested ANOVA in which litter of origin, litter of rearing, and their interaction were all nested within dyads (Riska et al. 1985). Variance due to the nest of origin (s^2_{origin}), rearing ($s^2_{rearing}$), and origin $\times$ rearing interaction ($s^2_{origin \times rearing}$) represented genetic, maternal, and genotype $\times$ environment interaction effects, respectively. Variance components were estimated using restricted maximum likelihood (REML) in the VARCOMP procedure in S-Plus (Mathsoft 1999a,b). The direct-indirect genetic covariance ($Cov[A_O, A_M]$) was estimated from two separate analyses as the difference between the among-litter variance of offspring who remained in their natal nest and the among-litter variance of cross-fostered offspring (Riska et al. 1985).

Maternal Effects

The cross-fostering design did not allow us to determine the genetic basis to maternal effects (V_{AM}) directly, but the composite term maternal performance is simply the combined effect of several maternal characteristics for which we may derive heritabilities individually (Wolf et al. 1998). We used multiple regression analysis to determine which maternal characteristics were correlated with maternal performance for offspring growth. Because offspring were distributed roughly equally between mothers within a dyad, differences between these mothers in the average growth rate of all nursed offspring (genetic and foster) represented differences in maternal performance independent of direct genetic effects. As a result, differences in maternal characteristics within dyads were correlated with differences in maternal performance (sample size: $n = 26$ complete dyads). Maternal characteristics included in the multiple regression were: litter size, parturition date, age, reproductive experience, body size, maternal mass at parturition and weaning, territory size, and the azimuth of the natal nest. Means for each characteristic were substituted for missing data, except for parturition date. Parturition dates could not be estimated accurately for six dyads, so these were deleted in a pairwise manner ($n = 20$ dyads).

Of these nine maternal characteristics, only litter size and parturition date were significantly correlated with maternal performance. The exclusive contributions of these two maternal characteristics to overall maternal performance were calculated using partial linear regression (Legendre and Legendre 1998). The contributions of litter size and parturition date to the heritability of maternal performance (h^2_m) were calculated as the product of the proportion of maternal performance explained by that trait (R^2_t) and its heritability (h^2_t). The heritability of maternal performance then was calculated as the sum of these products for litter size and parturition date ($h^2_m = R^2_{litter\ size} h^2_{litter\ size} + R^2_{parturition\ date} h^2_{parturition\ date}$). The product of variation in nestling growth due to maternal performance ($s^2_{rearing}$) and the heritability of maternal performance was used as an estimate of indirect genetic effects (V_{AM}).

Heritability

The heritability of growth in body mass and body size were calculated from the cross-fostering experiment as the proportion of total phenotypic variation within dyads that was due to additive genetic variation. Heritability estimates for litter size and parturition date were calculated from existing long-term data for this population (1988–1998; Berteaux and Boutin 2000) using derivative free restricted maximum likelihood techniques (DFREML 3.0; Meyer 1989; Réale et al. 1999), which used all available kin relationships within our mother-daughter pedigree (number of records per trait = 568, animals = 284, base animals = 68, records of dams with progeny = 132, records of grand dams with progeny = 55). Total heritabilities were calculated using equation (1).

Environmental Effects

Estimates of $Cov(A_O, A_M)$ derived from cross-fostering experiments are potentially confounded by persistent maternal environmental effects (bVE_M, Lynch and Walsh 1998; or m_m, Riska et al. 1985) or direct-maternal environmental covariances ($Cov[E_O, E_M]$). A direct-maternal environmental covariance will arise, for example, if environmental conditions early in life (e.g., food abundance) affect both a female's growth rate and her subsequent maternal performance for offspring growth (e.g., litter size). Persistent maternal environmental effects will occur if environmental conditions affect not only a mother's performance, but also the maternal performance of her daughter. Measures of maternal characteristics for the same mother in multiple years allowed the addition of a persistent environmental effect term in the DFREML model described above (Meyer 1989). We estimated the magnitude of $Cov(E_O, E_M)$ and $Cov(D_O, D_M)$ for growth in body mass by performing both a mother-offspring ($n = 604$) and a maternal grandmother–grandoffspring regression ($n = 195$) for all available combinations in our dataset (1990–1999). The difference between the mother-offspring covariance and twice the maternal grandmother–grandoffspring covariance estimates $Cov(E_O, E_M) + Cov(D_O, D_M) - bVE_M$ (Lynch and Walsh 1998).

Paternity and Dominance Variance

Levels of multiple paternity within litters are not known in red squirrels, and dominance variance cannot be adequately estimated with our experimental design. We initially assumed that offspring within litters were half-siblings ($V_{AO} =$

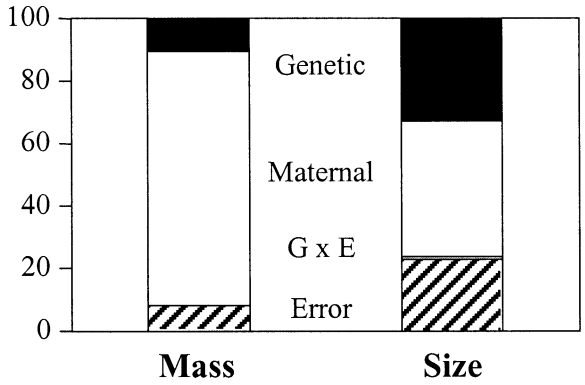

FIG. 1. Sources of variation as percentages of phenotypic variation in growth in body mass and body size in cross-fostered red squirrels. The black, white, gray, and hatched areas within each bar represent the percentage of total phenotypic variation within dyads that was due to genetic (V_{AO} assuming single paternity), maternal (V_M), interaction (genotype × environment, G × E), and error variances, respectively. Total phenotypic variation in growth of body mass and body size were 0.151 (g/day)^2 and 5.32 × 10^{-6} (day)$^{-2}$ respectively.

$2s^2_{origin}$), and that dominance variance was negligible, but later relaxed these assumptions and examined a range of possible values of relatedness and dominance variance. Variance components from the two-way nested ANOVA (s^2_{origin} and s^2_{error}) were used to calculate the minimum relatedness (r) and maximum levels of dominance variance (V_{DO}). In the ANOVA design $s^2_{origin} = rV_{AO} + 0.25V_{DO}$ and $s^2_{error} = (1 - r)V_{AO} + 0.75V_{DO} + e$ (Riska et al. 1985). We examined values of the average degree of relatedness within natal litters (r) ranging from single paternity (0.50) to complete multiple paternity (0.25), and values of V_{DO} from 0.00 to 0.05. Values of r and V_{DO} resulting in a negative value of e are not possible.

Values are presented throughout the text as means ± one standard error (SE). SEs for population parameters (e.g., V_{AO}, $Cov[A_O, A_M]$, h^2_t) were calculated by jackknifing at the level of the dyad.

RESULTS

Growth in nestling body mass averaged 1.91 g/day and appeared linear over this time period. There was, however, a positive correlation between linear growth rate (g/day) and the first mass measurement ($r^2 = 0.03$, $n = 177$, $P = 0.02$), so the residuals of this relationship were used for subsequent analyses (mean = −0.00073 g/day). The width of the zygomatic arch grew by an average of 0.30 mm/day but followed a Gompertz curve, where specific growth rates decreased with the geometric mean of the initial and final body size measurements (ln transformed). As a result, adjusted specific growth rates were used for subsequent analyses (mean = 0.017 day^{-1}). Growth in body mass and body size did not differ between male and female offspring (mass: $t_{175} = -0.89$, $P = 0.38$; size: $t_{175} = 0.52$, $P = 0.60$), so both sexes were examined together.

There were strong genetic and maternal effects on both growth in body mass (origin: $F_{33,26} = 2.08$, $P = 0.007$; rearing: $F_{27,26} = 16.74$, $P < 0.0001$), and body size (origin: $F_{33,26} = 2.06$, $P = 0.008$; rearing: $F_{27,26} = 4.25$, $P < 0.0001$), but no indication of genotype × environment interactions (mass: $F_{26,58} = 0.82$, $P = 0.71$; size: $F_{26,58} = 1.03$, $P = 0.44$; see Fig. 1). The heritability of nestling growth, calculated as the proportion of total phenotypic variance within dyads (V_{PO}) that was due to direct additive genetic variance (V_{AO}), was low for body mass (0.10 ± 0.001) and moderate for body size (0.33 ± 0.005; see Table 1).

The cross-fostering design revealed that 81% of the total phenotypic variation in growth in body mass was due to maternal performance (see Fig. 1), which is the sum of both genetic (V_{AM}) and environmental (V_{EM}) maternal effects. Both litter size at birth and parturition date were related negatively to maternal performance; females who gave birth to fewer offspring earlier in the season raised faster-growing offspring. These two maternal characters together explained 69% of the variation in maternal performance (paired multiple regression; $R^2 = 0.69$, $n = 20$, $P < 0.0001$). The heritabilities of litter size and parturition date, as calculated by the DFREML model, were 0.12 ± 0.04 and 0.29 ± 0.05, respectively. If we assume that the remaining 30% of unexplained variation in maternal performance has no genetic basis, we can estimate the heritability of maternal performance (h^2_m) conservatively as 0.13 (see Table 2). The cross-fostering design also revealed a large positive $Cov(A_O, A_M)$ for growth in body mass (0.02 ± 0.001), suggesting that

TABLE 1. The potential for evolution and sources of variation in growth in body mass and growth in body size of nestling red squirrels (±SE) based on direct genetic effects alone (h^2) and including maternal genetic effects (h_t^2). Sources of variation for growth in body mass and size are reported as (g/day)^2 and × 10^{-6} (day^{-2}), respectively. V_{PO}, V_{AO}, V_M, and Cov (A_0, A_M) were estimated from the cross-fostering experiment. V_{PO} represents the total phenotypic variance within dyads. V_{AM} was calculated as the product of the phenotypic variance due to maternal performance from the cross-fostering experiment (V_M) and the heritability of maternal performance (h_m^2; see Table 2). h_t^2 was calculated following Dickerson (1947). Standard errors were generated by jackknifing at the level of the dyad.

	Growth in body mass	Growth in body size
Total phenotypic variance (V_{PO})	0.151 ± 0.001	5.32 ± 0.04
Direct effects		
Direct genetic variance (V_{AO})	0.016 ± 0.0001	1.77 ± 0.01
Heritability (h^2)	0.10 ± 0.001	0.33 ± 0.005
Maternal effects		
Maternal variance (V_M)	0.123 ± 0.001	2.30 ± 0.04
Maternal genetic variance (V_{AM})	0.016 ± 0.0001	0.24 ± 0.004
Direct-maternal genetic covariance (Cov [A_O, A_M])	0.020 ± 0.001	0.02 ± 0.06
Total heritability (h_t^2)	0.36 ± 0.01	0.36 ± 0.02

TABLE 2. Maternal characters as components of maternal performance for growth in body mass. The heritability of maternal performance (h_m^2) was calculated as the sum of maternal character heritabilities weighted by their relative contribution to maternal performance. The exclusive contribution of each maternal character (R_i^2) to overall maternal performance was derived from partial multiple regression. Heritabilities (h_i^2 were estimated from an 11-year pedigree for this population (1988–1998) using DFREML. Unexplained variation in maternal performance is assumed to have a heritability of zero.

Maternal characters (i)	R_i^2	h_i^2	$R_i^2\ h_i^2$
Litter size	0.43	0.12	0.05
Parturition date	0.26	0.29	0.08
Unexplained	0.30	0.00	0.00
Total	1.00		$h_m^2 = 0.13$

genes for fast offspring growth in body mass were associated positively with genes for maternal performance leading to fast offspring growth (smaller litter sizes and earlier parturition dates).

The total heritability (h_t^2) of growth in body mass including indirect effects was calculated as 0.36 ± 0.01. This value represents a greater than three-fold increase in the potential response to a given amount of selection relative to that predicted by direct genetic effects alone (0.10).

For growth in body size, 43% of the total phenotypic variation within dyads was due to maternal effects. Litter size and parturition date remained the best predictors of maternal performance and explained 60% of the variation in maternal performance for growth in body size ($R^2 = 0.60$, $n = 20$, $P < 0.001$), but the heritability of maternal performance for growth in body size dropped slightly to 0.10. In addition, the $Cov(A_O, A_M)$ of growth in body size was not significantly different from zero ($2.2 \times 10^{-8} \pm 5.9 \times 10^{-8}$). As a result, direct genetic effects alone (0.33) adequately estimated the total heritability of growth in body size (0.36 ± 0.02).

We examined two possible sources of bias in our $Cov(A_O, A_M)$ estimate for growth in body mass: persistent maternal environmental effects and direct-maternal environmental and dominance covariances. The DFREML estimates for persistent environmental effects on litter size and parturition date were both small (as percentages of total variance: litter size 0.02 ± 0.02%, parturition date 0.00%) and the DFREML models including persistent environmental effects were not distinguishable from the previous models excluding these environmental effects (log-likelihood ratio test: litter size $G_1 = 0.000004$, $P = 0.99$; parturition date $G_1 = 0.0014$, $P = 0.97$). The difference between the mother-offspring covariance (0.033) and twice the maternal grandmother–grandoffspring covariance (0.083) was substantially negative (−0.05), suggesting that combined effects of $Cov(E_O, E_M)$ and $Cov(D_O, D_M)$ were negative. Persistent environmental effects or environmental covariances, therefore, did not inflate our estimate of the direct-maternal genetic covariance.

The effective partitioning of variance in growth in body mass allowed us to estimate maximum levels of paternity and dominance variance. Values of $r < 0.29$ or $V_{DO} > 0.024$ resulted in negative values of e and are, therefore, not possible.

DISCUSSION

In this study we quantified the contribution of both direct and indirect genetic effects to the potential for evolution in a natural population of animals. The heritability of nestling growth was low for body mass (0.10 ± 0.001) and moderate for body size (0.33 ± 0.005), but both estimates were similar to previously reported values for life-history traits (Mousseau and Roff 1987). Growth in body size, and particularly growth in body mass, were subject to large maternal effects, which accounted for more than 80% of the total phenotypic variation in growth in body mass. These large maternal effects were correlated with litter size and parturition date, which were themselves heritable ($h^2 = 0.13$ and 0.29, respectively). The combination of these two maternal traits resulted in a heritability of maternal performance (0.13), which is lower than most previous estimates of h_m^2 from laboratory animals (see Cheverud 1984), but provides evidence for a potential indirect contribution of maternal performance to the evolution of offspring traits in a natural population of animals. The consideration of both direct and indirect genetic effects revealed a greater than three-fold increase in the potential for evolution of growth in body mass relative to that predicted by direct genetic effects alone. As with previous heritability estimates for single traits, this represents the potential response to selection, which may not be realized if there is opposing selection on either genetically correlated offspring traits or maternal traits such as litter size and parturition date.

The large increase in the potential for evolution of body mass was the result of both a heritable basis to maternal performance and a large positive $Cov(A_O, A_M)$ (13% of V_{PO}) that was greater than most previous estimates for mice in the laboratory (Moore et al. 1970; Cheverud 1984; Riska et al. 1985). This covariance corresponds to a direct-maternal genetic correlation that is greater than one (1.25 ± 0.08) under the assumptions of single paternity and negligible dominance variance, but which is less than one for several possible values of these variables. We have no evidence to suggest that our estimate of $Cov(A_O, A_M)$ was inflated by either persistent maternal effects or maternal-offspring environmental covariances. Prenatal maternal effects lasting to 25 days of age could have confounded our estimate of additive genetic variation and $Cov(A_O, A_M)$, if prenatal and postnatal maternal effects were correlated positively. Prenatal maternal effects on postnatal growth rates in laboratory rodents are often thought to be relatively small (Riska et al. 1984; Roff 1997; but see Desai and Hales 1997; Rhees et al. 1999) and independent of postnatal maternal effects (Rhees et al. 1999), but their influence in natural populations is not known. Our analysis of the residuals of the relationship between growth in body mass and the initial weight measurement should have controlled for genetic and maternal effects acting prior to cross-fostering.

Estimates of V_{AO} depend on the degree of relatedness among siblings and in cross-fostering experiments the litter of origin term includes one-quarter of the dominance variance (V_{DO}; Riska et al. 1985). The effective partitioning of phenotypic variation by the cross-fostering design, however, allowed us to infer minimum levels of relatedness among siblings and the maximum amount of V_{DO} for growth in body

mass in this population of squirrels. The small amount of error variance in growth in body mass (see Fig. 1) allowed us to estimate the minimum relatedness among siblings as 0.29, which suggests that complete multiple paternity within litters is not common in this population. This contrasts with behavioral observations of female red squirrels mating with many males (range = 4–16, mean = 7.4, $n = 16$; H. Currie and S. Boutin, unpubl. data). The maximum level of dominance variance in growth in body mass was estimated as 0.024, or 16% of the total phenotypic variation. Our estimates of V_{AO}, therefore, were not biased substantially by V_{DO}, and dominance variance does not appear to be a likely mechanism by which genetic variation in growth in body mass of red squirrels can be maintained in the presence of strong selection (Crnokrak and Roff 1995).

A common extension of Fisher's (1930) fundamental theorem of natural selection suggests that traits more closely associated with fitness will have lower heritabilities than traits more distantly related to fitness (Mousseau and Roff 1987; Kruuk et al. 2000; Merilä and Sheldon 2000) given similar genetic architecture (Merilä and Sheldon 1999). The associations between fitness and growth in body size and growth in body mass in red squirrels are not known, but the consideration of indirect genetic effects eliminated what originally appeared to be a very large difference between the simple heritabilities (h^2) of these two traits. These data suggest that the relationship between the heritabilities of traits and their association with fitness can depend on whether indirect genetic effects are considered.

Positive covariances between direct and indirect genetic effects may arise from selection for offspring phenotypes that correspond to the environment that their mother can provide (Wade 1998). Positive correlations between traits subject to the same directional selection, however, are thought to be fixed rapidly, so negative correlations should predominate (Roff 1997). In fact, most previous estimates of $Cov(A_O, A_M)$ from captive animals raised in controlled environments have been negative (Cheverud 1984; Roff 1997). The large positive $Cov(A_O, A_M)$ and the small amount of dominance variation reported here for growth in body mass suggest that this trait may be particularly susceptible to allelic fixation under consistent directional selection. The occurrence of reasonable amounts of direct and indirect additive genetic variation suggest that selection on growth in body mass may instead vary spatially or temporally in this population of red squirrels. Periodic fluctuations in spruce cone production (Humphries and Boutin 2000) and the strong philopatry of red squirrels (Larsen and Boutin 1994) provide an ecological framework within which a positive $Cov(A_O, A_M)$ could be maintained through temporal and spatial variation in food resources.

The ability to predict evolutionary responses to selection and to infer previous levels of selection depends on the quantification of all sources of additive genetic variation. Our results clearly demonstrate the importance of indirect genetic effects on potential evolutionary trajectories in a natural population of animals. Furthermore, positive relationships between direct and indirect sources of genetic variation, which are maintained in inherently variable natural systems, may not be represented adequately by estimates derived from captive animals under controlled conditions.

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