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James M. Cheverud

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EVOLUTION BY KIN SELECTION: A QUANTITATIVE GENETIC MODEL ILLUSTRATED BY MATERNAL PERFORMANCE IN MICE

JAMES M. CHEVERUD

Departments of Anthropology, Cell Biology & Anatomy, and Ecology & Evolutionary Biology, Northwestern University, Evanston, Illinois 60201

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Models of evolution by kin selection have been developed primarily at the single locus level (Hamilton, 1964; Wade, 1978, 1980; Michod and Abugov, 1980; D. S. Wilson, 1980), even though most characters of evolutionary interest are polygenic. Recently, several quantitative genetic models have been derived for the evolution of altruism by kin or group selection (Yokoyama and Felsenstein, 1978; Aoki, 1982*a*, 1982*b*; Crow and Aoki, 1982; Engels, 1983). All of these models, both single locus and polygenic, have essentially confirmed the reliability of Hamilton's rule, that altruism will evolve when the additive genetic regression of the recipient on the altruist is greater than the ratio of selective costs to benefits.

Alternatively, the evolution of altruism through kin selection can be approached as a special case of the joint evolution of correlated characters. In such a quantitative genetic model, there are two traits, the target phenotype and kin performance for this phenotype. The target phenotype can be any trait which is affected by interaction with kin. In most previous models, the target phenotype has been fitness, but in the model presented here this is generalized so that it can be any trait of interest to the researcher, such as weight. Kin performance subsumes all aspects of an individual's phenotype which have an effect on the target phenotype in its relative. Thus kin performance is the source of kin effects on the target phenotype. These kin effects are the measured phenotypic effects of kin performance on the target phenotype. Kin performance is, by definition, only measurable in terms of its effects on the target phenotype and therefore only exists in

reference to a particular aspect of a relative's phenotype. Kin performance can be considered altruistic under certain selection regimes.

This view of kin selection as a special case of the joint evolution of correlated characters highlights previously unrecognized or underrated features of the models cited above. First, the evolution of altruism through kin selection is a special case of a more general model of the evolution of traits which are affected by and affect the phenotypes of relatives. This has been especially appreciated by those using "trait group" models (Wade, 1978, 1980; Yokoyama and Felsenstein, 1978; D. S. Wilson, 1980; Crow and Aoki, 1982). Second, the only genetic effects on the target phenotype which have been considered are kin effects, which are the phenotypic manifestations of kin performance. The effects of genes carried by the target on its own phenotype, usually referred to as the direct effects of genes on the target phenotype, have not been included in the models. This is because the models have concentrated on the evolution of the potentially altruistic kin performance trait to the exclusion of the evolution of the target phenotype itself. This second feature of the previous models has also led to an unstated assumption that there is no net pleiotropic effect of genes directly affecting the target phenotype and kin performance. In the model presented below, direct effects of genes on the target phenotype and pleiotropic effects on the target phenotype and kin performance will be taken into account.

The influence of kin performance on polygenic phenotypes has long been a

subject of both theoretical and practical interest in agricultural genetics, especially the interaction of mother with offspring (Dickerson, 1947; Willham, 1963, 1972). While quantitative genetic models of sib-sib interactions and interactions among other kinds of relatives can be developed, as discussed below, mother-offspring interactions and the consequent maternal (kin) effects on offspring (target) phenotypes are particularly well-known and therefore provide a potentially fruitful area for theoretical, experimental, and field studies of kin selection. Therefore, I will concentrate on a model for the joint evolution of offspring phenotypes and maternal performance as it affects these phenotypes. In analyzing mother-offspring interaction, the offspring's phenotype, such as weight, is the target phenotype and maternal performance for this phenotype is kin performance.

Maternal effects are the measured phenotypic effects of maternal performance on offspring phenotypes (Legates, 1972; Willham, 1972). They are known to occur throughout the animal kingdom from insects (Bondari et al., 1978) to fish (Reznick, 1981, 1982), reptiles (Bull, 1980), and mammals (Bradford, 1972; Cundiff, 1972; Legates, 1972; Robison, 1972). Mechanisms by which maternal performance can affect offspring phenotypes include choice of nest site, contributions to egg nutrients, milk production, preparation and form of the nest, protection from predators, etc. Maternal traits such as these are highly correlated with maternal performance for specific offspring phenotypes. However, outside of agricultural genetics, the role of maternal effects in the evolution of offspring phenotypes has generally not been considered. These effects are properly considered to be environmental with respect to the offspring (Willham, 1972) and "are a frequent and often troublesome source of environmental resemblance (among relatives)" (Falconer, 1981 p. 145). This view of maternal effects has resulted in attempts to remove them from estimates of heritability and response to

selection because they are environmental with respect to the offspring and heritabilities should only include genetic effects. However, it is recognized that maternal performance itself and its resulting maternal effect on the offspring may have a genetic basis (Dickerson, 1947; Willham, 1972; Falconer, 1981), and therefore may evolve through kin selection.

Model for the Evolution of Characters Influenced by Maternal Performance

A causal path analysis model for any target phenotype P , influenced by maternal performance M , is given in Figure 1. This model, which was first presented by Dickerson (1947) and subsequently elaborated on by Willham (1963, 1972), produces several unexpected results: (1) selection for increased value of an offspring phenotype may result in an expected evolutionary *decrease* in that phenotypic value; (2) selection for increased offspring phenotypic value may result in a *decrease* in maternal performance; (3) maternal performance may evolve towards more altruistic values even when selective costs exceed selective benefits; and (4) maternal performance may be prevented from evolving in an altruistic direction regardless of extensive benefits and minimal costs. None of these results is consistent with the kin selection models cited above because they have assumed a lack of pleiotropy and a lack of direct effects on the target phenotype. The model presented here will explain how these unexpected results come about.

In this model, the phenotype is broken down into several causal components as follows,

$$P = A_o + A'_m + E_o + E'_m, \quad (1)$$

where A signifies additive genetic value, E is an environmental deviation (Falconer, 1981), o refers to direct effects of genes or environment on the phenotype, m refers to genetic or environmental effects on the phenotype acting through maternal performance, or maternal effects, and "''" indicates a value in generation $t - 1$. Dominance deviations are

included in the environmental deviations.

The standardized variance of P as derived from the path analysis model in figure 1 is

$$1 = h_o^2 + m^2 h_m^2 + (\text{cov}(A_o, A_m)/s^2 p) + e_o^2 + m^2 e_m^2, \quad (2)$$

where h_o^2 is the proportion of variance accounted for by direct additive genetic effects, m^2 is the proportion of variance explained by maternal effects, h_m^2 is the proportion of variance in maternal performance accounted for by additive genetic effects, $s^2 p$ is the phenotypic variance, e_o^2 is the proportion of variance accounted for by direct environmental effects, and e_m^2 is the proportion of variance in maternal performance explained by environmental effects. The direct-maternal genetic covariance, $\text{cov}(A_o, A_m)$, is a measure of the net pleiotropic effects of genes which affect the offspring phenotype directly, since they are carried by the offspring, and also affect the offspring phenotype indirectly through their effect on maternal performance. So genes which affect maternal performance, which in turn affects the offspring phenotype, may also directly affect the offspring phenotype, leading to a non-zero direct-maternal genetic covariance for that offspring phenotype. Note that when the target is unrelated to the source of the "kin performance" phenotype, the path A'_o to A_o has a value of zero, and the genetic covariance term drops out of equation 2. The proportion of the total variance due to additive genetic effects is $(h_o^2 + m^2 h_m^2 + (\text{cov}(A_o, A_m)/s^2 p))$.

However, the response to selection on the offspring phenotype, P , does not directly depend on the percent additive genetic variance when a character is influenced by maternal effects. In general, the response to selection (R) is

$$R = b_{AP} s_P i_P, \quad (3)$$

where b_{AP} is the regression of an individual's additive genetic value, A , on its phenotype, P , s_p is the phenotypic standard deviation, and i_p is the selection inten-

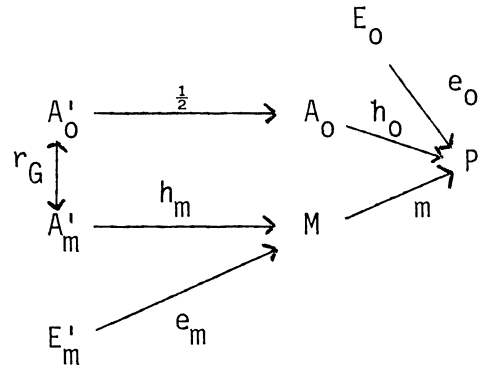


FIG. 1. Path model for an offspring phenotype impacted by maternal performance. P is the offspring phenotypic value; M is maternal performance; A represents an additive genetic value; E represents the environmental deviation; subscript o refers to direct effect; subscript m refers to maternal effects; path h is the square root of heritability; path e is the square root of the proportion due to environmental effects; path m is the maternal effect on the offspring phenotype, and r_G is the genetic correlation between direct effects and maternal effects. All other correlations are assumed to be zero and play no role in the expected response to selection.

sity. Usually b_{AP} equals the heritability (Falconer, 1981), but in this case, the genetic causal component of P is $A_o + A'_m$ (see equation 1) and the breeding value of an individual with phenotype P is $A_o + A'_m$. Therefore,

$$\begin{aligned} b_{AP} &= \text{cov}(A_o + A'_m, A_o + A_m)/s^2 p \\ &= (\text{cov}(A_o, A_o) + \text{cov}(A_o, A'_m) \\ &\quad + \text{cov}(A_m, A_o) + \text{cov}(A_m, A'_m))/s^2 p \\ &= h_o^2 + (1/2)m^2 h_m^2 + \\ &\quad (3/2)(\text{cov}(A_o, A_m)/s^2 p), \end{aligned} \quad (4)$$

(Dickerson, 1947; Willham, 1972; Hanrahan, 1976). The response of the offspring phenotype to selection is

$$R_p = (h_o^2 + (1/2)m^2 h_m^2 + (3/2)\text{cov}(A_o, A_m)/s^2 p) i_P s_P. \quad (5)$$

Note that b_{AP} is not the usual heritability and can even be negative when the direct-maternal covariance is a highly negative number, specifically when (h_o/h_m) is less than the absolute value of the direct-maternal genetic correlation (Hanrahan,

1976). Therefore, under certain specified conditions, selection for increased values of a phenotype may actually lead to an evolutionary decrease in that phenotype.

The overall response to selection (R_p) can be thought of as the sum of the correlated responses of the direct effects (CR_o) and maternal effects (CR_m) to selection as follows,

$$CR_m = \left(\frac{1}{2} m^2 h^2_m + (\text{cov}(A_o, A_m) / S^2_P) \right) i_p S_P \quad (6)$$

and

$$CR_o = \left(h^2_o + \frac{1}{2} (\text{cov}(A_o, A_m) / S^2_P) \right) i_p S_P \quad (7)$$

So the response of the offspring phenotype to selection can be partitioned into the separate responses of the maternal and direct effects.

In order to determine the evolutionary response to altruistic selection, it is necessary to also derive the direct response of maternal performance and the correlated response of the offspring phenotype to direct selection on maternal performance. The response of maternal performance to direct selection is

$$R_m = m h^2_m \left(\frac{1}{2} \right) i_m S_P, \quad (8)$$

where i_m is the selection intensity applied directly to maternal performance. The factor of $(1/2)$ is included because selection is only on females in this model. The correlated response of the offspring phenotype to this selection is

$$CR_p = m \left(h^2_m + (\text{cov}(A_o, A_m) / m^2 S^2_P) \right) \left(\frac{1}{2} \right) i_m S_P. \quad (9)$$

Maternal performance can be said to be under altruistic selection when direct selection on maternal performance (equation 8) is in the opposite direction from direct selection on the offspring phenotype (equation 5), or when i_m and i_p have opposite signs. Under this altruistic selection, maternal performance will evolve in an altruistic direction when $R_m + CR_m > 0$, given that i_p is positive and i_m is negative. Maternal performance evolves towards greater altruism when

$$m \left(\frac{1}{2} \right) + b_{Aom} > (|i_m| / 2 i_p), \quad (10)$$

where the $(1/2)$ in the left hand side of the equation represents the coefficient of relationship between mother and offspring, m is the partial correlation of maternal performance with the offspring phenotype, and b_{Aom} is the genetic regression coefficient of direct effects on maternal effects ($b_{Aom} = \text{cov}(A_o, A_m) / V_{Am}$). The ratio of selection intensities on the right hand side of equation 10 represents the cost ($i_m/2$)-benefit (i_p) ratio of Hamilton's rule. The factor of $(i_m/2)$ appears in the right hand side of the equation, rather than simply i_m , because direct selection on maternal performance only occurs in females, which constitute one-half of the breeding population. Note that when the correlation of the offspring phenotype and maternal performance (m) is 1.00 and the direct-maternal genetic covariance (b_{Aom}) is zero, as assumed in previous models, equation 10 reverts to the usual form of Hamilton's rule, that the coefficient of relationship must be greater than the cost-benefit ratio for maternal performance to evolve in an altruistic direction. The current formulation differs in that it allows for covariation between mother and offspring traits due to pleiotropy (b_{Aom}) in addition to the correlation due to relatedness.

Inspection of equation 10 shows that some unexpected results can be derived. While the offspring phenotype-maternal performance correlation, m , is restricted to the range zero to one by its measurement scale, the genetic regression between direct and maternal effects, b_{Aom} , can range from negative to positive infinity. If the regression is relatively large and positive, the left hand side of the equation can be greater than one, allowing evolution in an altruistic direction even when the selective costs ($|i_m|/2$), exceed the benefits, i_p . This occurs specifically whenever the regression exceeds $((2 - m)/2m)$. So a positive regression enhances the possibilities for evolution of altruism by kin selection. If the regression is a highly negative number, the left

hand side of equation 10 may be negative, in which case altruistic evolution is prevented, regardless of the cost-benefit ratio. This occurs when the absolute value of the negative direct-maternal genetic covariance is greater than one-half of maternal performance's additive genetic variance ($|\text{cov}(A_o, A_m)| > V_{Am}/2$). Altruistic evolution is prevented under these circumstances because the evolutionary response of maternal performance to selection for increased offspring phenotypic value (equation 6) is in a negative direction, which reinforces direct selection for decreased maternal performance rather than counteracting it.

The parameters which need to be estimated before the consequences of kin selection can be predicted include the direct effects heritability (h^2_o), the proportion of variance in the offspring phenotype due to maternal effects (m^2), the maternal performance heritability (h^2_m), the direct-maternal genetic covariance ($\text{cov}(A_o, A_m)$), the phenotypic variance of the offspring phenotype (s^2_p), and the direct selection intensities on the offspring phenotype (i_p) and maternal performance (i_m). The selection intensities can be measured by the covariance between the offspring phenotype or maternal performance and measures of survivorship and reproductive success. The maternal effects, heritabilities, and direct-maternal genetic covariance can be estimated through many different experimental designs using a variety of types of relatives and/or rearing designs (Willham, 1963, 1972). One method for estimating these parameters will be given in more detail below.

It remains to be seen whether the unexpected theoretical results discussed above have much practical biological importance. The biological importance of these results depends on typical empirically derived values for the parameters of importance in the model. Since quantitative genetic parameters refer to phenotypes rather than genotypes, they are more easily measured than parameters of single locus theory.

Experimental Evidence

Previous Empirical Results.—Most genetic research on maternal effects has been done on weight in the context of agricultural genetics, hence nearly all of the examples are taken from mammals and refer to body weight as the offspring phenotype. The values for all of the parameters given below may be different for other taxa and traits. There is overwhelming evidence that maternal effects play an important role in offspring phenotypes, even in adulthood (Young et al., 1966; El Oksh et al., 1967; Ahlschwede and Robison, 1971; Rutledge et al., 1972; Kuhlers et al., 1977; Herbert et al., 1979; Atchley and Rutledge, 1980; Cheverud et al., 1983). Maternal effects (m^2) in mammals typically decrease in importance with time since weaning from a high of about 50% at weaning to a low of about 10% in the adult. Therefore, maternal performance continues to have an effect on the offspring phenotype long after direct physical contact is broken. A reasonable value for the heritability of maternal performance (h^2_m), determined as the average of reported values, is about .40 (Hohenboken and Brinks, 1970; Ahlschwede and Robison, 1971; Hanrahan and Eisen, 1974; Hanrahan, 1976; Dillard et al., 1977; Kuhlers et al., 1977; Burfening et al., 1981). Not enough evidence is available to establish an ontogenetic trend for the heritability of maternal performance in mammals. Direct effects heritabilities are typically a bit lower than maternal effects heritabilities ($h^2_o \approx .30$) and are either constant or increase slightly with age (Cheverud et al., 1983).

The direct-maternal genetic covariance ($\text{cov}(A_o, A_m)$) is almost always negative (Dickerson, 1947; Ahlschwede and Robison, 1971; Hohenboken and Brinks, 1971; Vesey and Robison, 1972; Hanrahan and Eisen, 1973, 1974; Kuhlers et al., 1977; Bondari et al., 1978; Nagai et al., 1978; Burfening et al., 1981). Studies of ontogenetic change in standardized direct-maternal covariances are rare

(Ahlschwede and Robison, 1971; Kuhl-ers et al., 1977). Both studies used data from swine and found that this covariance decreased from about .30 at birth to a low of about $-.20$ soon after weaning and then increased to almost zero as the animals approached adulthood. Therefore, both studies showed a U-shaped pattern with age, the covariances being most negative at an intermediate age, just after weaning.

Although all of the authors cited above recognized the importance of these negative covariances in limiting the response to selection of an offspring phenotype, little attention has been paid to the possible causes of these values and their wider implications for evolutionary theory. In this paper I will present additional data bearing on ontogenetic trends in direct-maternal genetic covariances and relate this pattern to a possible cause for the trend. The implications of the model and the empirically derived parameter values for the theory of response to kin selection will also be considered.

EXPERIMENTAL MATERIALS AND METHODS

Males and females from a random-bred ICR strain of mice were randomly mated and longitudinal growth data collected on 60 four-member full-sib families (240 mice) (Cheverud et al., 1983) as part of a multivariate quantitative genetic study of growth and allometry. Litters obtained from two non-full-sib dams born on the same day were standardized at birth to eight pups, four males and four females, and a random half of each litter was exchanged between dams, forming a pair of cross-fostered litters. The offspring were weaned at 21 days and randomly assigned to single-sex cages containing four mice each. Four of the original eight pups, two males and two females, were used in this project. Five live body traits, weight (WT), head length (HL), trunk length (TRL), trunk circumference (TRC), and tail length (TL), will be analyzed here. The mice were measured weekly at 17, 24, 31, 45, 52, 59, and 66 days. A more

complete description and analysis of these data from the perspective of ontogenetic changes in variance components is contained in Cheverud et al. (1983).

The linear model used in this analysis is

$$Y_{ijkln} = \mu + S_i + P_j + d_{k(j)} + n_{l(j)} + dn_{kl(j)} + e_{nkl(j)}, \quad (11)$$

where Y_{ijkln} is the measurement on the n^{th} pup of the i^{th} sex, nursed by the l^{th} nurse, born of the k^{th} dam, nested in the j^{th} cross-fostered pair. Sex is included in the model to correct for its effects so all estimates are sex-corrected. Effects due to dam (d), nurse (n), dam by nurse interaction (dn), and the residual (e) were assumed to be random effects with zero means and variances V_d , V_n , V_{dn} , and V_R , respectively. Table 1 provides the expected mean squares for each factor identified in the model and used in the analysis. Separate analyses were also performed on those animals who were reared by an unrelated nurse and those who were reared by their own dam in order to calculate the direct-maternal genetic covariance from these separate between-group variances (see Table 2) (J. J. Rutledge and B. Riska, pers. comm.). Each of these between-group variances was based on 30 degrees of freedom.

A multivariate analysis of variance was performed on the 35 age-specific traits. However, only the univariate results will be presented here because only they are necessary for a simple illustration of the model. The traits measured here are correlated and therefore the results for any one trait are not independent of the results obtained for the others.

Each of the various components, those due to dams (V_d), nurses (V_n), residual plus dam \times nurse interaction (V_R), between-group variance due to pups reared by their own dam (V_{od}), and between-group variance due to pups reared by a foster dam (V_{fd}), is presented in terms of causal components in Table 2. The direct effects heritability will be estimated by

$$h^2_o = 2V_d/V_p, \quad (12)$$

TABLE 1. Sources of variation and expected mean squares for analysis of variance. V_d is the dam variance, V_n is the nurse variance, V_{dn} is the dam by nurse interaction variance, and V_R is the residual variance.

Source	Expected mean square
Dams within pairs	$V_R + 2V_{dn} + 4V_d$
Nurses within pairs	$V_R + 2V_{dn} + 4V_n$
Dams \times nurses within pairs	$V_R + 2V_{dn}$
Residual	V_R

the maternal effects by

$$m^2 = V_n/V_P, \quad (13)$$

the direct-maternal genetic covariance by

$$(\text{cov}(A_o, A_m)/V_P) = (V_{od} - V_{fd})/V_P \quad (14)$$

(J. J. Rutledge and B. Riska, pers. comm.), and the direct environmental effects (e^2_o) by

$$e^2_o = 1 - h^2_o - m^2 - (\text{cov}(A_o, A_m)/V_P). \quad (15)$$

Prenatal maternal effects are included in the estimate of direct effects heritability. However, these effects appear to be unimportant for weight by two weeks of age, probably due to compensatory growth (Monteiro and Falconer, 1966; Rutledge et al., 1972; Riska et al., 1984).

This cross-fostering design allows the estimate of many of the parameters in the kin selection model, including direct effects heritability (h^2_o), the proportion of variance in the offspring phenotype due to maternal effects (m^2), the direct-maternal genetic covariance ($\text{cov}(A_o, A_m)$), and the phenotypic variance (s^2_P). The maternal performance heritability (h^2_m) cannot be estimated with the data at hand. However, if the relatedness of the dams used in this breeding experiment was known, maternal effects heritabilities could have been estimated by dividing the nurse variance, V_n , into the components within and between sisters. For the purposes of illustration, a maternal heritability of .40 will be used here. This

TABLE 2. Coefficients describing the observational variance components in terms of causal variance components. The observational components are V_d , the dam variance, V_n , the nurse variance, V_{dn} , the dam by nurse interaction variance, V_R , the residual variance, V_{od} , the variance between groups of pups reared by their own dam, and V_{fd} , the variance between groups of pups reared by a foster dam. The causal components are V_{Ao} , the direct additive genetic variance, V_{Do} , the direct dominance variance, V_m , the maternal effects variance, V_E , the environmental variance, and $\text{cov}(A_o, A_m)$, the direct-maternal genetic covariance.

Variance	V_{Ao}	V_{Do}	V_m	V_E	$\text{cov}(A_o, A_m)$
V_d	1/2	1/4	0	0	0
V_n	0	0	1	0	0
$V_R + V_{dn}$	1/2	3/4	0	1	0
V_{od}	1/4	3/8	0	1/2	1
V_{fd}	1/4	3/8	0	1/2	0

value seems reasonable given values reported in the literature (see above). Selection intensities were not estimated in the current breeding experiment, because no selection was imposed. The parameters presented here will be used to illustrate the expected responses to various hypothetical selection schemes.

EXPERIMENTAL RESULTS

The direct effects heritabilities, maternal effects, direct environmental effects, and direct-maternal genetic covariances are presented in Table 3 for weight, head length, trunk length, trunk circumference, and tail length. As reported in Cheverud et al. (1983), the heritability estimates are either more or less constant with age, showing no linear trend (WT, TRL, TRC) or slightly increase with age (HL, TL) while maternal effects decrease with age for all traits from about .50 to .15.

The standardized direct-maternal covariances range from $-.37$ to $.29$. Negative covariances dominate at the earliest ages, from 17 to 45 days, and especially at 24 and 31 days, while positive covariances dominate at later ages, 52 to 66 days. This indicates a general trend for higher covariance at later ages. This

TABLE 3. Direct effects heritability (h^2_o), maternal effects (m^2), direct-maternal genetic covariance (cov), and residual environmental effects (e^2_o) for weight (WT), head length (HL), trunk length (TRL), trunk circumference (TRC), and tail length (TL) at ages 17, 24, 31, 45, 52, 59, and 66 days.

Trait	h^2_o	m^2	cov	e^2_o
WT17	.303**	.553**	-.073	.217
WT24	.316**	.417**	-.033	.300
WT31	.242**	.428**	.022	.308
WT45	.234**	.355**	.101	.310
WT52	.497**	.190**	.204*	.109
WT59	.208**	.200**	.215**	.377
WT66	.339**	.202**	.290**	.169
HL17	.313**	.345**	-.153	.495
HL24	.265**	.351**	-.231	.615
HL31	.432**	.359**	-.155	.364
HL45	.258**	.114*	-.323**	.951
HL52	.517**	.200**	.007	.276
HL59	.535**	.122**	-.329**	.672
HL66	.479**	.092*	-.128**	.557
TRL17	.237**	.400**	.226	.137
TRL24	.290**	.484**	-.143	.369
TRL31	.343**	.412**	-.020	.265
TRL45	.329**	.132**	-.113	.348
TRL52	.200*	.136**	.247**	.417
TRL59	.135	.166**	.163**	.536
TRL66	.325*	.213**	.103	.359
TRC17	.154	.462**	.289**	.095
TRC24	.353**	.335**	-.064*	.378
TRC31	.288**	.303**	-.224*	.367
TRC45	.245**	.113*	.053	.624
TRC52	.134	.049	.197**	.620
TRC59	.221**	.038	.216**	.525
TRC66	-.002	.116*	.264**	.622
TL17	.394**	.609**	-.317	.314
TL24	.392**	.545**	-.375*	.438
TL31	.292**	.440**	.051	.289
TL45	.292**	.440**	-.202	.470
TL52	.460**	.279**	-.050	.311
TL59	.396**	.266*	-.025	.363
TL66	.521**	.187**	-.223*	.515

* Significantly different from zero at the .10 level.
 ** Significantly different from zero at the .05 level.

ontogenetic trend is most clearly demonstrated by age-specific weights which show increased covariance at each age. Trunk length and trunk circumference show a U-shaped pattern with covariances high at age 17, decreasing to negative values at intermediate ages, and then increasing again to positive values at ages 52 to 66. Head length and tail lengths show no age-related trend in covariance values, but typically have negative co-

TABLE 4. Genotype-phenotype regression coefficients for the correlated responses of direct effects (CR_o), maternal effects (CR_m), and the direct response of the offspring phenotype (R_p) to selection on the offspring phenotype and the maximum cost-benefit ratio which allows altruistic evolution of maternal performance (Ratio)*.

Trait	CR_o	CR_m	R_p	Ratio*
WT24	.300	.050	.350	.195
HL24	.150	-.161	-.011	-.678
TRL24	.219	-.046	.173	-.166
TRC24	.321	.003	.324	.013
TL24	.205	-.266	-.061	-.901
WT45	.285	.172	.457	.721
HL45	.097	-.300	-.203	-2.223
TRL45	.273	-.087	.186	-.596
TRC45	.237	.076	.313	.562
TL45	.284	-.144	.140	-.618
WT59	.316	.255	.571	1.426
HL59	.371	-.305	.066	-2.180
TRL59	.217	.196	.413	1.204
TRC59	.329	.224	.553	2.868
TL59	.384	.028	.412	.137

* The heritability of maternal performance is assumed to be .40.

variances. There are no statistically significant differences among covariance estimates for the same trait at different ages. However, these estimates can be used to illustrate the model.

The genotype-phenotype regression coefficients, b_{AP} for the correlated responses of direct effects (CR_o , see equation 7), and maternal performance (CR_m , see equation 6), and the direct response of the offspring phenotype (R_p , see equation 5) to selection on the offspring phenotype are given in Table 4 for certain ages (25, 45, and 59 days) in order to illustrate the model. The responses to hypothetical directional selection on the offspring phenotypes may be obtained by multiplying the figures given by s_p and the selection intensity. The maximum cost-benefit ratio ($|i_m|/2i_p$) at which maternal performance will evolve in an altruistic direction, given that selection is altruistic, is also provided in Table 4. Any cost-benefit ratio greater than the one given will not result in evolution in an altruistic direction. These calculations are only illustrative because an assumed val-

ue (.40, see above) for maternal performance heritability had to be used. Decreased values of h^2_m result in smaller values for CR_m , R_p , and the maximum cost-benefit ratio, while increased values have the opposite effect.

All of the unexpected theoretical results noted above occur in the empirical estimates presented in Table 4. Head length at ages 24 and 45 would have a negative response to positive selection pressures. Selection for increased offspring phenotypic values would result in decreased maternal performance for many offspring phenotypes (HL24, HL45, HL59, TRL24, TRL45, TL24, TL45). Maternal performance would evolve towards more altruistic values even when selective costs exceeded benefits for WT59, TRL59, and TRC59. Maternal performance would be prevented from evolving in an altruistic direction regardless of the cost-benefit ratio for HL24, HL45, HL59, TRL24, TRL45, TL24, TL45.

DISCUSSION

The theoretical results presented above indicate that a more general theory of response to selection on target and kin performance phenotypes contains many phenomena which are not predicted by previous theories of kin selection. Furthermore, consideration of empirical data reported in the literature and presented above indicate that these "unusual" evolutionary results, not predicted by previous models, may be fairly common occurrences. Assuming a relatively high heritability for maternal effects of .40, many instances of negative maternal performance responses given selection for increased offspring phenotype would occur. This is due to the negative direct-maternal genetic covariances for these traits at the ages indicated. Also several cases are identified for which altruistic evolution of maternal performance might occur, even when costs exceed benefits.

The large number of traits and ages at which altruistic evolution for maternal performance cannot occur is due to the

large number of negative direct-maternal genetic covariances, which occur primarily at the earlier ages (24 and 31 days). These negative genetic covariances may be the result of strong selection in the past for weaning size and age at first reproduction. These two life history events may have high opportunities for selection and are probably related to one another (Montiero and Falconer, 1966). Strong selection for increased size during the period around 24 and 31 days would result in the fixation of alleles with a positive effect on both maternal performance and direct effects (+ + alleles) at the expense of alleles with a negative effect on both traits (- - alleles). This would only leave alleles with a positive effect on one trait and a negative effect on the other trait (+ - and - + alleles) segregating and contributing to the genetic covariance between direct and maternal effects (Dickerson, 1955; Falconer, 1981). Strong selection for the joint evolution of life history characters closely related to fitness is thought to produce the negative genetic covariances often found among these traits by the same process (Lande, 1982*a*, 1982*b*).

The maternal effects model is relevant to parent-offspring conflict theory (Trivers, 1974; Wilson, 1975). Trivers (1974) viewed parent-offspring relations at various ages as the result of selection operating in opposite directions in the two generations. Improved maternal performance, which is selected for because it contributes to higher offspring survivorship and earlier reproduction by the offspring, is also selected against due to the consequent decreased maternal fertility and survivorship. At the point where these two selection regimes balance in their effects, weaning occurs. However, as noted above, selection for increased offspring phenotype, especially at the age of weaning, may often lead to an evolutionary decrease in maternal performance for that phenotype. Therefore, these apparently opposed selection pressures may often result in complementary responses to selection. This is inconsistent

with Trivers' (1974) expectations, because in such a situation no balance of effects will occur.

While Trivers' (1974) analysis of parent-offspring conflict may properly identify the selection pressures acting on maternal performance and offspring phenotypes and their ecological causes, this analysis in itself is insufficient to predict the evolutionary outcome or response to this selection. Trivers did not consider the possibility of genetic covariance due to pleiotropy between maternal and offspring traits, and therefore implicitly assumed a value of zero. This assumption is not supported by the literature. The use of an evolutionarily dynamic quantitative genetic model makes such implicit assumptions explicit in contrast to the static optimization arguments employed by Trivers (1974). For this reason, models concerning the evolutionary response to selection pressures would benefit from including quantitative genetic analysis. This point has been well made by Lande (1982*a*, 1982*b*) in his analysis of life history evolution.

While the theory and results reported here pertain to maternal effects, similar models could be derived for phenotypic effects caused by interactions with other types of relatives. The extension to paternal effects is obvious and may be important in species where the father plays a role in caring for the young. Willham (1972) has extended the maternal effects model to include grandmaternal effects. Similar kinds of models could be derived where any kind of kin interaction plays an important role in phenotypic variability. When only one type of relative is considered at a time, the altruistic phenotype will evolve in an altruistic direction when,

$$k(r + (\text{cov}(A_o, A_q)/V_{Aq})) > |i_q|/i_p, \quad (16)$$

where k is the partial correlation between the altruistic phenotype, "kin performance," and the target phenotype and thus measures the relative importance of kin effects, r is the regression coefficient of the relative's genotype on that of the

altruist (Uyenoyama and Feldman, 1981), $\text{cov}(A_o, A_q)$, is the genetic covariance between direct and kin effects, V_{Aq} is the additive genetic variance in kin effects, and i_q is the direct selection intensity against increases in kin performance. Of course, quantitative genetic models can also be developed for more complex sets of interactants, involving more than one kind of kin effect. The quantitative genetic model of the evolution of characters impacted by interactions among relatives presented here provides new insights into the evolution of altruism not previously provided by kin selection models (Hamilton, 1964; E. O. Wilson, 1975; Wade, 1978, 1980; Wilson, 1980; Crow and Aoki, 1982) due to its inclusion of genetic constraints to evolution by selection.

SUMMARY

A quantitative genetic model for the evolution of traits by kin selection is presented, with special attention paid to mother-offspring interactions. The model derived produces several results which are not predicted by previous models, including the possibility of negative response to positive selection pressures, the possibility of evolution in an altruistic direction, even when the selective costs exceed benefits, and the identification of circumstances in which altruistic evolution is impossible regardless of the cost-benefit ratio. These results can be obtained with this quantitative genetic model because pleiotropic effects, measured by the direct-maternal genetic covariance, are taken into account instead of being disregarded, as in single locus models.

The biological importance of the possibilities noted above is evaluated by consideration of the agricultural genetic literature and data from a longitudinal growth study on random-bred mice. These data indicate that the possibilities for altruistic evolution of maternal performance are greatly affected by the direct-maternal genetic covariance. This covariance is often negative at early ages, near weaning, for all five traits consid-

ered and at all ages for head length and tail length, preventing altruistic evolution for maternal performance. The covariance is high and positive for weight, trunk length, and trunk circumference at later ages, allowing altruistic evolution of maternal performance even when selective costs exceed benefits.

It is proposed that the cause of direct-maternal genetic covariances being negative at ages near weaning is that there has been strong selection for increased values of offspring phenotypes, such as size, at weaning. The maternal effects model is also related to a sociobiological model of parent-offspring conflict, where direct selection on maternal performance and the offspring phenotype are in opposite directions. Due to the negative direct-maternal genetic covariances, selection for increased offspring phenotype may actually decrease maternal performance in many empirically studied situations, thus leading to no conflict in the evolutionary response to opposite selection pressures on mother and offspring and no altruistic evolution. The model for the evolution of maternal performance is generalized to account for evolution of any "kin performance" phenotype.

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Corresponding Editor: J. Felsenstein