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Evolution of intrafamilial interactions

(altruism/kin selection/Hamilton's rule/pleiotropy/quantitative genetics)

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ABSTRACT **A theory for the evolution of behavioral interactions among relatives is developed that allows for genetic correlations between the types of behavior that are expressed in different social contexts. Both theoretical and empirical considerations indicate that such genetic constraints will almost certainly be common in natural populations. It is shown that when genetic correlations between elements of social behavior exist, Hamilton's rule inaccurately describes the conditions for evolution by way of kin selection. The direction in which social organization evolves is a delicate function of the genetic covariance structure among behaviors expressed as an offspring, sibling, parent, etc. A change in this covariance structure caused by random genetic drift or by a change in environment for a population exhibiting genotype–environment interaction can cause the population to suddenly cross a threshold into a new selective domain. Consequently, radical changes in social organization may arise between closely related species without any major shift in selective pressures external to the population.**

Current thought on the evolution of social behavior is heavily influenced by the concept of inclusive fitness (1, 2). For the spread of an "altruism" gene at a single locus with additive effects, Hamilton's rule states that the cost in individual fitness (c) must be less than the gain in the fitness of the recipient (B) discounted by the coefficient of relationship between the two individuals (r): $c < rB$. On the other hand, a "selfish" gene can spread if the increase in fitness of the selfish individual exceeds the cost to the recipient discounted by r: $B > rc$. Several studies (3–7) have extended Hamilton's rule to polygenic characters with an additive genetic basis. These models are quite general in that they apply to descendent as well as nondescendent kin, but in the interest of tractability, they have ignored several biologically significant aspects of behavior.

First, individuals often act selfishly at one stage of their life and altruistically at another, and the recipients of these two extremes of behavior are often different members of the population (8, 9). Second, the various traits in an organism's behavioral repertoire will almost certainly be mutually constrained by pleiotropy (10). Models for the evolution of behavior should, therefore, be constructed in multivariate form. Third, the absolute baseline fitness of an individual (W_o) usually exhibits heritable variation (11). The failure to account for such variation would lead to serious inaccuracies in the prediction of the directional forces of selection operating on behavioral attributes if the latter were strongly correlated with W_o (12). The significance of these three factors for the evolution of social behavior is examined in this paper, and the practical difficulties of genetic analysis of social behavior are pointed out.

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Modification of Fitness by Family Members

Rather than present an exhaustively general model, I will focus on a specific but common type of kin group—a nuclear family consisting of a father, mother, and one or more progeny. With this type of population structure, five factors contribute to the fitness of an individual: (*i*) the direct effects of its own genes and the environment on its progeny production, its prior receipt of (*ii*) maternal and (*iii*) paternal care, and interactions with its (*iv*) siblings and (*v*) offspring. Behavioral interactions underlying all five factors are outlined in Table 1. It is by no means clear whether these classes of behavior are free to evolve independently.

In the following analysis, gene action is assumed to be additive both within and between loci, all of which are considered to be autosomal and in global linkage equilibrium. Mating is assumed to be random but monogamous. The generations are treated as being functionally discrete, except that parental care prior to the reproductive maturity of offspring is allowed.

The contribution of maternal effects to progeny performance was first considered by Dickerson (13) and by Willham (14, 15). Cheverud (16) utilized their model in the context of parent–offspring conflict. When the mother is the only relative that indirectly contributes to the absolute fitness (W) of an individual, x (denoted by a subscript x), $W_x = G_{ox} + M + E_{ox}$, where G_{ox} is the sum of the maternal and paternal contributions to the absolute direct additive genetic (G) value (denoted by a subscript o) for fitness in x, E_{ox} is the deviation in fitness resulting from the direct influence of the environment (E) on x, and M is the contribution to fitness from the maternal effect (Fig. 1). As a result of pleiotropy, a genetic correlation may exist between the direct and maternal components of fitness. Note that M may be further decomposed into genetic (G_{mw}) and environmental (E_{mw}) components (where the subscripts m and w stand for maternal effect and mother, respectively). Thus, while a maternal effect is an environmental effect from the perspective of x, it may have a genetic basis. The implications of this are 2-fold. First, if x is female, she will subsequently impose the genetic maternal effect G_{mx}, half of which is inherited from her mother, on her offspring. Second, although the father of x does not exhibit

Table 1. Summary of positive and negative contributions to the fitness of an individual with a family structure of mother, father, and offspring

Effect	Contribution	
	Positive	Negative
Direct	Survival Reproduction	Suicide
Sibling	Helping to raise siblings	Sibling competition Siblicide
Offspring	Care of parents	Matricide Patricide
Maternal or paternal	Care of offspring	Rejection Infanticide

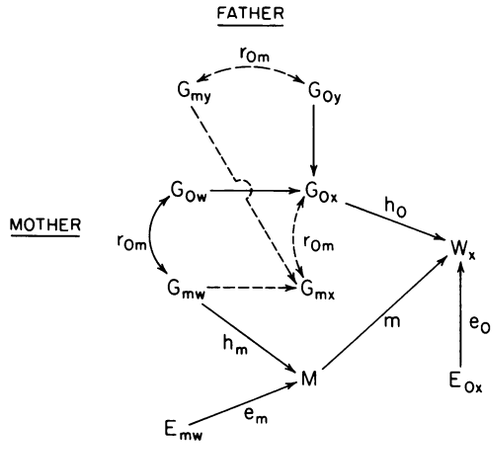

FIG. 1. Path diagram for the fitness of individual x with mother w and father y. Paternal effects and offspring effects are assumed to be unimportant, so that W_x is entirely determined by direct effects ($G_{ox} + E_{ox}$) and a maternal effect M. G and E refer to additive genetic and environmental effects, whereas subscripts o and m denote direct and maternal components of these effects. Quantities on single-headed arrows (h_o, e_o, m, e_m, and h_m) are path coefficients; the square of such a coefficient is the proportion of the variance of a dependent variable directly accounted for by an independent variable. All path coefficients not denoted are equal to $2^{-1/2}$. r_{om} is the additive genetic covariance between direct and maternal effects.

a maternal effect, he does carry genes for maternal effects that will be expressed through his daughters. Analogous arguments can be developed for paternal effects, which in the following will be denoted by a subscript p.

Van Vleck (17) first introduced offspring effects into quantitative genetic formulations. Although he was specifically concerned with the effects of a fetus on the traits of a mother in dairy cattle, his approach can be considered in a much broader context. Ignoring maternal and paternal effects for the time being, we now have $W_x = G_{ox} + F + E_{ox}$, where F is the effect of the behavior of offspring of x on the fitness of x. F has an environmental (E_f) as well as a genetic (G_f) component (denoted by a subscript f), and G_f is a function of the genes for offspring effect contributed by x (G_{fx}) and by the mate (denoted by a subscript v) of x (G_{fv}). In this sense, without investing in parental care, an individual can influence the progeny production of its mate through the genes for offspring effects (for example, parental manipulation) that it contributes to those progeny. Sibling–sibling interactions are readily incorporated into individual fitness expressions (18, 19). When these are present, individual fitness is determined partially by the effect of sibling(s), $S = G_{sz} + E_{sz}$ (where s and z are sibling effects and the sibling, respectively).

Joint Operation of Maternal, Paternal, Sibling, and Offspring Effects

In developing the concept of parent–offspring conflict, Trivers (8, 9) emphasized that both parental and offspring effects will be important in most social systems. The joint operation of these as well as direct effects are summarized in the path diagram in Fig. 2. For clarity, sibling effects are omitted from this figure. Individual fitness is now a linear function of five fitness components, $W_x = O + F + M + P + S$ (where O refers to the direct effects of x's genes and the environment on x's fitness), four of which are a consequence of interactions with close relatives and all of which are potentially constrained by pleiotropy.

Prediction of the response of quantitative traits to selection usually involves the regression of offspring phenotype on that of parents. In the current model, however, the effects of genes span two generations, and the response to selection is a function of the regression of individual fitness on the average fitness of its four grandparents. Letting the slope of this regression be h^2_W and the change in mean absolute fitness due to selection be S_W (the selection differential), the asymptotic response to selection is $\Delta\overline{W} = h^2_W S_W$ (where the subscript W is the absolute fitness). The selection differential is equivalent to the covariance of the character and relative fitness ($w_x = W_x/\overline{W}$) (20). Since the character of interest is fitness itself, the evolutionary rate of change of mean relative fitness caused by selection is equal to the sum of the covariances of an individual's relative fitness (w) with that of its grandparents:

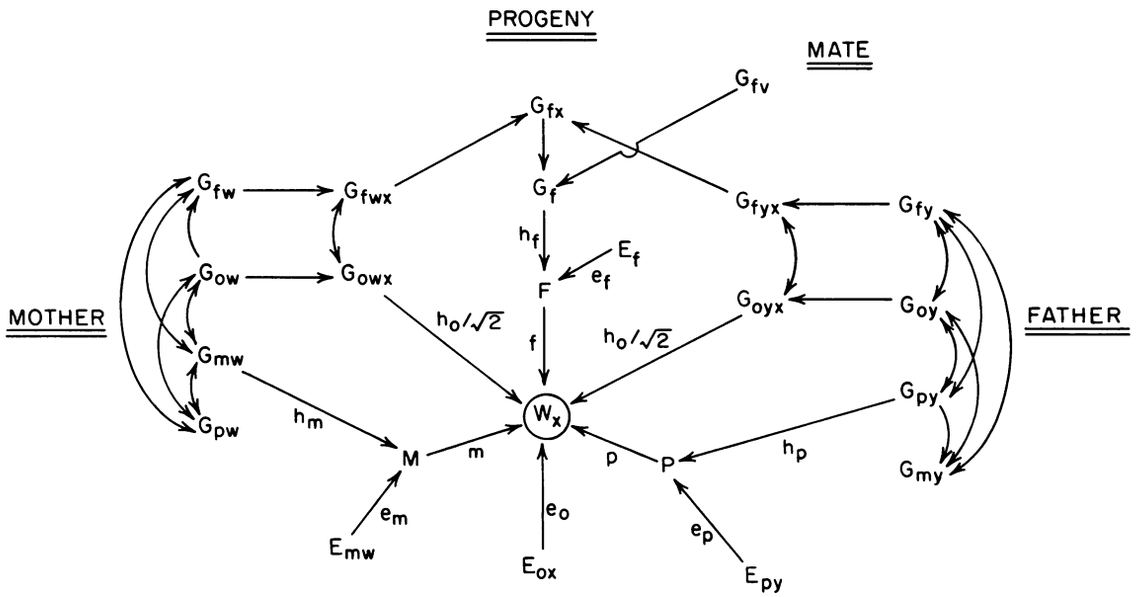

FIG. 2. Path diagram for the joint influence of direct (G_{owx}, G_{oyx}, and E_{ox}), maternal (M), paternal (P), and offspring (F) effects on an individual's fitness (W_x). Additive genetic correlations between the four fitness components are denoted by double-headed arrows. Labels on single-headed arrows (h_m, e_m, e_o, e_p, h_p, $h_o/\sqrt{2}$, e_f, and h_f) are path coefficients. All unlabeled, single-headed arrows have path coefficients equal to $2^{-1/2}$.

$$\Delta\bar{w} = \mathrm{Cov}(w_x, w_{w'}) + \mathrm{Cov}(w_x, w_{w''}) + \mathrm{Cov}(w_x, w_{y'}) + \mathrm{Cov}(w_x, w_{y''}), \quad [1]$$

where ′ and ″ denote the mothers and fathers, respectively, of the mother w and the father y.

In the following it is assumed that the resemblance between relatives is entirely attributable to genetic covariance. No distinction will be made between the effects of offspring on the fitness of fathers and mothers or the sibling effects between x and brothers or sisters. The former will be true if the offspring effect is sibling competition or cooperation, but not if it includes matricide, patricide, or helping parents of a specific sex. Both assumptions are made purely for simplicity of exposition and are readily eliminated with additional algebra. The extrapolation of Eq. 1 over multiple generations requires that selection is weak so that the linear relationship between selection response and selection differential is preserved.

The four covariances in Eq. 1 are functions of the five genetic components of fitness and the coefficients of relationship between contributors (14, 21). After some algebra, the evolutionary response of mean relative fitness to selection can be resolved into its five components, $\Delta\bar{w} = \Delta\bar{w}_o + \Delta\bar{w}_m + \Delta\bar{w}_p + \Delta\bar{w}_f + \Delta\bar{w}_s$, where

$$\Delta\bar{w}_o = \mathrm{Var}(g_o)[1 + 0.5b_{mo} + 0.5b_{po} + (1 + n)b_{fo} + 0.5(n - 1)b_{so}], \quad [2a]$$

$$\Delta\bar{w}_m = \mathrm{Var}(g_m)[b_{om} + 0.5 + 0.5b_{pm} + (1 + n)b_{fm} + 0.5(n - 1)b_{sm}], \quad [2b]$$

$$\Delta\bar{w}_p = \mathrm{Var}(g_p)[b_{op} + 0.5b_{mp} + 0.5 + (1 + n)b_{fp} + 0.5(n - 1)b_{sp}], \quad [2c]$$

$$\Delta\bar{w}_f = (0.5n)\,\mathrm{Var}(g_f)[b_{of} + 0.5b_{mf} + 0.5b_{pf} + (1 + n) + 0.5(n - 1)b_{sf}], \quad [2d]$$

$$\Delta\bar{w}_s = (n - 1)\mathrm{Var}(g_s)[b_{os} + 0.5b_{ms} + 0.5b_{ps} + (1 + n)b_{fs} + 0.5(n - 1)]. \quad [2e]$$

The terms Var($g_.$) are the additive genetic (g) variances for the relative fitness components. The $b_{..}$'s represent additive genetic regression coefficients. Each one defines the expected evolutionary change in the mean of one fitness component in response to a shift in another. For example, b_{mo} is the regression of g_m on g_o. The offspring effects on the grandparents have been partitioned into those from the parents (w and y) and each of the siblings of the latter ($n - 1$).

Evolutionary Consequences of Pleiotropy Between Components of Fitness Contributed to Relatives

If behaviors involving family members are mutually constrained by pleiotropy, as is often the case with morphological characters, Hamilton's rule needs to be modified. Consider, for example, the evolution of maternal care ($\Delta\bar{w}_m > 0$). Since mother and offspring are 50% related, one would ordinarily expect increased maternal investment to evolve if the genetic reduction in baseline fitness ($\bar{w}_o$) per unit increase in $\bar{w}_m$ is less than -0.5. Eq. 2b shows that if genes for maternal care have pleiotropic effects on paternal care, sibling interactions, and/or offspring effects, the criterion for the evolution of maternal care can be greatly modified. If $\{0.5[b_{pm} + (n - 1)b_{sm}] + (1 + n)b_{fm}\}$ is negative, the criterion for the evolution of maternal care becomes more stringent; and if it is less than -0.5, maternal care will not undergo an evolutionary advance unless it has a positive pleiotropic effect on the direct contribution to fitness.

Similarly, natural selection need not always favor an increase in the direct contribution of genes to fitness ($\Delta\bar{w}_o > 0$). If b_{mo}, b_{po}, b_{fo}, or b_{so} are sufficiently negative, selection on total fitness can lead to a decline in baseline fitness whereas other behavioral components of fitness undergo an evolutionary advance. Examples of such evolutionary change are progeny attributes, such as altriciality, that guarantee mortality in the absence of parental care or sibling cooperation. Contrary to what optimization arguments based on energetic constraints suggest, a reduction in baseline fitness is not a necessary outcome of the evolution of parental care. The two can evolve in parallel provided their genetic correlation is not too negative.

Lande (22) has argued that frequency-independent selection will always lead to an increase in the mean level of adaptation in a population. However, when fitness is partially determined by interactions with relatives, extreme negative pleiotropy can sometimes lead to a decline in mean relative fitness (23). Consider, for example, the situation in which individual fitness is a function of only direct and maternal effects, in which case $\Delta\bar{w} = \mathrm{Var}(g_o) + 1.5\,\mathrm{Cov}(g_o, g_m) + 0.5\,\mathrm{Var}(g_m)$. Provided $0.5 < [\mathrm{Var}(g_o)/\mathrm{Var}(g_m)]^{1/2} < 1$, an extremely negative genetic correlation between direct and maternal effects can result in $\Delta\bar{w} < 0$ (Fig. 3). Although the domain under which $\Delta\bar{w} < 0$ is small in this particular case, it will enlarge as interactions from more family members are added. The implications are that, under some circumstances, genetic trade-offs between fitness components contributed by different relatives can cause maladaptive evolution.

Explanations for the evolution of social systems are almost always based on ecological arguments (9, 24–26). Eq. 2 a–e, however, illustrates that when pleiotropy is present, the direction in which social interactions evolve is a delicate function of the genetic variances and covariances of the behavioral components of fitness. Any force external or internal to a population that changes this variance–covariance structure can potentially propel a population into a new selective domain. For example, a slight change in Var(g_o), Var(g_m), or Cov(g_o, g_m) for a population residing near the boundaries in Fig. 3 would suffice to induce a sudden shift in the direction of evolution of direct or maternal effects of fitness. Such a change in the direction of evolution could arise rapidly without any change in the genetic composition of a population if, through genotype–environment interaction, a shift in the environment induced changes in the genetic variance–covariance structure. Genetic drift resulting from a founder effect could also cause a population to cross a threshold into a new selection domain. By this means, significant and rapid divergence of social systems might occur in isolated populations despite their exposure to identical ecological pressures.

The long-term consequences of such excursions across a selection boundary will depend on the dynamics of genetic variance and covariance of the fitness components, which will be determined not only by the selection process itself, but also by mutation and random genetic drift. This subject is beyond the scope of this paper. Natural selection is expected to deplete the additive genetic variance for total fitness (27), but this need not be reflected at the component level. Thus, on the isocline $\Delta\bar{w} = 0$ in Fig. 3, the additive genetic variance for total fitness is zero, but the direct and maternal genetic contributions to fitness are variable and evolving in positive and negative directions, respectively.

Genetic Regressions of Behavioral Components of Fitness

Negative genetic correlations between nonbehavioral fitness components have been observed in many species (11, 28). Their common occurrence is probably due to the rapid fixation of genes with mutually favorable effects on different

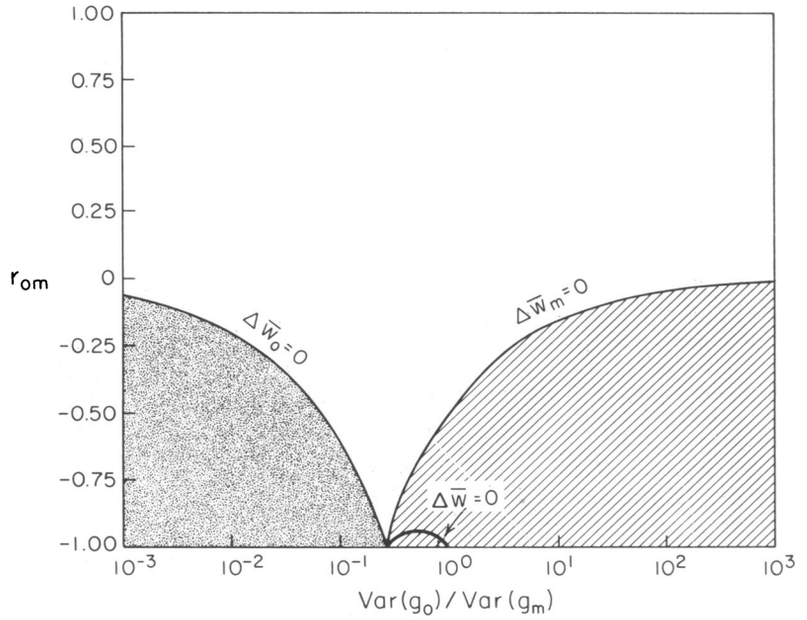

FIG. 3. Conditions for the evolution of the direct ($\bar{w}_o$) and maternal ($\bar{w}_m$) contributions to fitness when components due to all other relatives are absent. For populations in the unshaded area, both the direct and the maternal effects on fitness will evolve in a positive direction. In the lower left corner, maternal care evolves but the direct contribution to fitness declines as a consequence of negative genetic correlation between the two characters. The evolutionary change in mean total fitness is positive everywhere, except below the small isocline ($\Delta\bar{w} = 0$).

fitness traits and the approximate selective neutrality of alleles that exhibit negative pleiotropy. There are unfortunately no direct estimates for genetic covariances among behavioral components of fitness for any species, and the prospects for obtaining them are not very high.

Accurate determinations of genetic variances and covariances require very large sample sizes as well as phenotypic information on several classes of relatives (14, 29). For the type of social organization described above, 15 causal components of additive genetic variance and covariance contribute to the resemblance of the simplest relatives. Thus, no fewer than 15 sets of relatives would have to be observed for a complete evaluation of the significance of pleiotropy for social interactions. This poses obvious practical problems, but indirect evidence from the animal-breeding literature strongly suggests that pleiotropy should not be ignored.

For domesticated mammals, some insight into the significance of postnatal maternal effects is possible. Paternal care is absent for most such species, as are offspring effects for characters expressed prior to maturity. Many investigators have performed cross-fostering experiments (30) to obtain estimates of the variance of progeny phenotypes caused by mothers, mother–nurse interaction [Var(I)], and nurses [Var(N)]. These variance components usually are manipulated to give estimates of Var(G_o), Var(G_m), and Cov(G_o,G_m) for various traits, but such a treatment implicitly assumes the absence of sibling effects. Allowing for possible sibling effects, the ratio Var(I)/Var(N) has the reasonably simple interpretation of $b_{om} + b_{sm}$.

Fig. 4 summarizes data on Var(I)/Var(N) for age-specific weight from four experiments with laboratory mice. Var(I)/Var(N) consistently increases from zero at about 1 week to nearly 1.0 at 10 weeks. This indicates that genes that influence post-weaning growth through maternal care have substantial positive pleiotropic effects on direct or sibling effects on the same trait. Although body weight cannot be equated with fitness, for situations in which the two are positively correlated, such conditions would facilitate the joint evolution of maternal, direct, and sibling effects on fitness. Similar results have been obtained for rats (34) and sheep (35), but it is not clear that they can be generalized to other mammals. Several lines of evidence indicate that $b_{om} + b_{sm}$ is significantly negative in cattle (36) and swine (37).

A very large experiment on *Tribolium* (38) is noteworthy since it focused on a character close to fitness, number of viable offspring per 24-hr egg-laying period. Although individuals were raised in families, the analysis, based on phenotypic covariances of various types of relatives, implicitly assumes negligible sibling, offspring, and paternal effects. Subject to this constraint, the results indicate that $b_{mo} \approx -0.51$ and $b_{om} \approx -0.34$. Although a precise interpretation is difficult, these results imply the existence of substantial negative genetic covariances between direct effects on fitness and those due to intrafamilial interactions.

Methodology for estimating genetic variance and covari-

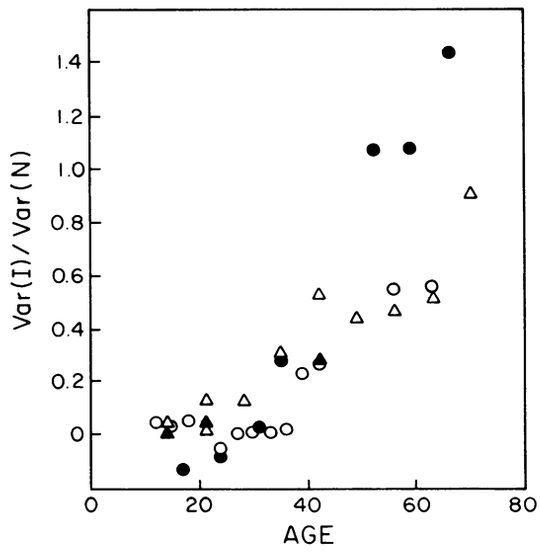

FIG. 4. Estimates of Var(I)/Var(N) $\approx b_{om} + b_{sm}$ for age-specific weights in four cross-fostering experiments with laboratory mice. Data are from references as follows: ●, ref. 16; ○, ref. 30; △, refs. 31 and 32; ▲, ref. 33.

ance from offspring effects has been discussed by Bar-Anan *et al.* (39) and Van Vleck (17). Estimation of $Var(G_f)$ is particularly straightforward, being simply four times the phenotypic covariance between unrelated individuals with the same mate. However, the recommended protocols for estimating $Cov(G_o, G_f)$ have the same limitations as that for evaluating $Cov(G_o, G_m)$; all other intrafamilial contributions to the phenotype (sibling, maternal, and paternal effects) are assumed to be negligible. Under this assumption, estimates of $b_{fo} \approx -0.53$ and $b_{of} \approx -0.29$ are obtained from data on calving difficulty in dairy cattle (39). Again while the composition of these parameter estimates is somewhat uncertain, such negative values imply substantial genetic tradeoffs between components of fitness contributed by different relatives.

Discussion

Since an individual plays the role of various relatives (offspring, sibling, parent, grandparent, etc.) at different stages of its life, it seems desirable to study social behavior among family members as an integrated and ontogenetic process. Thus, unlike previous approaches that rely on inclusive fitness functions, the approach taken here is based on the concept that the fitness of an individual is composed of components contributed by different relatives. By focusing on the individual as the unit of selection, a precise definition of the hereditary properties and evolutionary dynamics of mutually constrained characters underlying social organization is possible. This eliminates some of the ambiguities that have arisen in discussions on the evolution of behavioral conflicts between relatives such as parents and offspring (40).

If the types of behavior expressed by an individual in different social contexts are genetically correlated, a constraint will exist on the social interactions that can evolve. For example, if genes that contribute to an offspring effect, such as manipulation of parents, also have an effect on parental behavior later in life, the evolution of the parent–offspring interaction will depend on the direction of genetic correlation as well as the relative levels of genetic variance for the two behaviors. The need to recognize the genetic correlational structure for social behavioral traits raises a serious problem for the study of the evolution of social organization because of the enormous difficulties in performing an adequate genetic analysis. Cost/benefit ratios obtained in field studies of kin selection are almost always phenotypically based. Since genetic correlations are often very different than phenotypic correlations (28), it may be argued that the field work is often misdirected.

Certainly in many social systems, fitness will be influenced by a more diverse set of relatives than the nuclear family envisioned above. Nevertheless, the general approach taken in this paper can be extended to any degree of relationship, the major modification being the utilization of the proper coefficients of relationship for the various forms of genetic variance and covariance (15). It may, therefore, be of use in investigating the evolutionary consequences of matriarchal lineages in some primates, polyandry in certain birds, haplodiploidy in the social Hymenoptera, etc. The techniques can also be applied to the problem of group selection once the average degree of relatedness of group members has been derived from information on population structure (41). Although higher-order gene interactions have been ignored in the preceding analyses, the contribution of dominance and various forms of epistasis to the evolution of intrafamilial interactions is readily incorporated by use of the general expression of Willham (15). As pointed out above, however, the practical problems of measuring the parameters of the additive model are already formidable.

Finally, it should be emphasized that the theory developed here only considers the total components of fitness derived from the behavior of relatives. It does not specify the evolutionary dynamics of specific traits underlying these components. Such a refinement can be made by use of Robertson's "Secondary Theorem of Natural Selection" (42), which indicates that the response of a character to fitness is equal to the additive genetic covariance between the character and fitness.

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