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Effect of phenotypic variation on kin selection

(nepotism/quantitative genetics/trait groups/altruism)

ROBERT BOYD* AND PETER J. RICHERSON

Division of Environmental Studies, University of California, Davis, California 95616

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ABSTRACT **An expression for the equilibrium of the mean phenotypic value of a quantitative character is derived for a model in which the fitness of an individual depends on its own phenotype and the mean phenotypic value of a group of related individuals. When selection is weak the equilibrium mean is well predicted by Hamilton's $k > 1/r$ rule (k is the ratio of mean fitness gained by recipient of altruistic behavior to mean fitness lost by donor; r is mean coefficient of relationship between donor and recipient). When selection is strong, however, the equilibrium mean depends on the heritability of the character. Low heritability can lead to substantially more "altruism" than predicted by the $k > 1/r$ rule.**

With the exception of the work of Yokoyama and Felsenstein (1), the theoretical development of kin selection has been in terms of a discrete valued trait, and to the extent that genetic mechanisms have been considered, they have been limited to one (2, 3) or two loci (4). Continuous variation has been dealt with through the device of finding the evolutionary stable strategy (ESS)—i.e., finding the phenotypic value that, when common, is stable against mutation. Given that complex behavioral traits are likely to be characterized by both continuous variation and polygenic control, it seems likely that there will be both genetic and environmental variation in such traits at equilibrium. Because kin selection is a frequency-dependent process, one might suspect that this variation would affect the results in a way that is not predicted by the consideration of selection on a nearly monomorphic population.

In this paper, we analyze a model in which a quantitative character is subjected to two opposing selective processes. In the first, an individual's probability of survival depends on its own phenotypic value and in the second on the mean of the phenotypic values in its kin group. When selection is weak relative to the equilibrium phenotypic variance, the equilibrium mean of the population is well predicted by the ESS result. When selection is strong, however, heritabilities near one lead to less altruism than predicted by the $k > 1/r$ rule and heritabilities near zero lead to substantially more altruism (k is the ratio of mean fitness gained by a recipient of altruistic behavior to mean fitness lost by donor; r is mean coefficient of relationship between donor and recipient). These results differ somewhat from those of Yokoyama and Felsenstein (1).

THE MODEL

Recently, several authors (5–9) have proposed related models to describe the evolution of quantitative characters. These models share several general characteristics: Genotype is modeled as a continuous random variable and the entire probability density is iterated from generation to generation. Transmission, mutation, and selection schemes are assumed to be such that the genotypic value is always approximately normally distributed. Mutation maintains a finite variance at equilibrium. These models differ largely in the detail with which the underlying genetic system is modeled. Lande (8) and Felsenstein (9) consider the evolution of n linked loci, each with an infinite number of alleles. Bulmer's (7) model is a special case of this type, assuming an infinite number of unlinked loci. Slatkin (5) and Cavalli-Sforza and Feldman (6) use the more ad hoc assumption that the genotypic value of an offspring is the sum of the midparental value and a normally distributed error term.

Here we will use a version of the model of Cavalli-Sforza and Feldman to examine the case of kin and trait group selection. This model was chosen because of its relative computational simplicity. We will argue, however, that the qualitative conclusions should hold for all of the above described models. The quantitative results will depend on the magnitude of both the equilibrium genotypic and phenotypic variances, which differ somewhat among the various models.

Consider the evolution of a continuous trait, whose genotypic value will be labeled g. The inheritance system is defined by the function $T(g|g_1, g_2)$, which gives the probability that an offspring has a genotypic value g, given that the genotypic values of the parents were g_1 and g_2. If the probability density of genotypes before transmission is $\phi_t(g)$ and mating occurs randomly with respect to genotype, then the density of genotypes in the next generation after transmission, $\phi_t'(g)$, is given by $\phi_t'(g) = \iint \phi_t(g_1)\phi_t(g_2)T(g|g_1, g_2)\,dg_1\,dg_2$. We will assume $g = \frac{1}{2}(g_1 + g_2) + \delta$, in which δ is a normal random variable with mean zero and variance $\frac{1}{2}V_t + M$. If $\phi_t(g)$ is also normal with mean μ_t and variance V_t, then it can be shown (6) that $\phi_t'(g)$ is also normal with mean μ_t and variance $V_t' = V_t + M$.

Let the expected fitness of an individual with genotypic value g be given by the function $\tilde{W}(g)$. Then, the complete recursion is given by:

$$\phi_{t+1}(g) = \phi'(g)\tilde{W}(g)/\int \tilde{W}(g)\phi_t'(g)dg. \qquad [1]$$

To model ordinary natural selection, it is usually assumed that the fitness of an individual depends only on the individual's own phenotype. For example, if $P(f|g)$ is the probability that an individual with genotypic value g has phenotypic value f and $W(f)$ is the fitness of such an individual, then to compute $\tilde{W}(g)$ one averages over the fitness of all phenotypes that can result from a genotype g—i.e., $\tilde{W}(g) = \int W(f)\,P(f|g)\,df$.

The essence of kin selection is that the fitness of an individual depends on the phenotypes of a group of other individuals in the population with whom the individual interacts socially as well as its own phenotype. Recent theoretical treatments of kin selection (1–4) make this group nature of kin selection explicit. This suggests we generalize the usual approach in the following way. Let

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Abbreviation: ESS, evolutionary stable strategy.

* Present address: School of Forestry and Environmental Studies, Duke University, Durham, NC 27706.

$$\tilde{W}(g_1) = \int\int \dots \int W(f_1|f_2, \dots f_n)P(f_1, \dots f_n|g_1)\, df_1 \dots df_n, \quad [2]$$

in which g_1 and f_1 are the genotypic and phenotypic values of a focal individual and $f_2, \dots, f_n$ are the phenotypic values of the other $n-1$ individuals in the socially interacting kin group. The function $W(f_1|f_2 \dots f_n)$ gives the fitness of an individual with phenotypic value f_1 in a kin group whose other $n-1$ numbers have phenotypes $f_2 \dots f_n$. $P(f_1 \dots f_n|g_1)$ gives the probability that an individual with genotypic value g_1 has phenotypic value f_1 and finds itself in such a kin group. Thus the function $W(f_1|f_2, \dots, f_n)$ represents the nature of the social interaction between kin group members and $P(f_1, \dots f_n|g_1)$ the population structure.

To determine $P(\cdot|\cdot)$ we make the following assumptions: First, we will assume that there is a constant correlation, r, between the genotypic values of individuals in a kin group. That is, we assume that the genotypic values of the n individuals in the kin group are random variables jointly distributed according to an n-variate normal distribution with means equal to the population mean and the covariance matrix

$$V_t' \begin{pmatrix} 1. & \cdot & r \\ r & \cdot & .1 \end{pmatrix}. \quad [3]$$

This assumption is equivalent to supposing that kin groups are formed from a single class of relatives—for example, all sibs. Each individual may belong to several such groups simultaneously, each composed of other individuals with whom it interacts socially in a uniform way. Yokoyama and Felsenstein (1) make use of a similar assumption. We then assume that the phenotype of each individual, i, is given by $f_i = g_i + \epsilon_i$, in which the ϵ_i are independent, identically normally distributed random variables with mean zero and variance E, representing environmental variation. Thus $P(f_1|g_1)$ is normal with mean g_1 and variance E.

Clearly there are many forms that $W(\cdot|\cdot)$ might take, depending on the details of the ecological situation and social interaction of interest. We have chosen the following form because it provides a plausible caricature of many situations of interest and has the mathematical advantage of preserving the normality of $\phi_t(g)$, which in turn allows the model to be solved analytically. Let $W(f_1|f_2, \dots, f_n) = W_s(f_1)W_\gamma(\bar{f})$, in which

$$W_s(f_1) = \exp[-(f_1 - \mu_s)^2/2S], \; W_\gamma(\bar{f}) = \exp[-(\bar{f} - \mu_\gamma)^2/2\gamma], \quad [4]$$

and $\bar{f} = 1/n \sum_{i=1}^{n} f_i$. This form of $W(\cdot|\cdot)$ implies that there are two selective processes that act on the population sequentially. In one process, which we will call "own phenotype selection," the probability that an individual with phenotype f_1 survives is $W_s(f_1)$. Individuals with phenotypic value μ_s have the highest probability of surviving own phenotype selection. The parameter S is inversely related to the strength of this selection. In the second process, which we will call "group phenotype selection," the probability that an individual survives depends on the mean phenotype of its kin group and is given by $W_\gamma(\bar{f})$. Individuals who are members of a group whose mean phenotype is μ_γ have the highest probability of surviving this stage of selection. The strength of group phenotype selection is inversely related to the parameter γ.

Suppose that $\mu_\gamma \neq \mu_s$. In this case the two selective processes are competitive. Own phenotype selection will tend to move the phenotypic mean of the population towards μ_s and group phenotype selection will move it toward μ_γ. Our goal is to derive the equilibrium result of these two forces. In this context it is useful to compare the equilibrium to two standards:

$$\mu_m = (\gamma\mu_s + S\mu_\gamma)/(\gamma + S), \; \mu_k = (\gamma\mu_s + \bar{r}S\mu_\gamma)/(\gamma + \bar{r}S), \quad [5]$$

in which $\bar{r} = (1 + (n-1)r)/n$. A population monomorphic for phenotypic value μ_m is the configuration with the highest mean fitness, and is sometimes thought to represent the result of pure group selection. A population monomorphic for μ_k can resist rare invading types and therefore is the ESS value. μ_k is also the value that maximizes inclusive fitness in a monomorphic population. Note that $\bar{r}$ can be interpreted as the average relatedness of an individual to the group.

This model represents some situations found in nature: f might represent the rate of excretion of a toxic metabolic byproduct by an organism that lives in an aquatic environment. There is some value of f that optimizes individual physiology; this is μ_s. The fitness of the individual also depends, however, on how polluted its environment is, which in turn depends on its own excretion rate and the rate of neighboring individuals. Each individual is better off if, up to a certain point, their environment is less polluted; thus $\mu_\gamma < \mu_s$. The character represented by f might also be individual predation rate. At a constant prey density, the optimal rate is μ_s. But the prey density depends on the average predation rate of the local group in the usual way. The average fitness of the group is increased by harvesting at the maximal sustained yield and again $\mu_\gamma < \mu_s$. This model will not represent many other situations. For example, if f represents the threshold for giving alarm calls, then the group effect might depend on the minimal value of f in the group rather than the mean value.

EQUILIBRIUM VALUES OF μ_t AND V_t

Given the above assumptions, Eq. 2 can be rewritten

$$\tilde{W}(g_1) = \int\int W_s(f_1)W_\gamma(\bar{f})P(f_1, \bar{f}|g_1)\, d\bar{f}\, df_1. \quad [6]$$

Integrating Eq. 6 shows that $\tilde{W}(\cdot)$ is normal with mean $\tilde{\mu}$ and variance $\tilde{V}$, in which:

$$\tilde{\mu} = (\mu^I(E + S) + \mu_s V^I)/(E + S + V^I) \quad [7]$$

and

$$\tilde{V} = V^I(E + S)/(V^I + E + S), \quad [8]$$

in which

$$\mu^I = \frac{n(S + E)(\mu_\gamma - (1 - \bar{r})\mu_t) - E\mu_s}{S + n(S + E)(\bar{r} - 1/n)} \quad [9]$$

and

$$V^I = (SE + SG_t + G_tE)(S + E)/(S + n(S + E)(\bar{r} - 1/n))^2, \quad [10]$$

and in turn in which

$$G_t = n^2(\gamma + (1 - \bar{r})\bar{r}V_t' + (n - 1)E/n^2). \quad [11]$$

Given $\tilde{u}$ and $\tilde{V}$, the recursion for the mean and variance of the genotypic values in the population are (from Eq. 1):

$$\mu_{t+1} = (\mu_t\tilde{V} + \mu V_t')/(\tilde{V} + V_t) \quad [12]$$

and

$$V_{t+1} = \tilde{V}V_t'/(V_t' + \tilde{V}). \quad [13]$$

Note that the recursion for the variance is independent of the mean. This result holds for all of the models of quantitative characters discussed above. It can be shown that Eq. 13 results in a unique stable positive equilibrium genetic variance. The different models would differ only in the magnitude of this variance. Therefore, the qualitative conclusions discussed below are insensitive to the choice of model.

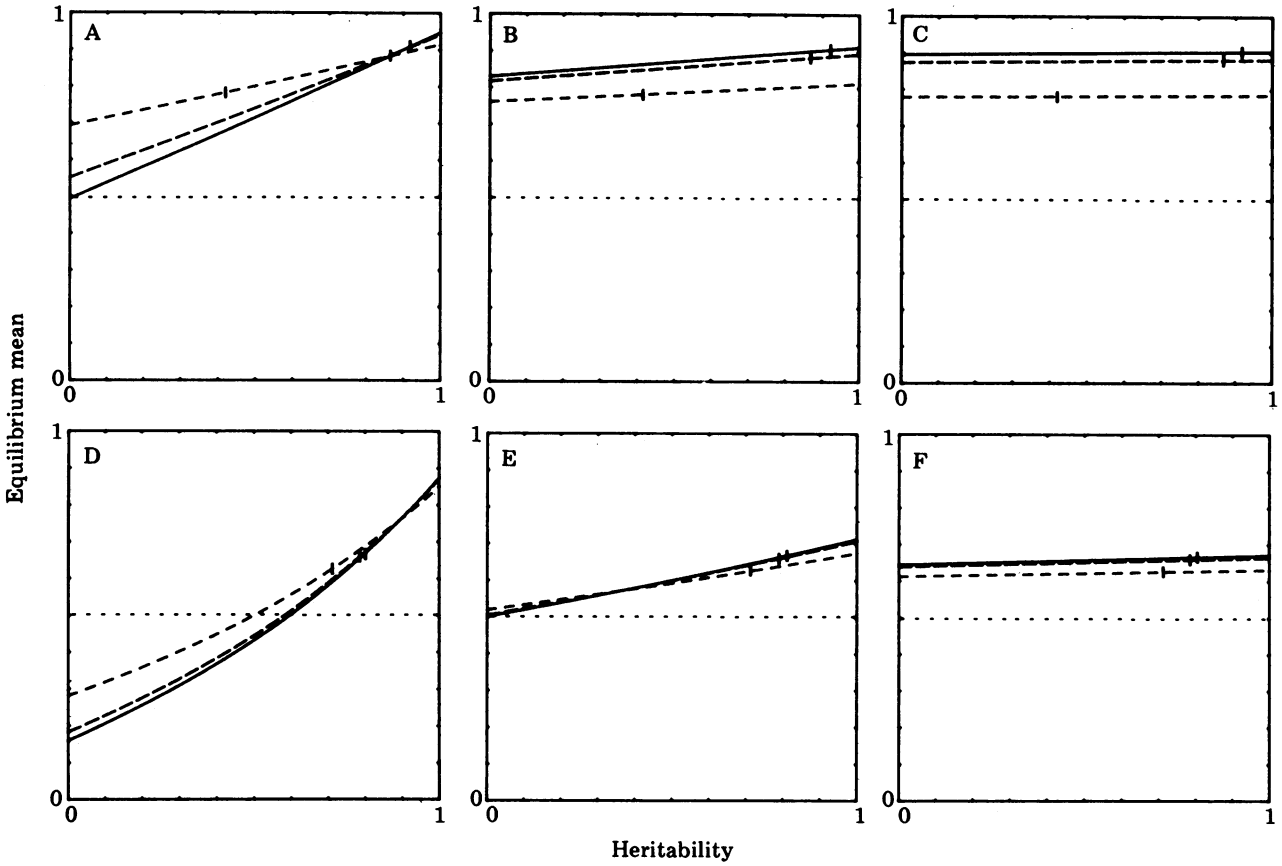

FIG. 1. Equilibrium mean as a function of the heritability. In all cases $\mu_\gamma = 0$, $\mu_s = 1$, and $S = \gamma$ for the following values of W_n and r: $W_n = 0.1, r = 0.1$ (*A*); $W_n = 1, r = 0.1$ (*B*); $W_n = 10, r = 0.1$ (*C*); $W_n = 0.1, r = 0.5$ (*D*); $W_n = 1, r = 0.5$ (*E*); $W_n = 10, r = 0.5$ (*F*). —, $n = 5$; – –, $n = 25$; - - -, $n = 125$. The value of h^2 for which $\hat{\mu} = \mu_k$ is indicated by a vertical hash mark and $\mu_m = \frac{1}{2}$ by a horizontal broken line.

Solving Eq. **12** for the equilibrium mean, $\hat{\mu}$, yields

$$\hat{\mu} = \frac{\mu_s\{\gamma + a(1-\bar{r})\bar{r}\hat{F}'\} + \mu_\gamma\bar{r}\{S + (1-a)\hat{F}'\}}{\{\gamma + a(1-\bar{r})\bar{r}\hat{F}'\} + \bar{r}\{S + (1-a)\hat{F}'\}}, \quad [14]$$

in which the parameter a,

$$a = h^2/h_f^2 = (1 + (n-1)h^2r)/(1 + (n-1)r), \quad [15]$$

is the ratio of individual heritability ($h^2 = \hat{V}'/(\hat{V}' + E)$) to the heritability of group phenotypic mean (h_f^2), which is defined as "the proportion of the variation in means of group phenotypes that is made up of additive genetic variance" (10). With our assumptions $h_f^2 = (1 + (n-1)r)h^2/(1 + (n-1)rh^2)$. $\hat{F}' = (\hat{V}' + E)$ is the variance of phenotypic values in the population at equilibrium.

A comparison of Eq. **14** and Eq. **5** shows that the existence of phenotypic variance decreases the strength of both own phenotype and group phenotype selection relative to a monomorphic population. The impact of phenotypic variation on the relative strength of the two processes depends on the parameters $\bar{r}$ and a. Decreasing a increases the coefficient of μ_γ and decreases that of μ_s, thus moving $\hat{\mu}$ closer to μ_γ. Note that a is a decreasing function of h^2, and thus decreasing h^2 decreases $\hat{\mu}$. For values of h^2 near 1, we have $a \approx 1$ and therefore $\hat{\mu} > \mu_k$; for h^2 near 0 we have $a \approx 1/\bar{r}$ and therefore $\hat{\mu} < \mu_k$. In other words, complete heritability leads to less altruism than predicted by the $k > 1/r$ rule, but for low enough heritability there will be more altruism than predicted by the $k > 1/r$ rule. Also note that a is a decreasing function of n, thus one would expect that increasing n would move $\hat{\mu}$ closer to μ_γ than predicted by the ESS result. This is quite contrary to the ESS result, in which decreasing group size increases the direct effect of an individual's phenotype on the group mean, and therefore increases the amount of altruism.

The values of $\hat{\mu}$ that result from various parameter combinations are shown in Fig. 1. This figure illustrates the quantitative impact of the different parameters. We have assumed (without loss of generality) that $\mu_\gamma = 0$ and $\mu_s = 1$. We have also assumed that $\gamma = S$. In each panel of the figure the equilibrium mean is plotted as a function of the heritability $h^2 = \hat{V}'/\hat{F}'$ for $n = 5$, 25, and 125. The value of μ_k (the conventional ESS result) appropriate to the values of n and r is marked by a vertical hash mark. *A–F* of Fig. 1 assume different values of r and $W_n = S/\hat{F}'$. W_n is a measure of selection intensity.

Several qualitative features are evident. When selection is weak ($W_n = 10$), changes in the heritability of the character have almost no effect on the equilibrium mean of the phenotype, a value that is well predicted by the $k > 1/r$ rule. Increasing group size has the usual effect of decreasing the amount of altruism. As selection becomes stronger, the effect of h^2 on $\hat{\mu}$ becomes more apparent. For very strong selection ($W_n = 0.1$) this effect can be quite substantial. For example in Fig. 1*A*, for small values of h^2, $\hat{\mu} \approx \mu_m$, the group selection result even though $r = 0.1$. Also note that here the effect of increasing n is to strongly increase the amount of altruism. Increasing r increases the sensitivity of $\hat{\mu}$ to changes in h^2 but decreases it to changes in n.

Because the amount of genetic variance at equilibrium depends on the strength of selection, the values of h^2 and W_n are not independent. As is shown in Table 1, however, h^2 is much more sensitive to the mutation rate than to selection strength. At any assumed selection intensity, a wide range of heritabilities is consistent with reasonable values of M/E (8). Table 1 also

Table 1. Equilibrium values of $h^2 = \hat{V}'/\hat{F}'$ and the selective mortality, S_m, for $r = 0.5$, $n = 5$, and different combinations of M/E and W_n

	$M/E = 1$		$M/E = 0.1$		$M/E = 0.01$		$M/E = 0.001$	
W_n	h^2	S_m	h^2	S_m	h^2	S_m	h^2	S_m
0.1	0.60	0.85	0.21	0.83	0.05	0.82	0.01	0.81
1	0.71	0.38	0.32	0.40	0.08	0.40	0.01	0.41
10	0.90	0.07	0.57	0.07	0.14	0.07	0.01	0.07

These values are relatively insensitive to changes in r and n.

gives the selection intensities associated with $W_n = 0.1$, 1, and 10, shown for different values of M/E. These values are also relatively insensitive to changes in r and n. It is important to note that these results, unlike those shown in Fig. 1, require solution of Eq. 13 for the equilibrium variance and thus depend on the particular quantitative character model chosen.

DISCUSSION

In the model analyzed in this paper a quantitative character was subjected to two opposing selective processes, one culling on the basis of individual phenotypes and the other on the basis of mean phenotypes of local groups. When the mutation rate and amount of environmental variance are small enough so that the population is approximately monomorphic, the resulting equilibrium is consistent with the $k > 1/r$ rule derived for discrete characters. Increasing the phenotypic variance can have one of two effects, depending on the relative importance of environmental and (additive) genetic variance. Heritabilities near one lead to less altruism than predicted by the $k > 1/r$ rule and those near zero lead to substantially more altruism. Both of these effects, however, become important only when selection is very strong.

These results provide a good example of the usefulness of considering kin selection as a kind of group selection. The response of a quantitative character to ordinary individual selection is proportional to the heritability of that character. Similarly, the response of a character to selection based on the phenotypic mean of genetically related groups depends on the proportion of additive genetic variation that underlies the variation in the phenotypic means of kin groups, the family heritability. Assuming that the environmental effects on different individuals are uncorrelated, the family heritability can be larger than the individual heritability because the phenotypic mean "averages out" the environmental effects. Thus at low individual heritabilities and large family sizes group phenotype selection can be stronger than individual phenotype selection. This fact has long been known to animal breeders, who sometimes find it more efficient to select whole sibships on the basis of the mean value of some trait than to select individuals (10). In essence what we are arguing here is that in nature these processes will often select in different directions. The nature of this selection process is perhaps made even more clear if one notes in Fig. 1 *A* and *D* that for small h^2, $\hat{\mu} < \mu_m$; that is, the mean of the phenotype is nearer to that favored by selection between families (μ_γ) than the value that maximizes mean fitness (μ_m)!

Although the model presented here and that of Yokoyama and Felsenstein (1) are similar in the assumed population structure, the results obtained are somewhat different. Yokoyama and Felsenstein consider an exponential fitness function of the form

$$W(f_1,\bar{f}) = \exp(-\alpha f_1 + \beta\bar{f}) \qquad [16]$$

and ask for what values of α, β, and n will the mean value of f in the population increase—i.e., favor the increase of altruism. If one assumes, as we have done here, that the phenotypic correlations result solely from genetic correlation, then their condition for altruism to spread is independent of h^2, contrary to our result for strong selection. We believe that this independence results from a special property of exponential fitness functions, namely, the expected fitness of an individual of genotype g_1 is independent of both the mean and the variance of the trait in the population.

It would clearly be of interest to extend these results to include the case in which phenotypic correlations are due to nongenetic causes. It will be important to distinguish interaction based on phenotypic assortment and correlated environments from that due to maternal effects or cultural transmission. In the latter case, it seems likely that a cultural analog of kin selection will also act to increase the frequency of altruistic phenotypes in the population.

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