ON THE THEORY OF PARENT-OFFSPRING CONFLICT: A TWO-LOCUS GENETIC MODEL

Trivers (1974) suggested that although natural selection should operate to increase the ability of parents to care for their young, parent-offspring conflict may arise and be explained on the basis of Hamilton's inclusive fitness rule (Hamilton 1964). Thus, while the relatedness of a parent to each offspring, or of one offspring to its sib is one-half, the relatedness of an offspring to itself is one. (For a detailed study of the appropriate definition of relatedness, see Uyenoyama and Feldman [1981] and Uyenoyama et al. [1981].) An event which increases an offspring's viability by η, say, and decreases that of its sibs by less than 2η will increase the former's inclusive fitness and therefore be desired by him. However, a decrease in the parents' total reproductive success may occur and the result is the parent-offspring conflict in fitness.

Alexander (1974) challenged this argument, claiming that such "selfish" alleles (in the offspring) would not spread because they would reduce the fitness of their carriers when the latter became parents and had selfish offspring. Alexander further suggested (see also West Eberhard 1975) that such parent-offspring conflict would be reduced by the asymmetry between the parties to the conflict; parents can force offspring to behave in a way which would increase brood survival thereby increasing the parent's fitness. However, Zahavi (1977) pointed out that such punishment by parents of their selfish offspring, either active or by withdrawal of resources, would be difficult to accomplish without a reduction of the parents' reproductive success.

Standard models of sib-to-sib altruism which usually involve brood selection, can be equivalently rephrased in terms of offspring-to-parent altruism. Such altruism may be considered outside of the usual context that originated with consideration of the social Hymenoptera. Thus, for example, increased efficiency in using a parent's limited resources at the expense of higher viability or reproductive success in the future might be regarded as altruism. Conversely, the delayed attainment of maturity at the expense of present or future sibs may be regarded as selfish. In this paper the terms "handicapped" and "selfish" are used interchangeably although it is realized that, in a behavioral sense, a selfish individual may not be handicapped. It has been suggested by Zahavi (1977) that parents of such handicapped offspring may allocate a disproportionate share of their resources to them, to reduce the mortality of the brood containing such progeny.

The selfish phenotype in the offspring and the modified parental behavior in the parent would be complex traits for which models developed in terms of only a few genes may not apply. Nevertheless, some aspects of the conflict between the selection of genes which act in offspring (but are carried and passed by parents) and those which act in parents (but are also carried by offspring) are worth investigation, as long as the disparity between the simplicity of these models and

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the complexity of the behavior is understood. Thus if the handicapped phenotype is the result of a single gene we can ask in terms of an exact population genetic model whether the absolute number of viable offspring (either selfish or altruistic) is the trait selected for among the parents. In the same way, we can focus on parental interactions with the offspring genotypes to ask how genes controlling such interactions with selfish offspring might evolve.

Here the term sib-to-sib altruism is used in the sense of offspring-to-parent altruism, with the selfish type of offspring reducing its parents' fitness. Parents who respond to selfish offspring by withdrawing resources will be termed *interfering*. These parents are less active in their assistance of handicapped offspring which, as a result, suffer a fitness reduction. Selfish offspring of *noninterfering* parents do not suffer from this withdrawal of parental resources. In this sense, interference is tantamount to active punishment (Alexander 1974). Our model of parental interference will involve a single locus linked to that which determines the progeny phenotype. Our central question is whether an allele which increases parental interference against selfish offspring can succeed. Such an allele effectively increases parental immunity to manipulation by handicapped offspring. Thus the question might also be asked in terms of how much of the parents' resources previously allocated to the handicapped offspring must be redistributed among the whole brood before interference can succeed. In order to answer this question exact population genetic recursions for the two loci simultaneously are used. We ask whether an allele causing parental interference in its heterozygote carriers will increase in frequency when it is rare. This allele could arise when the locus governing the offspring phenotype is fixed on one phenotype or is polymorphic. We show that the evolution of the parental behavior depends on which of these latter starting conditions is assumed.

THE MODEL

Consider two loci with alleles A,a at the first and B,b at the second in a diploid random mating population. The first locus determines sib-to-sib interactions which result in kin selection. The genotype at the second, which at the outset is fixed on the allele b, determines the parental phenotype which can be called interference. A parent of genotype bb does not affect the outcome of the sib-to-sib interactions due to the first locus. Bb parents, however, cause a reduction δ in the fitness of each selfish offspring, a fraction $\theta\delta$ of which is then redistributed among all of the offspring in the brood. The kin selection among the offspring is of the additive type, with loss γ in fitness incurred by altruists and gain proportional to β enjoyed by the recipients (see Cavalli-Sforza and Feldman 1978; Uyenoyama and Feldman 1981).

Table 1 defines the fitness of the phenotypes as offspring according to the phenotype of a randomly chosen one of its parents, where ℓ is the frequency of altruists in the brood. Thus by helping the selfish offspring less, the parent reduces the latter's fitness by δ. Since there is a fraction $1 - \ell$ of selfish offspring in the brood, $(1 - \ell)\delta$ of the parental contribution is assumed to be redistributed, with a loss measured by θ, among all of the offspring.

TABLE 1

FITNESS EFFECTS OF PARENT INTERFERENCE

	PARENT STATUS	
	Without Parent Interference	With Parent Interference
Offspring altruistic	$1 - \gamma + \beta\ell$	$1 - \gamma + \beta\ell + \theta\delta(1 - \ell)$
Phenotype selfish	$1 + \beta\ell$	$1 - \delta + \beta\ell + \theta\delta(1 - \ell)$

In the terminology of Uyenoyama and Feldman (1981) the genotypes AA, Aa, and aa have probabilities h_1, h_2, h_3 of performing altruism. Using table 1 the fitness of an offspring whose probability of performing altruism is h_i, if born into a brood in which a fraction ℓ of the offspring are altruists and the parent interferes in the sense of table 1, is

$$\begin{aligned} w(h_i,\ell;\theta,\delta) &= h_i[1 - \gamma + \beta\ell + \theta\delta(1 - \ell)] \\ &\quad + (1 - h_i)[1 - \delta + \beta\ell + \theta\delta(1 - \ell)] \qquad (1) \\ &= 1 + \beta\ell - \gamma h_i + \theta\delta(1 - \ell) - \delta(1 - h_i). \qquad (2) \end{aligned}$$

At the outset the population is all bb so that the first column of table 1 applies, and the only selection operating is sib-to-sib altruism. The rare mutation B induces interference on the part of heterozygote Bb parents and the resulting fitnesses in their offspring are given by the second column of table 1. Our object is to study the initial increase properties of B when it is introduced near an equilibrium of the sib-to-sib altruism model assumed to operate with all parents bb. First we consider a fixation point of the sib-to-sib altruism model, and ask for conditions allowing simultaneous increase at both loci. The second situation follows the fate of B when it arises near a polymorphic equilibrium at the A/a locus.

Analysis at Fixation of the A/a *Locus*

Suppose that initially the population is fixed on Ab/Ab. (The argument for fixation on ab/ab is the same if h_3 is substituted for h_1.) We ask under what conditions will this equilibrium be locally unstable, in particular, so that B will enter the population. For this the full two-locus transformation in the 10 genotypes, including the recombination fraction r between the loci, is linearized in the neighborhood of the fixation of Ab/Ab and the eigenvalues of the local linear transformation determined. The significant eigenvalues are

$$\begin{aligned} \lambda_1 &= [1 + (\beta - \gamma)h_1 - \delta(1 - \theta)(1 - h_1)]/[1 + (\beta - \gamma)h_1], \\ \lambda_2 &= [1 + \beta(h_1 + h_2)/2 - \gamma h_2]/[1 + (\beta - \gamma)h_1], \qquad (3) \\ \lambda_3 &= (1 - r)[1 + \beta(h_1 + h_2)/2 - \gamma h_2 + \delta\theta\left(1 - \frac{h_1 + h_2}{2}\right) \\ &\quad - \delta(1 - h_2)]/[1 + (\beta - \gamma)h_1]. \end{aligned}$$

Here λ_1 governs the rate of increase of B when it is rare, λ_2 that of a, and λ_3 that of the aB combination. (This and the other local analyses reported here are technically similar to the two-locus boundary analyses of Bodmer and Felsenstein [1967].)

Consider first the case $h_1 < h_2$. This entails that AA is less altruistic than Aa and in terms of two-locus theory the initial fixation is described as a "selfish corner." In the absence of parental interference, $\lambda_3 < \lambda_2$ and there can be no geometric increase of either a or B if $\gamma > \beta/2$, i.e., if $\delta = 0$, $\lambda_3 < \lambda_2 < 1$ if $\gamma > \beta/2$. This is the classical result from one-locus kin selection theory, that the altruistic allele, a, cannot succeed if the loss in fitness is more than half of the gain. Now consider the case $r = 0$ and $\theta = 1$; the latter equality means that all of the fitness loss resulting from punishment of the selfish is redistributed among the brood, while the former gives the greatest opportunity for the double heterozygote to influence the stability properties. With these assumptions $\lambda_3 > 1$ if

$$\left(\frac{\beta}{2} + \frac{\delta}{2} - \gamma\right)\left(h_2 - h_1\right) > 0. \tag{4}$$

If $\beta/2 + \delta/2 > \gamma$, then both the altruistic allele a and the parental interference allele B will increase. In other words, parental interference can mediate the increase of "altruism" when this would not otherwise have occurred. By continuity, $\lambda_3 > 1$ for small positive r and θ close to but less than unity.

Now consider the opposite situation $h_2 < h_1$, fixation at the "altruistic corner", and again take $r = 0$, $\theta = 1$. If $\beta/2 - \gamma < 0$ then $\lambda_2 > 1$ and the selfish allele will increase. In this case, even if $(\beta/2 - \gamma + \delta/2) > 0$, so that $\lambda_3 < 1$ we still have $\lambda_2 > 1$ so that parental interference cannot force the stability of the otherwise unstable fixation. If the selfish allele would have increased without parental interference, the latter, even though it operates against the selfish phenotype, cannot prevent its increase.

Analysis at Polymorphic Equilibria of the A/a *Locus*

Under the conditions of the model the genotypes Ab/Ab, Ab/ab, ab/ab are maintained in equilibrium prior to the arrival of B at frequencies $\hat{u}$, $\hat{v}$, and $\hat{w}$ with gene frequency $\hat{p} = \hat{u} + \hat{v}/2$. The rare genotypes, all heterozygous Bb are AB/Ab, AB/ab, Ab/aB, aB/ab with frequencies ϵ_1, ϵ_2, ϵ_3, ϵ_4, respectively, which we denote by the vector $\boldsymbol{\epsilon}$. In the neighborhood of $(\hat{u},\hat{v},\hat{w})$ the linearized recursion system breaks into two parts, one of which controls the stability of $(\hat{u},\hat{v},\hat{w})$ as a one-locus polymorphism while the other controls the initial increase of B. The latter is a 4×4 matrix which has the form $\boldsymbol{\epsilon}' = \mathbf{A}\boldsymbol{\epsilon}$ where $\mathbf{A} = \|a_{ij}\|_{i,j=1}^{4}$. In general, the stability of $(\hat{u},\hat{v},\hat{w})$ to the increase of B is governed by the spectral properties of $\mathbf{A}$. For $r > 0$ we have been unable to make much progress with the eigenvalues of $\mathbf{A}$. But when $r = 0$, $\mathbf{A}$ splits into two 2×2 blocks each of which is strictly positive matrix and the analysis can proceed.

Uyenoyama and Feldman (1981) showed that there are two classes of equilibria for the sib-to-sib altruism model with only b present described above. The first has the form

$$p^* = (h_2 - h_3)/(2h_2 - h_1 - h_3), \tag{5}$$

and has been termed *viability-analogous* by Uyenoyama et al. (1981), because of the analogy with the one-locus selection model with viabilities h_1, h_2, and h_3 for AA, Aa, and aa. The second is a more complicated class for which the relation

$$pq(\beta - \gamma) = \beta v/4 \tag{6}$$

is satisfied, together with the quadratic in the gene frequency,

$$p^2[(\beta - \gamma)^2(2h_2 - h_1 - h_3)] + p[(\beta - \gamma)^2(h_1 + h_3 - 2h_2) + (h_1 - h_3)(\beta - \gamma)(2\gamma - \beta)] + (2\gamma - \beta)[1 + h_3(\beta - \gamma)] = 0. \tag{7}$$

This second class has been called *structural* to suggest its relations to the interactive fitness structure of the kin selection models and, as first discussed by Cavalli-Sforza and Feldman, can include stable equilibria.

The fate of the mutation B depends on which of these two equilibrium classes describes the population before its arrival. We assume that the equilibrium prior to the arrival of B is stable. Analytic conditions for this stability were given by Uyenoyama and Feldman (1981, eq. 52) for the viability-analogous but not for the structural equilibria.

Result 1. Initial increase from the viability-analogous equilibrium.—A sufficient condition for the instability of the viability-analogous equilibrium to invasion by B with $r = 0$ is

$$\delta(1 - \theta)(1 - h^*)\{1 + (\beta - \gamma)h^* + (2h_2 - h_1 - h_3)(\beta - \delta\theta)[4p^*(1 - p^*) - v^*]/8\} < 0 \tag{8}$$

where $h^* = h_1(p^*)^2 + 2h_2p^*(1 - p^*) + h_3(1 - p^*)^2$ and v^* is the equilibrium heterozygote frequency corresponding to p^*. Note that the left side of (8) vanishes when $\theta = 1$. Qualitatively this means that when the fitness loss δ to the selfish offspring resulting from parent interference is just compensated for by increase in fitness to the whole brood there can be no geometrically fast change of B frequency. Further, a careful comparison of the left side of (8) with the equilibrium value of v^* given by Uyenoyama and Feldman (1981, eq. 50) reveals that if $\delta < 1$, B cannot increase when rare if $\theta < 1$. The assumption $\delta < 1$ (and $\gamma < 1$) ensures that the fitnesses remain positive.

Result 2.—Initial increase from the structural equilibrium.—The analysis in this case is rather tedious and for ease of writing we concentrate on the relatively simple case $h_1 = h_2$. Recall that when $r = 0$ the local stability matrix for initial increase of B decomposes into two 2×2 matrices corresponding to the rare genotype AB/Ab and AB/ab for the first with Ab/aB and aB/ab for the second. Considering the first of these matrices after some algebra it is found that one of its two eigenvalues is larger than unity if

$$\frac{\gamma}{\beta}(1 - h_2)\delta^2\theta(\theta - 1)\hat{p}\hat{q}(h_2 - h_3)/2 - \delta(\theta - 1)(1 - h_2)[1 + (\beta - \gamma)\bar{h} + \gamma\hat{p}\hat{q}(h_2 - h_3)/2] + \theta\delta(h_3 - h_2)\hat{q}^2[1 + (\beta - \gamma)(\hat{p}h_2 + \hat{q}h_3) + \frac{(\beta - \gamma)^2}{\beta}\hat{p}\hat{q}(h_2 - h_3)] < 0. \tag{9}$$

Since our qualitative interest resides in whether B can increase even when it causes a fitness loss to the brood, $\theta < 1$, we can examine (9) near $\theta = 1$. The dominant contribution to the left side of (9) near $\theta = 1$ comes from the last term on the left. Thus, if $h_1 = h_2 > h_3$, near $\theta = 1$ the inequality (7) holds and selection operates in favor of intervention by parents. If $h_1 = h_2 < h_3$ then the first matrix has both eigenvalues less than unity. Now consider the second matrix which corresponds to the case where the mutation affecting the parents is linked to the recessive allele for offspring altruism. Under the condition $h_1 = h_2$, the second matrix has its largest eigenvalue greater than unity at $\theta = 1$ if

$$\delta(h_2 - h_3)\frac{(\beta - \gamma)\hat{p}\hat{q}}{\beta}[1 + (\beta - \gamma)(\hat{q}h_3 + \hat{p}h_2) + \frac{\hat{q}(h_2 - h_3)}{2}(\delta - \beta)] < 0. \tag{10}$$

If $h_1 = h_2 < h_3$, (10) is satisfied under the usual assumptions and we conclude that the structural equilibrium is unstable for an interval of values of $\theta < 1$.

DISCUSSION

The quantitative results obtained above are not as decisive as might have been desired in view of previous claims about the evolution of parent-offspring conflict. Under specified circumstances we have shown that altruism and parental interference may evolve in association while under other specified conditions they may not.

The distinction between the structural and viability-analogous equilibria in their stability to invasion by alleles causing parental interference is of considerable interest. Only from the structural equilibrium can parental interference evolve. Although this is rigorously proven only under the assumption of dominance at the offspring altruism locus we conjecture that this difference between the two classes of equilibria is more generally true. The reason for this conjecture is that Uyenoyama et al. (1981) have shown that at the viability-analogous equilibrium the additive genetic variance is zero which means, of course, that the parent-offspring covariance vanishes. Thus selection based on parent-offspring interaction, with $\theta = 1$, would intuitively be expected to have no effect. On the other hand at the structural equilibrium there is additive genetic variance and parental interference is likely to interact with the kin selection among offspring.

The inclusive fitness method (i.e., analysis in terms of gene frequencies alone) can produce the viability-analogous equilibria (Michod and Abugov 1980) but not the structural equilibria, which require analysis in terms of genotype frequencies (Uyenoyama and Feldman 1981). Thus the inclusive fitness method cannot be expected to lead to accurate analysis of the parent-offspring conflict problem. For example from the structural equilibrium an allele will increase which causes a reduction of the parent's expected number of offspring. No other relatives compensate at this generation for this loss of parental genes represented in the next generation. Thus, by definition, there is a loss of parents' inclusive fitness.

Passage to third generation arguments may resolve this apparent paradox. Near the selfish fixation and the structural equilibrium the positive parent-offspring covariance entails that an altruistic offspring will have, on average, more altruists in its own brood and therefore, more surviving offspring of its own. Thus helping

an altruistic offspring, even when this results in a slight decrease in the parent's total number of viable offspring, may increase its expected number of viable grand-offspring. This heuristic argument is similar to Fisher's argument on the evolution of the sex ratio (Fisher 1930). Eshel and Cohen (1976; see also Motro 1981) suggest that the very concept of inclusive fitness should be modified to take account of differences in expected contribution to future generations made by the various recipients of the help. However, it is unlikely that even this modified concept can help make predictions in a multidimensional situation like that modeled here. In the same way, departures from single-locus or nonepistatic multilocus theory (Eshel, MS) result in the failure of ESS arguments. Departure from Hardy-Weinberg frequencies and linkage introduce complications, intrinsic to the exact genetical model, and which profoundly influence even the way in which the questions are asked. For example, with free recombination fixation of selfish offspring (AA) and noninterfering parent (bb) will usually be stable, apparently in agreement with Zahavi's verbal argument. But this equilibrium may be unstable when the loci are tightly linked, apparently in agreement with the verbal arguments of Alexander and West Eberhardt.

Finally, we draw attention to the possibility of coadaptive evolution from the Ab/Ab fixation. Here the altruistic allele is permitted to evolve by virtue of its linkage to the allele which induces parents to withhold resources from their selfish offspring. On the other hand, parental interference cannot induce the stability of an otherwise unstable fixation in an altruistic allele. Together these two remarks entail that parental interference itself can, under conditions that would otherwise have resulted in fixation of the selfish allele, induce protected polymorphism at the locus for offspring behavior.

The reasons for restriction of the analysis to $r = 0$ are purely technical, and our answers extend by continuity to small positive values of the recombination. It is generally true, however, that initial increase of a rare allele at a locus linked to a polymorphism becomes less likely when linkage is looser (Bodmer and Felsenstein 1967). Similarly, initial increase of rare alleles at both loci from a state of fixation in one chromosome is less probable when linkage is looser. Thus although we set $r = 0$ to avoid analysis of high order polynomials, it is a situation most favorable to the increase of parent-interference.

In the same way, $\theta = 1$ should be most favorable to the evolution of parental interference because it entails redistribution among a brood of all the fitness removed from the handicapped individuals. However, the results will remain qualitatively true for θ close to but less than unity, that is when the allele determining parent interference causes a decrease in its carrier's fitness.

The complex dependence of these results on assumptions about initial conditions and parameters values in both offspring and parents may be disagreeable to some. However, this complexity lends emphasis to our admonitions elsewhere against the acceptance of verbal theory without careful quantitative evaluation.

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