The Evolution of Altruism by Kin Selection: New Phenomena With Strong Selection

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The development of a theory of kin selection has proceeded along two lines. Inclusive-fitness models have implicitly assumed that selection is weak, whereas exact-population genetic models place no constraints on the strength of selection. Several examples are presented showing that qualitatively new behavior has emerged from the exact models. However, for many problems, the exact-population and inclusive-fitness models often yield identical results. Unfortunately, it is not possible to identify a priori those problems that can be handled sufficiently by the simpler inclusive-fitness models. The initial increase of cooperative behavior in a population of egoists involves difficulties similar to the initial increase of altruism. Clustering of cooperatives produces dynamics for the increase of cooperation that are formally similar to population models of inbreeding. Here, an increase in the tendency to cluster is equivalent to increasing the "relationship" among cooperatives, and therefore augments the chance for cooperation to increase.

KEY WORDS: Inclusive fitness; Exact population models.

INTRODUCTION

Altruistic behavior appears to be common among humans and other species of animals. Although the meaning of "altruism" may vary, we first illustrate the phenomena with examples from several diverse animal groups, and later we present a precise definition that forms the basis of our population genetic models.

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Several bird species among the swifts, kingfishers, and bee eaters exhibit cooperative breeding in large colonies (Emlen 1978). Studies of the white-fronted bee eater (*Merops bulockoides*) show that more than 70% of nests are tended by helpers. These helpers take part in nest excavation, incubation and feeding and defending the young.

Perhaps the most extreme examples of cooperation and altruism are the social insects. In many species of bees and ants, female workers forego reproduction in order to raise the queen's offspring. Honeybees will often die upon stinging a perceived intruder. Such heroic acts extend to raising young such that worker honeybees will metabolize their own tissue protein to feed larvae when fed only sugar water (Wilson 1971).

To many, altruistic behavior of the sort just described seemed contrary to the theory of natural selection. How could such sacrifice result from a process favoring genotypes that leave large number of offspring? Haldane (1955), Williams and Williams (1957), Hamilton (1964) and Maynard Smith (1964) all recognized that individuals can transmit their genes to the next generation by having their own offspring or by helping close relatives have many offspring. Hamilton quantified these ideas in what has become known as Hamilton's rule,

$$\beta b_{AR} > \gamma. \tag{1}$$

This rule states that behavior directed from altruist to recipient will be favored by natural selection if the gain in fitness (β) experienced by the recipients discounted by a coefficient of relatedness (b_{AR}), between altruist and recipient, is greater than the loss in fitness (γ) suffered by the altruists. If b_{AR} is interpreted as the probability that the recipient contains an allele identical to one in the altruist, equation (1) can be restated: if the behavior causes relatives to produce βb_{AR} more "altruist" alleles, and altruists to produce γ fewer "altruist" alleles, the behavior will be favored by natural selection if there is a net gain in "altruist" alleles, for example, $\beta b_{AR} - \gamma > 0$, which is simply equation (1).

The major qualitative inference from Hamilton's rule is that altruistic behavior is more likely to evolve among closely related individuals than among distant relatives. This has been invoked to explain the unusual concentration of sociality among the insect *Hymenoptera*. The genetic system of these insects is haplo–diploid. Females emerge from fertilized, diploid eggs, whereas males are haploid and develop from unfertilized eggs. Sisters in diploid species have a coefficient of relatedness of $\frac{1}{2}$, whereas this quantity is $\frac{3}{4}$ in haplo–diploid species. Multiple insemination, however, may lower this figure substantially.

The appearance of sociality among diploid termite species might be explained by similar phenomena, although this is still a matter of contention (Crozier and Luykx 1985). In several species of termites, nearly 50% of the genome occurs in translocations of the X chromosome (Lacy 1980). This

makes it more likely for sisters to carry the same altruistic alleles, since they all inherit the same X chromosome from their father.

Although Hamilton's rule and the heuristic justification we have provided are intuitively attractive, we know that natural selection in general does not simply maximize the number of copies of the "best" allele or even the mean fitness of a population. To actually quantify the action of natural selection, we need to take into account such things as Mendelian genetics and differences in the fitness of alternative genotypes.

Two different approaches have been used in the development of population genetic models of kin selection. The inclusive fitness models (Charnov 1977; Charlesworth 1978; Wade 1979; Michod and Abugov 1980; Abugov and Michod 1981) implicitly assume that selection is weak and, therefore, genotype frequencies will remain in Hardy–Weinberg proportions after selection. The exact population genetic models (Levitt 1975; Cavalli-Sforza and Feldman 1978; Uyenoyama and Feldman 1981; Uyenoyama et al. 1981; Toro et al. 1982; Matessi and Karlin 1984) can accommodate selection of any intensity. The exact models are substantially more difficult to analyze and have yielded results that are sometimes at odds with Hamilton's rule. This state of affairs has lead Maynard Smith (1983) to suggest that "the main service that population geneticists can perform is to specify the circumstances in which inclusive fitness methods can safely be applied."

The theory of reciprocal altruism, originated by Trivers (1971), was certainly eclipsed as an explanatory force for behavioral ecologists by the theory of kin selection. This hiatus lasted about 10 years but has given way to a new wave of activity connected with the evolution of cooperation. This renewed focus of interest on reciprocal altruism derives from the application of rational choice models to animal behavior by Axelrod and Hamilton (1981). The framework for this is the continued prisoner's dilemma game, and does not include genetic contributions to the cooperation–noncooperation dichotomy. There are, however, ways in which genetics might be included in these models for the evolution of cooperation. Axelrod (1981) suggested that clustering of cooperative individuals would increase the chance of their invasion. If the dichotomy were genetic, this would be equivalent to assortative mating (Eshel and Cavalli-Sforza 1982) or possibly some form of inbreeding.

An alternative genetic framework would take one or more of the parameters in the prisoner's dilemma model to be under genetic control, namely, the payoffs or the discount rate. Then one might ask whether genes that result in a higher (or lower) discount rate would be favored in evolution. The analysis of such issues could involve integration of the approach we use in this article in studying the evolution of parameters of kin selection, with some of the ecological models of cooperation discussed by the other authors in this volume.

In this article, we review published exact-population genetic models and present a new model whose object is to determine if their predictions

could have been anticipated from the simpler inclusive fitness models. Two major conclusions follow from these examples. First, qualitatively new behavior is seen in the exact-population models that is not present in inclusive-fitness models. The models differ in ways other than just their quantitative predictions of allele frequencies (Grafen 1985). Second it is nearly impossible to predict a priori which problems can be completely understood with inclusive-fitness models. We conclude that the development of exact models of kin selection must be viewed as an important and necessary step for progress in this field.

POPULATION GENETIC MODELS

Notation

We allow for n different alleles at a single locus. These alleles are denoted $A_1, A_2, \ldots, A_n$. The frequencies of these alleles in the population are p_1, $p_2, \ldots, p_n$. Lastly, we let genotype A_iA_j occur with frequency g_{ij}.

Life Cycle

The organism reproduces at discrete intervals, in which there is random mating and an infinite population size. The life cycle is given below.

$$\underset{g_{ij} = 2p_ip_j}{\text{Zygotes}} \xrightarrow[\text{Viability Selection}]{\text{Altruism}} \underset{g'_{ij}}{\text{Adults}} \xrightarrow[\text{mating}]{\text{random}} \underset{g'_{ij} \times g'_{ij}}{\text{Zygotes}}$$
$$\text{etc.}$$

Initially, the zygotic genotypes are in Hardy–Weinberg equilibrium. However, after selection, the adult genotypes are not in Hardy–Weinberg proportion and, hence, genotype frequencies must be specified in the exact models so that the precise frequencies of all families can be computed. The inclusive fitness models approximate adult genotype frequencies, g'_{ij}, with their Hardy–Weinberg expectations.

Genotypes and Phenotypes

Hamilton's original model assumes, implicitly, that genotypes are either always selfish or altruistic. Here, we allow genotype A_iA_j to be altruistic with probability h_{ij} where $0 \leq h_{ij} \leq 1$.

Fitness

Each genotype will have two independent components to its net fitness. Fitness will be incremented by altruism received by the genotype and decremented as a result of altruism performed by the genotype. This increment

will be equal to βf_{ij}, where f_{ij} is the probability that genotype A_iA_j has an altruistic relative (sister, brother, sib, etc.) and will be decremented by γh_{ij}. As pointed out by Cavalli-Sforza and Feldman (1978) there are two natural ways in which fitness might be computed. The additive model sets the fitness of genotype A_iA_j, w_{ij}, proportional to

$$w_{ij} \propto 1 - \gamma h_{ij} + \beta f_{ij},$$

whereas the multiplicative model has

$$w_{ij} \propto (1 - \gamma h_{ij})(1 + \beta f_{ij}).$$

These formulations are not equivalent, and Hamilton's rule is most consistent with the additive formulation.

RESULTS

Abugov and Michod (1981) have shown that identical expressions are obtained for allele frequency dynamics from Hamilton's model and the inclusive-fitness models. Thus, these two models can be considered equivalent with respect to important predictions they might make.

Two basic types of questions have been addressed using the exact-population genetic models of kin selection. The initial increase question asks under what conditions a rare mutant allele, which affects altruism, will be able to increase in frequency. Cavalli-Sforza and Feldman (1978), Uyenoyama and Feldman (1981) and Uyenoyama et al. (1981) drew attention to polymorphic equilibria and to the importance of fixation on the altruistic allele.

For the initial increase problems, Hamilton's theory predicts that a rare genotype that is always altruistic should increase in frequency in a selfish population if (1) holds. When genotypes are altruistic to varying degrees, equation (1) can be modified to: $(h_{12} - h_{11})\beta b_{AR} > 0$. Cavalli-Sforza and Feldman (1978) have shown that the multiplicative, exact-population-genetic models yield initial increase conditions that are not simply expressible in terms of relationship coefficients. Cavalli-Sforza and Feldman have also shown that initial increase conditions for the additive model are more often consistent with Hamilton's theory.

The exact single locus models yield two types of polymorphic equilibria (Uyenoyama and Feldman 1981; Uyenoyama et al. 1981). The *viability analogous equilibrium* is characterized by equilibrium allele frequencies that can be determined by treating the h_{ij}s as viabilities and using the standard results from single locus viability models (Ewens 1979, chap 2). Equilibrium allele frequencies for the *structural equilibria* are usually solutions to quadratic or higher order polynomials and depend on the h_{ij}s, β and γ. Only the viability analogous equilibria can be extracted from the inclusive-fitness models. Regardless of this similarity, the stability conditions for the viability analogous

equilibria depend on the population genetic model used. For an inclusive-fitness sib–sib model, Michod and Abugov (1980) show that viability analogous equilibrium for two alleles is stable when $\beta/2 > \gamma$ and $h_{12} > h_{11}, h_{22}$. The conditions for its stability from the exact-population genetic models are more complicated (Uyenoyama and Feldman, 1981). In addition to the previous result from the inclusive-fitness model, the exact models also predict stability in special cases of the viability analogous equilibrium when,

$$\beta/2 < \gamma \text{ and } h_{12} > h_{11}, h_{22}, \tag{2a}$$

or

$$\beta/2 > \gamma \text{ and } h_{12} < h_{11}, h_{22}. \tag{2b}$$

It should be noted that when conditions (2a) and (2b) hold, in addition to the stable polymorphic equilibrium, there are states corresponding to fixation of the A_1 or A_2 allele that are locally stable. Thus, it would seem that in a monomorphic population, these polymorphic equilibria could never be reached unless some stochastic process leads to the introduction of large numbers of the alternative allele. We see below that this is not the only possibility.

It is clear that exact-population genetic models predict different stability conditions for the viability analogous equilibrium than do the inclusive-fitness models and yield additional polymorphic equilibria. We next address this very important question: at a theoretical level, do the additional complications of the exact-population genetic model result in interesting biological phenomena that could not have been predicted or anticipated from the simpler inclusive-fitness models? Our answer is a resounding yes.

NEW PHENOMENA FROM THE EXACT POPULATION GENETIC MODELS

Altruism With Inbreeding

It has been suggested (Hamilton, 1964) that inbreeding may facilitate the evolution of altruism. Uyenoyama (1984) has examined a number of exact models that incorporate regular systems of inbreeding such as selfing, parthenogenesis, and sibmating. Although her primary finding has been that inbreeding does not always promote altruism, and indeed may actually make it more difficult to evolve, she also shows that genetic polymorphisms are possible with inbreeding. Nevertheless, we find that these polymorphic equilibria are all structural and thus, presumably, would not be present in the analogous inclusive-fitness models. The exact-population genetic models have thus lead to a qualitatively new result concerning the existence of polymorphisms with inbreeding.

Parental Interference

The arguments presented previously to study the evolution of altruism can be reversed to study the evolution of selfish behavior. Thus, for interactions between full sibs, selfish behavior should evolve if $\beta/2 < \gamma$. Alexander (1974) claimed that such selfish behavior among sibs could be prevented by parental interference. Since it is not in the best interests of the parent to produce a selfish brood, resources could be withheld from the selfish offspring and the consequent decrease in fitness would prevent the spread of these selfish alleles. To study this problem, Feldman and Eshel (1982) constructed a two-locus model in which one locus controlled the propensity of an individual to be altruistic as a sib and the second locus determined whether a parent would interfere with its offspring. Feldman and Eshel then show that the evolution of parental interference may depend on the particular equilibrium at the altruism locus. When the population is at a viability analogous equilibrium for the altruist alleles, parental interference will not evolve under the conditions examined. However, it is possible for parental interference to evolve when the population is at a structural equilibrium. Since inclusive-fitness models do not produce the structural equilibria, they would fail to uncover this interesting phenomena.

Two-Locus Kin Selection

The first detailed analysis of an exact two-locus, sib–sib, model of kin selection was conducted by Mueller and Feldman (1985). This model assumes that genotypes are altruistic with probability h_2, h_1 or h_0, depending on whether the genotype is heterozygous at both loci, one, or neither locus. In that article it was shown that a monomorphic selfish population could become polymorphic and more altruistic even when $\beta/2 < \gamma$. In particular, a population that is monomorphic at both loci may be unstable to the introduction of alternative alleles at each locus when $\beta/2 < \gamma$, $h_1 > h_0$ and linkage is neither too tight or too loose. Consider the following example: $\beta = 1.9$, $\gamma = 1.0$, $h_0 = 0$, $h_1 = 1$, $h_2 = 0.1$ and $0.001 < r < 0.47$, where r is the recombination fraction. In a population fixed for the A and B alleles, the introduction of small numbers of a and b alleles results in an initial increase. This population ultimately converges to a viability analogous equilibrium at one locus, and fixation at the second. Recall the earlier summary of the single locus theory. For the parameter values given above, the fixation states and the viability analogous equilibrium were locally stable and the population could not move from an allele fixation to the viability analogous equilibrium. The addition of a second locus controlling altruism provides such a mechanism, and thus the stable equilibrium unattainable from the single locus theory is in fact an attainable state under a more complicated genetic system. Needless to say, none of this behavior could be observed with the inclusive-fitness models.

Uyenoyama (1987) has recently shown how initial increase conditions for two locus models may be summarized by a rule analogous to equation (1). However, the value of the coefficient of relatedness depends on current genotype frequencies and thus could not be properly predicted from inclusive-fitness models. Furthermore, Uyenoyama has shown that the ability of new alleles to increase in these two locus models can differ markedly depending on whether the population was initially at a single-locus structural or viability analogous equilibrium.

Loss Modification

All models considered so far have treated the loss (γ) and gain (β) components of fitness as fixed constants. It seems reasonable to assume that changes in physiology or behavioral repertoire may make certain altruistic acts less costly. To examine this problem, we have constructed a two-locus model in which the genotypes at the first locus (*AA*, *Aa*, aa) determine the probability of performing altruistic acts (h_1, h_2, h_3) and the genotypes at the second locus (*BB*, *Bb*, *bb*) determine the loss suffered by altruists ($k_1\gamma$, $k_2\gamma$, $k_3\gamma$). If we consider a population fixed for the *B* allele, we can determine the conditions necessary for the *b* allele to increase when rare, if the population is either monomorphic at the *A* locus, at a viability analogous equilibrium or a structural equilibrium at the *A* locus. The actual analysis of these problems is quite complicated (see Appendix) but in all cases examined, we find that the *b* allele will increase when rare, if $k_2 < k_1$. That is, if the loss suffered by the *Bb* heterozygotes is less than that suffered by the common *BB* homozygotes, the *b* allele will increase. Such a result is quite intuitive and probably could have been predicted from an inclusive-fitness model. The example serves to illustrate two points. First, as noted by Cavalli-Sforza and Feldman (1978) the exact models do not always produce results differing from the inclusive-fitness models. However, forcasting *a priori* which problems can be satisfactorily handled by the inclusive-fitness models does not seem to be possible. Examination of the detailed analysis of this loss modification model shows that certain results are intuitive only with hindsight.

These examples have demonstrated the usefulness of exact-population genetic models of kin selection for detecting phenomena of biological interest. In most cases, the inclusive-fitness models would not have uncovered the phenomena just mentioned and thus any extra work involved in developing exact-population genetic models would seem to be more than justified.

The Evolution of Altruism and Cooperation with Cultural Transmission

Cavalli-Sforza and Feldman (1973a, 1973b, 1981) and Feldman and Cavalli-Sforza (1975) developed a theory for the evolution of cultural transmission.

Pulliam (1982) showed how this theory could be used to model the evolution of cooperation and Werren and Pulliam (1981) used classical accounts of the degree of genetic relationship in a model that included a genetic component to cooperation. Boyd and Richerson (1982) extended these models to a situation of interdemic selection. Feldman et al. (1985) addressed the problem of culturally transmitted altruistic traits using the population genetic theory outlined above. Their model assumes that altruism may be learned from a parent and that the probability that an offspring is altruistic depends on the parents' phenotype and the offspring genotype. Their results show that nongenetic transmission may involve substantial departures from the predictions of the simple inclusive-fitness approach. Such results forcefully argue for the continued development of such models before any generalizations are made concerning the evolution of altruism in human populations.

A Comparison with Theory for the Evolution of Cooperation

The classical theory of kin selection is essentially one of initial increase of an allele that enhances altruistic behavior. There has been, by comparison, much less focus on conditions that produce fixation of such alleles, even though there is little or no evidence for variation in the behavior studied in most species to which the theory is applied. The polymorphic equilibria we have described above are important because they define parameter sets and delimit domains of attraction that allow progress to fixation. If the parametric conditions for initial increase and final fixation are the same, it does not follow that fixation always occurs.

It is interesting that the evolutionary theory of cooperation, via the prisoner's dilemma is primarily one of final fixation, not initial increase. In fact, Axelrod (1981) is clear that with random encounters, the cooperative strategy tit-for-tat (TFT) cannot invade a population of egoists. Nevertheless, empiricists have accepted the TFT paradigm in the same way they accepted the kin-selection theory regardless of the fact that they apply to opposite ends of the phenotype frequency spectrum.

Axelrod (1981) pointed out that, if intense enough, clustering of cooperatives can allow initial increase of TFT in an egoistical world. Feldman and Thomas (1987) have developed a dynamic theory for the prisoner's dilemma framework in which the probability of continuing to play depends on the history of the game. Polymorphisms arise naturally in this case and TFT may increase when rare. In the analysis, it is shown that clustering is formally analogous to population genetic models of mixed self-fertilization and random mating, except that the "fitnesses" are asymmetric. Thus, "relationship" among cooperatives is ensured and if this is close enough (i.e., the propensity to cluster is great enough), initial increase of TFT may occur for sets of payoffs that preclude the advance of TFT in the absence of clustering.

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APPENDIX

Loss Modification Model

We assume a genetic system with two alleles at each of two autosomal loci. Locus A determines the probability that a genotype is altruistic. Thus, genotypes AA, Aa, and aa are altruistic with probabilities by h_1, h_2, and h_3. The performance of an altruistic act involves a loss in fitness proportional to $h_j k_i \gamma$. The value of k_i is determined by the genotype at the B locus. The k parameters for genotypes BB, Bb, and bb are k_1, k_2, and k_3, respectively. Altruistic interactions will be assumed to be between full sibs. Consequently, an individual will experience an increase in fitness equal to β times the probability of encountering an altruistic sib. As in our previous kin selection models (Uyenoyama et al. 1981; Mueller and Feldman 1985) we assume that the loss and gain components of fitness are combined in an additive fashion. We will label the gametes AB, Ab, aB, and ab, 1 to 4 respectively.

Then, the fitnesses of the various two locus genotypes are proportional to the quantities given below:

	AA	Aa	aa
BB	$1 - h_1 k_1 \gamma + \beta\phi_{11}$	$1 - h_2 k_1 \gamma + \beta\phi_{13}$	$1 - h_3 k_1 \gamma + \beta\phi_{33}$
Bb	$1 - h_1 k_2 \gamma + \beta\phi_{12}$	$1 - h_2 k_2 \gamma + \beta\phi_{14}$	$1 - h_3 k_2 \gamma + \beta\phi_{34}$
bb	$1 - h_1 k_3 \gamma + \beta\phi_{22}$	$1 - h_2 k_3 \gamma + \beta\phi_{24}$	$1 - h_3 k_3 \gamma + \beta\phi_{44}$

where ϕ_{ij} is the conditional probability that genotype ij has an altruistic sib. Let the frequencies of the four gametes AB, Ab, aB, and ab be x_1, x_2, x_3, and x_4 respectively and r, the recombination fraction between the A and B locus. We can now write the recursions for the ten two-locus genotypes below.

$$\bar{W}g'_{11} = \tilde{x}_1^2(1 - h_1 k_1 \gamma) + \beta f_{11}, \tag{A1a}$$

$$\bar{W}g'_{12} = 2\tilde{x}_1\tilde{x}_2(1 - h_1 k_2 \gamma) + \beta f_{12}, \tag{A1b}$$

$$\bar{W}g'_{13} = 2\tilde{x}_1\tilde{x}_3(1 - h_2 k_1 \gamma) + \beta f_{13}, \tag{A1c}$$

$$\bar{W}g'_{14} = 2\tilde{x}_1\tilde{x}_4(1 - h_2 k_2 \gamma) + \beta f_{14}, \tag{A1d}$$

$$\bar{W}g'_{22} = \tilde{x}_2^2(1 - h_1 k_3 \gamma) + \beta f_{22}, \tag{A1e}$$

$$\bar{W}g'_{23} = 2\tilde{x}_2\tilde{x}_3(1 - h_2 k_2 \gamma) + \beta f_{23}, \tag{A1f}$$

$$\bar{W}g'_{24} = 2\tilde{x}_2\tilde{x}_4(1 - h_2 k_3 \gamma) + \beta f_{24}, \tag{A1g}$$

$$\bar{W}g'_{33} = \tilde{x}_3^2(1 - h_3 k_1 \gamma) + \beta f_{33}, \tag{A1h}$$

$$\bar{W}g'_{34} = 2\tilde{x}_3\tilde{x}_4(1 - h_3 k_2 \gamma) + \beta f_{34}, \tag{A1i}$$

$$\bar{W}g'_{44} = \tilde{x}_4^2(1 - h_3 k_3 \gamma) + \beta f_{44}, \tag{A1j}$$

where

$$\tilde{x}_1 = x_1 + rL/2,$$
$$\tilde{x}_2 = x_2 - rL/2,$$
$$\tilde{x}_3 = x_3 - rL/2,$$
$$\tilde{x}_4 = x_4 + rL/2,$$
$$L = g_{23} - g_{14},$$

and $\overline{W}$ is a normalizer equal to the sum of the right hand sides of equations (A1).

The joint probability of being genotype ij and having an altruistic sib is denoted by f_{ij}. Calculation of the various f_{ij}s is done in a manner identical to that described in Mueller and Feldman (1985). Each f_{ij} expression contains terms multiplied by h_1, h_2, and h_3. For simplicity Table A1 contains the f_{ij} expressions grouped as coefficients of h_1, h_2 and h_3.

We now proceed with the analysis of this model. We are particularly interested in studying the evolution of the loss parameter (k_is) from various initial equilibria. First, we assume the population is fixed for the A and B alleles (corner equilibrium). We then find conditions that guarantee stability of the corner equilibrium. We also examine the initial increase conditions for alleles at the B locus when the A locus is at a stable polymorphism. As mentioned previously, there are two kinds of single locus equilibria: viability analogous and structural. We consider each of these equilibria in our analysis.

Stability of a Corner Equilibrium

We assume the population is initially fixed for the *AB/AB* genotype. Our problem is to find conditions under which the corner remains stable in the presence of a small number of *ab* haplotypes. The analysis reduces to examining the linear dynamics of the three rare genotypes, *AB/ab*, *AB/aB*, and *AB/Ab* in the neighborhood of the equilibrium point $g_{11} = 1$. If $\mathbf{g} = (g_{12}, g_{13}, g_{14})^T$, then the linear dynamics are given by $\mathbf{g}' = \mathbf{Ag}$, where,

$$\mathbf{A} = \frac{1}{T}\begin{bmatrix} 1 - k_2h_1\gamma + \beta h_1, & 0, & r(1 - k_2h_1\gamma) + \beta r(h_1 + h_2)/2 \\ 0, & 1 - k_1h_2\gamma + \beta(h_1 + h_2)/2, & r(1 - k_1h_2\gamma) + \beta r(h_1 + h_2)/2 \\ 0, & 0, & (1 - r)(1 - k_2h_2\gamma) + \beta(1 - r)(h_1 + h_2)/2 \end{bmatrix}$$

and $T = 1 + \beta h_1 - k_1h_1\gamma$.

Table A1. Joint Probabilities, f_{ij}, of Being Genotype ij and Having an Altruistic Sib. Each f_{ij} is the Sum of Three Terms Given Below, Which Are Multiplied by h_0, h_1, and h_2, Respectively. The Following Identities Are Used Throughout: $L = g_{23} - g_{14}$, $V = g_{14}(1 - r) + g_{23}r$, $M = g_{14}r + g_{23}(1 - r)$.

h_1 – terms

$$f_{11} = x_1(2g_{11} + g_{12})/2 + g_{13}(g_{13} + 2g_{14})/16 + rL(4g_{11} + 2g_{12} + g_{13})/8 + V^2/16$$

$$f_{22} = x_2(2g_{22} + g_{12})/2 + g_{24}(g_{24} + 2g_{23})/16 - rL(4g_{22} + 2g_{12} + g_{24})/8 + M^2/16$$

$$f_{33} = g_{13}(g_{13} + 2g_{23})/16 - g_{13}rL/8 + M^2/16$$

$$f_{44} = g_{24}(g_{24} + 2g_{14})/16 + g_{24}rL/8 + V^2/16$$

$$f_{12} = x_1(2g_{22} + g_{12})/2 + x_2(2g_{11} + g_{12})/2 + (g_{13}g_{23} + g_{13}g_{24} + g_{14}g_{24})/8 + rL[4(g_{22} - g_{11}) + g_{24} - g_{13}]/8 + VM/8$$

$$f_{13} = (g_{13} + g_{23})(2g_{11} + g_{12})/4 + g_{13}(g_{13} + g_{14} + g_{23})/8 - rL(2g_{11} + g_{12})/4 + MV/8$$

$$f_{24} = (g_{14} + g_{24})(2g_{22} + g_{12})/4 + g_{24}(g_{14} + g_{23} + g_{24})/8 + rL(2g_{22} + g_{12})/4 + MV/8$$

$$f_{34} = (g_{13}g_{14} + g_{13}g_{24} + g_{23}g_{24})/8 + rL(g_{13} - g_{24})/8 + MV/8$$

$$f_{14} = (g_{14} + g_{24})(2g_{11} + g_{12})/4 + (g_{13}g_{14} + g_{13}g_{24} + g_{14}g_{24})/8 + rL(4g_{11} + 2g_{12} + g_{13} + g_{24})/8 + V^2/8$$

$$f_{23} = (g_{13} + g_{23})(2g_{22} + g_{12})/4 + (g_{13}g_{23} + g_{13}g_{24} + g_{23}g_{24})/8 - rL(4g_{22} + 2g_{12} + g_{13} + g_{24})/8 + M^2/8$$

h_2 – terms

$$f_{11} = (g_{13} + g_{14})(2g_{11} + g_{12})/4 + g_{13}(2g_{14} + g_{13})/8 + rL(2g_{11} + g_{12} + g_{13})/4) + V^2/8$$

$$f_{22} = (g_{23} + g_{24})(2g_{22} + g_{12})/4 + g_{24}(2g_{23} + g_{24})/8 - rL(2g_{22} + g_{12} + g_{24})/4 + M^2/8$$

$$f_{33} = (g_{13} + g_{23})(2g_{33} + g_{34})/4 + g_{13}(2g_{23} + g_{13})/8 - rL(2g_{33} + g_{13} + g_{34})/4 + M^2/8$$

$$f_{44} = (g_{14} + g_{24})(2g_{44} + g_{34})/4 + g_{24}(2g_{14} + g_{24})/8 + rL(2g_{44} + g_{24} + g_{34})/4 + V^2/8$$

$$f_{12} = g_{13}x_2/2 + g_{24}(x_1 - g_{13}/2)/2 + g_{23}(2g_{11} + g_{12})/4 + g_{14}(2g_{22} + g_{12})/4 + rL[2(g_{22} - g_{11}) + g_{24} - g_{13}]/4 + MV/4$$

$$f_{13} = g_{33}x_1 + x_3(2g_{11} + g_{12})/2 + g_{13}(g_{13} + g_{14} + g_{23} + g_{34})/4 + g_{34}(2g_{11} + g_{12} + g_{14})/4 + rL(2g_{33} + g_{34} - g_{12} - 2g_{11})/4 + MV/4$$

$$f_{24} = g_{22}x_4 + x_2(2g_{44} + g_{34})/2 + g_{24}(g_{12} + g_{14} + g_{23} + g_{24})/4 + g_{12}(2g_{44} + g_{14} + g_{34})/4 + rL[2(g_{22} - g_{44}) + g_{12} - g_{34}]/4 + MV/4$$

$$f_{34} = g_{13}x_4/2 + g_{24}(x_3 - g_{13}/2)/2 + g_{23}(2g_{44} + g_{34})/4 + g_{14}(2g_{33} + g_{34})/4 + rL[2(g_{33} - g_{44}) + g_{13} - g_{24}]/4 + MV/4$$

$$f_{14} = x_1(2g_{44} + g_{34})/2 + x_4(2g_{11} + g_{12})/2 + (g_{13}g_{14} + g_{13}g_{24} + g_{14}g_{24})/4 + rL(2g_{11} + 2g_{44} + g_{12} + g_{13} + g_{24} + g_{34})/4 + V^2/4$$

$$f_{23} = x_2(2g_{33} + g_{34})/2 + x_3(2g_{22} + g_{12})/2 + (g_{13}g_{23} + g_{13}g_{24} + g_{23}g_{24})/4 - rL(2g_{22} + 2g_{33} + g_{12} + g_{13} + g_{24} + g_{34})/4 + M^2/4$$

Table A1. ***(Continued)***

h_3 – terms
$f_{11} = g_{13}(g_{13} + 2g_{14})/16 + g_{13}rL/8 + V^2/16$
$f_{22} = g_{24}(g_{24} + 2g_{23})/16 - g_{24}rL/8 + M^2/16$
$f_{33} = x_3(2g_{33} + g_{34})/2 + g_{13}(g_{13} + 2g_{23})/16 - rL(4g_{33} + 2g_{34} + g_{13})/8 + M^2/16$
$f_{44} = x_4(2g_{44} + g_{34})/2 + g_{24}(g_{24} + 2g_{14})/16 + rL(4g_{44} + 2g_{34} + g_{24})/8 + V^2/16$
$f_{12} = (g_{13}g_{23} + g_{13}g_{24} + g_{14}g_{24})/8 + rL(g_{24} - g_{13})/8 + MV/8$
$f_{13} = (g_{13} + g_{14})(2g_{33} + g_{34})/4 + g_{13}(g_{13} + g_{14} + g_{23})/8 + rL(2g_{33} + g_{34})/4 + MV/8$
$f_{24} = (g_{23} + g_{24})(2g_{44} + g_{34})/4 + g_{24}(g_{14} + g_{23} + g_{24})/8 - rL(2g_{44} + g_{34})/4 + MV/8$
$f_{34} = x_4(2g_{33} + g_{34})/2 + x_3(2g_{44} + g_{34})/2 + (g_{13}g_{14} + g_{13}g_{24} + g_{23}g_{24})/8$ $+ rL[4(g_{33} - g_{44}) + g_{13} - g_{24}]/8 + MV/8$
$f_{14} = (g_{13} + g_{14})(2g_{44} + g_{34})/4 + (g_{13}g_{14} + g_{14}g_{24} + g_{13}g_{24})/8 + rL(4g_{44} + 2g_{34} + g_{13} + g_{24})/8 + V^2/8$
$f_{23} = (g_{23} + g_{24})(2g_{33} + g_{34})/4 + (g_{13}g_{23} + g_{13}g_{24} + g_{23}g_{24})/8 - rL(4g_{33} + 2g_{34} + g_{13} + g_{24})/8 + M^2/8$

The matrix **A** has three unique eigenvalues:

$$\lambda_1 = \frac{1 - k_2h_1\gamma + \beta h_1}{1 + \beta h_1 - k_1h_1\gamma},$$

$$\lambda_2 = \frac{1 - k_1h_2\gamma + \beta(h_1 + h_2)/2}{1 + \beta h_1 - k_1h_1\gamma},$$

$$\lambda_3 = \frac{(1 - r)(1 - k_2h_2\gamma) + \beta(1 - r)(h_1 + h_2)/2}{1 + \beta h_1 - k_1h_1\gamma}.$$

The *AB* fixation is stable if $\lambda_1, \lambda_2, \lambda_3 < 1$. These conditions reduce to

$$k_2 > k_1, \tag{A2a}$$

$$(h_2 - h_1)(\beta/2 - k_1\gamma) < 0, \tag{A2b}$$

$$\gamma(k_1h_1 - k_2h_2) + \beta(h_2 - h_1)/2 - r[2(1 - k_2h_2\gamma) + \beta(h_1 + h_2)]/2 < 0. \tag{A2c}$$

If inequalities (A2a) and (A2b) are satisfied, then (A2c) is always true. Since conditions (A2a) and (A2b) are also the conditions for the protection of single locus monomorphism, we can conclude that if both the *a* and *b* alleles fail to increase at their separate loci, they will also fail to increase in the linked two locus systems.

Stability at the *A* Locus; Increase of Loss Alleles Linked to a Polymorphism

We now consider a population that is at a stable viability analogous equilibrium and fixed for the *B* allele. Consequently, $\hat{x}_1 = (h_2 - h_3)/(2h_2 - h_1$

$- h_3)$. To study the conditions necessary for the initial increase of b alleles, we need to examine the dynamics of the four rare genotypes AB/Ab, AB/ab, Ab/aB, and aB/ab in the vicinity of the viability analogous equilibrium. If $\mathbf{g} = (g_{12}, g_{14}, g_{23}, g_{34})^T$, then the dynamics of the rate genotypes may be summarized as $\mathbf{g}' = \mathbf{Bg}$. The largest eigenvalue of $\mathbf{B}$ is

$$\lambda_1 = \frac{1 + (\beta - k_2\gamma)(h_2^2 - h_1h_3)/(2h_2 - h_1 - h_3)}{1 + (\beta - k_1\gamma)(h_2^2 - h_1h_3)/(2h_2 - h_1 - h_3)}.$$

For λ_1 to be greater than 1, we must have $k_1 > k_2$. Thus, b alleles can get into the population only if the loss suffered by the Bb heterozygotes is less than the loss BB homozygotes experience.

The previous result is true when the A locus is at a viability analogous equilibrium. We now derive initial increase conditions assuming the A locus is at the structural equilibrium. The same matrix, $\mathbf{B}$, determines stability in this problem. The difference between the two problems is that the values of $\hat{x}_1$ and $\hat{g}_{13}$ are not the same. In particular there is no simple expression for $\hat{x}_1$, since it is the solution of a quadratic equation (Uyenoyama and Feldman, 1981). Finding the largest eigenvalue of $\mathbf{B}$ at the structural equilibrium has proven to be quite difficult. We can make progress for the case of $r = 0$. With absolute linkage, the matrix $\mathbf{B}$ breaks into two 2×2 matrices.

The first 2×2 matrix for the $r = 0$ case is given below.

$$\frac{1}{T}\begin{Bmatrix} (1 - k_2h_1\gamma)\hat{x}_1 + \frac{\beta}{4}[4\hat{x}_1h_1 + \hat{g}_{13}(h_2 - h_1)], & (1 - k_2h_1\gamma)\hat{x}_1 + \frac{\beta}{8}[4\hat{x}_1(h_1 + h_2) + \hat{g}_{13}(h_3 - h_1)] \\ (1 - k_2h_2\gamma)(1 - \hat{x}_1) + \frac{\beta}{4}[4(1 - \hat{x}_1)h_2 + \hat{g}_{13}(h_1 - h_2)], & (1 - k_2h_2\gamma)(1 - \hat{x}_1) + \frac{\beta}{8}[4(1 - \hat{x}_1)(h_2 + h_3) + \hat{g}_{13}(h_1 - h_3)], \end{Bmatrix} \quad \text{(A3)}$$

where $T = 1 + (\beta - k_1\gamma)[[\hat{x}_1^2h_1 + 2\hat{x}_1(1 - \hat{x}_1)h_2 + (1 - \hat{x}_1)^2h_3]$. After some algebra, we find that the largest eigenvalue of (A3) is less than 1 if

$$\begin{aligned} \hat{x}_1(1 - \hat{x}_1)\beta\gamma[k_1(h_1h_3 - h_2^2) + k_2(h_2 - h_1)(\bar{h}_2 - \bar{h}_1)] - k_1\gamma\bar{h}(1 - k_2\gamma\bar{h}_1) \\ - k_1\gamma[h_1\hat{x}_1 + h_3(1 - \hat{x}_1)][(\beta - k_1\gamma) + k_2\gamma\bar{h}_1] \\ - T[\beta(\bar{h}_1 - \bar{h}) - \gamma(k_2\bar{h}_1 - k_1\bar{h}) - \gamma k_2\bar{h}_1] > 0, \end{aligned} \quad \text{(A4)}$$

where

$$\begin{aligned} \bar{h}_1 &= \hat{x}_1h_1 + (1 - \hat{x}_1)h_2, \\ \bar{h}_2 &= \hat{x}_1h_2 + (1 - \hat{x}_1)h_3, \\ \bar{h} &= \hat{x}_1\bar{h}_1 + (1 - \hat{x}_1)\bar{h}_2. \end{aligned}$$

By itself, this inequality is not very informative. We can, however, obtain some simple results by noticing the following features of (A4). The inequality (A4) is a linear function of k_2. Thus, if we can determine the value of this function for two values of k_2 we may be able to derive a simple relationship between the magnitude of the largest eigenvalue and k_2. We first start with $k_2 = k_1 = k$. Again, after some algebra we find that the expression on the left of equation (A4) is equal to

$$(\bar{h}_1 - \bar{h})\{-(k\gamma)^2[h_1\hat{x}_1 + h_3(1 - \hat{x}_1) + \bar{h}] + \beta k\gamma[h_1\hat{x}_1 + h_3(1 - \hat{x}_1) + 2h] - \beta^2\bar{h} - \beta + 2k\gamma\}. \quad \text{(A5)}$$

From Uyenoyama and Feldman (1981) we know that the allele frequencies at the structural equilibrium satisfy the following quadratic equation:

$$x_1^2[(\beta - \gamma)^2(2h_2 - h_1 - h_3)] + x_1[(\beta - \gamma)^2(h_1 + h_3 - 2h_2) + (h_1 - h_3)(\beta - \gamma)(2\gamma - \beta)] + (2\gamma - \beta)[1 + h_3(\beta - \gamma)] = 0. \quad \text{(A6)}$$

If we note that γ in (A6) is equal to $k\gamma$ in our notation, equation (A6) can be rearranged to yield

$$-(k\gamma)^2[h_1\hat{x}_1 + h_3(1 - \hat{x}_1) + \bar{h}] + \beta k\gamma[h_1\hat{x}_1 + h_3(1 - \hat{x}_1) + 2\bar{h}] - \beta^2\bar{h} - \beta + 2k\gamma = 0.$$

Thus, (A5) is equal to 0 when $k_2 = k_1$. We next set $k_2 = 2k_1 = 2k$ in the left side of equation (A4). Rearranging this expression yields

$$k\gamma\overline{hh}_2(\beta - k\gamma) + h_3(1 - \hat{x}_1)\bar{h}_2k\gamma(\beta - k\gamma) + k\gamma\overline{hh}_2(1 - k\gamma) + \hat{x}_1k\gamma[h_1h_2\hat{x}_1(\beta - k\gamma) + (1 - \hat{x}_1)\beta h_2^2] + 2k\gamma h_2\hat{x}_1 + (1 - \hat{x}_1)k\gamma h_3(2 - h_1k\gamma\hat{x}_1). \quad \text{(A7)}$$

If $\beta > k\gamma$, then each element of (A7) is positive, hence the whole expression is positive. We can now conclude that for the inequality (A4) to hold requires $k_2 > k_1$. Analysis of the second 2×2 matrix from **B** yields the same result.

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