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POPULATION GENETIC THEORY OF KIN SELECTION: A TWO-LOCUS MODEL

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Conditions that permit the success of a genotype that increases the fitness of relatives while suffering a reduction of its own were first suggested by Hamilton (1964*a*, 1964*b*). If r is the probability that a relative carries that same "altruistic" allele as the altruist (i.e., the coefficient of relatedness defined by Crozier [1970]) then these alleles will be favored if the loss in fitness suffered by altruists, γ, is more than compensated for by the gain in fitness to the recipients, β, multiplied by the coefficient of relatedness, r:

$$\beta r > \gamma. \tag{1}$$

These ideas were later developed as single-locus two-allele population genetic models by several authors (Charnov 1977; Charlesworth 1978; Wade 1979; see Michod [1982] for a review). These models, which essentially confirm Hamilton's basic results, all assume that adults occur in Hardy-Weinberg proportions. We call these models *inclusive fitness models*. Clearly, if the types of interactions subsumed by altruism occur among pre-adults and these interactions affect viability, then adults will not be in Hardy-Weinberg proportions. Consequently, exact models of kin selection have been developed that do not assume Hardy-Weinberg proportions among the adults (Levitt 1975; Cavalli-Sforza and Feldman 1978; Uyenoyama and Feldman 1981; Uyenoyama et al. 1981). If selection is weak then adult genotypic proportions will not deviate substantially from Hardy-Weinberg proportions (Charlesworth 1980). Although inclusive fitness models can provide approximations to the dynamics of genotypes under the appropriate conditions, it should be emphasized that the difference between the inclusive fitness and exact models is not merely in the quantitative prediction of genotype frequencies each generation. As shown by Cavalli-Sforza and Feldman (1978) the loss and gain components of fitness must be combined in an additive fashion to recover any of Hamilton's results. Even with an additive formulation, however, exact models yield initial increase conditions that differ from equation (1) (using Crozier's definition of r) if altruism is directed from sister to brother or sister to any sibling

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and the genetic system is haplodiploid (see also Uyenoyama and Feldman 1981). For single-locus models there exist two classes of polymorphic equilibria we have named *viability analogous* and *structural* (Uyenoyama and Feldman 1981; Uyenoyama et al. 1981; Toro et al. 1982). In inclusive fitness models only the viability-analogous equilibrium can be extracted (as in Michod and Abugov 1980).

Despite the fact that exact analysis and the Hardy-Weinberg assumption both predict the existence of the viability-analogous equilibrium, the conditions for its stability are quite different in the two treatments. For instance, Michod and Abugov (1980) show that for a diploid, sib-sib inclusive fitness model the viability-analogous equilibrium will be stable if $\beta/2 > \gamma$ and there is overdominance in the propensity to be "altruistic." Using equation (52) of Uyenoyama and Feldman (1981), the viability-analogous equilibrium can be stable locally either if (1) $\beta/2 > \gamma$ and there is underdominance in altruism or if (2) $\beta/2 < \gamma$ and altruism is overdominant.

If relatedness is defined as the regression of the additive genotypic value of the recipient on the altruist, $b_{A\to R}$ (see Uyenoyama et al. [1981] for definition), then equation (1) can be rewritten as $\beta b_{A\to R} > \gamma$. This condition accurately describes the initial increase of altruistic alleles. In cases in which altruist and recipient differ in sex, the above condition must be modified by the mean fitness of each sex. The viability-analogous equilibrium corresponds to the condition when the variance of the additive genotypic value is zero (Uyenoyama et al. 1981). In addition the structural equilibria were shown to satisfy $\beta b_{A\to R} = \gamma$.

In this paper we continue our development of a population genetic theory of kin selection by examining a two-locus, two-allele model. There have been two previous two-locus kin-selection models by Wade (1979) and Aoki and Moody (1981). Wade's model made the Hardy-Weinberg assumptions and assumed the two loci were in linkage equilibrium. Thus all the complicating effects of linkage were ignored. Aoki and Moody (1981) studied the origin of worker behavior in the Hymenoptera and consequently used a rather detailed life history which limits the generality of the model. In this paper we use analytic and numerical methods to study the behavior of some exact two-locus models of sib-to-sib altruism near certain fixation states and at internal polymorphic equilibria. The behavior of these two-locus kin-selection models is compared to their viability selection analogs. We see a number of similarities between the single-locus kin-selection models and the two-locus models and point out important cases in which Hamilton's conditions at a single locus are misleading when interpreted for two loci simultaneously.

PHENOTYPIC KIN-SELECTION MODEL

We assume adults mate at random and that the population size is infinite. Altruistic interactions are between full sibs. The altruistic behavior of interest is assumed to be under the control of two linked autosomal loci with two alleles (A, a; B, b). Associated with the performance of each altruistic act is a loss in fitness proportional to γ; likewise, recipients of altruism experience an increase in fitness proportional to β. Genotypes are characterized by their probability of being

altruistic. In this model these probabilities take on three values, h_0, h_1, or h_2, depending on whether an individual is heterozygous at 0, 1, or 2 loci. Because of this simple characterization of altruistic genotypes we call the model *phenotypic kin selection* by analogy to the two-locus viability models (Lewontin and Kojima 1960; Karlin and Feldman 1970*a*; Karlin and Liberman 1976). The h's for the 10 two-locus genotypes are given below.

	AB	Ab	aB	ab
AB	h_0	h_1	h_1	h_2
Ab		h_0	h_2	h_1
aB			h_0	h_1
ab				h_0

The net fitness of each genotype is composed of two components which we have described previously as the loss and gain. As first discussed by Cavalli-Sforza and Feldman (1978), even if these components are assumed to be independent there are still at least two natural ways in which they can be combined, additively or multiplicatively. In the model treated here fitness will be computed in an additive fashion. Our reasons for this are twofold. (1) The original formulation of kin selection (Hamilton 1964*a*, 1964*b*) and the majority of subsequent elaborations of these ideas (see Michod [1982] for review) correspond most closely to the additive assumption. (2) We have previously developed the equilibrium theory for single-locus exact population genetic models of kin selection most extensively for additive models (Uyenoyama and Feldman 1981; Uyenoyama et al. 1981; but see Uyenoyama and Feldman 1982).

It was pointed out previously (Cavalli-Sforza and Feldman 1978; Uyenoyama and Feldman 1981; Uyenoyama et al. 1981) that proper accounting of the dynamics of kin-selection models must be in terms of genotype frequencies since the randomly mating adults are not in Hardy-Weinberg equilibrium. As emphasized most recently by Feldman and Eshel (1982), the difference between these exact population genetic models and those that use gene frequencies under the implicit assumption of weak selection (see, e.g., Charlesworth 1980) can be qualitatively important.

To construct the basic genotype recursions needed for this model, the probability that each genotype has an altruistic sib must be specified in addition to the parameters γ, β, and h_i. Index the four chromosomes AB, Ab, aB, and ab by 1, 2, 3, and 4. Then the frequency of chromosome i is x_i and the frequency of genotype ij will be represented by g_{ij} with f_{ij} the joint probability of being genotype ij and having an altruistic sib (see Cavalli-Sforza and Feldman 1978). With this notation the 10 single-generation genotype recursions are

$$\bar{W}g'_{11} = \bar{x}_1^2 (1 - \gamma h_0) + \beta f_{11}, \tag{2a}$$

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

$$\bar{W}g'_{13} = 2\bar{x}_1\bar{x}_3 (1 - \gamma h_1) + \beta f_{13}, \tag{2c}$$

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

$$\bar{W}g'_{22} = \tilde{x}_2^2\,(1 - \gamma h_0) + \beta f_{22}, \tag{2e}$$

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

$$\bar{W}g'_{24} = 2\tilde{x}_2\tilde{x}_4\,(1 - \gamma h_1) + \beta f_{24}, \tag{2g}$$

$$\bar{W}g'_{33} = \tilde{x}_3^2\,(1 - \gamma h_0) + \beta f_{33}, \tag{2h}$$

$$\bar{W}g'_{34} = 2\tilde{x}_3\tilde{x}_4\,(1 - \gamma h_1) + \beta f_{34}, \tag{2i}$$

$$\bar{W}g'_{44} = \tilde{x}_4^2\,(1 - \gamma h_0) + \beta f_{44}, \tag{2j}$$

where

$$\tilde{x}_1 = x_1 + rL/2,$$

$$\tilde{x}_2 = x_2 - rL/2,$$

$$\tilde{x}_3 = x_3 - rL/2,$$

$$\tilde{x}_4 = x_4 + rL/2,$$

$$L = g_{23} - g_{14},$$

r is the recombination fraction, and $\bar{W}$ is a normalizer equal to the sum of the right-hand sides of (2).

To calculate f_{ij} we must first determine the frequency of offspring from all 55 possible matings of the two-locus genotypes. Then from each family where genotype ij occurs, the contribution to f_{ij} can be added to all previous contributions. Thus, for instance, the contribution to f_{12} from the 23 $\times$ 23 family is $g_{23}^2\, r(1 - r)/2\,\{h_0\,[r^2/2 + (1 - r)^2/2] + h_1[2r(1 - r)] + h_2[r^2/2 + (1 - r)^2/2]\}$. To this term are added similar values from all families which produce AB/Ab offspring. It should be noted that each f_{ij} expression will contain terms multiplied by h_0, h_1, and h_2. For simplicity, table 1 contains the f_{ij} expressions grouped as coefficients of h_0, h_1, and h_2.

The analysis will focus on three general topics. First, conditions for the evolution of new gamete types or alleles from a "corner" equilibrium, i.e., where the population is fixed for one chromosome only, will be determined. Second, we examine the case where one locus is at a stable polymorphic equilibrium with the second locus fixed, and determine the conditions which allow for the increase of a new allele at the second locus. Third, we look for internal equilibria at which all four chromosomes are segregating.

Stability of a Corner Equilibrium in the Symmetric Model: Increase of Altruistic Chromosomes

Suppose the population is fixed for the genotype AB/AB. The conditions for stability of this equilibrium to the introduction of Ab, aB, and ab are obtained from the dynamics of the three rare genotypes, AB/ab, AB/aB, AB/Ab in the neighborhood of the equilibrium point $\hat{g}_{11} = 1$.

TABLE 1

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:
$L = g_{23} - g_{14}$, $U = g_{14}r^2 + g_{23}(1 - r)^2$, $M = g_{23} + g_{14}$, $V = g_{14}(1 - r)^2 + g_{23}r^2$

h_0 Terms

$$f_{11} = g_{12}(x_1 + g_{11})/4 + g_{11}(2g_{11} + g_{13})/2 + g_{13}(g_{13} + g_{14})/8 + rL(g_{12} + g_{13})/8 + g_{11}V/2 + (1 - r)^2r^2M^2/8 + V^2/8$$

$$f_{22} = g_{12}(x_2 + g_{22})/4 + g_{22}(2g_{22} + g_{24})/2 + g_{24}(g_{23} + g_{24})/8 - rL(g_{12} + g_{24})/8 + g_{22}U/2 + (1 - r)^2r^2M^2/8 + U^2/8$$

$$f_{33} = g_{13}(x_3 + g_{33})/4 + g_{33}(2g_{33} + g_{34})/2 + g_{34}(g_{23} + g_{34})/8 - rL(g_{13} + g_{34})/8 + g_{33}U/2 + (1 - r)^2r^2M^2/8 + U^2/8$$

$$f_{44} = g_{24}(x_4 + g_{44})/4 + g_{44}(2g_{44} + g_{34})/2 + g_{34}(g_{14} + g_{34})/8 + rL(g_{24} + g_{34})/8 + g_{44}V/2 + (1 - r)^2r^2M^2/8 + V^2/8$$

$$f_{12} = g_{12}(x_1 + x_2 + g_{11} + g_{22})/4 + (g_{13}g_{23} + g_{14}g_{24})/8 + rL(g_{24} - g_{13})/8 + (1 - r)rM\{g_{11} + g_{22} + M[(1 - r)^2 + r^2]/2\}/2$$

$$f_{13} = g_{13}(x_1 + x_3 + g_{11} + g_{33})/4 + (g_{12}g_{23} + g_{14}g_{34})/8 + rL(g_{34} - g_{12})/8 + (1 - r)rM\{g_{11} + g_{33} + M[(1 - r)^2 + r^2]/2\}/2$$

$$f_{14} = [g_{12}(g_{14} + g_{24}) + g_{13}(g_{14} + g_{34}) + g_{14}(g_{24} + g_{34})]/8 + rL(g_{12} + g_{24} + g_{13} + g_{34})/8 + V(g_{11} + g_{44})/2 + (1 - r)^2r^2M^2/4 + V^2/4$$

$$f_{23} = [g_{34}(g_{23} + g_{24}) + g_{23}(g_{12} + g_{24}) + g_{13}(g_{12} + g_{23})]/8 - rL(g_{12} + g_{13} + g_{24} + g_{34})/8 + U(g_{22} + g_{33})/2 + (1 - r)^2r^2M^2/4 + U^2/4$$

$$f_{24} = g_{24}(x_2 + x_4 + g_{22} + g_{44})/4 + (g_{12}g_{14} + g_{23}g_{34})/8 + rL(g_{12} - g_{34})/8 + (1 - r)rM\{g_{22} + g_{44} + M[(1 - r)^2 + r^2]/2\}/2$$

$$f_{34} = g_{34}(x_3 + x_4 + g_{33} + g_{44})/4 + (g_{13}g_{14} + g_{23}g_{24})/8 + rL(g_{13} - g_{24})/8 + (1 - r)rM\{g_{33} + g_{44} + M[(1 - r)^2 + r^2]/2\}/2$$

h_1 Terms

$$f_{11} = (g_{12} + g_{13})(x_1 + g_{11} + g_{14}/2)/4 + rL(g_{12} + g_{13})/4 + r(1 - r)M(2g_{11} + V)/2$$

$$f_{22} = (g_{12} + g_{24})(x_2 + g_{22} + g_{23}/2)/4 - rL(g_{12} + g_{24})/4 + r(1 - r)M(2g_{22} + U)/2$$

$$f_{33} = (g_{13} + g_{34})(x_3 + g_{33} + g_{23}/2)/4 - rL(g_{13} + g_{34})/4 + r(1 - r)M(2g_{33} + U)/2$$

$$f_{44} = (g_{24} + g_{34})(x_4 + g_{44} + g_{14}/2)/4 + rL(g_{24} + g_{34})/4 + r(1 - r)M(2g_{44} + V)/2$$

$$f_{12} = x_2(g_{12} + g_{13})/2 + g_{11}(4g_{22} + g_{12} + g_{24})/2 + g_{14}(g_{12} + g_{24})/4 + rL(g_{24} - g_{13})/4 + (1 - r)^2r^2M^2/2 + g_{11}U + g_{22}V + UV/2$$

$$f_{13} = x_3(g_{12} + g_{13})/2 + g_{11}(4g_{33} + g_{13} + g_{34})/2 + g_{14}(g_{13} + g_{34})/4 + rL(g_{34} - g_{12})/4 + (1 - r)^2r^2M^2/2 + g_{11}U + g_{33}V + UV/2$$

$$f_{14} = x_1(g_{24} + g_{34})/2 + (g_{12} + g_{13})(2g_{44} + g_{14})/4 + rL(g_{12} + g_{13} + g_{24} + g_{34})/4 + (1 - r)rM(g_{11} + g_{44} + V)$$

$$f_{23} = x_2(g_{13} + g_{34})/2 + (g_{12} + g_{24})(2g_{33} + g_{23})/4 - rL(g_{12} + g_{13} + g_{24} + g_{34})/4 + (1 - r)rM(g_{22} + g_{33} + U)$$

$$f_{24} = x_2(g_{24} + g_{34})/2 + g_{44}(4g_{22} + g_{12} + g_{24})/2 + g_{14}(g_{12} + g_{24})/4 + rL(g_{12} - g_{34})/4 + (1 - r)^2r^2M^2/2 + g_{22}V + g_{44}U + UV/2$$

$$f_{34} = x_3(g_{24} + g_{34})/2 + g_{44}(4g_{33} + g_{13} + g_{34})/2 + g_{14}(g_{13} + g_{34})/4 + rL(g_{13} - g_{24})/4 + (1 - r)^2r^2M^2/2 + g_{33}V + g_{44}U + UV/2$$

(*continued*)

TABLE 1 (*Continued*)

h_2 Terms
$f_{11} = (g_{12}g_{13} + g_{13}g_{14} + g_{12}g_{14})/8 + rL(g_{12} + g_{13})/8 + (1 - r)^2r^2M^2/8 + g_{11}V/2 + V^2/8$
$f_{22} = (g_{12}g_{24} + g_{12}g_{23} + g_{23}g_{24})/8 - rL(g_{12} + g_{24})/8 + (1 - r)^2r^2M^2/8 + g_{22}U/2 + U^2/8$
$f_{33} = (g_{13}g_{23} + g_{13}g_{34} + g_{23}g_{34})/8 - rL(g_{13} + g_{34})/8 + (1 - r)^2r^2M^2/8 + g_{33}U/2 + U^2/8$
$f_{44} = (g_{14}g_{24} + g_{14}g_{34} + g_{24}g_{34})/8 + rL(g_{24} + g_{34})/8 + (1 - r)^2r^2M^2/8 + g_{44}V/2 + V^2/8$
$f_{12} = g_{24}(x_1 + g_{11})/4 + g_{13}(x_2 + g_{22})/4 + g_{12}M/8 + rL(g_{24} - g_{13})/8 + (1 - r)rM\{2(g_{11} + g_{22}) + M[(1 - r)^2 + r^2]\}/4$
$f_{13} = g_{34}(x_1 + g_{11})/4 + g_{12}(x_3 + g_{33})/4 + g_{13}M/8 + rL(g_{34} - g_{12})/8 + (1 - r)rM\{2(g_{11} + g_{33}) + M[(1 - r)^2 + r^2]\}/4$
$f_{14} = (x_1 + g_{11})(g_{24} + g_{34})/4 + g_{44}(4g_{11} + g_{12} + g_{13})/2 + g_{12}(g_{14} + g_{34})/8 + g_{13}(g_{14} + g_{24})/8 + rL(g_{12} + g_{13} + g_{24} + g_{34})/8 + (1 - r)^2r^2M^2/4 + V(g_{11} + g_{44})/2 + V^2/4$
$f_{23} = (x_2 + g_{22})(g_{13} + g_{34})/4 + g_{33}(4g_{22} + g_{12} + g_{24})/2 + g_{24}(g_{13} + g_{23})/8 + g_{12}(g_{23} + g_{34})/8 - rL(g_{12} + g_{13} + g_{24} + g_{34})/8 + (1 - r)^2r^2M^2/4 + U(g_{22} + g_{33})/2 + U^2/4$
$f_{24} = g_{34}(x_2 + g_{22})/4 + g_{12}(x_4 + g_{44})/4 + g_{24}M/8 + rL(g_{12} - g_{34})/8 + (1 - r)rM\{2(g_{22} + g_{44}) + M[(1 - r)^2 + r^2]\}/4$
$f_{34} = g_{24}(x_3 + g_{33})/4 + g_{13}(x_4 + g_{44})/4 + g_{34}M/8 + rL(g_{13} - g_{24})/8 + (1 - r)rM\{2(g_{33} + g_{44}) + M[(1 - r)^2 + r^2]\}/4$

The conditions for fixation of AB to be stable reduce to

$$(h_1 - h_0)\,(\beta/2 - \gamma) < 0, \tag{3a}$$

and

$$(h_2 - h_0)(1 - r)(\beta/2 - \gamma) - r[1 + (\beta - \gamma)h_0] - r(1 - r)\beta(h_0 + h_2 - 2h_1)/2 < 0. \tag{3b}$$

From these results we can see the first complications which make the evolution of altruistic alleles in two-locus systems qualitatively different from the single-locus models. Clearly (3a) corresponds to the stability of AB to the single-locus perturbations, and if $h_1 > h_0$ and $\beta/2 > \gamma$, (3a) fails and the corner equilibrium is unstable, the usual one-locus result. If $h_0 > h_1$ and $\beta/2 > \gamma$, then single-locus theory predicts that a or b alleles introduced separately at each locus will not increase. For the full two-locus model, however, it is possible that even if a or b introduced separately would not have increased, ab would increase if the double heterozygotes were very altruistic. In other words if $h_2 > h_0 > h_1$ and $\beta/2 > \gamma$ then (3a) holds but (3b) may not, and indeed, under these conditions, at $r = 0$ (3b) is false. As $r \to \frac{1}{2}$, the left side of (3b) decreases although for extreme parameter values it is possible that (3b) fails even at $r = \frac{1}{2}$. For $h_2 > h_0 > h_1$ and $\beta/2 > \gamma$ there will usually be some r^* such that for all $r < r^*$ the AB fixation will be unstable. However, if $\beta \gg \gamma$ and $h_2 \gg h_0$ and h_1, this equilibrium can be unstable for all values of r. Clearly this requires nonadditivity of the alleles and genes contributing to the h's. When $\beta/2 > \gamma$, the eigenvalue corresponding to condition (3b) changes monotonically over the range of r. Consequently, the region $r \in [0, \frac{1}{2}]$ will contain at most one critical value, r^*, which separates stable from unstable fixation states.

When $\beta/2 < \gamma$ the situation is quite different. In this case it is possible for the AB fixation to be locally stable when $r = 0$ and $r = \frac{1}{2}$ but unstable for intermediate values of r. Consider the following example: $\beta = 1.9$, $\gamma = 1.0$, $h_0 = 0$, $h_1 = 1$, and $h_2 = 0.1$. Equation (3b) predicts all fixations will be unstable if $5.6 \times 10^{-4} < r < 0.47$ and otherwise stable. When this corner is unstable, numerical results have shown the genetic system converges to a single-locus boundary equilibrium. Single-locus theory predicts the local stability of the chromosome fixations so that with single-locus theory alone we would conclude that the viability-analogous equilibrium could be attained only with the help of some stochastic force, e.g., genetic drift, founder effect. A second epistatic locus enables this viability-analogous equilibrium to be reached via a purely deterministic mechanism. Thus an initially selfish population ($h_0 = 0$) can become more altruistic even when $\beta/2 < \gamma$. Of course this result depends critically on the rate of recombination.

Remark.—The conditions (3) are special cases of the analogous conditions for general altruistic propensities, h_{ij}. In general the conditions which ensure stability of the AB fixation are

$$(h_{12} - h_{11})(\beta/2 - \gamma) < 0, \tag{4a}$$

$$(h_{13} - h_{11})(\beta/2 - \gamma) < 0, \tag{4b}$$

and

$$(h_{14} - h_{11})(\beta/2 - \gamma)(1 - r) - \frac{\beta r(1 - r)}{2}(h_{11} + h_{14} - h_{12} - h_{13}) - r[1 + (\beta - \gamma)h_{11}] < 0. \tag{4c}$$

These (and the special case [3]) are analogous to conditions for initial increase in the sex-linked case found by M. K. Uyenoyama (personal communication).

Stability at One Locus: Increase of Altruistic Allele Linked to a Polymorphism

Consider now the case of a population that is at a stable polymorphic equilibrium for one locus, say A, and fixed at the second locus, B. We seek the conditions for the increase of a new allele b at the second locus. In our previous work on single-locus kin-selection models (Uyenoyama et al. 1981), we pointed out that there are two possible classes of polymorphic equilibria which we called *viability analogous* and *structural*. Allele frequencies at the viability-analogous equilibrium are those calculated as if the h_i's were viabilities in standard viability models of natural selection. In the present model the three genotypes AB/AB, AB/aB, and aB/aB are altruistic with probabilities h_0, h_1, and h_0, respectively. Thus the frequency of the AB gamete at the viability-analogous equilibrium will be $\frac{1}{2}$. For single-locus, two-allele, sib-sib models of kin selection, Uyenoyama and Feldman (1981) have shown that allele frequencies at the structural equilibrium are solutions to a certain quadratic equation, but because of the symmetry imposed by the present model there are no feasible allele frequencies that satisfy their quadratic (Uyenoyama and Feldman 1981, eq. [51]) when $\beta/2 > \gamma$ and $h_1 > h_0$.

To determine the fate of the b allele at the second locus we study the linearized dynamics of the rare genotypes AB/Ab, AB/ab, Ab/aB, and aB/ab in the vicinity of the viability-analogous equilibrium. It can be shown that if

$$\mu_1 = \frac{1 - \gamma(h_1 + h_2)/2 + \beta\,[2h_1 + (h_0 + h_2)]/4}{1 + (\beta - \gamma)(h_0 + h_1)/2} \tag{5}$$

is less than unity, then b cannot increase when rare for any recombination value, while if $\mu_1 > 1$ it increases for all $r \leq \frac{1}{2}$. Now $\mu_1 < 1$ if

$$(h_2 - h_0)(\beta/2 - \gamma) < 0, \tag{6}$$

so that this result is independent of the recombination fraction. Condition (6) is identical to that expected for a single-locus three-allele model; if the chromosomes AB and aB are considered to be the segregating alleles and Ab (or ab) is the introduced allele, then in the terminology of Uyenoyama et al. (1981) condition (6) is the same as $(h_3 - \bar{h})(\beta/2 - \gamma) < 0$, namely, the condition for the protection at a two-allele viability-analogous equilibrium against invasion by a third rare allele.

That (6) does not involve r is a consequence of the symmetry of this model (Bodmer and Felsenstein 1967). In other two-locus models that do not have this symmetry, the fate of new alleles at the second locus is quite complicated and does involve the recombination fraction.

Internal Equilibria

It is obvious from table 1 that the recursions 1 are quite complicated and therefore the exploration of internal equilibria is liable to be a tedious task. This can be simplified somewhat if we take advantage of the natural symmetry of this model. With absolute linkage, genetic polymorphisms exist with either $\hat{x}_1 = \hat{x}_4 = \frac{1}{2}$ or $\hat{x}_2 = \hat{x}_3 = \frac{1}{2}$. Polymorphisms of this kind, which entail maximum linkage disequilibrium, have been termed *high complementarity equilibria* (Franklin and Lewontin 1970; Feldman et al. 1974). With $r = 0$ these equilibria are special cases of the four-allele model encompassed by the theory of Uyenoyama et al. (1981). By a straightforward application of this theory, we see that the high complementarity points are stable at $r = 0$ (and hence, by continuity, for small r) when

$$\beta/2 > \gamma \tag{7a}$$

with

$$h_2 > h_0 \tag{7b}$$

and

$$h_0 + h_2 > 2h_1, \tag{7c}$$

or when

$$\beta/2 < \gamma \tag{8a}$$

with

$$h_2 < h_0 \tag{8b}$$

and

$$h_0 + h_2 < 2h_1. \tag{8c}$$

Thus at $r = 0$ the altruistic propensities in this two-locus system, as special cases of the one-locus model, exhibit the properties of two-locus fitnesses (see, e.g., Karlin and Feldman 1970*b*).

In view of the symmetry assumptions on h_0, h_1, h_2 it is natural to attempt to transform the genotype frequencies g_{ij} into new coordinates that facilitate the analysis. A natural system used by Feldman and Liberman (1985) in fertility models is given in table 2, with the reverse transformation in table 3. This coordinate change simplifies the local stability analysis and in addition suggests the form that equilibria might take. The first step is to search for symmetric equilibria with the following structure:

$$\text{All double homozygotes equal: } \hat{g}_{11} = \hat{g}_{22} = \hat{g}_{33} = \hat{g}_{44} = \hat{g}_0, \tag{9a}$$

$$\text{All single homozygotes equal: } \hat{g}_{12} = \hat{g}_{13} = \hat{g}_{24} = \hat{g}_{34} = \hat{g}_1, \tag{9b}$$

$$\text{Double heterozygotes equal: } \hat{g}_{14} = \hat{g}_{23} = \hat{g}_2, \tag{9c}$$

that is,

$$\hat{g}_{11} + \hat{g}_{12}/2 + \hat{g}_{14}/2 = \tfrac{1}{4}. \tag{9d}$$

Clearly these entail that all chromosome frequencies are equal: $\hat{x}_1 = \hat{x}_2 = \hat{x}_3 = \hat{x}_4 = \frac{1}{4}$ and that there is genotypic linkage equilibrium. This equilibrium is analogous to the central equilibrium that has been described for a variety of two-locus viability models (Lewontin and Kojima 1960; Bodmer and Felsenstein 1967; Karlin and Feldman 1970*a*, 1970*b*). The frequency dependence in our kin-selection models, however, will cause the equilibrium genotype frequencies ($\hat{g}_0$, $\hat{g}_1$, $\hat{g}_2$) to differ from those of the corresponding viability model. Thus the similarity between the kin-selection and viability models is not complete. By inspection of table 2 it is clear the conditions (9) leave all u_i's equal to 0 except u_1, u_6, u_{11}, and u_{16}. Since $u_1 = 1$ and $u_6 = u_{11} = \tilde{u}$, say, we need only consider the two equilibrium equations given below,

$$T\tilde{u} = (h_0 - h_2)\,[-\gamma/4 + \beta\,(1 + \tilde{u})^2/16] \tag{10a}$$

$$\begin{aligned} Tu_{16} = (h_0 + h_2 - 2h_1)\{&-\gamma/4 + \beta\,[1 + 2\tilde{u} + u_{16} \\ &+ (1 - 2r)^2(1 + u_{16} - 2\tilde{u})]^2/64\} \end{aligned} \tag{10b}$$

where $T = 1 + (\beta - \gamma)(h_0 + 2h_1 + h_2)/4$. Equation (10a) has a single root in the range $(-1,1)$ of validity of $\tilde{u}$, and this root, when substituted into (10b), produces a unique valid root for u_{16}. Although the equilibrium value of $\tilde{u} = 4\hat{g}_{11} - 2\hat{g}_{14}$ is independent of r, the value of $u_{16} = 4\hat{g}_{11} + 2\hat{g}_{14} - 4\hat{g}_{12}$ does vary with r. Thus, although there is gametic and genotypic linkage equilibrium the genotype frequencies depend on the recombination fraction. Since the equilibrium chromosome and gene frequencies are exactly the same as those arising from the corresponding symmetric viability model this equilibrium can be regarded as viability analogous.

The local stability of (10) has so far defied analysis. We have made an extensive numerical survey of its stability which we summarize later in our more general

TABLE 2

THE LINEAR TRANSFORMATIONS OF GENOTYPE FREQUENCIES LISTED BELOW DEFINE A NEW COORDINATE SYSTEM, WHICH IS USED TO STUDY THE EQUILIBRIUM BEHAVIOR OF THE MODEL AS DESCRIBED IN THE TEXT

$$
\begin{aligned}
u_1 &= g_{11} + g_{12} + g_{13} + g_{14} + g_{22} + g_{23} + g_{24} + g_{33} + g_{34} + g_{44} \\
u_2 &= g_{11} + g_{13} - g_{22} - g_{24} + g_{33} - g_{44} \\
u_3 &= g_{11} + g_{12} + g_{22} - g_{33} - g_{34} - g_{44} \\
u_4 &= g_{11} + g_{14} - g_{22} - g_{23} - g_{33} + g_{44} \\
u_6 &= g_{11} - g_{12} + g_{13} - g_{14} + g_{22} - g_{23} + g_{24} + g_{33} - g_{34} + g_{44} \\
u_7 &= g_{11} - g_{14} - g_{22} + g_{23} - g_{33} + g_{44} \\
u_8 &= g_{11} - g_{12} + g_{22} - g_{33} + g_{34} - g_{44} \\
u_{11} &= g_{11} + g_{12} - g_{13} - g_{14} + g_{22} - g_{23} - g_{24} + g_{33} + g_{34} + g_{44} \\
u_{12} &= g_{11} - g_{13} - g_{22} + g_{24} + g_{33} - g_{44} \\
u_{16} &= g_{11} - g_{12} - g_{13} + g_{14} + g_{22} + g_{23} - g_{24} + g_{33} - g_{34} + g_{44}
\end{aligned}
$$

TABLE 3

EQUILIBRIA DESCRIBED IN TERMS OF THE COORDINATES GIVEN IN TABLE 2 ARE TRANSFORMED TO GENOTYPE FREQUENCIES USING THE FOLLOWING IDENTITIES

$$
\begin{aligned}
g_{11} &= (u_1 + 2u_2 + 2u_3 + 2u_4 + u_6 + 2u_7 + 2u_8 + u_{11} + 2u_{12} + u_{16})/16 \\
g_{12} &= [(u_1 + 2u_3 + u_{11}) - (u_6 + 2u_8 + u_{16})]/8 \\
g_{13} &= [(u_1 + 2u_2 + u_6) - (u_{11} + 2u_{12} + u_{16})]/8 \\
g_{14} &= [(u_1 + 2u_4 + u_{16}) - (u_6 + 2u_7 + u_{11})]/8 \\
g_{22} &= [(u_1 + 2u_3 + u_6 + 2u_8 + u_{11} + u_{16}) - (2u_2 + 2u_4 + 2u_7 + 2u_{12})]/16 \\
g_{23} &= [(u_1 + 2u_7 + u_{16}) - (u_6 + 2u_4 + u_{11})]/8 \\
g_{24} &= [(u_1 + 2u_{12} + u_6) - (u_{11} + 2u_2 + u_{16})]/8 \\
g_{33} &= [(u_1 + 2u_2 + u_6 + u_{11} + 2u_{12} + u_{16}) - (2u_3 + 2u_4 + 2u_7 + 2u_8)]/16 \\
g_{34} &= [(u_1 + 2u_8 + u_{11}) - (u_6 + 2u_3 + u_{16})]/8 \\
g_{44} &= [(u_1 + 2u_4 + 2u_7 + u_6 + u_{11} + u_{16}) - (2u_2 + 2u_3 + 2u_8 + 2u_{12})]/16
\end{aligned}
$$

discussion of the results of numerical iteration of the system (2). However, two special cases are amenable to analysis and serve to illustrate some of the interesting stability properties of the central equilibrium (10). The first of these has $h_0 + h_2 = 2h_1$ and the second $h_2 = h_0$.

Special Case 1: $h_0 + h_2 = 2h_1$ (Symmetric Additivity)

Here, from (10b) $u_{16} = 0$, so that

$$\hat{g}_1 = \tfrac{1}{8},\ \hat{g}_2 = \tfrac{1}{4} - 2\hat{g}_0, \tag{11a}$$

where $\hat{g}_0$ is the solution to the quadratic equation

$$g_0^2\,\beta(h_0 - h_2)/2 + g_0[\gamma(h_0 + h_2)/2 - \beta(7h_0 + 9h_2)/16 - 1] + \beta(17h_0 + 15h_2)/8^3 + (1 - \gamma h_0)/16 = 0. \quad (11b)$$

Note that this equilibrium is independent of the recombination fraction.

In studying the local stability of (11) we note that at $r = 0$ there is a unit eigenvalue. For $r > 0$ the leading eigenvalue, which governs the local stability, is

$$\nu = \frac{1 - \gamma(h_2 + 3h_0)/4 + \beta[h_0(9/16 + \hat{g}_0) + h_2(7/16 - \hat{g}_0)]}{1 + (\beta - \gamma)(h_0 + h_2)/2}, \quad (12)$$

and $\nu < 1$ if

$$(h_0 - h_2)[\beta(\tfrac{1}{4} + 4\hat{g}_0) - \gamma] < 0. \quad (13)$$

It is helpful to recall that with symmetric additivity the four chromosome fixation states are stable if $\beta/2 > \gamma$, $h_2 < h_0$ or $\beta/2 < \gamma$, $h_2 > h_0$ and unstable if $\beta/2 > \gamma$, $h_2 > h_0$ or $\beta/2 < \gamma$, $h_2 < h_0$. In addition, when $\beta/2 > \gamma$ and $h_2 > h_0$ or $\beta/2 < \gamma$ and $h_2 < h_0$, there are no structural equilibria on the gene fixation edges. The other two cases, $\beta/2 > \gamma$, $h_0 > h_2$ and $\beta/2 < \gamma$, $h_2 > h_0$, allow structural equilibria on the gene fixation edges provided that γ and $\beta/2$ are not too far apart. For example, if $h_2 > h_0$ then when γ is larger than and close to $\beta/2$, structural equilibria exist, but if γ is close to β, they do not. Similarly, if $h_0 > h_2$ then for γ less than and close to $\beta/2$ structural equilibria exist but for γ close to $\beta/4$ they do not.

These boundary considerations are relevant to interpretation of (13). If $h_2 > h_0$, $\beta/2 > \gamma$ then if $r > 0$ (11) is locally stable, the corners are unstable and the gene fixation viability-analogous equilibria are unstable in the two-locus sense. If $h_2 > h_0$ and $\gamma > \beta/2$ then the corners are stable and the viability-analogous gene-fixation-edge equilibria may also be stable. If γ is not too much larger than $\beta/2$ the interior polymorphism (11) may also be stable according to (13) if $r > 0$.

When $h_2 < h_0$, $\beta/2 < \gamma$ the corners are unstable, the viability-analogous edge equilibria are unstable and the polymorphism (11) is locally stable. If $h_2 < h_0$, $\beta/2 > \gamma$, then provided r is not too small, the corners, edges, and polymorphism (11) may all be stable simultaneously if $r > 0$.

A numerical example of additive symmetry is the following table of h values:

	AA	Aa	aa
BB	1	0.5	1
Bb	0.5	0	0.5
bb	1	0.5	1 .

Here $h_0 = 1$, $h_1 = 0.5 = (h_0 + h_2)/2$. For example, with $\beta = 0.81$, $\gamma = 0.4$, $r = 0.5$, all corners, all viability-analogous edge equilibria, and the central point are stable. Similar equilibrium patterns can be observed with $\gamma > \beta/2$ and $h_2 > h_0$, e.g., $h_0 = 0$, $h_2 = 1$, $\beta = 0.78$, $\gamma = 0.4$, and $r = .02$.

Remark.—Note that the $D = 0$ equilibrium under additive viabilities can never

be simultaneously stable with boundary equilibria. In the present case the frequency-dependent selection results in a more intricate equilibrium pattern.

Special Case 2: $h_2 = h_0$

This case is clearly degenerate in several respects. First, from (6) it is seen that the external eigenvalue for a viability-analogous edge equilibrium is unity, while from (3b), at $r = 0$ the chromosome fixations also have a unit eigenvalue. This suggests that equilibrium surfaces may exist in the interior of the frequency simplex. It is clear, however, that under the condition $h_2 = h_0$, $\tilde{u} = 0$ solves (10a) and an equilibrium is given by

$$\hat{g}_2 = \tfrac{1}{2} - 2\hat{g}_1 - 2\hat{g}_0, \tag{14a}$$

$$\hat{g}_0 = (1 - 4\hat{g}_1)/8, \tag{14b}$$

$$\hat{g}_1 = (1 - \hat{u})/8, \tag{14c}$$

where $\tilde{u}$ solves the quadratic

$$u^2\beta(h_0 - h_1)(1 - 2r + 2r^2)^2/8 - u[\beta(h_1 - h_0)(1 - 2r + 2r^2)^2/4 + 1 + (\beta - \gamma)(h_0 + h_1)/2] + (h_0 - h_1)[\beta(1 - 2r + 2r^2)^2/8 - \gamma/2] = 0. \tag{15}$$

For all r, (15) has a unique valid root, but the local stability analysis for this equilibrium produces a unit eigenvalue. In fact, from the other eigenvalues, u is locally unstable if

$$\gamma[h_1/2 - h_0(\tfrac{1}{2} - r)] - r + \beta\{h_1[2g_1(1 - r) + (1 - 4g_1)r(1 - r)^2 - \tfrac{1}{2}] + h_0[g_1(1 - 2r) + (\tfrac{1}{4} - g_1)(3 - 8r + 8r^2 - 4r^3) - \tfrac{1}{4}] > 0. \tag{16}$$

When (16) is violated, the unit eigenvalue is the largest, and linear analysis is inadequate. Numerical analysis indicates that when (16) holds there is convergence to chromosome fixation, while when it is reversed there appears to be extremely slow convergence to a surface whose nature is as yet unclear.

Numerical Results.—The following remarks should not be regarded as a complete description of the many iterations of (2) with various values of β, γ, h_0, h_1, and h_2 that have been carried out. Rather, we point out some of the important similarities and differences between the present frequency-dependent symmetric altruism model and symmetric viabilities, as studied by, e.g., Karlin and Feldman (1970*b*) and Feldman and Liberman (1979). For tight linkage, as (7) and (8) indicate, with $\beta/2 > \gamma$, $h_2 > h_0$ and $h_0 + h_2 > 2h_1$, as well as the case where all of these are reversed, there are stable high complementarity equilibria. These are not, however, of the form $\hat{x}_1 = \hat{x}_4$, $\hat{x}_2 = \hat{x}_3$ as might have been expected from the symmetric viability theory. As the recombination rate is increased under these conditions, the central point (11) becomes stable. Whether these high complementarity equilibria should be termed structural in the sense of Uyenoyama et al. (1981) is an open question.

With the completely symmetric viabilities of Lewontin and Kojima (1960), the central point $D = 0$ cannot be stable if the double homozygote is fitter than the double heterozygote. This condition is not required of the h's in the symmetric

kin-selection framework. For example, if $\beta = .81$, $\gamma = .4$, $h_0 = 1$, $h_1 = .51$, and $h_2 = 0$, we have $h_0 + h_2 < 2h_1$ (negative epistasis) but $h_2 < h_0$. Nevertheless, there is a domain of attraction to the equilibrium (10).

Surprisingly, the high complementarity equilibria may overlap in stability with the central point (10). Corners may overlap in stability with the high complementarity points and with the central point. If the first two of the above three inequalities hold but $h_0 + h_2 - 2h_1 < 0$, the situation is analogous to negative epistasis in viability and the central point is stable for all recombination values. Recall, however, that if $h_0 + h_2 = 2h_1$, additive symmetry, there can be simultaneous stability of chromosome and gene fixation as well as the central polymorphism.

These findings suggest that an "inclusive fitness" gene frequency approach is unlikely to reveal much of the dynamic structure when the kin selection is caused by two linked loci. It is worth noting with respect to single-locus kin-selection theory that Michod and Abugov (1980) suggest that $\beta/2 > \gamma$ and simple overdominance in h's (i.e., Aa > AA, aa) are sufficient for stability of the viability-analogous equilibrium. The exact condition for its stability is more complicated, however, and it can be stable with (1) $\beta/2 > \gamma$ and underdominance or (2) $\beta/2 < \gamma$ and overdominance. For example, if $\beta = .81$, $\gamma = .4$, AA and aa have $h = 1.0$ and Aa 0.5, both gene fixations and the viability-analogous equilibrium are stable with unstable structural equilibria separating them (see also Uyenoyama and Feldman 1981).

DISCUSSION

In order to compare the present results with those for one locus obtained most generally by Uyenoyama et al. (1981) we define

$$\tilde{x}_1 = g_{11} + (g_{12} + g_{13} + g_{14})/2 - r(g_{14} - g_{23})/2$$
$$\tilde{x}_2 = g_{22} + (g_{12} + g_{23} + g_{24})/2 + r(g_{14} - g_{23})/2$$
$$\tilde{x}_3 = g_{33} + (g_{13} + g_{23} + g_{34})/2 + r(g_{14} - g_{23})/2$$
$$\tilde{x}_4 = g_{44} + (g_{14} + g_{24} + g_{34})/2 - r(g_{14} - g_{23})/2.$$

Let $\alpha_i = \Sigma_j \tilde{x}_j(h_{ij} - \bar{h})$, where $\Sigma_j \tilde{x}_j\alpha_j = 0$. Then α_i are analogous to the additive genotypic values, and the variance among siblings is

$$\text{Var(sib)} = \sum_i \sum_j \hat{x}_i\hat{x}_j(\alpha_i - \alpha_j)^2.$$

Following Uyenoyama et al., we then compute the covariance between the additive genotypic value and fitness of sibs:

$$\frac{\text{Cov(sib G, sib F)}}{\bar{W}} = 2\sum_i \alpha_i\hat{x}_i' - 2\sum_i x_i\alpha_i + r(g_{14}' - g_{23}')(\alpha_1 + \alpha_4 - \alpha_2 - \alpha_3) \quad (17)$$

where $\bar{W}$ is the normalizer from (2).

Heuristically, we expect equilibria to be given by Cov(sib G, sib F) = 0, which

is seen to be true only if (1) $r = 0$ or (2) $g_{14} - g_{23} = D = 0$, or (3) $\alpha_1 + \alpha_4 - \alpha_2 - \alpha_3 = 0$. The last condition clearly is met only when the h's are truly additive, as when $h_0 + h_2 = 2h_1$ above. The $D = 0$ points, however, will also satisfy the heuristic, while with $r = 0$ the multiple-allele theory of Uyenoyama et al. is sufficient.

We can rewrite (17) at equilibrium

$$\beta \text{ Cov(sib,sib)} - \gamma \text{ Var(sib)}$$
$$= r(g'_{14} - g'_{23})(\alpha_1 + \alpha_4 - \alpha_2 - \alpha_3)\,\bar{W}.$$

The equilibria have either Var(sib) = 0 or, writing b(sib,sib) as the regression,

$$\beta \text{ b(sib,sib)} - \gamma = \frac{r(g'_{14} - g'_{23})(\alpha_1 + \alpha_4 - \alpha_2 - \alpha_3)\,\bar{W}}{\text{Var(sib)}}.$$

In the symmetric model studied here the central point given by (10) always satisfies Var(sib) = 0 and might therefore properly be called a viability-analogous two-locus equilibrium. Under only one of the three above conditions does Hamilton's heuristic equilibrium criterion, β b(sib $\rightarrow$ sib) = γ, predict any other equilibria. These findings agree with those of M. K. Uyenoyama (personal communication) in the sex-linked case.

Insofar as the initial increase conditions at boundary equilibria are concerned, inspection of (4) and computation of the covariances reveals that only if $r(\alpha_1 + \alpha_4 - \alpha_2 - \alpha_3) = 0$ does a Hamilton-type condition emerge. Thus only if $r(\alpha_1 + \alpha_4 - \alpha_2 - \alpha_3) = 0$ is stability at a corner predicted by the positivity of lim [Cov(sib G,sib F)]/$\bar{W}$ at the corner. Again this result has also been shown by M. K. Uyenoyama (personal communication) for the sex-linked case. It should be emphasized that epistasis between the loci involved in the kin selection can result in initial increase of alleles at both loci under conditions which, according to classical one-locus theory, would prevent their increase at either loci. Further, the dependence of this sort of anomalous behavior on the linkage between the loci may not be monotonic.

SUMMARY

The dynamics of a two-locus, two-allele model of kin selection with sib-to-sib interactions are analyzed. The initial increase of altruistic alleles at each locus separately can be predicted from Hamilton's criteria for single-locus, sib-sib kin-selection models. The addition of a second locus controlling altruistic behavior will permit the increase of altruistic alleles even when Hamilton's condition is not satisfied. In addition, there may exist certain polymorphic equilibria which cannot be reconciled with Hamilton's theory. In general it is only under rather special conditions that the dynamics of this two-locus genetic model can be considered in terms of Hamilton's theory.

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LITERATURE CITED

Aoki, K., and M. Moody. 1981. One- and two-locus models of the origin of worker behavior in Hymenoptera. J. Theor. Biol. 89:449–474.

Bodmer, W. F., and J. Felsenstein. 1967. Linkage and selection: theoretical analysis of the deterministic two-locus random mating model. Genetics 57:237–265.

Cavalli-Sforza, L. L., and M. W. Feldman. 1978. Darwinian selection and "altruism." Theor. Popul. Biol. 14:263–281.

Charlesworth, B. 1978. Some models of the evolution of altruistic behavior between siblings. J. Theor. Biol. 72:297–319.

———. 1980. Models of kin selection. Pages 11–26 *in* H. Markl, ed. Evolution of social behavior: hypotheses and empirical tests. Dahlem Konferenzen, Berlin.

Charnov, E. L. 1977. An elementary treatment of the genetical theory of kin selection. J. Theor. Biol. 66:541–550.

Crozier, R. H. 1970. Coefficients of relationship and the identity of genes by descent. Am. Nat. 104:216–217.

Feldman, M. W., and I. Eshel. 1982. On the theory of parent-offspring conflict: a two-locus genetic model. Am. Nat. 119:285–292.

Feldman, M. W., and U. Liberman. 1979. On the number of stable equilibria and the simultaneous stability of fixation and polymorphism in two-locus models. Genetics 92:1355–1360.

———. 1985. A symmetric two-locus fertility model. Genetics (in press).

Feldman, M. W., I. Franklin, and G. J. Thomson. 1974. Selection in complex genetic systems. I. The symmetric equilibria of the three-locus symmetric viability model. Genetics 76:135–162.

Franklin, I., and R. C. Lewontin. 1970. Is the gene the unit of selection? Genetics 65:701–734.

Hamilton, W. D. 1964*a*. The genetical evolution of social behavior. I. J. Theor. Biol. 1:1–16.

———. 1964*b*. The genetical evolution of social behavior. II. J. Theor. Biol. 1:17–52.

Karlin, S., and M. W. Feldman. 1970*a*. Convergence to equilibrium of the two locus additive viability model. J. Appl. Prob. 7:262–271.

———. 1970*b*. Linkage and selection: two locus symmetric viability model. Theor. Popul. Biol. 1:39–71.

Karlin, S., and U. Liberman. 1976. A phenotypic symmetric selection model for three loci, two alleles: the case of tight linkage. Theor. Popul. Biol. 10:336–364.

Levitt, P. R. 1975. General kin selection models for genetic evolution of sib altruism in diploid and haplodiploid species. Proc. Natl. Acad. Sci. USA 72:4531–4535.

Lewontin, R. C., and K. Kojima. 1960. The evolutionary dynamics of complex polymorphisms. Evolution 14:458–472.

Michod, R. E. 1982. The theory of kin selection. Annu. Rev. Ecol. Syst. 13:23–55.

Michod, R. E., and R. Abugov. 1980. Adaptive topographies in family structured models of kin selection. Science 210:667–669.

Toro, M., R. Abugov, B. Charlesworth, and R. E. Michod. 1982. Exact versus heuristic models of kin selection. J. Theor. Biol. 97:699–713.

Uyenoyama, M. K., and M. W. Feldman. 1981. On relatedness and adaptive topography in kin selection. Theor. Popul. Biol. 19:87–123.

———. 1982. Population genetic theory of kin selection. II. The multiplicative model. Am. Nat. 120:614–627.

Uyenoyama, M. K., M. W. Feldman, and L. D. Mueller. 1981. Population genetic theory of kin selection: I. Multiple alleles at one locus. Proc. Natl. Acad. Sci. USA 78:5036–5040.

Wade, M. J. 1979. The evolution of social interactions by family selection. Am. Nat. 113:399–417.