Population genetic theory of kin selection: Multiple alleles at one locus

(additive genetic variance/regression among relatives)

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ABSTRACT   Exact population genetic models of one-locus sib-to-sib kin selection with an arbitrary number of alleles are studied. First, a natural additive scaling is established for the genotypic value associated with probabilities of performance of altruism. Two classes of polymorphic equilibria are possible, one corresponding to the usual one-locus viability equilibria and the other reflecting the kin-selection assumptions of the model. At both, the covariance between additive genotypic value and genotypic fitness vanish. Further, the sign of this covariance determines the fate of rare alleles introduced near the first class of equilibria. In addition, the covariance explains the differences between Hamilton's rule, which results from Hardy–Weinberg assumptions, and exact initial increase conditions.

Our previous papers introduced exact population genetic models of kin selection among first-degree relatives (1–3). These models were constructed in the spirit of Hamilton (4) but were expressed and analyzed in terms of genotype rather than of gene frequencies. Gene-frequency treatments (5–7) with Hardy–Weinberg assumptions produce conditions for initial increase of an altruistic allele that agree with the rule first stated by Hamilton. Hamilton's rule is usually understood to entail that an altruistic allele should increase in frequency when rare if $\beta r_H > \gamma$, where β is the gain in fitness to recipients of altruism, γ is the fitness loss of altruists, and r_H is a measure of the genetic relationship between these participants.

In their exact population genetic treatment, Cavalli-Sforza and Feldman (1) showed that additive composition of losses and gains to form genotypic fitnesses usually produced initial increase conditions in agreement with Hamilton's rule. Further examination of the exceptions found in ref. 1 led us to suggest (3) that within the context of additive combination of losses and gains an appropriate value of r_H is the regression of the recipient's additive genotypic value on that of the altruist.

More generally, Hamilton's rule suggests that, if r_H is frequency dependent, polymorphic equilibria should entail $\beta r_H = \gamma$. With the correct frequency-dependent interpretation of r_H, this equation was shown (3) to produce a class of polymorphic equilibria in models of sib-to-sib altruism except in those cases in which the donor was diploid and the recipients included members of the opposite sex to the altruists. In fact, two classes of polymorphic equilibria may coexist and, even when the same condition entails initial increase and local stability of fixation of the altruistic allele, fixation may not be globally stable (1).

In this paper, we extend the exact population genetic theory with additive combination of losses and gains to include an arbitrary number of alleles. We develop a suggestion made by Li (8), Price (9), and Hamilton (10) concerning the covariance be-

tween additive genotypic value and fitness and use it to produce a unified theory for initial increase and polymorphism.

MODELS OF SIB-TO-SIB ALTRUISM

Consider a set of n alleles $\{A_i\}$ at a single locus. When all individuals are diploid, the genotypes A_iA_j have frequencies u_{ij}. When it is necessary to differentiate the sexes in the diploid case, the male and female frequencies are denoted m_{ij} and f_{ij}, respectively. In the haplodiploid case, diploid A_iA_j females have frequency f_{ij} while that of A_i males is m_i. In the diploid case, the frequency of A_i (without regard to sex) is $p_i = u_{ii} + 1/2 \sum_{j \neq i} u_{ij}$. Where it is necessary to differentiate the sexes in the diploid case and in the haplodiploid case, the frequency of A_i in females is $f_i = f_{ii} + 1/2 \sum_{j \neq i} f_{ij}$, with m_i the corresponding value in males. In models in which A_iA_j can perform altruism, it does so with probability h_{ij} and, for males of genotype A_i, the corresponding probability is denoted g_i. The losses and gains to the fitnesses of each genotype follow the composition of Cavalli-Sforza and Feldman (1) for their *additive model*, as extended by Uyenoyama and Feldman (3). We present the resulting recursions of the genotypic frequencies for the cases of diploid sib-to-sib altruism and haplodiploid sister-to-brother altruism. Recursions for the other cases of sib altruism can be derived in the same way as in Uyenoyama and Feldman (3).

Recursions. Diploid, sib-to-sib altruism:

$$Tu'_{ii} = u_{ii} \left\{ u_{ii}[1 + (\beta - \gamma)h_{ii}] + \sum_{j \neq i} u_{ij} \right.$$

$$\times \left[1 + \frac{1}{2}\beta(h_{ii} + h_{ij}) - h_{ii}\gamma \right] \right\} + \frac{1}{2}\sum_{k \neq i} u_{ik} \left\{ \frac{1}{2}\sum_{j \neq i} u_{ij} \right. \quad [1]$$

$$\times \left[1 + \frac{1}{4}\beta(h_{ii} + h_{ij} + h_{ik} + h_{kj}) - h_{ii}\gamma \right] \right\}$$

and, if $\ell \neq i$,

$$Tu'_{i\ell} = 2u_{ii} \left\{ u_{\ell\ell}[1 + (\beta - \gamma)h_{i\ell}] + \frac{1}{2}\sum_{j \neq \ell} u_{\ell j} \right.$$

$$\times \left[1 + \frac{1}{2}\beta(h_{ij} + h_{i\ell}) - h_{i\ell}\gamma \right] \right\} + \sum_{k \neq i} u_{ik} \left\{ u_{\ell\ell} \left[1 \right. \right. \quad [2]$$

$$\left. + \frac{1}{2}\beta(h_{i\ell} + h_{\ell k}) - h_{i\ell}\gamma \right] + \frac{1}{2}\sum_{j \neq \ell} u_{\ell j} \left[1 + \frac{1}{4}\beta(h_{i\ell} + h_{ij} \right. \right.$$

$$\left. \left. + h_{\ell k} + h_{jk}) - h_{i\ell}\gamma \right] \right\},$$

where

$$T = 1 + (\beta - \gamma)\sum_i \sum_j p_i p_j h_{ij}. \quad [3]$$

Haplodiploid, sister-to-brother altruism:

$$T_f f'_{ii} = f_i m_i (1 - h_{ii} \gamma) \qquad [4]$$

and, if $\ell \neq i$,

$$T_f f'_{i\ell} = (m_i f_\ell + m_\ell f_i)(1 - h_{i\ell} \gamma), \qquad [5]$$

$$T_m m'_i = f_{ii} \sum_j m_j (1 + h_{ij} \beta) + \frac{1}{2} \sum_{k \neq i} f_{ik} \sum_j m_j$$
$$\times \left[1 + \frac{1}{2} \beta (h_{ij} + h_{jk}) \right], \qquad [6]$$

where

$$T_f = 1 - \gamma \sum_i \sum_j f_i m_j h_{ij} \qquad [7a]$$

and

$$T_m = 1 + \beta \sum_i \sum_j f_i m_j h_{ij}. \qquad [7b]$$

Construction of Variances and Covariances. Before regression coefficients among relatives can be defined, an appropriate quantitative value must be assumed to correspond with each genotype. Following the usual formulation from the theory of quantitative inheritance (11–13), we assign to the diploid genotype $A_i A_j$ the genotypic value $(\alpha_i + \alpha_j)$ and to the haploid genotype A_i the genotypic value α_i. The genotypic scale coefficients $\{\alpha_i\}$ are chosen such that the mean-squared deviation between the genotypic value and the phenotypic value—namely, the propensity to perform altruism—is minimized in the following way. In those cases in which the altruists are diploid, the mean-squared deviation, calculated at birth, before selection, is

$$\sum_i \sum_j m_i f_i [(h_{ij} - \bar{h}) - (\alpha_i + \alpha_j)]^2 = \overline{D^2}, \qquad [8]$$

where, for example, m_i and f_i are the gene frequencies in the parental generation at the time of mating. Here, $A_i A_j$ performs altruism with probability h_{ij}, which entails a phenotypic deviation $h_{ij} - \bar{h}$ from the average in the population $\bar{h} = \Sigma_i \Sigma_j m_i f_j h_{ij}$. To find expressions for the α_i in terms of gene frequencies and h_{ij}, we differentiate $\overline{D^2}$ with respect to α_i and determine the genotypic values that minimize $\overline{D^2}$. This produces the normal equations for the α_i,

$$\sum_j (m_i f_j + m_j f_i)[(h_{ij} - \bar{h}) - (\alpha_i + \alpha_j)] = 0; \, i = 1,2,\ldots,n. \qquad [9]$$

These linear equations can then be solved for the α_i. For the case of diploid sib-to-sib altruism, in which the sexes are not distinguished with respect to the performance or reception of altruism, $m_i = f_i = p_i$ and Eq. 9 reduces to the familiar formula

$$\sum_j p_j (h_{ij} - \bar{h}) = \alpha_i. \qquad [10]$$

When altruism is performed by males in the haplodiploid system, the mean-squared deviation is

$$\sum_i f_i [(g_i - \bar{g}) - \alpha_i]^2 = \overline{D^2_M}, \qquad [11]$$

where $\bar{g} = \Sigma_i f_i g_i$, from which we immediately have $\alpha_i = g_i - \bar{g}$. The α_i obtained according to the above procedure are the "average effects" defined by Fisher (13).

By using the genotypic values obtained in this way, variances and covariances of genotypic values among siblings can be com-

puted for each model. These quantities for the cases of diploid sib-to-sib and haplodiploid sister-to-brother altruism are as follows. Diploid, sib-to-sib altruism: (*i*) sexes not distinguished,

$$\text{var(sib)} = \sum_i \sum_j p_i p_j (\alpha_i - \alpha_j)^2 \qquad [12]$$

$$\text{cov(sib,sib)} = \sum_i \sum_j \left(p_i p_j - \frac{1}{4} u_{ij} \right)(\alpha_i - \alpha_j)^2. \qquad [13]$$

(*ii*) Sexes distinguished,

$$\text{var(sib}_{\text{male}}) = \text{var(sib}_{\text{female}})$$

$$= \frac{1}{2} \sum_i \sum_j (m_i m_j + f_i f_j)(\alpha_i - \alpha_j)^2 \qquad [14]$$

$$\text{cov(sib}_{\text{male}}, \text{sib}_{\text{female}})$$

$$= \frac{1}{2} \sum_i \sum_j \left[m_i m_j + f_i f_j - \frac{1}{4}(m_{ij} + f_{ij}) \right](\alpha_i - \alpha_j)^2. \qquad [15]$$

Haplodiploid, sister-to-brother altruism:

$$\text{var(S)} = \frac{1}{2} \sum_i \sum_j [m_i m_j + f_i f_j](\alpha_i - \alpha_j)^2, \qquad [16]$$

$$\text{cov(SS)} = \frac{1}{2} \sum_i \sum_j \left(m_i m_j + f_i f_j - \frac{1}{4} f_{ij} \right)(\alpha_i - \alpha_j)^2 \qquad [17]$$

$$\text{cov(SB)} = \frac{1}{2} \sum_i \sum_j \left(f_i f_j - \frac{1}{4} f_{ij} \right)(\alpha_i - \alpha_j)^2, \qquad [18]$$

where S and B refer to haplodiploid sister and brother, and sib refers to diploids.

Note specifically that, because u_{ij} and f_{ij} refer to genotype frequencies in the *parental* generation after selection, these quantities cannot properly be replaced by Hardy–Weinberg combinations of gene frequencies (see refs. 1 and 3).

Covariance Between Fitness and Genotypic Value. Li (8) and Price (9) have suggested that the covariance between genotypic value and genotypic fitness provides a useful device for describing initial increase conditions and the structure of polymorphic equilibria under viability selection at a single locus. In the present context, we can write, for example,

$$T_f f'_{ii} = f_i m_i \phi_{ii} \qquad [19]$$

where ϕ_{ii} is a representation of the fitness of $A_i A_i$ and $f_i m_i$ is the frequency of $A_i A_i$ at birth. By using the fitnesses of the genotypes defined in this way and the genotypic distribution at birth, the covariance between female (sister) genotype (i.e., its additive genotypic value) and female fitness, in the diploid case is written cov(SG,SF), and

$$\text{cov(SG,SF)} = \sum_i 2\alpha_i T_f f'_{ii} + \frac{1}{2} \sum_i \sum_{j \neq i} (\alpha_i + \alpha_j) T_f f'_{ij}$$
$$- T_f \sum_i f_i \sum_j m_j (\alpha_i + \alpha_j) \qquad [20]$$
$$= 2 T_f \sum_i \alpha_i f'_i - T_f \sum_i (m_i + f_i) \alpha_i.$$

In Eq. 13, $T_f f'_{ii}$ and $T_f f'_{ij}$ represent the right sides of genotypic recursions for the appropriate model, for example Eqs. 1 and 2 above. Similarly, the covariance between male (brother) genotype and fitness for diploids is written cov(BG, BF) with

$$\mathrm{cov(BG,BF)} = \sum_i 2\alpha_i T_m m'_{ii} + \frac{1}{2}\sum_i \sum_{j\neq i} (\alpha_i + \alpha_j)T_m m'_{ij}$$
$$- T_m \sum_i (m_i + f_i)\alpha_i. \quad [21]$$

Note that Eqs. 20 and 21 together imply that for diploids in which the sexes are distinguished

$$\frac{\mathrm{cov(SG,SF)}}{T_f} + \frac{\mathrm{cov(BG,BF)}}{T_m}$$
$$= 2\sum_i [\alpha_i(m'_i + f'_i) - \alpha_i(m_i + f_i)] \quad [22]$$

while, when sexes are not distinguished, we write cov(sib G, sib F) with

$$\frac{\mathrm{cov(sib\ G, sib\ F)}}{T} = 2\sum_i \alpha_i(p'_i - p_i). \quad [23]$$

For haplodiploids, the covariance between male (brother) genotype and fitness is

$$\mathrm{cov(BG,BF)} = \sum_i \alpha_i T_m m'_i - T_m \sum_i f_i\alpha_i. \quad [24]$$

Then Eq. 24 can be combined with Eq. 20 to form

$$\frac{\mathrm{cov(SG,SF)}}{T_f} + \frac{\mathrm{cov(BG,BF)}}{T_m}$$
$$= \sum_i \alpha_i [(m'_i + 2f'_i) - (m_i + 2f_i)] \quad [25]$$

for haplodiploids.

Heuristically, we expect genotypes with positive genotypic values to increase if the total covariance between genotype and fitness (Eqs. 22, 23, and 25) is positive. Further, equilibria should exist at points for which the total covariance is zero.

RESULTS OF ANALYSIS OF THE MODELS

Model I: Diploid; Sib-to-Sib Altruism. *Polymorphic equilibria.* The equilibrium condition $p'_i = p_i$, which, from Eq. 16, entails cov(sib G, sib F) = 0 can be expanded as

$$\beta\left\{\sum_i \sum_j \left[p_i p_j - \frac{1}{4}u_{ij}\right](\alpha_i - \alpha_j)^2\right\} - 2\gamma\sum_i p_i\alpha_i^2 = 0 \quad [26]$$

or

$$\beta\mathrm{cov(sib,sib)} - \gamma\mathrm{var(sib)} = 0. \quad [27]$$

Two classes of equilibria may then exist. The first is defined by var(sib) = 0, which is satisfied only if $\alpha_i = \alpha_j$ for all i and j. From the calculation of α_i, at these equilibria we have for all i

$$h_i = \sum_j p_j h_{ij} = \bar{h}. \quad [28]$$

The equilibrium gene frequencies are therefore exactly those that would be obtained in a one-locus viability selection model, with viability h_{ij} for A_iA_j. For this reason, we term this first class *viability–analogous* equilibria. In the two-allele case, these are the points denoted p^* in ref. 3.

The second class of equilibria is such that var(sib) $\neq$ 0 and satisfies the relationship

$$\beta b_{\mathrm{sib}\to\mathrm{sib}} = \gamma, \quad [29]$$

where $b_{\mathrm{sib}\to\mathrm{sib}}$ is the regression of the recipient sib's additive genotypic value on that of the altruistic donor:

$$b_{\mathrm{sib}\to\mathrm{sib}} = \mathrm{cov(sib,sib)/var(sib)}. \quad [30]$$

For the multiple-allele situation, we have not obtained explicit existence conditions for the second class of equilibria, although in ref. 3, they were obtained explicitly for the two-allele case. The second class of equilibria will be termed *structural* to indicate their greater dependence on the detailed interaction between β, γ, and the h_{ij}.

In the diploid case, where we identify one sex (e.g., sisters) as the donor of altruism to the other (brothers), both sides of Eq. 22 are zero at equilibrium and expansion of either produces

$$\beta\left\{\sum_i \sum_j \alpha_i(m_i f_j + m_j f_i)(h_{ij} - \bar{h}) - \frac{1}{4}\sum_k \sum_i \sum_j \alpha_i (f_{ij}m_k \right.$$
$$\left. + m_{ij}f_k)(h_{ij} - h_{jk})\right\}\bigg/ T_m - \gamma\mathrm{var(sib_{female})}/T_f = 0,$$
$$[31]$$

where the α_i are derived from Eq. 9. As before, the viability–analogous equilibria are given by var(sib_{female}) = 0, satisfying $\alpha_i = \alpha_j$ for all i and j and the structural equilibria are characterized by the ratio of mean fitnesses of males to females at equilibrium, T_m/T_f given by rearranging Eq. 31. In the special case of no dominance, $h_{ij} - \bar{h} = \alpha_i + \alpha_j$, Eq. 24 reduces to

$$\beta(b_{\mathrm{sib_{female}}\to\mathrm{sib_{male}}})/T_m = \gamma/T_f, \quad [32]$$

where $b_{\mathrm{sib_{female}}\to\mathrm{sib_{male}}}$ is the regression of the recipient (male) genotypic value on that of the donor female. Similar results are obtained for the case where one sex performs altruism to both sexes.

Initial increase of a new allele. (*i*) Introduction of A_i near fixation of A_1: The analogous result to the two-allele case treated by Uyenoyama and Feldman (3) is that A_i will increase if introduced near the fixation of A_1 if

$$(h_{i1} - h_{11})[\beta/2 - \gamma] > 0. \quad [33]$$

Condition 33 is equivalent to

$$(\alpha_i - \alpha_l)[\beta\lim b_{\mathrm{sib}\to\mathrm{sib}} - \gamma] > 0, \quad [34]$$

where $\lim b_{\mathrm{sib}\to\mathrm{sib}}$ represents the limiting value of the regression near fixation of A_1. The condition analogous to [33] in the case in which female diploid sibs are donors to male diploid sibs is

$$(h_{i1} - h_{11})\left(\frac{\beta/2}{1 + \beta h_{11}} - \frac{\gamma}{1 - \gamma h_{11}}\right) > 0. \quad [35]$$

(*ii*) Introduction of A_{n+1} near a viability–analogous equilibrium at which alleles $A_1, A_2, \ldots, A_n$ are segregating: For this it is assumed that the viability–analogous equilibrium is stable in the frequency simplex of $A_1, A_2, \ldots, A_n$. The condition for the initial increase of A_{n+1} on its introduction near this equilibrium is

$$[(\beta/2) - \gamma](h_{n+1\cdot} - \bar{h}) > 0, \quad [36]$$

where $h_{n+1\cdot} = \sum_{i=1}^n p_i h_{n+1,i}$ and $\bar{h} = \sum_i\sum_j p_i p_j h_{ij}$. Condition 36 can be written in a form analogous to [34]—namely,

$$(\alpha_{n+1} - \alpha)(\beta\lim b_{\mathrm{sib}\to\mathrm{sib}} - \gamma) > 0, \quad [37]$$

where $\alpha_i = \alpha_j = \alpha = 0$ for $i = 1, 2, \ldots, n$ at the viability–analogous equilibrium, and $\lim b_{\mathrm{sib}\to\mathrm{sib}}$ denotes the limit of the regression of recipient's additive genotypic value on the donor's.

In the diploid case with sexes distinguished, A_{n+1} increases when rare if

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$$(h_{n+1\cdot} - \bar{h})\left(\frac{\beta/2}{1 + \beta\bar{h}} - \frac{\gamma}{1 - \gamma\bar{h}}\right) > 0, \qquad [38]$$

which, as with [35], can be represented

$$(\alpha_{n+1} - \alpha)\left(\frac{\beta \lim b_{\text{sib}_{\text{female}} \to \text{sib}_{\text{male}}}}{1 + \beta\bar{h}} - \frac{\gamma}{1 - \gamma\bar{h}}\right) > 0 \qquad [39]$$

with the interpretations already given.

Model II. Haplodiploid Sibling Altruism. *Polymorphic equilibria.* Both sides of Eq. 25 vanish at equilibrium and, in the model of sister-to-brother altruism, the resulting identity reduces to

$$\beta\left[\sum_i \sum_j f_i m_j \alpha_i (h_{ij} - \bar{h}) - \frac{1}{4}\sum_i \sum_j \sum_k \alpha_i m_j f_{ik}\right.$$
$$\left. \times (h_{ij} - h_{jk})\right]\bigg/ T_m - \gamma\text{var}(S)/T_f = 0, \qquad [40]$$

where the α_i are derived from Eq. 9. Eq. 40 describes two classes of equilibria. The first is characterized by var(S) = 0, which is satisfied if and only if $\alpha_i = \alpha_j$ for all i and j. In this first equilibrium class, $m_i = f_i$ and $\Sigma_j f_j(h_{ij} - \bar{h}) = 0$ for all i. Such equilibria are extensions to the multiple-allele case of the points denoted p^* in the haplodiploid models of ref. 3. As in the diploid case, these equilibrium gene frequencies are exactly those of a one-locus viability selection model with genotypic fitnesses h_{ij}. We term this class of equilibria *viability–analogous* in the haplodiploid case as well.

The structural equilibria emerge if var(S) $\neq$ 0. Then the ratio of the mean fitnesses of males and female at equilibrium, T_m/T_f, is obtained by rearranging Eq. 40. Under the special assumption that there is no dominance in the phenotypic value—i.e., $h_{ij} - \bar{h} = \alpha_i + \alpha_j$—Eq. 40 reduces to

$$\frac{\beta b_{S \to B}}{T_m} - \frac{\gamma}{T_f} = 0 \qquad [41]$$

where $b_{S \to B}$ is the regression of the brothers' genotypic values on those of the sisters':

$$b_{S \to B} = \text{cov}(SB)/\text{var}(S).$$

Other cases of sib altruism in haplodiploids exhibit a similar equilibrium structure. As in models I and II, there is a viability–analogous equilibrium of the sister-to-sister model if the h_{ij} viewed as one-locus viabilities would allow a valid polymorphism. With sister-to-sister altruism, all equilibria of the second equilibrium class, the structural equilibria, are described by

$$\beta b_{S \to S} = \gamma, \qquad [42]$$

even if the no-dominance restriction on h_{ij} does not hold. Under brother-to-sister altruism, the viability–analogous equilibrium does not exist and the only equilibrium class is described by

$$\frac{\beta b_{B \to S}}{T_f} = \frac{\gamma}{T_m}. \qquad [43]$$

It can be shown that no polymorphic equilibria exist in the model of brother-to-brother altruism.

Initial increase of a new allele. (*i*) Introduction of A_i near fixation of A_1: Fixation of A_1 is unstable to the introduction of A_i in the sister-to-brother case if

$$(h_{i1} - h_{11})\left[\frac{1}{4}\beta(1 - h_{i1}\gamma) - \gamma(1 + h_{11}\beta)\right] > 0. \qquad [44]$$

Condition 44 is equivalent to

$$(\alpha_i - \alpha_1)\left[\frac{\beta\lim b_{S \to B}}{1 + h_{11}\beta} - \frac{\gamma}{1 - h_{11}\gamma}\right] > 0,$$

where $\lim b_{S \to B}$ represents the limiting value of the regression near the fixation.

The condition corresponding to [44] for the case of sister-to-sister altruism is

$$(h_{i1} - h_{11})\left(\frac{3}{4}\beta - \gamma\right) > 0. \qquad [45]$$

For the case of brother-to-sister altruism, the condition is

$$(g_i - g_1)\left[\frac{1}{2}\beta(1 - \gamma g_1) - \gamma(1 + g_1\beta)\right] > 0 \qquad [46]$$

and, for brother-to-brother altruism, it is

$$(g_i - g_1)\left(\frac{1}{2}\beta - \gamma\right) > 0. \qquad [47]$$

Condition 45 is equivalent to

$$(\alpha_i - \alpha_1)[\beta\lim b_{S \to S} - \gamma] > 0,$$

condition 46 is equivalent to

$$(\alpha_i - \alpha_1)\left(\frac{\beta\lim b_{B \to S}}{1 + g_1\beta} - \frac{\gamma}{1 - g_1\gamma}\right) > 0,$$

and condition 47 is equivalent to

$$(\alpha_i - \alpha_1)[\beta\lim b_{B \to B} - \gamma] > 0.$$

(*ii*) Introduction of A_{n+1} near a viability–analogous equilibrium at which alleles $A_1, A_2, \ldots, A_n$ are segregating: Under the assumption that the viability–analogous equilibrium is itself stable, A_{n+1} will increase in frequency from an initially rare state in the sister-to-brother case if

$$(h_{n+1\cdot} - \bar{h})\left[\frac{1}{4}\beta(1 - h_{n+1\cdot}\gamma) - \gamma(1 + \bar{h}\beta)\right] > 0, \qquad [48]$$

where $h_{n+1\cdot} = \Sigma_{j=1}^n \hat{f}_j h_{n+1,j}$, $\bar{h} = \Sigma_{j=1}^n \Sigma_{k=1}^n \hat{f}_j \hat{f}_k h_{jk}$, and the $\hat{f}_j$ represent the equilibrium gene frequencies at the viability–analogous equilibrium. Condition 48 is equivalent to

$$(\alpha_{n+1} - \alpha)\left[\frac{\beta\lim b_{S \to B}}{1 + \bar{h}\beta} - \frac{\gamma}{1 - \gamma\bar{h}}\right] > 0, \qquad [49]$$

where $\alpha_j = \alpha_k = 0$ for j and k from $1, 2, \ldots, n$ as computed at the viability–analogous equilibrium point, and $\lim b_{S \to B}$ is the limiting value of the regression near the equilibrium.

With sister-to-sister altruism, A_{n+1} will increase in frequency near the viability–analogous equilibrium point if

$$(h_{n+1\cdot} - \bar{h})\left(\frac{3}{4}\beta - \gamma\right) > 0, \qquad [50]$$

which is equivalent to

$$(\alpha_{n+1} - \alpha)(\beta\lim b_{S \to S} - \gamma) > 0, \qquad [51]$$

where $\alpha_j = \alpha_k = \alpha$ for $j, k \in \{1, 2, \ldots, n\}$ and $\lim b_{S \to S}$ is the limiting value of the regression near the viability–analogous equilibrium. In models involving altruism by brothers, the viability–analogous equilibrium does not exist.

DISCUSSION

We have extended the analysis of exact population genetic models of sibling altruism to include an arbitrary number of alleles at a single locus. Interior equilibria of the models studied fall into two classes. The first class, termed viability–analogous,

corresponds to the p^* point that we found in the case of two alleles (ref. 3; see also ref. 14). The second class, termed structural, corresponds to points at which relatedness is equal to the cost/benefit ratio in several of our models. Both equilibrium classes arise naturally from consideration of the covariance between fitness and genotypic value at equilibrium.

The viability–analogous equilibrium corresponds directly to the equilibrium obtained in the traditional multiple-allele viability selection model in which the h_{ij} are regarded as viability parameters. All genotypic scale factors are identical and equal to zero at this equilibrium. The structure of the viability–analogous equilibrium is quite different from the second equilibrium class at which the connection to a generalized version of Hamilton's rule is apparent. This justifies our use of the term structural for the latter equilibria.

The equality $r_H = \gamma/\beta$ describes a class of genotype frequencies, rather than a special relationship between the parameters β and γ. We have shown that structural equilibria corresponding to $r_H = \gamma/\beta$ do in fact exist in a number of our models. In model I with diploid sib-to-sib altruism in which the sexes are not distinguished, structural equilibria are described by $\mathrm{cov(sib,sib)/var(sib)} = \gamma/\beta$, where the left side represents the regression of sib on sib or relatedness in our models. However, in model II of altruism by sisters toward their brothers under haplodiploidy, identity between the structural equilibria of our models and the relationship $\mathrm{cov}(SB)/\mathrm{var}(S) = \gamma/\beta$ fails except in the case of phenotypes for which no dominance among alleles exists. Nevertheless, all equilibria in this model and the other models studied here correspond to points at which the covariance between genotypic value and fitness is equal to zero without the necessity of imposing restrictions on dominance among the alleles.

The covariance between genotypic value and fitness has been shown (8, 9) to be equal to differences between generations of gene frequencies in one-locus models of viability selection, and the method has been applied directly to kin selection theory (ref. 10; see also ref. 15). In our models, this covariance is equal to differences between generations of averages of the additive allelic effects. Of course, differences between generations of any function of gene or genotype frequencies will be intimately related to initial increase conditions and reduce to zero at equilibrium, but the particular differences (Eqs. 22, 24, and 25) have heuristic appeal as covariances between genotypic value and fitness.

The covariance approach possesses a number of advantages over the adaptive topography concept suggested in our previous paper (ref. 3; see also ref. 14). With the covariance approach, differences in reproductive value between the sexes, as represented by separate normalization of male and female fitnesses by the mean fitness in each sex, arise in a more natural way. Further, in the models studied here of additive composition of gains and losses due to altruism, Hamilton's rule emerges as a special case within the general covariance framework. Restriction of Hamilton's rule to the case of no dominance in phenotypic values (i.e., $\bar{h}_{ij} = \alpha_i + \alpha_j$) was noted for models involving performance of altruism by diploids and reception of benefits by members of the opposite sex in our earlier paper (3). In that paper, we suggested that the discrepancies might be due to either of two reasons: (*i*) some aspect of our representation of the verbal theory may have been incorrect or (*ii*) the models showing these departures in fact represented limits within which the verbal theory applies. In view of the results reported here in which a distinct heuristic framework has been shown to predict all of the results of the exact models, we now favor the latter interpretation.

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