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THE POPULATION GENETICS OF SOCIAL INTERACTIONS

By

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SIGNED: 

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ABSTRACT

The concept of inclusive fitness plays a key role in much of sociobiology. Yet most theoretical studies concerning the evolution of social behavior circumvent inclusive fitness by mobilizing the concept of frequency dependent individual fitness. Given certain assumptions, it is shown that models based on these two different concepts are dynamically equivalent. The models do differ, however, in bookkeeping methods which are advantageous under different circumstances. A knowledge of these circumstances should prove of value to students of social behavior.

It is then shown that evolution acts according to an adaptive landscape based on Hamilton's inclusive fitness in the absence of strong selection and inbreeding. This yields an inclusive fitness analogue to much of traditional population genetics. For example, heterozygote superiority in inclusive fitness yields stable polymorphisms, while intermediate dominance results in fixation of one of the alleles. When individuals do not affect one another's fitnesses, the inclusive fitness topography collapses to one based on individual fitness. A general rule for the evolution of social behavior under intermediate dominance is shown to yield Hamilton's Rule as a special case.

Next, a general model for examining the evolution of social behavior is developed which, unlike inclusive fitness models, does not require that benefits received be linear functions of the number of

social donors encountered. The subsocial route for the evolution of eusociality in haplodiploid organisms is then examined within the context of this model. Nonlinearities render conditions for frequency independent fixation or loss of sister-helping alleles more stringent than expected from models based on the assumption of linear benefits. In particular, both stable polymorphisms and frequency dependent selective thresholds for sister-helping behavior may commonly obtain.

CHAPTER 1

THE RELATION OF FAMILY STRUCTURED MODELS TO INCLUSIVE FITNESS MODELS FOR KIN SELECTION

Inclusive Fitness and Current Research

In 1963, Hamilton proposed his $c/b < r$ rule for the evolution of altruism. This rule has provided both the motivation and direction for many studies concerning the evolution of social behavior.

An equally important result of Hamilton's work lies in a subtle shift of focus. Population genetics has traditionally associated the evolutionary potential of a trait with its effects on individual fitness. Instead, as Hamilton pointed out, the fundamental determinant of how selection acts on a trait lies in the trait's effects on genic rather than individual fitness. Thus, he defined "inclusive fitness" to include the effects an individual has on copies of its genes in other individuals, as well as its effects on its own genes.

Because inclusive fitness plays a major role in thought concerning the evolution of social behavior (see, for example, empirical studies by Eberhard, 1972; Alexander, 1974; Wilson, 1975; Trivers and Hare, 1976; Metcalf and Whitt, 1978a and 1978b; Noonan, 1978; Greenberg, 1979), it is surprising to note that it has not been mobilized in recent theoretical investigations of social behavior. Some reasons given for this pattern are that Hamilton's model is not rigorous (Levitt, 1975), that it is difficult to explain to others (Charnov, 1977), that it is a strategic,

rather than a genetical model (Cavalli-Sforza and Feldman, 1978; Roughgarden, 1979), and that the coefficient of relationship Hamilton used is "ambiguous" (Harpending, 1979). Thus, most present investigators of kin selection (e.g., Levitt, 1975; Charnov, 1977, Cavalli-Sforza and Feldman, 1978; Charlesworth, 1978; Wade, 1978; Michod, 1980; Uyenoyama and Feldman, 1980) have circumvented the inclusive fitness concept by constructing family structured models in which individual fitness is made a function of the number of altruists in the various family types.

Below, I examine the relation of Hamilton's model to the family structured approach. There are at least three reasons for doing so: (1) to obviate the need for rederiving Hamilton's original results (e.g., portions of Levitt, 1975; Charnov, 1977; Cavalli-Sforza and Feldman, 1978; Charlesworth, 1978; Wade, 1978) should Hamilton's model prove equivalent to the family structured models; (2) to help relate Hamilton's model to parameters and concepts commonly used in classical population genetics; and (3) to justify the use of a modelling tool, inclusive fitness, which may facilitate future theoretical studies of social behavior.

The analysis consists of showing that simple algebraic rearrangements of a typical (see Appendix A) individual fitness model (i.e., Charnov, 1977) for kin selection yield Hamilton's (1964) model. The key to this study lies in the use of measures of genetic identity to generate the conditional interaction probabilities used in family structured models (see Cotterman, 1940, in Crow and Kimura, 1970; and see also Li and Sacks, 1954). The use of these measures, of course, depends on the

assumption of weak selection which pervades much of the literature on the theory of kin selection.

Comparison of Dynamics of Inclusive Fitness and Family Structured Models

Two genetic models are dynamically equivalent if and only if they predict the same changes in gene frequency. To see when this holds, note that:

$$p(t+1) = \frac{N_1(t+1)}{N_1(t+1) + N_2(t+1)} = \frac{N_1(t)W_1}{N_1(t)W_1 + N_2(t)W_2} \qquad (1.1)$$

where $N_i(t)$ is the number of A_i alleles at time t, and W_i is the fitness of the A_i allele. Consequently, two genetic models are dynamically equivalent if they produce the same W_i's. I now derive these allelic fitnesses for Charnov's (1977) individual fitness model. Later, it is shown that these are, indeed, the same allelic fitnesses obtained from Hamilton's model.

The general formulation of Charnov's (1977) family structured model is given as Equation A.5 in Appendix A. Since $\sum_k P(ij/k)P(k/uv) = P(ij/uv)$, Equation A.5 can be written as:

$$W_{ij} = 1 + cH_{ij} + \frac{1}{P(ij)} \sum_{uv} bH_{uv}P(uv)\ P(ij/uv) \qquad (1.2)$$

Thus, assuming weak selection, Equation 1.2 becomes:

$$W_{11} = 1 + H_{11}c + b[H_{11}P(11/11) + \frac{2(1 - p)}{p} H_{12}P(11/12) +$$

$$\frac{(1 - p)^2}{p^2} H_{22}P(11/22)], \qquad (1.3a)$$

$$W_{12} = 1 + b[\frac{p}{2(1 - p)} H_{11}P(12/11) + H_{12}P(12/12) +$$

$$\frac{(1 - p)}{2p} H_{22} P(12/22)] + H_{12}c, \qquad (1.3b)$$

and

$$W_{22} = 1 + b[\frac{p^2}{(1 - p)^2} H_{11}P(22/11) + \frac{2p}{(1 - p)} H_{12}P(22/12) +$$

$$H_{22}P(22/22)] + H_{22}c. \qquad (1.3c)$$

Since $W_1 = p(W_{11}) + (1 - p)W_{12}$, and $W_2 = pW_{12} + (1 - p)W_{22}$, Equations 1.3a, 1.3b, and 1.3c yield:

$$W_1 = 1 + pH_{11}[bP(11/11) + \frac{b}{2} P(12/11) + c] +$$

$$2(1 - p)H_{12}[bP(11/12) + \frac{b}{2} P(12/12) + c] +$$

$$\frac{(1 - p)^2}{p} H_{22}[bP(11/22) + \frac{b}{2} P(12/12)] \qquad (1.4a)$$

and

$$W_2 = 1 + \frac{p^2}{1 - p} H_{11}[bP(22/11) + \frac{b}{2} P(12/11)]$$

$$2pH_{12}[bP(22/12) + \frac{b}{2} P(12/12) + c] +$$

$$(1 - p)H_{22}[bP(22/22) + \frac{b}{2} P(12/22) + c].$$

These are the equations for marginal allelic fitnesses from Charnov's model. As an interesting aside, we may relate Equations 1.4a and 1.4b to an intuitive view of genic fitness in Appendix B. To compare Equations 1.4a and 1.4b to Hamilton's model, I now turn to re-expressing them in Hamilton's notation.

The first difference between Hamilton's and the family structured models is in the methodology used to calculate the interaction probabilities P(ij/uv). In the family structured models, this quantity is calculated by taking the average interaction probabilities over all family types. Thus, in a family structured model,

$$P(ij/uv) = \sum_{k=1}^{d} P(k/uv)\, P(ij/uv \text{ and } k)$$

where P(d) is the frequency of a given family type, and P(ij/uv and k) is the P(ij/uv) for that family type. An example of the calculation of P(ij/uv) for sib-sib interactions random with respect to genotype within a sibship is given in Table 1.

An alternate method which yields the same results as the family structured approach is to use genetic identity coefficients to generate these conditional probabilities. Following Cotterman (1940), in Crow and Kimura (1970), and Li and Sacks (1954), let C_0, C_1, and C_2 be the probilities that a recipient has zero, one, and two alleles identical by descent to those of the altruist donator. These three coefficients

Table 1. Calculations of P(ij/uv) via family structured approach. -- Note the equivalence of these results with those from Li and Sacks (1954) approach outlined in Table 2, part B. $P(ij/uv) = \sum_k P(uv/k)P(ij/uv \text{ and } K)$ is calculated assuming that family sizes are large, and that interactions within families are randomly distributed. See text for further details.

Family Types: Parents	Expected Frequencies	Prob(Family Type/A_1A_1) [=P(k/A_1A_1)]	Prob(Family Type/A_1A_2) [=P(k/A_1A_2)]	Interaction Probabilities Given Each Family Type [=P(ij/k)]		
				P(11/k)	P(12/k)	P(22/k)
$A_1A_1 \times A_1A_1$	p^4	p^2	0	1	0	0
$A_1A_1 \times A_1A_2$	$4p^3(1-p)$	$2p(1-p)$	p^2	1/2	1/2	0
$A_1A_1 \times A_2A_2$	$2p^2(1-p)^2$	0	$p(1-p)$	0	1	0
$A_1A_2 \times A_1A_2$	$4p^2(1-p)^2$	$(1-p)^2$	$p(1-p)$	1/4	1/2	1/4
$A_1A_2 \times A_2A_2$	$4p(1-p)^3$	0	$(1-p)^2$	0	1/2	1/2
$A_2A_2 \times A_2A_2$	$(1-p)^4$	0	0	0	0	1

Therefore, since $P(ij/uv) = \sum_k P(k/uv)P[ij/uv \text{ and } k]$

$$P(11/11) = p^2 + p(1-p) + (1/4)(1-p)^2 = 1/4 + \frac{p}{2} + \frac{p^2}{4}$$

$$P(12/11) = p(1-p) + (1/2)p(1-p) = (1/2)(1-p) + (1/2)p(1-p)$$

$$P(11/12) = (1/2)p^2 + (1/4)(1-p) = (1/4)p^2 + (1/4)p$$

$$P(12/12) = (1/2)p^2 + p(1-p) + (1/2)p(1-p) + (1/2)(1-p)^2 = 1/2 + (1/2)p(1-p)$$

$$P(22/11) = (1/4)(1-p)^2 = (1/4)(1-p)^2$$

$$P(22/22) = (1/4)p(1-p) + (1/2)(1-p)^2 = (1/4)(1-p) + (1/4)(1-p)^2$$

are a special case of the nine condensed identity coefficients which must be used in cases involving inbreeding to generate the interaction probabilities (Michod, 1979). It should be stressed that the use of such coefficients have meaning only for neutral genes. When outbreeding and weak selection obtain, the proportion of alleles identical by descent in two individuals is given by the coefficient of relationship r, which is $C_2 + (1/2)C_1$ (Li and Sacks, 1954; and Cotterman, 1940, in Crow and Kimura, 1970). Since C_0, C_1, and C_2 are independent of gene frequency, r is independent of gene frequency.

Table 2 shows how the probabilities of interaction in Equations 1.4a and 1.4b can be described in terms of C_0, C_1, and C_2. For example, $P(11/12) = (1/2)C_1p + C_0p^2$, since if the interactants share two alleles identical by descent, they cannot, barring mutation, be of different genotypes. If they share one allele by descent, the probability that our recipient of genotype A_1A_1 is $(1/2)\,p$. Similarly, if the interactants have no alleles identical by descent, the probability that the recipient is A_1A_1 is simply p^2, the frequency of A_1A_1 in the population at large. In Table 2 the calculation of P(ij/uv) for sibling interactions is demonstrated, and the results are shown to be equivalent to those for the family structured approach.

By using the identities in Table 2, we can perform some useful rearrangements. For example, part of Equation 1.4a may be expressed as:

$$2bP(11/12) + bP(12/12) = 2b[(1/2)C_1p + C_0p^2] + b[C_2 + (1/2)C_1 + C_02p(1 - p)]$$

Table 2. Probabilities of interactions between genotypes in outbred population in terms of identity coefficients and gene frequency -- See text for further details.

(A) General Model	(B) Example: Sib-sib interactions: $C_0=1/4, C_1=1/2, C_2=1/4$
$P(11/11) = C_2 + pC_1 + p^2C_0$	$P(11/11) = 1/4 + (1/2)p + (1/4)p^2$
$P(11/12) = (1/2)pC_1 + C_0p^2$	$P(11/12) = (1/4)p + (1/4)p^2$
$P(11/22) = p^2C_0$	$P(11/22) = (1/4)p^2$
$P(12/11) = (1-p)C_1 + 2p(1-p)C_0$	$P(12/11) = (1/2)(1-p) + (1/2)p(1-p)$
$P(12/12) = C_2 + (1/2)C_1 + C_02p(1-p)$	$P(12/12) = 1/4 + 1/4 + (1/2)p(1-p)$
$P(12/22) = pC_1 + 2P(1-p)C_0$	$P(12/22) = (1/2)(1-p) + (1/2)p(1-p)$
$P(22/11) = (1-p)^2C_0$	$P(22/11) = (1/4)(1-p)^2$
$P(22/12) = (1/2)(1-p)C_1 + (1-p)^2C_0$	$P(22/12) = (1/4)(1-p) + (1/4)(1-p)^2$
$P(22/22) = C_2 + (1-p)C_1 + (1-p)^2C_0$	$P(22/22) = 1/4 + (1/2)(1-p) + (1/4)(1-p)^2$

$$= b[C_1(p + 1/2) + C_0(2p^2 +$$

$$2p - 2p^2) + C_2]$$

$$= b[(1/2)C_1 + C_2) + (C_1p + 2C_0P)]$$

Noting that $(1/2)C_1 + C_2 = r$, and $(1/2)\,C_1 + C_0 = (1 - r)$, we may obtain from the above equation:

$$2bP(11/12) + bP(12/12) = b[r + 2p(1 - r)]. \tag{1.5a}$$

Similarly,

$$2bP(11/11) + bP(12/11) = b[2r + 2p(1 - r)], \tag{1.5b}$$

$$2bP(11/22) + bP(12/22) = b[2p(1 - r)], \tag{1.5c}$$

$$2bP(22/11) + bP(12/11) = b[2(1 - p)(1 - r)] \tag{1.5d}$$

$$2bP(22/12) + bP(12/12) = b[r + 2(1 - p)(1 - r)], \tag{1.5e}$$

and

$$2b(22/22) + bP(12/22) = b[2r + 2(1 - p)(1 - r)] \tag{1.5f}$$

Substituting Equations 1.5a, 1.5b, and 1.5c into Equation 1.4a yields:

$$W_1 = 1 + pH_{11}[b(r + p(1 - r) - c] +$$

$$(1 - p)H_{12}[b(r + 2p(1 - r)) - c] +$$

$$(1 - p)^2H_{22}[b(1 - r)] \tag{1.6}$$

Recall now that $H_{ij}b$ is the effect of an individual of genotype A_iA_j on other individuals, and $H_{ij}c$ is the effect of genotype A_iA_j's altruistic acts on its own fitness. These are symbolized in Hamilton's terminology by $(\delta a_r)_{ij}$, where A_iA_j is the genotype of the altruist and δa_r is the effect of the altruist on individuals of relatedness r. Equation 1.6 may, therefore, be written as:

$$W_1 = 1 + \sum_j p_j \sum_r r(\delta a_r)_{ij} + \sum\sum_{ij} p_i p_j \sum_r (1 - r)(\delta a_r)_{ij} \qquad (1.7)$$

We may now define $\Sigma r(\delta a_r)_{ij}$ as the "inclusive fitness effect" of an individual of genotype A_iA_j, δR_{ij}, which acts preferentially on individuals of genotypes similar to A_iA_j, and we may define $\Sigma(1 - r)(\delta a_r)_{ij}$ as the "diluting effect," δS_{ij}, which returns additional alleles to the population in their original frequencies.

Equation 1.7 may thus be written as:

$$W_1 = 1 + R_{1.} + S_{..} \qquad (1.8a)$$

where $\delta R_{1.} = \sum_j p_j \sum_r r(\delta a_r)_{ij}$ is the mean of δR for allele A_1 and $\delta S_{11..}$ $\sum\sum_{ij} p_i p_j \sum_r (1 - r)(\delta a_r)_{ij}$ is the mean δS over the population regardless of genotype.

A similar treatment of Equation 1.4b gives us:

$$W_2 = 1 + \delta R_{2.} + \delta S_{..} \qquad (1.8b)$$

Therefore, by Equations 1.8a and 1.8b,

$$p(t+1) = \frac{P(t)W_1}{\bar{W}} = p(t)\,\frac{1 + \delta R_{1\cdot} + \delta S_{\cdot\cdot}}{p(1+\delta R_{1\cdot}+\delta S_{\cdot\cdot})+(1-p)(1+\delta R_{2\cdot}+\delta S_{\cdot\cdot})}$$

$$= p(t)\,\frac{1 + \delta R_{1\cdot} + \delta S_{\cdot\cdot}}{1 + \delta T_{\cdot\cdot}} \qquad (1.9)$$

where

$$\delta T_{\cdot\cdot} \equiv \sum_i p_i(\delta R_{1\cdot} + \delta S_{\cdot\cdot}).$$

Equation 1.9 is simply an expression of Hamilton's model (see Equation 2 in Hamilton, 1964). We, therefore, conclude that Hamilton's model is, indeed, equivalent to family structured individual fitness models for kin selection. The individual fitness models appear to differ from Hamilton's inclusive fitness model only in trivial details of bookkeeping, not in genetic dynamics.

Relative Advantages of Different Kin Selection Models

It is of interest here to briefly contrast the two approaches to kin selection examined above. First, Hamilton's model gives both quantitative and qualitative results in terms of the coefficients of relationship. Family structured approaches, on the other hand, yield specific results which can then be interpreted in terms of coefficients of relationship of the interactants. Since the interactants must be redefined for each case examined, family structured models lack the generality of Hamilton's model. Second, Hamilton's model takes into account the

evolution of multiple alleles coding for different social traits at a single locus. In general, family structured models are analytically tractable only when one of the two alleles at the locus considered is non-social. The single exception to this is provided by Uyenoyama and Feldman (1980); but the expression of sociality in their model may vary only in intensity (h_{ij}). In Hamilton's model, on the other hand, the c_{ij}'s and b_{ij}'s may vary independently of one another. Also, the genetic relationship of recipient to donor may vary in Hamilton's model.

These advantages of Hamilton's model appear to stem from the factoring of interactions into two categories. In the first category, which occurs in proportion (1 - r), alleles are added or removed from the population in their original frequencies. Effects on this category generally do not affect the direction of selection. In the second category are interactions with genes which, due to some factor, are not in the same frequencies as those in the population at large. Interactions affecting the fitnesses of genes in this category can affect the direction of selection.

The measure of relative importance of these categories used by Hamilton, r, is valid only in outbred populations. The problems associated with this fact are discussed in Michod and Anderson (1979) and in Michod (1979).

Family structured models, on the other hand, may be particularly well suited for relating kin selection to traditional concepts in population genetics. For example, these models have proved of great value in examining the relative roles of intra- and inter-family selection in

the evolution of social behavior (Michod, submitted manuscript; Wade and Breden, personal communication).

Another major advantage of family structured models lies in their adaptability, since the population processes underlying the interactions are explicitly represented. For example, when strong selection occurs, adult genotypic frequencies are not those expected under Hardy-Weinberg conditions. The only successful analyses of evolution in this situation have been via family structured framework (Levitt, 1975; Cavalli-Sforza and Feldman, 1978; Uyenoyama and Feldman, 1980; Michod, submitted manuscript). That the Hardy-Weinberg assumption affects the genetic dynamics only negligible, provided selection is weak, is claimed in Charlesworth (1979) and is proven in Appendices C and D for sib-sib helping in diploid and haplodiploid organisms. Consideration of the effects of non-random interactions within sibships can also be examined with family structured models (Charlesworth, 1978); but the topic has not been broached by an inclusive fitness model. Hamilton's (1964) model also assumes that the benefit accrued to a recipient of altruistic acts is a linear function of the number of altruists in its interaction group: the family structured model of Levitt (1975) and Chapter 3 of this dissertation provide the only means of examining the effects of violating this assumption.

In summary, it appears that the different theoretical approaches to the study of kin selection presently discussed are equivalent under the circumstances assumed here. The need for rederivation of results from different modelling approaches, therefore, seems minimal. The

models do differ, however, in bookkeeping methods, which appear to be advantageous under different circumstances. A knowledge of these circumstances will certainly prove rewarding to students of social behavior.

CHAPTER 2

ADAPTIVE TOPOGRAPHIES BASED ON INCLUSIVE FITNESS

The Adaptive Topography Concept

Sewall Wright's (1942) concept of adaptive topographies based on mean individual fitness has provided a simple means of visualizing evolution. When selection pressures are independent of gene frequency, evolution is expected to cause changes in gene frequency which cause natural populations to "climb" to the tops of "adaptive peaks" in mean individual fitness. A given adaptive peak may represent fixation or loss of an allele, or it may represent the maintained presence of more than one allele at a given locus in a population.

In more complicated cases, however, our intuitive view of the evolutionary process often becomes blurred: the adaptive topography becomes based on "fitness functions" which are often intuitively and analytically difficult. Below, I show that, for social behavior, the adaptive topography can be based on an intuitively simple notion which Hamilton (1964) has called "inclusive fitness." This construct stresses that an individual may pass copies of his genes on to future generations by aiding neighbors who are genetically similar to himself. I further show that the mean inclusive fitness topography collapses into Wright's more familiar individual fitness topography when social interactions are absent.

The present results are of interest for at least two reasons: (1) they provide a means of utilizing classical studies concerning the adaptive landscape to extend Hamilton's theory of social behavior; and (2) they clearly delineate conditions under which evolution maximizes inclusive fitness. This is important, since the maximization of inclusive fitness provides the basis for much of sociobiology (e.g., Eberhard, 1972; Alexander, 1974; Wilson, 1975; Trivers and Hare, 1976; Brown, 1978; Metcalf and Whitt,1977a; Noonan, 1978; Oster and Wilson, 1978; Greenberg, 1979); yet, contrary to common belief, proof of maximization of inclusive was never proved by Hamilton.

Hamilton's Model and the Maximization of Inclusive Fitness

Before demonstrating maximization of inclusive fitness, I will provide a brief explication of the original derivation (Hamilton, 1964) of Hamilton's model. This is useful since, as mentioned in Chapter 1, the original derivation is not well understood but is more general in that it provides a means of allowing b and c to be independent of one another for the different genotypes considered. Also, this derivation provides an intuitively simple means of viewing kin selection.

As before, let C_0, C_1, and C_2 be the probabilities that a recipient has zero, one, and two alleles at a given locus which are identical by descent to those of the social donor. Then given a donor of genotype A_iA_j, $C_1 + 2C_2$ is the number of genes a given recipient has which are of type A_i or A_j due to identity by descent with genes in the donor. The probability that a single allele drawn from the recipient is

identical by descent to one of the donor's alleles is, therefore, $1/2C_1 + C_2$, which is called r in the present treatment.

Let $(\delta a_r)_{ij}$ represent the total effect social interactions by an average A_iA_j individual have on the survival of individuals related to him by r. Then the total effect an A_iA_j individual has is $\sum_r (\delta a_r)_{ij}$.

Now:

$$\sum_r (\delta a_r)_{ij} = \sum_r r(\delta a_r)_{ij} + \sum_r (1 - r)(\delta a_r)_{ij}.$$

This simple step is used repeatedly in the present derivation, and it divides the effects of social interactions by a donor of genotype A_iA_j into two components. The $\sum_r (1 - r)(\delta a_r)_{ij}$ component reflects interactions affecting genes randomly sampled from the population, while the $\sum_r r(\delta a_r)_{ij}$ component reflects interactions affecting genes which are identical by descent to one of the genes in the donor.

Now, let $p_i(t + 1)$ be the frequency of allele A_i at time $t + 1$. Then if we let N be the population size and N_i be the number of A_i alleles,

$$P_i(t + 1) = \frac{N_i(t + 1)}{2N(t + 1)} \qquad (2.1)$$

Let N(t) and p(t) be abbreviated as N and p. Then,

$$N_i(t + 1) = N \{[2p_i^2 + 2p_i(1 - p_i)] + p_i^2 \sum_r r2(\delta a_r)_{ii} +$$

$$\sum_{j \neq i} 2p_i p_j \sum_r (1/2) r 2(\delta a_r)_{ij} +$$

$$p_i \sum_k \sum_j p_k p_j \sum_r (1 - r) 2(\delta a_r)_{kj} \}. \qquad (2.2)$$

To see how this equation works, note first that $N[2p_i^2 + 2p_i(1 - p_i)]$ is the number of A_i alleles which would occur at time $t + 1$ if no social interactions occurred. Thus the last three terms in the curved brackets represent the effects of social interactions on $N_i(t + 1)$. The second two terms in the brackets represent effects on alleles identical by descent to the social donor, and are given by the product of donor number and per donor effect. The 2 preceding each (δa_r) term reflects the fact that each increment to individual survivorship affects two alleles. The one-half preceding the r in the third term within the brackets accounts for the fact that if the donor is heterozygous for the A_i allele, then the probability is only one-half that the allele identical by descent in the recipient is A_i.

The last term within the brackets represents the effects of interactions distributed randomly. Thus, the number of A_i genes added via such interactions is the product of the total number of genes added via these random interactions and p_i, the frequency of A_i among these genes.

The first three terms in the brackets may be merged, and p_i may be factored out, to rewrite Equation 2.2 as:

$$N_i(t + 1) = 2Np_i \{ \sum_j p_j [1 + \sum_r r(\delta a_r)_{ij}] +$$

$$\sum_k \sum_j p_k p_j \sum_r (1 - r)(\delta a_r)_{kj} \qquad (2.3)$$

Noting that the denominator of Equation 2.1 could be rewritten as $2N\left[1 + \sum_i \sum_j p_i p_j \sum_r (\delta a_r)_{ij}\right]$, and substituting Equation 2.3 into the numerator of Equation 2.1 yields:

$$p_i(t+1) = p_i(t)\frac{\sum_j p_j[1+\sum_r r(\delta a_r)_{ij}]+\sum\sum_{kj} p_k p_j \sum_r (1-r)(\delta a_r)_{kj}}{1 + \sum_k \sum_j p_k p_j \sum_r (\delta a_r)_{kj}} \qquad (2.4)$$

Now, define the "inclusive fitness effect" of genotype A_iA_j, $\delta R_{ij} = \sum_r r(\delta a_r)_{ij}$, as the total effect an individual of genotype A_iA_j has on the fitness of genes related to his own, and define the "diluting effect" of genotype A_iA_j as $\delta S_{ij} = \sum_r (1 - r)(\delta a_r)_{ij}$ to represent the total effect of an individual of genotype A_iA_j on genes in the same proportions as they occur in the population at large.

Then Equation 2.4 may be rewritten as:

$$p_i(t + 1) = p_i(t)\,\frac{\sum_j p_j(1 + \sum_r \delta R_{ij}) + \sum\sum_{kj} p_k p_j \sum_r \delta S_{kj}}{1 + \sum\sum_{kj} p_k p_j (R_{kj} + S_{kj})},$$

which is the same as:

$$p_i(t + 1) = p_i(t)\,\frac{(1 + \delta R_{i.} + \delta S_{..})}{1 + \delta R_{..} + \delta S_{..}}, \qquad (2.5)$$

where $x_{i.} = \sum_j p_j x_{ij}$ is the mean of variable x for individuals carrying the A_i allele, and $x_{..} = \sum_i \sum_j p_i p_j x_{ij}$ is the mean of variable x for

individuals regardless of their genotype. It is important to note that selection in the present model must be weak, and that no inbreeding occur (Michod, 1979), since r is an insufficient measure of genetic correlation when these conditions are violated.

As in Hamilton (1964), define the "inclusive fitness" of genotype A_iA_j as $R_{ij} = 1 + \delta R_{ij}$. Then we may rewrite Equation 2.5 as:

$$\Delta p_i = p_i(t) \frac{(R_{i.} - R_{..})}{1 + \delta R_{..} + \delta S_{..}} \tag{2.6}$$

It is not immediately clear from this equation exactly what determines the direction of evolution. Indeed, Hamilton (1964) felt that the denominator could be negative, causing natural selection to decrease p_i even when $R_{i.}$ is higher than the population average of R.

Therefore, an analysis of factors affecting the sign of the denominator of Equation 2.6 is needed. Note that, by definition:

$$1 + \delta R_{..} + \delta S_{..} = 1 + \sum_r (\delta a_r)_{..} \tag{2.7}$$

Call the effect $(\delta a_1)_{ij}$ an individual has on his own fitness c_{ij}, and call the total effect an individual has on other individuals' fitness b_{ij}, which is equal to $\sum_{r \neq 1} (\delta a_r)_{ij}$.

Then Equation 2.6 becomes:

$$1 + \delta R_{..} + \delta S_{..} = 1 + c_{..} + b_{..}, \tag{2.8}$$

where $b_{..}$ and $c_{..}$ may be positive, negative, or zero.

The term $b_{..}$ is the total benefit (which may be positive or negative) an average individual gives his neighbors. Thus the total benefits dispensed in the entire population of size N is $Nb_{..}$; and since the benefit received per individual is the total dispensed divided by the number of potential recipients N, I conclude that the benefit an average individual receives is $b_{..}$.

Therefore, Equation 2.8 says that $(1 + \delta R_{..} + \delta S_{..})$ is [the baseline fitness of an average individual] + [the fitness an average individual gains from others in social interactions] + [the fitness an average individual loses from social actions he contributes]. But this means that $(1 + \delta R_{..} + \delta S_{..})$ is $\bar{w}$, the fitness of an average individual. $\bar{w}$ can never be negative, since negative numbers of individuals do not exist in nature.

Equation 2.6 may, therefore, be rewritten as:

$$\Delta p_i = \frac{p_i(t)}{\bar{w}} (R_{i.} - R_{..}), \qquad (2.9)$$

which assures that as long as the population exists, the denominator for Equation 2.5 is positive. Analogues of Equation 2.9 have been derived both for continuous and discrete time kin selection models by Harpending (1979) and Charlesworth (1979), respectively, although the coefficients used by Harpending are frequency dependent for non-recessive social alleles.

The meaning of Equation 2.9 is clarified by rearrangement of $(R_{i.} - R_{..})$. Lump all $A_{k \neq i}$ alleles into a single group such that $p_k = (1 - p_i)$. Some algebraic rearrangements then yield:

$$(R_{i.} - R_{..}) = (1 - p_i)(p_i R_{ii} + (1 - 2p_i)R_{ik} + (p_i - 1)R_{kk} \quad (2.10)$$

where R_{ik} and R_{kk} represent the mean fitnesses of the A_iA_k heterozygote and A_kA_k homozygote genotypes respectively, and are assumed constant relative to R_{ii}.

Since the right-hand side of Equation 2.6 is $(1/2)\partial R_{..}/\partial p_i$, Equation 2.9 may be rewritten as:

$$\Delta p_i = \frac{p_i(1 - p_i)}{2\bar{w}} \left(\frac{\partial R_{..}}{\partial p_i}\right) \quad (2.11)$$

For the case of sex-linked alleles, see Appendix E.

Equation 2.11 is the inclusive fitness analogue of Wright's adaptive surface equation $p_i = \frac{p_i(1 - p_i)}{2\bar{w}} \frac{\partial \bar{w}}{\partial p_i}$. The reason an adaptive surface is implied is that, since $p_i(1 - p_i)/2\bar{w}$ is always positive, the change in gene frequency Δp_i depends only on the sign of $\partial R_{..}/\partial p_i$. Thus, since selection favors only those genes which increase mean inclusive fitness, it must ultimately maximize inclusive fitness.

A more graphic way to visualize this phenomenon is to visualize a rugged terrain on which elevation is really mean inclusive fitness, and horizontal location represents gene frequency. Let us now place a population at a particular location on the landscape. The adaptive surface equation tells us that selection will cause the population to climb the peak of any hill on which it is placed. Evolution (= change in gene frequency) stops only after the population reaches the top of a peak.

Comparison with Some Previous Studies

The similarity of Equation 2.11 to Wright's adaptive surface equation suggests that the classical work concerning its dynamics are applicable to evolutionary problems in social behavior. In fact, this turns out to be true.

Consider evolution in the simplest case of two alleles, A_1 and A_2, at a single locus, where the inclusive fitness of an individual of genotype A_1A_1 is greater than that of genotype A_2A_2. When the inclusive fitness of the heterozygous genotype A_1A_2 lies between R_{11} and R_{22} or equals one of them, evolution will always cause fixation of the A_1 allele. When the heterozygote has a higher inclusive fitness than either of the homozygotes, both alleles remain in the population since there is an inclusive fitness peak at $\hat{p}_1 = R_{12} - R_{22}/-R_{11} + R_{12} - R_{22}$ and no valleys occur anywhere else. When the heterozygote is inferior to both homozygotes, $\hat{p}_1$ becomes the bottom of a valley: A_1 is lost if its initial frequency is less than $\hat{p}_1$ and is fixed if its initial frequency is greater than $\hat{p}_1$. The factors affecting heterozygote inclusive fitness are more complex than simple penetrance, as discussed in Appendix F, which also gives some insight into factors affecting inclusive fitness. To demonstrate the potential power of this approach, an analysis of the evolutionary dynamics for intermediate dominance is presented below.

An assumption of intermediate dominance guarantees that if Δp_1 is positive at any one gene frequency it is positive at all gene frequencies, and will, therefore, become fixed. Thus, if Δp_1 is positive

at $p_1 = 0.5$, one may infer fixation of A_1. I will exploit this fact by examining Δp_1 at $p_1 = 0.5$ for analytical simplification.

Recall that $\partial R_{..} / p_i = 2[pR_{11} + (1 - 2p)R_{12} + (p - 1)R_{22}]$. Substituting $p_1 = 0.5$ into this identity shows that the results of selection depend only on the sign of $(R_{11} - R_{22})$. Thus A_1 is fixed if and only if:

$$\sum_r r(\delta a_r)_{11} > \sum_r r(\delta a_r)_{22}. \tag{2.12}$$

As mentioned before, it is usual to call $(\delta a_1)_{ij}$ the change in the survivorship of the donor c_{ij}, and to call the effects of the donor on the survivorship of others b_{ij}. If, as in Hamilton (1964), we define the mean coefficient of relationship of recipients as:

$$\bar{r}_{ij} = \frac{\sum_{r \neq 1} r(\delta a_r)_{ij}}{\sum_{r \neq 1} (\delta a_r)_{ij}} \tag{2.13}$$

then condition 2.12 becomes:

$$\bar{r}_{11} b_{11} + c_{11} > \bar{r}_{22} b_{22} + c_{22} \tag{2.14}$$

Let us now examine the special case which gives rise to "Hamilton's Rule" as defined in the first chapter. Let A_1 code for an altruistic behavior directed towards relatives, and let A_2 code for completely asocial behavior. Then, by condition 2.14, A_1 will be fixed when $r > -c/b$. This corresponds to Hamilton's Rule.

A closer parallel to classical population genetics theory is obtained by defining the cost to the donor in terms of number of offspring rather than in terms of lost donor survival (West-Eberhard, 1975). Let our former c_{ij} be independent of genotype, A_2 code for offspring raising, and let A_1 code for aiding relatives other than offspring. Then the condition for increase in A_1 becomes:

$$\frac{b_{11}}{b_{22}} > \frac{r_{22}}{r_{11}}$$

where b_{22} is the number of offspring raised by each A_2A_2 individual.

This result corroborates that of West-Eberhard's (1975) study. For example, in the special case of sister- versus offspring-raising in the Hymenoptera, the model predicts that a female should raise sisters rather than daughters when:

$$\frac{b_s}{b_d} > \frac{2}{3}\ , \qquad (2.15)$$

in which $2/3 = 1/2 \div 3/4$, where 1/2 and 3/4 are the respective coefficients of relatedness for a female Hymenopteran to her daughters and sisters, and where b_s and b_d are the number of sisters or daughters she can potentially raise.

As mentioned in Chapter 1, this type of treatment requires that the benefit accrued to a recipient be a linear function of the number of donors it encounters. The next chapter concerns the construction of a framework for studying the effects of relaxing this assumption of linearity.

CHAPTER 3

NONLINEAR BENEFITS AND THE EVOLUTION OF EUSOCIALITY IN THE HYMENOPTERA

Introduction: The Assumption of Linearity

Consider the following evolutionary scenario. A long-lived Hymenopteran mother continues to lay eggs at the same nest from which her earlier offspring have been hatched. A mutation causes one of her female offspring to forego raising daughters in favor of raising sisters who will later reproduce. Fixation of this new allele for facultative altruism results in the formation of a eusocial species.

That this scenario has held for most eusocial Hymenoptera is the prevalent view among most students of insect sociality (e.g., recent reviews by Evans and West Eberhard, 1970; Wilson, 1975; and Starr, 1979), although it seems plausible that this "subsocial" route to eusociality was not taken by most non-allodapine bees (Michener, 1974). The purpose of the present study is to examine the evolutionary dynamics of the allele for sister-raising.

A rather fragile assumption upon which previous studies addressing this topic depend (West Eberhard, 1975; after Hamilton, 1964) is relaxed in this study. In particular, this treatment will not require that the benefit accrued to a recipient be a linear function of the number of social donors with which she interacts. Relaxation of this requirement permits the inclusion of social interactions which are

biologically more realistic. As an example, consider food donations by altruists. In a bad year, a recipient catered to by only one altruist may gain only a negligible increment in fitness, while a recipient with two altruists may gain more than double that increment. This makes the increment gained as a function of the number of altruists encountered concave upward. If recipients with sufficient donors are satiated, this increment function becomes sigmoidal. If, on the other hand, satiation begins to occur even with small numbers of altruists, the increment function may be concave downward. Other factors which potentially affect the shape of these increment functions are discussed by Oster and Wilson (1978).

A General Model in Which the Linearity Assumption is Relaxed

The present analysis is based on the following simple genic model, and the notation used is summarized in Table 3. Consider alleles A_1 and A_2 in relative frequencies p and (1 - p), and in absolute frequencies N_1 and N_2, respectively. Let B be the baseline fitness of the alleles independent of social behavior. Also, let c_{ij} be the effect of donating social acts on the survival of an individual donor of genotype A_iA_j, and let b_{ij} be the total effect of social acts donated by an individual of genotype A_iA_j on the individuals who receive them. Then if the population size is N, and if P(ij/km) is the probability that a given social act by an A_kA_m individual is directed toward an individual of genotype A_iA_j,

Table 3. Notation used in Chapter 3.

A_1	Dominant allele for daughter-raising behavior
A_2	Recessive allele for sister-raising behavior
B	Baseline allelic fitnesses independent of social behaviors
b(D)	Number of extra daughters accrued to parents with proportion D A_2A_2 individuals among daughters
b_d	Number of own daughters raised by each daughter-raising first brood female
$b_s(D)$	Number of extra sisters raised by each sister-raiser with proportion D A_2A_2 individuals among her female sibs
b_k	Number of extra individuals saved by each potential altruist within subgroup k
b_{ij}	Benefit donated by average individual of genotype A_iA_j
c_{ij}	Cost of social behavior to survival by average individual of genotype A_iA_j
$c_{ij,m}$	Number of A_m alleles lost due to altruistic acts by each average A_iA_j individual
D	Proportion of A_2A_2 individuals among sibs of potential sister-raisers
e	Expressivity of the A_2 allele
F	1/4 - p
n	$b_{s(1/2)} - b_{s(1)}$
N_i	Number of A_i alleles in the population
N	Number of individuals in the population
p	Relative frequency of the A_1 allele

Table 3 -- Continued

P(ij/km)	Probability that a social act by genotype A_kA_m is directed toward an individual of genotype A_iA_j
P(i/km)	Probability that a gamete from the recipient of a social act by an individual of genotype A_kA_m has the A_i allele
P(i in km)	Probability that a gamete from an A_kA_m individual contains the A_i allele
P(i/k)	Probability that a gamete randomly drawn from the recipients in subgroup k contains the A_i allele
P(k/ij)	Probability that a given individual of genotype A_iA_j is in subgroup k
z	Number of first brood daughters raised by each pair of founder parents

$$N_1(t+1) = N_1(t)B + \{Np^2[2b_{11}P(11/11) + b_{11}P(12/11) - 2c_{11}] + 2Np(1-p)[2b_{12}P(11/12) + b_{12}P(12/12) - c_{12}] + N(1-p)^2(2b_{22}P(11/22) + b_{22}P(12/22)\} \quad (3.1)$$

To see what this means, note than $N_1(t)B$ is what $N_1(t+1)$ would be if no social interactions occurred. The rest of the left-hand side (LHS) of Equation 3.1 describes the effects of social interactions. Np^2, $2Np(1-p)$, and $N(1-p)^2$ are the numbers of A_1A_1, A_1A_2, and A_2A_2 individuals, respectively, at the start of a generation given random mating. Each of these A_iA_j individuals may donate social acts. As for the rest of the terms, note that each time an individual of genotype A_1A_1 is helped, (2b) extra A_1 alleles are saved, while helping an individual of genotype A_1A_2 saves only (b)A_1 alleles. Similarly, the number of A_1 alleles lost via the donation of social acts by A_1A_1, A_1A_2, or A_2A_2 individual are $2c_{11}$, c_{12} and zero, respectively.

By similar reasoning, one may show that:

$$N_2(t+1) = N_2(t)B + \{Np^2(2b_{11}P(22/11) + b_{11}P(12/11) + 2Np(1-p)(2b_{12}P(22/12) + b_{12}P(12/12) - c_{12}) + N(1-p)^2(2b_{22}P(22/22) + b_{22}P(12/22) - 2c_{22})\} \quad (3.2)$$

That models in the form of Equations 3.1 and 3.2 are formally equivalent to those mobilized by other students of kin selection is shown in Chapter 1.

It is of interest to define the conditions under which the A_1 allele increases in frequency. This occurs when $p_{t+1} > p_t$, which may be rewritten as:

$$\frac{N_1(t+1)}{N_1(t+1) + N_2(t+1)} > \frac{N_1(t)}{N_1(t) + N_2(t)} \tag{3.3}$$

Rewrite LHS of Equation 3.1 and LHS of Equation 3.2 as $N_1(t)B + a$ and $N_2(t)B + d$, respectively. Substituting these into Equation 3.3 and rearranging yields the following result: A_1 increases in frequency when:

$$\frac{a}{N_1} > \frac{d}{N_2} \tag{3.4}$$

Equation 3.4 makes intuitive sense: evolution favors A_1 when social behaviors increase the fitness of an average A_1 allele more than they increase the fitness of an average A_2 allele.

A series of rearrangements render inequality 3.4 analytically tractable. First, note that $N_1 = 2Np$ and $N_2 = 2N(1 - p)$, and also that $2P(ii/km) + P(ij/km) = 2P(i/km)$, where $i \neq j$. Also, define $c_{ij,m} = 2C_{ij}P(m \text{ in } ij)$, where $P(m \text{ in } ij)$ is the relative frequency of gametes from A_iA_j individuals which contain A_m. Thus, for example, $P(1 \text{ in } 11) = 1$. Substituting these equalities and definitions into Equation 3.1 yields $P_{t+1} > p_t$ when:

$$\frac{1}{p} \sum_i \sum_j p_i p_j \left[b_{ij}2P(1/ij) - c_{ij,1}\right] > \frac{1}{1-p} \sum_i \sum_j p_i p_j \left[b_{ij}2P(2/ij) - c_{ij,2}\right]. \tag{3.5}$$

Now, $P(2/ij) = 1 - P(1/ij)$, and $P(m/ij) = \sum_{k=1}^{x} P(k/ij)P(m/k)$, where the summation over all k = 1 to x indicates that the population is divided into x subpopulations.

These definitions may be used to rewrite Equation 3.5 as:

$$\frac{1}{p} \sum_i \sum_j p_i p_j \left[\sum_k b_{ij,k}\, 2P(k/ij)P(1/k) - c_{ij,1}\right] >$$

$$\frac{1}{(1-p)} \sum_i \sum_j p_i p_j \left[\sum_k b_{ij,k}\, 2P(k/ij)\right.$$

$$\left.[1 - P(1/ij)] - c_{ij,2}\right],$$

which is rearranged to yield:

$$\frac{1}{p} \sum_i \sum_j p_i p_j \sum_k b_{ij,k}\, 2P(k/ij)P(1/k) - \frac{1}{(1-p)} \sum_i \sum_j p_i p_j \sum_k b_{ij,k}$$

$$2P(k/ij)[1 - P(1/k)] > \frac{1}{p} \sum_i \sum_j p_i p_j c_{ij,1} -$$

$$\frac{1}{1-p} \sum_i \sum_j p_i p_j c_{ij,2},$$

which, in turn, rearranges to:

$$\sum_i \sum_j p_i p_j \sum_k b_{ij,k}\, P(k/ij)\,[P(1/k) - p] > \sum_i \sum_j p_i p_j c_{ij}$$

$$[P(1 \text{ in } ij) - p] \qquad (3.6)$$

The LHS of Equation 3.6 can be simplified. Note that $P(k/ij) = P(ij/k)p(k)/P(ij)$. Therefore, the LHS of Equation 3.6 $= \sum_k P(k)\,\{P(1/k) - p\} \sum_{ij} b_{ij}(k)P(ij/k)$, where $\sum_{ij}$ indicates that $b_{ij} = b_{ji}$ is summed over only once. We may substitute this into Equation 3.6 to obtain:

$$\sum_k P(k)[P(1/k) - p] \sum_{ij} b_{ij,k} P(ij/k) > \sum_i \sum_j p_i p_j c_{ij}$$

$$[P(1 \text{ in } ij) - p] \qquad (3.7)$$

As a final notational flourish, note that $\sum_{ij} b_{ij,k} P(ij/k) = b_k$ is the mean benefit donated by the potential social donors within subgroup k. Thus Equation 3.7 may be rewritten as:

$$\sum_k P(k)[P(1/k) - p]\, b_k > \sum_i \sum_j p_i p_j c_{ij}\, [P(1 \text{ in } ij) - p]. \qquad (3.8)$$

Inequality 3.8 defines conditions under which selection favors the A_1 allele. Further, if Equation 3.8 is written as an equality, it defines conditions for genetic equilibrium, and a reversal of the direction of Equation 3.8 defines conditions under which selection favors allele A_2.

An Application of the Model: Eusociality in Haplodiploid Organisms

I now use Inequality 3.8 to examine the evolutionary scenario concerned with sister- versus daughter-raising behavior envisioned in the first paragraph of this paper. During each season, a haplodiploid organism is assumed to produce two broods. The first brood consists of young raised in the spring by overwintering 'foundress' adults. Females from the first brood are assumed to raise females from the second brood.

The genetic mother of a given second brood female, however, depends on the behavior of the first brood female who raised her. This stems from the fact that a first brood female may raise her sisters instead of raising her own daughters.

Individuals from the second brood overwinter to become foundresses the following spring. First brood individuals may also overwinter: in the case presently considered, their survival rate is assumed to be independent of whether they decide to raise daughters or sisters. The behavior is assumed neutral with respect to the production and actions of males.

The time unit in the present model is the season. Thus, $N_i(t)$ and $N_i(t + 1)$ in Inequality 3.3 are the numbers of A_i alleles in the founders at two successive springtimes.

The setup for the application of Inequality 3.8 to discover when sister-raising behavior will evolve is given in Table 4, which is explained in detail below.

Assume that the monogamous foundress adults randomly mate. Then the first brood female genotypes A_1A_1, A_1A_2, and A_2A_2 are produced in frequencies p^2, $2p(1 - p)$, and $(1 - p)^2$, respectively. All A_1A_1 and A_1A_2, and proportion (1-e) A_2A_2 first brood females mate: each then raises b_d daughters. Proportion e of the A_2A_2 first brood females, however, forego raising daughters: instead each raises $b_{s(D)}$ sisters. The D in $b_{s(D)}$ and in Table 4 represents the proportion of a sister-raiser's sibs who are of genotype A_2A_2.

Recall that the benefits doled out can be divided into x subgroups. This division is accomplished in Table 4, in which the subgroup to which a particular benefit belongs depends on the genotype and behavior of the female dispersing that benefit. More specifically, let P(k) represent the proportion of first brood females who

Table 4. Calculation of variables relevant to the evolutionary dynamics of eusociality. -- Each k represents a trait group type composed of the individuals a given first brood daughter may raise. The female parents in k = 1 - 6 represent first brood daughters who raise their own daughters, while the female parents in k = 7 and 8 represent parents of first brood daughters who help raise their sisters. The P(k) are the probabilities that the young raised by any given first brood daughter are from the indicated parental genotypes. p is the frequency of the dominant A_1 daughter-raising allele, and e is the expressivity of the recessive A_2 allele for sister-raising behavior. See text for further details.

k	1	2	3	4	5	6	7	8
Parental Genotypes	$A_1A_1xA_1$	$A_1A_2xA_1$	$A_1A_2xA_2$	$A_1A_1xA_2$	$A_2A_2xA_1$	$A_2A_2xA_2$	$A_1A_2xA_2$	$A_2A_2xA_2$
P(k)	p^3	$2p^2(1-p)$	$2p(1-p)^2$	$p^2(1-p)$	$(1-e)p(1-p)^2$	$(1-e)(1-p)^3$	$ep(1-p)^2$	$e(1-p)^3$
P(1/k)-p	1-p	3/4 - p	1/4 - p	1/2 - p	1/2 - p	-p	1/4 - p	-p
D							1/2	1

raise females from subgroup k. Thus, subgroups k = 1-6 in Table 4 represent daughters raising their own daughters. For example, $P(1) = p^3$, since the probability that a given first brood daughter is A_1A_1 is p^2, and the probability that her mate is of genotype A_1 is p.

Similarly, subgroups denoted by k = 7 and 8 represent first brood daughters raising their own sisters. P(7) and P(8) are calculated as follows. A given first brood A_2A_2 female will raise sisters instead of daughters with probability e. The frequency of sister-raisers among first brood females is therefore $e(1 - p)^2$. The parent of a first brood sister-raiser, and therefore the genetic parents of the individuals she raises, are $A_1A_2 \times A_2$ with probability p, and they are $A_2A_2 \times A_2$ with probability 1 - p. Thus P(7) is the probability that a first brood female is a sister-raiser with parents of genotype $A_1A_2 \times A_2$, and this probability is $P(7) = pe(1 - p)^2$. Similarly, $P(8) = (1 - p)e(1 - p)^2$. In the above calculations, it is assumed that the gene frequencies in the male and female portions of the population are the same, and that the foundress population genotypes are distributed according to Hardy Weinberg expectations. These assumptions should not significantly affect the genetic dynamics provided e is relatively small.

The parental genotypes of each subgroup determine the recipient genotypes, and therefore determine P(1/k) - p. For example, P(1/k) - p when k = 1 is 1 - p, since the probability that an allele randomly drawn from a second brood female from subgroup 1 is unity.

If we now substitute the values from Table 4 into Inequality 3.8, divide both sides by p(1 - p), and rearrange the result, we find

that allele A_2 for the sister-raising behavior increases in frequency when:

$$p^2 [b_1 - 2b_2 + 2b_3 - b_4 + (1 - e)(b_5 - b_6) + e(b_7 - b_8)] +$$

$$p[\tfrac{3}{2}b_2 - \tfrac{5}{2}b_3 + \tfrac{1}{2}b_4 - (1 - e)(\tfrac{3}{2}b_5 - 2b_6) -$$

$$e(\tfrac{5}{4}b_7 - 2b_8)] + \tfrac{1}{2}b_3 + (1 - e)(\tfrac{1}{2}b_5 - b_6) +$$

$$e(\tfrac{1}{4}b_7 - b_8) < pc_{11} + c_{12} - 2pc_{12} - c_{22} + pc_{22}. \qquad (3.9)$$

Note that if the personal costs to survival are independent of behavior, then $c_{11} = c_{12} = c_{22}$, and the RHS of Equation 3.9 becomes zero. Note also that $b_1 = b_2 = b_3 = b_4 = b_5 = b_6 = b_d$, that $b_7 = b_{s(1/2)}$, and that $b_8 = b_{s(1)}$. These observations, coupled with rearrangements of Equation 3.9 yield increases in the frequency of allele A_2 when:

$$\{b_{s(1/2)} - b_{s(1)}\} \{p^2 - \tfrac{5}{4}p + 1/4\} + (p - 1)$$

$$(\tfrac{3}{4}b_{s(1)} - \tfrac{1}{2}b_d) < 0 \qquad (3.10)$$

Nonlinearities in benefits donated by sister raisers are introduced as follows. Let D represent the proportion within a sibship of the first brood females of genotype A_2A_2 for sister-raising behavior, and let z be the number of first brood females raised by each founder family. Then the number of extra offspring accrued to the founder parents due to sister raising is $b(D) = Dzeb_{s(D)}$, where Dze is the number of sister raisers in the family, and $b_{s(D)}$ is the number of sisters each raises.

If $b_{s(D)}$ is independent of D then b(D) is a linear function of D. b(0) = 0, and in the present treatment it is assumed that b(1) is fixed. Therefore, nonlinearities only affect the value of $b_7 = b_{s(1/2)}$. More compactly, let $n = b_{s(1/2)} - b_{s(1)}$. Then $n > 0$ implies either that b(D) is concave downward or sigmoidal with b(D) crossing $Dzeb_{s(1)}$ at $D < 1/2$. An $n < 0$, on the other hand, implies either than b(D) is concave up or that b(D) is sigmoidal with b(D) crossing $Dzeb_{s(1)}$ at $D > 1/2$. Some potential biological reasons for nonlinearities in b(D) are given in the third paragraph of this text. Of course, since e is assumed small, nonlinearities such as saturation, etc., may obtain in the present treatment only when family size z is relatively large.

Now, let $F = (p^2 - \frac{5}{4}p + \frac{1}{4})/(1 - p)$, which reduces algebraically to $1/4 - p$. Then substituting F and n into Equation 3.10 shows that A_2 increases in frequency when:

$$nF < \frac{3}{4} b_s - \frac{1}{2} b_d \ , \tag{3.11}$$

where $b_s = b_{s(1)}$.

For the linear case, $n = 0$, and sister-raising behavior evolves when $\frac{3}{4} b_s - \frac{1}{2} b_d > 0$. This result corroborates West Eberhard's (1975) study of Hamilton's (1964) inclusive fitness theory.

Characterization of equilibria for the nonlinear case is more complex than in the previous example, since the value of n is dependent on p. Furthermore, simple solution for $\hat{p}$ yields little insight into the genetic dynamics. A global genetic analysis is therefore given.

Figure 1 shows that internal equilibria obtain when $nF = \frac{3}{4} b_s - \frac{1}{2} b_d$ for $0 > p > 1$. Now, F monotonically decreases with increasing p. This has implications concerning the location, number, and stability characteristics of potential equilibria. If n is negative, then loss of A_2 occurs if $\frac{3}{4} b_s - \frac{1}{2} b_d \leq 0.25n$. If, on the other hand, $\frac{3}{4} b_s - \frac{1}{2} b_d \geq -0.75n$, fixation of A_2 occurs. For $-0.75n \geq \frac{3}{4} b_s - \frac{1}{2} b_d \geq 0.25n$, an internal equilibrium ($1 - \hat{p} = \hat{q}$) obtains. This internal equilibrium is unstable, since when the frequency of allele A_2 is below $\hat{q}$ it decreases, and when it is above $\hat{q}$ allele A_2 increases in frequency.

For $n > 0$, fixation or loss of A_2 occurs if $\frac{3}{4} b_s - \frac{1}{2} b_d > 0.25n$ or $\frac{3}{4} b_s - \frac{1}{2} b_d < -0.75n$, respectively. The single internal equilibrium point, which occurs if $0.25\ n > \frac{3}{4} b_s - \frac{1}{2} b_d > -0.75n$, is stable. This verdict of stability is, of course, subject to the usual caveat concerning the potentially destabilizing effects of overdamping in discrete time models. However, for the weak selection presently assumed, the potential importance of such overdamping is minimal.

It is interesting to note that survival and reproduction by foundresses and first brood adults which occurs independently of their genotypes will not affect the present predictions. To see why, note that for benefits which occur independently of genotype, the frequency of A_1 among the extra survivors and offspring is simply p_1 and therefore their p(1/k) - p and their p(1 in ij) terms in Equation 3.8 are zero. Thus, the RHS of Equation 3.8 remains zero. The LHS of Equation 3.8, however, will change in magnitude; but it does not change in sign, and therefore the direction of evolution is not altered. To see why it

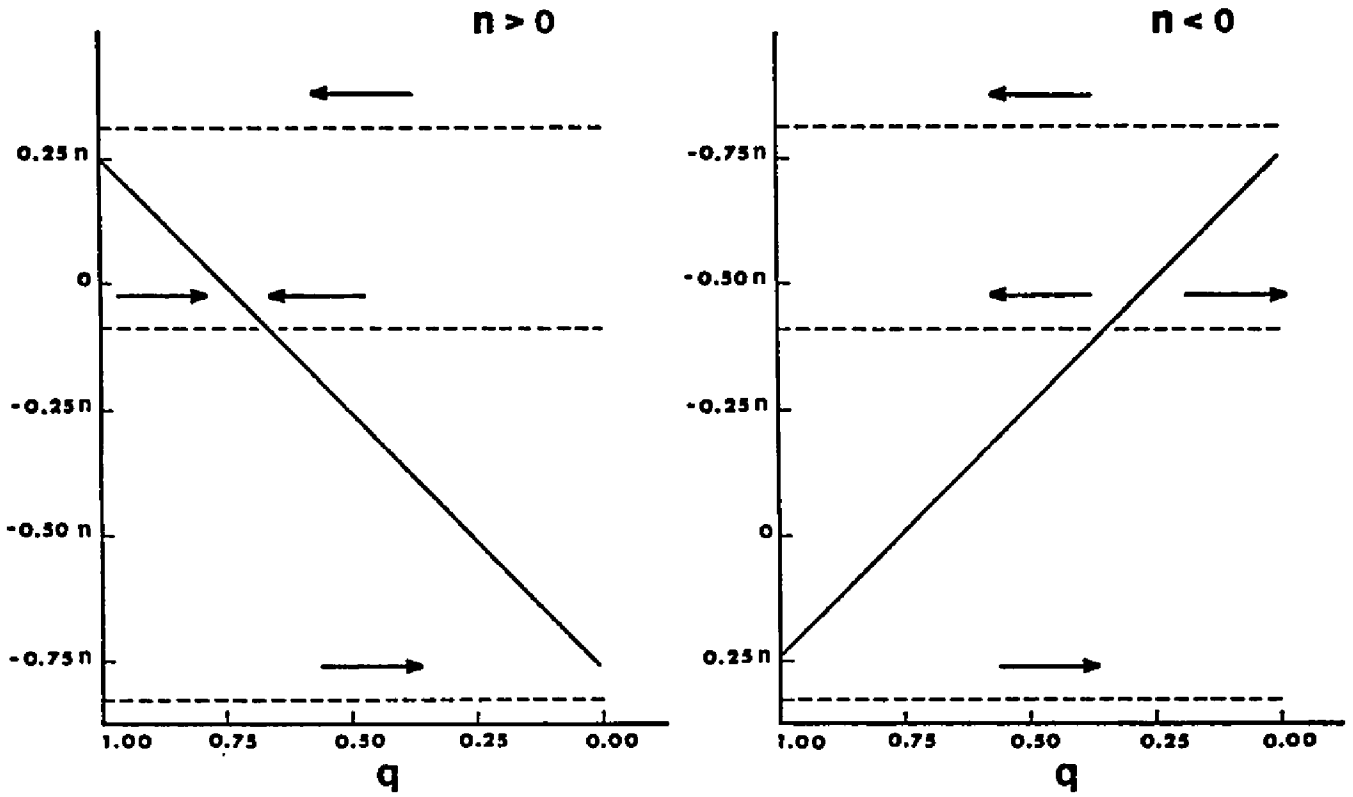

Figure 1. Evolution of eusociality for nonlinear benefits . -- Arrows (→) point toward direction of evolution. q is frequency of A_2 allele for sister-sister social interactions. $n = b_{s(1/2)} - b_{s(1)}$, and $F = \frac{1}{4} - p^2$. Solid line (—) is value of nF, and dashed lines (--) are some potential values of $(3/4)b_s - (1/2)b_d$. See text for further details.

doesn't change in sign, note that p(k) = number of type k benefits/total number of benefits. The total number of benefits will change as individuals raise new offspring, and therefore all p(k) will change. However, since the total number of benefits is independent of k, it can be factored out of the LHS of Equation 3.8; and since it must always be positive, its change cannot affect the sign of the LHS of Equation 3.8.

Eusociality and Nonlinear Benefits

For linear b(D), the present model predicts the fixation of eusocial behavior when $2/3 < b_s/b_d$. This corroborates the result of West Eberhard's (1975) study of Hamilton's (1964) inclusive fitness model.

Uncritical application of this result, however, may be misleading. In particular, when benefits received are nonlinear functions of the number of donors encountered, the direction of evolution may depend on gene frequency. For example, when $n > 0$, stable polymorphic equilibria occur within a certain range of benefit values.

The unstable polymorphic equilibria which may occur for $n < 0$ may prevent the appearance of eusociality even when its fixation is predicted by Hamilton's model. Such equilibria represent selective thresholds in gene frequency which must be passed before fixation can occur. This suggests that historical factors such as genetic drift during bottlenecks in population size or fluctuations in the benefit functions may have played a crucial role in the evolution of eusociality for many species. Consequently, this study may help to explain why many Hymenopteran species lack eusociality.

Fixation of sister-raising behavior when the linear model predicts its loss should occur only rarely, since the selective threshold, when it exists, is greater or equal to $q = 0.75$.

APPENDIX A

THE EQUIVALENCE OF FAMILY-STRUCTURED KIN SELECTION MODELS

I now compare family-structured models for the commonly examined situation of diploidy, random interactions within family groups, random mating, and weak selection. Wade (1978, Equation 4) and Michod (1980) define the fitness of genotype A_iA_j as:

$$W_{ij} = \sum_{k=1}^{d} W_{ij}(k)P(k/ij), \tag{A.1}$$

where k is one of d possible family types and (P(k/ij) is the probability of being in family k given genotype A_iA_j.

By noting that $P(k/ij) = \frac{P(k)P(ij/k)}{P(ij)}$, multiplying both sides of Equation A.1 by P(ij), and multiplying the right side of Equation A.1 by (N÷d)/(N÷d), we obtain Levitt's (1975, equation 3) model, i.e.,

$$P_{ij}(t+1) = \sum_{k=1}^{d} \frac{W_{ij}(k)P(k)P(ij/k)\ N\div d}{N\div d}. \tag{A.2}$$

Equation A.1 may also be written as:

$$W_{ij} = \sum_{k=1}^{d} 1 + \gamma h_{ij} + \beta[h_{11}P(11/k) + h_{12}P(12/k) + h_{12}P(22/k)]\ \frac{P(k)P(ij/k)}{p(ij)}\ . \tag{A.3}$$

γ is the cost over an A_1A_1 individual's entire lifetime due to altruistic acts, and β is the benefit to fitness due to receiving altruistic acts obtained when an individual interacts only with altruists. In Equation A.2, the actual benefit received is a linear function of the

proportion of altruists in a group of interactants. h_{ij} is a measure of dominance.

We may modify Equation A.3 by taking $1 + \gamma h_{ij}$ out of the summation sign and rewriting $[h_{11}P(11/k) + h_{12}P(12/k) + h_{22}P(22/k)] \equiv \sum_{\mu\nu} h_{\mu\nu}P(\mu\nu/k)$ to obtain:

$$W_{ij} = 1 + \gamma h_{ij} + \sum_{k=1}^{d} \sum_{\mu\nu} \beta h_{\mu\nu} P(\mu\nu/k) P(k) \frac{P(ij/k)}{P_{ij}}$$

$$= 1 + \gamma h_{ij} + \sum_{\mu\nu} \beta h_{\mu\nu} P(\mu\nu/ij). \qquad (A.4)$$

This is the model used by Cavalli-Sforza and Feldman (1978, Equation 11) and by Uyenoyama and Feldman (1980).

Let c and b be cost and benefit of each altruistic act, and let $H_{\mu\nu}$ be the number of altruistic acts per individual of genotype $A_\mu A_\nu$. Then Equation A.4 can be rewritten as:

$$W_{ij} = 1 + cH_{ij} + \sum_{\mu\nu} bH_{\mu\nu} P(\mu\nu/ij).$$

This rearranges to:

$$W_{ij} = 1 + cH_{ij} + \sum_{\mu\nu} bH_{\mu\nu} \sum_{k} P(\mu\nu/k) P(k/ij),$$

which in turn rearranges to:

$$W_{ij} = 1 + cH_{ij} + \sum_{\mu\nu} bH_{\mu\nu} P(\mu\nu) \sum_{k} \frac{P(k/\mu\nu)}{P(k)} P(k/ij).$$

After noting that $P(k/ij) = \frac{P(ij/k)P(k)}{P(ij)}$, this equation becomes:

$$W_{ij} = 1 + cH_{ij} + \sum_{\mu\nu} bH_{\mu\nu}P(\mu\nu)\Sigma_k P(k/\mu\nu)\frac{P(ij/k)}{P(ij)}, \qquad (A.5)$$

which can be rewritten as:

$$W_{ij} = 1 + \frac{NP(ij)cH_{ij} + \sum_{\mu\nu} bH_{\mu\nu}NP(\mu\nu)\Sigma_k P(k/\mu\nu)P(ij/k)}{NP(ij)}, \qquad (A.6)$$

where N is the population size.

Equation A.5 is Charnov's model (1977, Equations 5, 6, and 7) for family-structured kin selection. The $NP(ij)cH_{ij}$ term represents the total number of A_iA_j individuals lost due to the cost of donating social acts. Each $\sum_{\mu\nu} bH_{uv}NP(uv)P(k/uv)$ term represents the total benefit dispersed in a family of type k: the factor P(ij/k) by which it is multiplied gives the proportion of this benefit which accrues to A_iA_j individuals. Both the cost and benefit terms are divided by NP(ij) to normalize these increments to a per A_iA_j individual basis.

In most of the studies mobilizing the above models $H_{11} = 1$, $H_{22} = 0$, and $1 \geq H_{12} \geq 0$. The single exception is the study of Uyenoyama and Feldman (1980).

To summarize this appendix, the above frameworks for the examination of kin selection are analytically interconvertible, and are therefore equivalent. In the rest of this study we compare this class of models to that of Hamilton (1964) by attempting to derive Hamilton's (1964) model from that of Charnov (1977).

APPENDIX B

A GENIC FITNESS MODEL FOR SOCIAL INTERACTIONS

From Equation 1.4a we have:

$$W_1 = \frac{N_1(t+1)}{N_1(t)} = 1 + pH_{11}[bP(11/11) + \frac{b}{2}P(12/11) + c] +$$

$$(1-p)H_{12}[2b\ (11/12) + bP(12/12) + c] +$$

$$+ \frac{(1-p)^2}{p} H_{22}[bP(11/12) + \frac{b}{2}P(12/22)]$$

Multiplying both sides of this equation by $N_1(t)$, and substituting 2NP for $N_1(t)$ yields:

$$N_1(t+1) = N_1(t) + Np^2H_{11}[2bP(11/11) + bP(12/11) - 2c)] +$$

$$2NP(1-p)H_{12}[2bP(11/12) + bP(12/12) - c] +$$

$$N(1-p)^2H_{22}[2bP(11/12) + bP(12/22)] \qquad (B.1)$$

To see how Equation B.1 relates directly to genic fitness, note that $NpH_{11}2NP(1-p)H_{12}$ and $N(1-p)^2$ are the number of altruistic acts by A_1 homozygotes and heterozygotes, and A_2 homozygotes, respectively. As for the terms in parentheses, note that each time an individual of genotype A_1A_1 is helped, 2b A_1 alleles are saved, while helping an A_1 heterozygote saves only b A_1 alleles per altruistic act. Similarly, each donation of an altruistic act results in the loss of c and 2c A_1 alleles from A_1A_2 heterozygotes and A_1A_1 homozygotes, respectively.

By similar means one can show that Equation 1.4b yields:

$$N_2(t + 1) = N_2(t) + N(1 - p)^2 H_{22}[2bP(22/22) + bP(12/22) -$$

$$2c] + Np^2 H_{11}[2bP(22/11) + bP(12/11] +$$

$$2Np(1 - p)H_{12}[2bP(22/12) + bP(12/12) - c]$$

The interpretation of this equation parallels that of Equation B.1.

APPENDIX C

DIPLOID SIBLING INTERACTIONS, WEAK SELECTION, AND THE HARDY-WEINBERG ASSUMPTION

The purpose of this and the next appendix is to demonstrate that interaction frequencies may be calculated as if Hardy-Weinberg genotypic frequencies obtain provided selection is weak. The key to these proofs lies in showing that deviations from Hardy-Weinberg expectations due to selection result in changes in Δp which are second or higher order in s, e.g., $O(s^2)$= terms in $s^2+s^3+ \ldots+s^n$. Assuming ℓ is small, these higher order terms can be ignored with minimal loss of precision since they are negligible compared to the O(s) term which results from calculations according to Hardy-Weinberg expectations.

Let $p_{\mu\nu}$ be the frequency of genotype $A_\mu A_\nu$ among mating adults, where $A_\mu A_\nu \neq A_\nu A_\mu$. Then,

$$p_{i\ell}p_{mj} = P(ij \cap A_iA_\ell \times A_mA_j) \qquad (C.1)$$

is the frequency of individuals among the offspring which are either of genotype A_iA_j or A_jA_i, and which are also from any of families $A_iA_\ell \times A_mA_j$, $A_\ell A_i \times A_mA_j$, $A_\ell A_i \times A_jA_m$, or $A_iA_\ell \times A_jA_m$.

Thus, for example, the probability of drawing an offspring which is of genotype A_1A_1 and which is from families of type $A_1A_1 \times A_1A_2$ or $A_1A_1 \times A_2A_1$ is $P(11 \cap A_1A_1 \times A_1A_2) = \frac{1}{2} p_{11}p_{12} + \frac{1}{2} p_{11}p_{21}$. Now, if we define p_{12} as the probability that we draw an A_1 and then an A_2 allele from a given adult, then $p_{12} = p_{21}$, and we find that $P(11 \cap A_1A_1 \times A_1A_2) = p_{11}p_{12}$.

Now,

$$W_{ij} = \sum_{k} x_{ij}(k)p(k/ij) \qquad (C.2)$$

(Wade, 1978; Michod, 1980; also, see Equation A.1), where $x_{ij}(k)$ represents the fitness of an individual of genotype A_iA_j in a family of type k.

Also, from standard population genetics,

$$p_{ij}(t + 1) = p^{\circ}_{ij} \frac{w_{ij}}{\bar{w}} \qquad (C.3)$$

where p°_{ij} is the frequency of A_iA_j among the offspring before selection, where $A_iA_j \neq A_jA_i$ for $i \neq j$, and where $p_{ij}(t + 1)$ is this same frequency after selection but before mating.

We may now use the identity $P(k/ij) = P(k \cap ij)/p_{ij}$ to combine Equations C.1 and C.2 to demonstrate that:

$$W_{ij} = \sum_{\ell m} \frac{p_{i\ell}p_{mj}}{p^{\circ}_{ij}} x_{ij/i\ell,mj} , \qquad (C.4)$$

and Equation C.4 may be substituted into Equation C.3 to show that, in the absence of interfamily selection (i.e. all $w_{i\ell,mj}$ = the constant $\bar{w}$).

$$p_{ij}(t + 1) = \sum_{\ell m} p_{i\ell}p_{mj} x_{ij/i\ell,mj} w^{-1}_{i\ell,mj} .$$

(Nagylaki, 1977). Here, $x_{ij/ik,\ell j}$ represents the fitness of an A_iA_j or A_jA_i individual when it is in one of the families $A_iA_\ell \times A_mA_j$, $A_\ell A_i \times A_mA_j$, $A_\ell A_i \times A_jA_m$, or $A_iA_\ell \times A_jA_m$, and $w^{-1}_{i\ell,mj}$ is the inverse of the mean fitness of offspring within these families.

The parental genotypes in the $P(ij \cap A_iA_\ell \times A_jA_m)$, $x_{ij/i\ell,mj}$, and $w^{-1}_{i\ell,mj}$ terms are completely specified by the ℓ and m in the summation sign. Therefore, the $i\ell,mj$ portion of $x_{ij/i\ell,mj}$ may be suppressed, and the above equation may be rewritten as:

$$p_{ij}(t+1) = \sum_{\ell m} p_{i\ell}p_{mj}x_{ij}w^{-1}_{i\ell,mj} ,$$

with the $/i\ell,mj$ of x_{ij} understood.

The step to kin selection is provided by adding interfamily selection to this model, i.e., by letting:

$$p_{ij}(t+1) = \sum_{\ell m} p_{i\ell}p_{mj}x_{ij}w^{-1}_{i\ell,mj}\frac{w_{i\ell,mj}}{\bar{w}} . \qquad (C.5)$$

In the classical terminology of kin selection, we have $x_{ij/i\ell,mj} = 1 + b_{i\ell,mj} + c_{ij} \equiv 1 + s_{ij/i\ell,mj}$; $w^{-1}_{i\ell,mj} = 4(1 + 4b_{i\ell,mj} + c_{ij} + c_{i\ell} + c_{m\ell} + c_{mj})^{-1} \equiv (1 + s_{i\ell,mj})^{-1}$; and $\bar{w} = \sum_{ij\ell m} p_{i\ell}p_{mj}w_{i\ell,mj} = 1 + \bar{b} + \bar{c} \equiv 1 + \bar{s}$, in which the b's and c's are benefits and costs accrued. The benefits received depend on the family which the recipient is in, and the costs depend solely on the donor. If we again suppress the $/i\ell,mj$ of $x_{ij/i\ell,mj}$ and note that $x_{ij}/\bar{w} = 1 - [(\bar{s} - s_{ij})/1 + \bar{s}] = 1 + (s_{ij} - \bar{s}) + [\bar{s}(s_{ij} - \bar{s})/(1 + \bar{s})]$, then Equation C.5 rearranges into:

$$p_{ij}(t+1) = \sum_{\ell m} p_{i\ell}p_{mj} + \sum_{\ell m} p_{i\ell}p_{mj}(s_{ij} - \bar{s}) + \sum_{\ell m} p_{i\ell}p_{mj}\frac{\bar{s}(s_{ij} - \bar{s})}{1 + \bar{s}} , \qquad (C.6)$$

which can be rewritten as:

$$p_{ij}(t + 1) = p_i p_j + O(s) \qquad (C.7)$$

where p_i is the frequency of the A_i allele among offspring in generation t.

Equation C.6 can be used to obtain Δp_i:

$$\Delta p_i = \sum_{j\ell m} p_{i\ell} p_{mj}(1 + s_{ij} - \bar{s} - 1) + O(s^2) ,$$

or, assuming weak selection,

$$\Delta p_i \simeq \sum_{j\ell m} p_{i\ell} p_{mj}(s_{ij} - \bar{s}) \qquad (C.8)$$

The genotypic frequencies in this equation deviate from Hardy-Weinberg expectations due to selection in the previous generation. According to Equation C.7, $p_{ij}(t + 1) = p_i p_j + O(s)$. Substituting this into Equation C.8 and ignoring terms which are second or higher order in s yields:

$$\Delta p_i \simeq p_i(s_i - \bar{s})$$

where s_i and $\bar{s}$ are calculated according to Hardy-Weinberg expectations.

APPENDIX D

HAPLODIPLIOD SIBLING INTERACTIONS, WEAK SELECTION, AND THE HARDY-WEINBERG ASSUMPTION

Let p_{ij} and p_i be the frequencies of genotypes A_iA_j and A_i among the female and male parents, respectively. Also, let w_{ij} be the fitness of an A_iA_j daughter in its family, and let $w_{ik,j}$ be the mean fitness of an individual in a family of parental genotypes $A_iA_k \times A_j$. Then given no interfamily selection:

$$p_{ij}(t+1) = \sum_k p_{ik}p_j \frac{w_{ij}}{w_{ik,j}} + (1 - \delta_{ij}) \sum_k p_{jk}p_i \frac{w_{ij}}{w_{jk,i}} \qquad (D.1)$$

where the Kronecker delta, δ_{ij}, is defined as unity if $i = j$, and as zero if $i \neq j$. Adding interfamily selection to Equation D.1 yields:

$$p_{ij}(t+1) = \sum_k p_{ik}p_j \frac{w_{ij}}{w_{ik,j}} \frac{w_{ik,j}}{\bar{w}} + (1 - \delta_{ij}) \sum_k p_{jk}p_i \frac{w_{ij}}{w_{ik,j}} \frac{w_{ik,j}}{\bar{w}},$$

which simplifies to:

$$p_{ij}(t+1) = \sum_k p_{ik}p_j \frac{1 + s_{ij}}{1 + \bar{s}} + (1 - \delta_{ij}) \sum_k p_{jk}p_i \frac{1 + s_{ij}}{1 + \bar{s}} \qquad (D.2)$$

where $\bar{w}$ is mean female fitness, and $w = 1 + s$.

Now, $1 - \frac{(1 + s_{ij})}{(1 + \bar{s})} = \frac{(\bar{s} - s_{ij})}{(1 + \bar{s})}$. Therefore, Equation D.2 may be rewritten as:

$$p_{ij}(t+1) = \sum_k p_{ik}p_j \left(1 - \frac{\bar{s} - s_{ij}}{1 + \bar{s}}\right) + (1 - \delta_{ij}) \sum_k p_{jk}p_i \left(1 - \frac{\bar{s} - s_{ij}}{1 + \bar{s}}\right),$$

which can be rearranged to:

$$p_{ij}(t+1) = \sum_k p_{ik}p_j + (1 - \delta_{ij}) \sum_k p_{jk}p_i + O(s), \qquad (D.3)$$

where $O(s) = [\sum_k p_{ik}p_j(s_{ij} - \bar{s})/(1 + \bar{s})] + [(1 - \delta_{ij}) \sum_k p_{jk}p_i (s_{ij} - \bar{s})/(1 + \bar{s})]$.

Equation D.3 rearranges to yield:

$$p_{ij}(t+1) = p_i^f p_j^m + (1 - \delta_{ij})p_j^f p_i^m + O(s) \qquad (D.4)$$

where p_i^f and p_i^m are the frequencies of A_i among the females and males in the population, respectively.

Equations D.3 and D.4 may be used to find $p_i^f(t+1)$. First, note that $p_i^f = (1/2)(2p_{ii} + p_{ij})$, where $j \neq i$. If we replace the δ_{ij} in Equation D.4 with zero, we obtain $2p_{ii}(t+1)$ instead of $p_{ii}(t+1)$ when $j = i$, and we may write:

$$p_i^f(t+1) = \frac{1}{2} \sum_j [p_i^f p_j^m + p_j^f p_i^m + \sum_k p_{ik}p_j(\frac{s_{ij} - \bar{s}}{1 + \bar{s}}) + \sum_k p_{jk}p_i(\frac{s_{ij} - \bar{s}}{1 + \bar{s}})]$$

which simplifies to:

$$p_i^f(t+1) = \frac{1}{2} [p_i^f(\frac{s_i - \bar{s}}{1 + \bar{s}})^m + p_i^m(\frac{s_i - \bar{s}}{1 + \bar{s}})^m + p_i^f + p_i^m] \qquad (D.5)$$

where the f's and m's over the right parentheses indicate that the $\bar{s}$'s and s_i's are from families in which the A_i allele originates from mother or father, respectively.

Now, since $s_i - \bar{s} = \frac{\bar{s}(s_i - \bar{s})}{1 + \bar{s}} - \frac{s_i - \bar{s}}{1 + \bar{s}}$, Equation D.5 may be rewritten as:

$$p_i^f(t + 1) = \frac{1}{2}[p_i^f + p_i^m + p_i^f(s_i - \bar{s})^f + p_i^m(s_i - \bar{s})^m + O(s^2)], \quad (D.6)$$

where $O(s^2) = \sum_{m,f} \frac{\bar{s}(s_i - \bar{s})}{1 + \bar{s}}$.

A similar treatment can be used to obtain $p_i(t + 1)$ for males. First, all of a male's genes are given to him by his mother. Therefore,

$$p_i^m = \sum_j \sum_k p_j p_{ik} \frac{w_i}{\bar{w}},$$

which can be rewritten as:

$$p_i^m = \sum_j \sum_k p_j p_{ik} \left(\frac{1 + s_i}{1 + \bar{s}}\right),$$

or

$$p_i^m = \sum_j \sum_k p_j p_{ik} \left(1 + \frac{s_i - \bar{s}}{1 + \bar{s}}\right), \quad (D.7)$$

which yields:

$$p_i^m = p_i^f + p_i^f(s_i - \bar{s}) + O(s^2) \quad (D.8)$$

The $O(s^2)$ term in Equation D.8 comes from two sources. First, an $(s_i - \bar{s}) - \frac{\bar{s}(s_i - \bar{s})}{1 + \bar{s}}$ is substituted into Equation D.7 for $\frac{s_i - \bar{s}}{1 + \bar{s}}$. Second, as we noted in Equation D.3, p_{ik} in females mating after selection occurs differs from Hardy-Weinberg expectations by an $O(s)$ term.

Next, we now wish to know whether $\bar{p}$ can be used rather than p^f and p^m in our dynamical equation for gene frequency. Let p be the frequency of A_i. Then, by definition:

$$\bar{p}(t + 1) = \frac{2}{3} p^f(t + 1) + \frac{1}{3} p^m(t + 1) \qquad (D.9)$$

Substituting Equations D.6 and D.8 into D.9 yields:

$$p(t + 1) = \frac{2}{3}\frac{1}{2}[p^f + p^m + p^f(s_i - \bar{s})^f + p^m(s_i - \bar{s})^m] +$$

$$\frac{1}{3} [p^f + p^f(s_i - \bar{s})]$$

which rearranges to:

$$\bar{p}(t + 1) = \frac{2}{3} p^f + \frac{1}{3} p^m + \frac{1}{3} [p^f(s_i - \bar{s})^f + p^f(s_i - \bar{s}) +$$

$$p^m(s_i - \bar{s})^m].$$

Therefore, if $\Delta\bar{p} = \bar{p}(t + 1) - \bar{p}(t)$,

$$\Delta\bar{p} = \frac{1}{3} [p^f(s_i - \bar{s})^f + p^f(s_i - \bar{s}) + p^m(s_i - \bar{s})^m]. \qquad (D.10)$$

I shall now show that $\bar{p} - p^f$ and $\bar{p} - p^m$ are on the order of O(s). Thus, substituting $\bar{p}$ for p^f and p^m in Equation D.10 will have little effect on the genetic dynamics since the $\bar{p} - p^f$ and $\bar{p} - p^m$ come out of the Δp equation as $O(s^2)$ terms.

Let $D = p^m - p^f$. Then, since the male and female gene frequencies move by O(s) each generation, the increment added to $|D|$ via selection each generation is an O(s) term.

Random mating also affects D each generation. In particular, it halves $|D|$. To see this, note that;

$$D' = p'^{m} - p'^{f} \tag{D.11}$$

where the prime indicates we are examining newborns.

Now, we may rewrite the right-hand side of Equation D.11 in terms of the parental gene frequencies, i.e.,

$$D' = p^{f} - \frac{1}{2} p^{f} - \frac{1}{2} p^{m} = 1/2 \ |D|. \tag{D.12}$$

Thus, $|D|$ is halved each generation.

To summarize, then, selection adds an $O(s)$ term to D each generation, while mating halves D each generation. Therefore,

$$D_1 = \frac{D_0 + O(s)}{2}$$

$$D_2 = \frac{D_1 + O(s)}{2} \rightarrow \frac{D_0 + 3\,O(s)}{4}$$

$$D_3 = \frac{D_2 + O(s)}{2} \rightarrow \frac{D_0 + 7\,O(s)}{8}$$

$$D_4 = \frac{D_3 + O(s)}{2} \rightarrow \frac{D_0 + 15\,O(s)}{16}$$

or, in general, $D_t = \dfrac{D_0 + (2^t - 1)O(s)}{2^t}$. (D.13)

As t becomes large, $D_0/2^t$ becomes negligible, and equation D.13 becomes

$$D_t \simeq \frac{(2^t - 1)O(s)}{2^t},$$

which implies that:

$D_t < O(s)$.

This implies that $|\bar{p} - p^m|$ and $|\bar{p} - p^f|$ are less than $O(s)$, since $|D_t| = |p^m - p^f| > |\bar{p} - p^m|$ and $|\bar{p} - p^f|$ holds because $\bar{p}$ is between p^m and p^f.

Therefore, Equation D.10 may be rewritten as:

$$\Delta\bar{p} \simeq \frac{1}{3}\bar{p}\,[(s_i - \bar{s})^f + (s_i - \bar{s})^m + (s_i - \bar{s})],$$

with all other terms, caused by sex linkage or deviations from Hardy-Weinberg expectations have been proven negligible provided selection is weak.

APPENDIX E

AN INCLUSIVE FITNESS TOPOGRAPHY FOR SEX-LINKED ALLELES

For sex-linked alleles, as in haplodiploidy, it is possible to obtain the analogue to Equation 2.11 which delineates the conditions under which sexual polymorphisms in social behavior may evolve. From Hamilton (1964),

$$P_i(t+1) = p_i(t)\frac{(1 + \delta R_{1.} + \delta S_{..})}{1 + \delta R_{..} + \delta S_{..}} \qquad (E.1)$$

We have already shown that $1 + R_{11} + S_{..} = \bar{w}$. By noting also that $P_i(t) = N_i(t)/2N(t)$, where N_i is the number of A_i alleles and N is the number of individuals in the population, Equation E.1 becomes:

$$p_i(t+1) = \frac{N_i(t)}{2N(t)} \frac{1 + R_{1.} + S_{..}}{\bar{w}} . \qquad (E.2)$$

Since $2p_i(t+1)N(t+1) = N_i(t+1)$, the number of A_i alleles at time $(t+1)$ may be written as:

$$N_i(t+1) = N_i(t)[1 + \delta R_{..} + \delta S_{..}] \qquad (E.3)$$

We may now divide the term in brackets into two groups: (1) actions by which females increase the number of A_i alleles; and (2) actions by which males increase the number of A_i alleles. If we assume that a given chromosome has an equally likely chance of being passed down to the next generation regardless of the sex of the carrier, Equation E.3 becomes:

$$N_i(t+1) = N_i(t)[P_f(1 + \delta R_{1.}{}^{f} + \delta S_{..}{}^{f}) +$$

$$P_m(1 + \delta R_{1.}{}^{m} + \delta S_{..}{}^{m})], \qquad \text{(E.4)}$$

where P_f and P_m are the respective proportions of females and males in the population. By noting additionally that $\Delta p_i = p_i(t+1) - P_i(t)$, and that $\bar{w} = \sum_r (\delta a_r)_{..} + 1$, Equation E.2 becomes:

$$\Delta p_i = p_i\left[\frac{P_f(1+\delta R_{1.}{}^{f}+\delta S_{..}{}^{f})+P_m(1+\delta R_{1.}{}^{m}+\delta S_{..}{}^{m})-(1+\sum_r(\delta a_r)_{..})}{\bar{w}}\right] \qquad \text{(E.5)}$$

By definition, $1 + (\delta a_r)_{..} = 1 + \sum_r r(\delta a_r)_{..} + \sum_r (1 - r)(\delta a_r)_{..} = 1 + \delta R_{..} + \delta S_{..}$. We may use this fact to rearrange the $(1 + \sum_r(\delta a_r)_{..}$ term so that it fits into Equation E.5 more naturally. Divide effects into those given by the average male and those given by the average female. Thus,

$$1 + \sum_r(\delta a_r)_{..} = 1 + P_f \sum_r (\delta a_r)_{..}{}^{f} + P_m \sum_r (\delta a_r)_{..}{}^{m},$$

or

$$1 + \sum_r(\delta a_r)_{..} = P_f[1 + \sum_r(\delta a_r)_{..}{}^{f}] + P_m(1 + \sum_r(\delta a_r)_{..}{}^{m}.$$

Substituting this identity into Equation E.3 yields:

$$\Delta p_i = p_i\left[\frac{P_f[1 + \delta R_{1.}{}^{f} + \delta S_{..}{}^{f} - 1 - \sum_r(\delta a_r)_{..}{}^{f}]}{\bar{w}} + \right.$$

$$\left.\frac{P_m[1 + \delta R_{1.}{}^{m} + \delta S_{..}{}^{m} - 1 - \sum_r(\delta a_r)_{..}{}^{m}]}{\bar{w}}\right] \qquad \text{(E.6)}$$

Since $\delta S_{..} - \sum_r(\delta a_r)_{..} = \delta R_{..}$, and since $1 + \delta R_{ij} = R_{ij}$, Equation E.6 may be rewritten as:

$$\Delta p_i = p_i \frac{P_f(R_{1.}^{\ f} - R_{..}^{\ f}) + P_m(R_{1.}^{\ m} - R_{..}^{\ m})}{\bar{w}} . \qquad (E.7)$$

By Equation E.7, and the subsequent argument leading to Equation 2.11, it is found that:

$$\Delta p = \frac{p(1 - p)}{2\bar{w}} \left[P_f\left(\frac{\partial R_{...}^{\ f}}{\partial p_i}\right) + P_m\left(\frac{\partial R_{...}^{\ m}}{\partial p_i}\right)\right] \qquad (E.8)$$

Equation E.8 is of interest since it compartmentalizes the effects of selection into two components of inclusive fitness, both of which (within natural constraints) evolution may be expected to independently maximize. Since this compartmentalization is based on sex, one may expect the evolution of modifier alleles promoting sexual similarities or differences which maximize the inclusive fitness of both sexes simultaneously.

APPENDIX F

CONDITIONS AFFECTING INCLUSIVE FITNESS FOR HETEROZYGOTES

It is not immediately clear why social behavior alleles may exhibit heterozygote superiority or inferiority. To discuss this, assume $R_{11} > R_{22}$. Then we have overdominance when $R_{12} > R_{11}$ and heterozygote inferiority when $R_{12} < R_{22}$. If we break these R_{ij}'s into $1 + \partial R_{ij}$'s, it initially seems correct to define ∂R_{12} as $h(\partial R_{11})$, where $0 \leq h \leq 1$.

But, as Cockerham, Burrows, Young and Prout (1972) have pointed out, there may be two components to fitness: b_{12} and c_{12} may be related to b_{11} and c_{11} by different proportionality constants. We may therefore define the inclusive fitness effect of A_1A_2 as $\delta R_{12} = ec_{11} + \sum_{r\neq 1} rg\,(\delta a_r)_{11}$.

Thus, if we assume that the alleles do not affect r, that $\delta R_{11} > \delta R_{22}$, and that g is independent of r, we have overdominance and its potential for balance polymorphism if $\delta R_{12} > R_{11}$, i.e., if $ec_{11} + g\sum_{r\neq 1} r(b_r)_{11} > c_{11} + \sum_{r\neq 1} r(b_r)_{11}$. This leads to the conclusion that overdominance occurs when:

$$\frac{c_{11}}{\sum_{r=1} r(b_r)_{11}} > \frac{1 - g}{e - 1} \qquad \text{(F.1)}$$

if $\sum_{r\neq 1} r(b_r)_{11}$ and $(e - 1)$ are of the same signs. If they are of different signs, overdominance occurs when:

$$\frac{c_{11}}{\sum_{r\neq 1} r(b_r)_{11}} < \frac{1 - g}{e - 1} \qquad \text{(F.2)}$$

Heterozygote inferiority, on the other hand, obtains when $\delta R_{12} < \delta R_{22}$, which I rewrite as $xc_{22} + y \sum_{r \neq 1} r(b_r)_{22} < c_{22} + \sum_{r \neq 1} r(b_r)_{22}$. This means that there is heterozygote inferiority when:

$$\frac{c_{22}}{\sum_{r \neq 1} r(b_r)_{22}} < \frac{1 - y}{x - 1} \tag{F.3}$$

if $\sum_{r \neq 1} r(b_r)_{11}$ and $(x - 1)$ are of the same signs. If they are of different signs, the heterozygote is inferior when:

$$\frac{c_{22}}{\sum_{r \neq 1} r(b_r)_{22}} > \frac{1 - y}{x - 1} . \tag{F.4}$$

A similar analysis which includes effects on r can be made using this approach.

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