EVOLUTION OF INTERACTIONS IN FAMILY-STRUCTURED POPULATIONS: MIXED MATING MODELS

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ABSTRACT

The effect of inbreeding on sociality is studied theoretically for the evolution of interactions between siblings in certain mixed mating systems that give rise to inbreeding: sib with random mating and selfing with random mating. Two approaches are taken. First, specific models of altruism are studied for the various mating systems. In the case of the additive model, inbreeding facilitates the evolution of altruistic genes. Likewise, for the multiplicative model this is usually the case, as long as the costs of altruism are not too great. Second, the case of total altruism, in which the gene has zero individual fitness but increases the fitness of associates, is studied for a general fitness formulation. In this case, inbreeding often retards the ability of such genes to increase when rare, and the equilibrium frequency of those recessive genes that can increase is totally independent of the mating system and, consequently, of the amount of inbreeding. It appears from the results presented that inbreeding facilitates most forms of altruism, but retards extreme altruism. These results stem from the fact that inbreeding increases the within-family relatedness by increasing the between-family variance in allele frequency. In most cases this facilitates altruism. However, in the case of total altruism, only heterozygotes can pass on the altruistic allele, and inbreeding tends to decrease this heterozygote class. In either case, the important effect of inbreeding lies in altering the genotypic distribution of the interactions.

INBREEDING may have important effects on the evolution of social behavior. Indeed, WILSON (1976) has referred to the effects of inbreeding on sociality as one of the "central problems" of sociobiology. In his pioneering work, HAMILTON (1964) noted that "it does seem necessary to invoke at least mild inbreeding if we are to explain some of the phenomena of the social insects—and indeed of animal sociability in general—by means of this theory." This theme was pursued more explicitly (HAMILTON 1972), where he argued the following points: (1) Before socialization has begun, inbreeding may facilitate the evolution of sociality by increasing the likelihood of associations between individuals. (2) Once haploidiploid species have become social, inbreeding may diminish the asymmetries in relationship that exist between a female and her daughters and sisters. This may induce workers to breed, thereby increasing the reproductive efficiency of Hymenopteran nests. (3) Inbreeding may offset the problems posed by polygyny.

These hypothesized effects of inbreeding on sociality are based on the fact that, if an individual is inbred, its parents must have been related. Consequently, the degree of inbreeding measures the degree of relationship between parents. However, another well-known result of inbreeding is an increase in homozygosity, which in turn tends to increase the genetic variance in the population. For this reason it is not necessarily the case that interacting individuals are related in an inbred population, although by definition the parents must be. However, most discussions in the sociobiological literature treat inbreeding and relatedness as synonymous.

Selection poses an additional problem. The theoretical tool most often used to study kin selection in inbreeding populations is Hamilton's Rule:

$$c/b < \text{“}r\text{”} \ . \tag{1}$$

In this rule, c and b are the additive decrement and increment to the fitness of altruist and recipient, respectively, and "r" is a measure of their degree of genetic relationship. This rule is often used to study inbreeding by asking how "r" is affected if the individuals involved are inbred (HAMILTON 1972; SEGER 1976; FLESNESS 1978; MICHOD and ANDERSON 1979; BARTZ 1979). However, the rule was derived (HAMILTON 1964; CHARNOV 1977) for *outbred* populations, along with the additional assumptions of weak selection and an additive model of fitness.

Various rules analogous to (1) have been derived for inbred populations under these assumptions (MICHOD 1979; FLESNESS and HOLTZMAN 1980). In addition, it recently has been shown (MICHOD and HAMILTON 1980) that the right-hand side (RHS) of these inbred rules [MICHOD 1979, equation (3); FLESNESS and HOLTZMAN 1980, equation (10)], although independently derived, are identical. They equal Cov (X_p, Y_g) /Cov (X_p, X_g), which in turn equals ORLOVE and WOOD's (1978) ρ in outbreeding populations. Here, X and Y denote altruist and recipient, respectively, and X_p, X_g, Y_p, Y_g denote the phenotype and genotype of X and Y. These independent derivations suggest that the rule for inbred populations is fairly robust.

The RHS of this inbred rule depends upon gene frequency and the degree of dominance, in addition to genetic relationship. In the outbred case, these complications do not arise, and the RHS of (1) depends solely on genetic relationship (at least in the derivations based on identity coefficients and the additive model). Consequently, the dynamics of altruistic genes in inbred populations may be expected to differ from the outbred case. From the discussions of inbreeding in the sociobiological literature, one would expect the RHS of the inbred rule to increase as inbreeding proceeds, facilitating the evolution of sociality. However, as pointed out by MICHOD (1979), this is not necessarily true. Consequently, there is the possibility that inbreeding may have qualitatively different effects on sociality in different situations.

These derivations of Hamilton's Rule in inbred populations (MICHOD 1979; FLESNESS and HOLTZMAN 1980; MICHOD and HAMILTON 1980) rely on two important assumptions. (1) Selection is weak so that traditional genetic identity

coefficients can be used to generate the genotypic distribution of interactions (for more discussion on this use of identity coefficients see JACQUARD 1974; MICHOD 1979; MICHOD and HAMILTON 1980; ABUGOV and MICHOD 1980). (2) These derivations do not attend to the population and mating processes that must ultimately give rise to the inbreeding. Both of these limitations stem from models that use genetic identity coefficients. The approach used below circumvents the need for identity coefficients, since the mating system and population structure is specified explicitly. This, in turn, implicitly specifies the genetic context for selection.

The purpose of the work reported here is to study the effects of inbreeding on sociality by studying the evolution of interactions in explicitly specified mixed mating systems. In order to study the effects of the mating system *per se*, it will be assumed that inbreeding has no direct effect on fitness (such as inbreeding depression).

THE MODEL

The model used here is an extension of the family-structured, frequency-dependent model previously studied for the case of random mating (WILLIAMS and WILLIAMS 1957; LEVITT 1975; SCUDO and GHISELIN 1975; CHARNOV 1977; CAVALLI-SFORZA and FELDMAN 1978; CHARLESWORTH 1978; WADE 1978, 1979; TEMPLETON 1979; MICHOD and ABUGOV 1980; ABUGOV and MICHOD 1980; MICHOD, submitted). For heuristic purposes (MICHOD 1980), consider a single diploid locus with two alleles, A_1 and A_2. The 6 family frequencies, offspring arrays and fitnesses are given in Table 1. $W_{i,m}$ is defined to be the fitness of genotype i in family $m (i = 1,2,3$ corresponding to A_1A_1, A_1A_2, A_2A_2, respectively). It is important to note that, since fitness is family specific, the overall fitness of a genotype will vary with the frequencies of the various families within which it exists. The family-specific fitness could arise either from (a) the interactions of the various genotypes within families, or (b) an effect mediated through the parents. In either case, the fitness of a genotype will vary with the genotypes of its parents. In the present paper, the family-specific fitness will be interpreted as in (a). In a subsequent paper (MICHOD, submitted for publication), I will contrast the 2 interpretations, since they are central to the current controversy concerning the role of kin selection and parental manipulation in the evolution of sociality.

In case (a), the family-specific fitnesses $W_{i,m}$ are given in Table 2, where $w_{i,j}$ is the fitness of genotype i when in association with genotype j. The family specific fitness, $W_{i,m}$, is obtained by taking the arithmetic average of the association fitnesses of the i genotype over the various associations it has in family m. Other averages besides the arithmetic may be appropriate if the interactions are separated in time or in space. The association specific fitness, $w_{i,j}$, can be further decomposed into specific models of behavior. Assume that the A_1 allele codes for an "altruistic" behavior. Altruism is usually considered to involve a cost in fitness, c, to the altruist and a benefit in fitness, b, to the recipient. These costs and benefits are generally assumed to be independent, resulting either in the additive

TABLE 1

Model of selection in a family structured population

Mating	Frequency	Offspring array	Offspring fitness	Family fitness
$A_1A_1 \times A_1A_1$	f_1	A_1A_1	$W_{1,1}$	$\bar{W}_1 = W_{1,1}$
$A_1A_1 \times A_1A_2$	f_2	$\frac{1}{2}A_1A_1$, $\frac{1}{2}A_1A_2$	$W_{1,2}$, $W_{2,2}$	$\bar{W}_2 = \frac{1}{2}W_{1,2} + \frac{1}{2}W_{2,2}$
$A_1A_1 \times A_2A_2$	f_3	A_1A_2	$W_{2,3}$	$\bar{W}_3 = W_{2,3}$
$A_1A_2 \times A_1A_2$	f_4	$\frac{1}{4}A_1A_1$, $\frac{1}{2}A_1A_2$, $\frac{1}{4}A_2A_2$	$W_{1,4}$, $W_{2,4}$, $W_{3,4}$	$\bar{W}_4 = \frac{1}{4}W_{1,4} + \frac{1}{2}W_{2,4} + \frac{1}{4}W_{3,4}$
$A_1A_2 \times A_2A_2$	f_5	$\frac{1}{2}A_1A_2$, $\frac{1}{2}A_2A_2$	$W_{2,5}$, $W_{3,5}$	$\bar{W}_5 = \frac{1}{2}W_{2,5} + \frac{1}{2}W_{3,5}$
$A_2A_2 \times A_2A_2$	f_6	A_2A_2	$W_{3,6}$	$\bar{W}_6 = W_{3,6}$

TABLE 2

Correspondence between fitness in family structured model and fitness in COCKERHAM et al. *(1972)*

$W_{1,1} = w_{1,1}$	$W_{2,4} = \frac{1}{4}w_{2,1} + \frac{1}{2}w_{2,2} + \frac{1}{4}w_{2,3}$
$W_{1,2} = \frac{1}{2}(w_{1,1} + w_{1,2})$	$W_{3,4} = \frac{1}{4}w_{3,1} + \frac{1}{2}w_{3,2} + \frac{1}{4}w_{3,3}$
$W_{2,2} = \frac{1}{2}(w_{2,1} + w_{2,2})$	$W_{2,5} = \frac{1}{2}(w_{2,2} + w_{2,3})$
$W_{2,3} = w_{2,2}$	$W_{3,5} = \frac{1}{2}(w_{3,2} + w_{3,3})$
$W_{1,4} = \frac{1}{4}w_{1,1} + \frac{1}{2}w_{1,2} + \frac{1}{4}w_{1,3}$	$W_{3,6} = w_{3,3}$

$W_{i,m}$ is the fitness of genotype i in family m (Table 1). $w_{i,j}$ is the fitness of genotype i in association with genotype j as defined by COCKERHAM *et al.* (1972).

model given in Table 3a (HAMILTON 1964; WILSON 1980; CHARNOV 1977; WADE 1979; CAVALLI-SFORZA and FELDMAN 1978; MICHOD 1979), or the multiplicative model given in Table 3b (CAVALLI-SFORZA and FELDMAN 1978, CHARLESWORTH 1978). In Table 3, h is the probability with which a heterozygote is an altruist and reflects the penetrance of the gene.

In order to develop recurrence equations for the various family frequencies, one must specify the mating process. Here, I will consider 2 mating models: (1) selfing and random mating, and (2) sib and random mating. In both cases, it is assumed that an individual mates at random with probability t and either selfs or mates with a sib with probability 1-t.

The mixed-selfing system applies to the evolution of interactions in many plant species; the mixed-sib system is relevant to wolves (MECH 1970), Mexican jays (J. H. BROWN, personal communication) and other animals, but is most common in insects. Sibling matings are suspected in the harvester ant *Pogonomyrex badius* (CROZIER 1977) and also in various species that possess wingless males or partially winged microgyne females (DONISTHORPE 1927). Sib-sib or sib-parent mating is usual in the Pharaoh's ant *Monomorium pharaonis* (WILSON 1971) and is probably common in other ant species that have abandoned the nuptial flight and mate in or close to the nest (*e.g.*, *Myrmica schencki*, TALBOT 1945). HAMILTON (1967) provides a list of more than 20 species of parasitic

TABLE 3

Specific models of altruism

(a) Additive model		
$w_{1,1} = 1 + b - c$	$w_{1,2} = 1 - c + hb$	$w_{1,3} = 1 - c$
$w_{2,1} = 1 - hc + b$	$w_{2,2} = 1 - hc + hb$	$w_{2,3} = 1 - hc$
$w_{3,1} = 1 + b$	$w_{3,2} = 1 + hb$	$w_{3,3} = 1$
(b) Multiplicative model		
$w_{1,1} = (1 + b)(1 - c)$	$w_{1,2} = (1 - c)(1 + hb)$	$w_{1,3} = 1 - c$
$w_{2,1} = (1 - hc)(1 + b)$	$w_{2,2} = (1 - hc)(1 + hb)$	$w_{2,3} = 1 - hc$
$w_{3,1} = 1 + b$	$w_{3,2} = 1 + hb$	$w_{3,3} = 1$

In these models, c and b are positive increments in fitness; h is the probability that a heterozygote performs an altruistic act.

insects and mites that have sib mating (see also JAYAKAR and SPURWAY 1966; ASKEW 1968; WILSON 1971; HAMILTON 1972; CROZIER 1977). In addition, isolation by distance may be an important factor promoting inbreeding in many insect species (see, for example HAMILTON 1964, 1972; ALEXANDER and SHERMAN 1977; PAMILO *et al.* 1978; GREENBERG 1979). In termites, sib-sib or sib-parent mating certainly exists and is probably common. Colonies are monogamous and, when a king or queen dies, they are replaced by resident eggs or larvae. This neotene then mates with his or her parent or sib (depending on whether one or both of the original pair had died). Indeed, it has been suggested that termite eusociality is due to their high degree of inbreeding (HAMILTON 1972; LIN and MICHENER 1972). In conclusion, mixed mating models may be widely appropriate as tools to study the effects of inbreeding on sociality.

Mixed selfing: Let g_i be the genotypic frequency in the total population of i adults at time t. The random-mating portion of the population mates according to $(g_1 + g_2 + g_3)^2$, while the selfing portion mates according to $(g_1 + g_2 + g_3)$. This gives rise to the following mating scheme, where a prime superscript, $'$, denotes the $t + 1$ generation:

$$\begin{aligned} f'_1 &= tg_1^2 + (1-t)g_1 \\ f'_2 &= t2g_1g_2 \\ f'_3 &= t2g_1g_3 \\ f'_4 &= tg_2^2 + (1-t)g_2 \\ f'_5 &= t2g_3g_2 \\ f'_6 &= tg_3^2 + (1-t)g_3 \ . \end{aligned} \tag{2}$$

Note that families 2, 3 and 5 result from heterogenotypic matings and clearly cannot arise from selfing.

The offspring-parent transition is given by

$$\begin{aligned} g'_1\overline{W} &= f'_1W_{1,1} + \tfrac{1}{2}f'_2W_{1,2} + \tfrac{1}{4}f'_4W_{1,4} \\ g'_2\overline{W} &= \tfrac{1}{2}f'_2W_{2,2} + f'_3W_{2,3} + \tfrac{1}{2}f'_4W_{2,4} + \tfrac{1}{2}f'_5W_{2,5} \\ g'_3\overline{W} &= \tfrac{1}{4}f'_4W_{3,4} + \tfrac{1}{2}f'_5W_{3,5} + f'_6W_{3,6} \ , \end{aligned} \tag{3}$$

with

$$\overline{W} = \sum_m f_m\overline{W}_m \ . \tag{4}$$

Equation (2) can be substituted in (3) to yield the genotypic recurrence equations for selection in family-structured, mixed-selfing populations. In the case of the mixed-sib mating system to be considered shortly, the recurrence equations are in terms of the family frequencies (5).

Special cases of this system [(2) and (3)] that have been previously studied in the literature assume that a genotype's fitness is independent of the family context and hence constant across families. In the case of pure selfing (t=0), one obtains the model first studied by HAYMAN and MATHER (1953, with notation $W_{1,1} = W_{1,4} = x$, $W_{3,6} = W_{3,4} = y$, and $W_{2,4} = 1$). This model has, in turn, been studied by many others (REEVE 1955; HALDANE 1956, but see HAYMAN and

MATHER 1956; KARLIN 1968). The mixed mating model ($0<t<1$) was first studied by HAYMAN (1953) for the case of constant fitness (see also KIMURA and OHTA 1971). This model has been repeatedly studied from different points of view and is now an extremely important tool for the study of selection in plant populations (for example, WORKMAN and JAIN 1966; JAIN and WORKMAN 1967; WEIR 1970; ALLARD and WORKMAN 1963; CLEGG, KAHLER and ALLARD 1978).

Mixed sib mating: The family frequencies for the random-mating portion of the population are again given by $(g_1+g_2+g_3)^2$; however, the adult frequencies are no longer sufficient to generate the sib matings. Let $M_{i,k}$ be the expected Mendelian frequency of genotype i in family k. The sib matings are assumed to occur at random within family k and are given by

$$\left(\frac{M_{1,k}W_{1,k}}{\bar{W}_k}+\frac{M_{2,k}W_{2,k}}{\bar{W}_k}+\frac{M_{3,k}W_{3,k}}{\bar{W}_k}\right)^2 .$$

The relative contribution of these sib matings to the next generation is $f_k\bar{W}_k/\bar{W}$, so that the mating array for the sib-mated portion of the population is

$$\sum_k f_k\frac{\bar{W}_k}{\bar{W}}\left(\frac{M_{1,k}W_{1,k}}{\bar{W}_k}+\frac{M_{2,k}W_{2,k}}{\bar{W}_k}+\frac{M_{3,k}W_{3,k}}{\bar{W}_k}\right)^2 .$$

This leads to the following family recurrence equations under mixed-sib random mating:

$$\bar{W}f'_1=(1-t)\{f_1W_{1,1}+f_2\frac{W_{1,2}^2}{4\bar{W}_2}+f_4\frac{W_{1,4}^2}{16\bar{W}_4}\}+\frac{t}{\bar{W}}\{f_1W_{1,1}+\tfrac{1}{2}f_2W_{1,2}+\tfrac{1}{4}f_4W_{1,4}\}^2$$

$$\bar{W}f'_2=(1-t)\{f_2\frac{W_{1,2}W_{2,2}}{2\bar{W}_2}+f_4\frac{W_{1,4}W_{2,4}}{4\bar{W}_4}\}+\frac{2t}{\bar{W}}\{f_1W_{1,1}+\tfrac{1}{2}f_2W_{1,2}+\tfrac{1}{4}f_4W_{1,4}\}$$
$$\times\{\tfrac{1}{2}f_2W_{2,2}+f_3W_{2,3}+\tfrac{1}{2}f_4W_{2,4}+\tfrac{1}{2}f_5W_{2,5}\}$$

$$\bar{W}f'_3=(1-t)f_4\frac{W_{1,4}W_{3,4}}{8\bar{W}_4}+\frac{2t}{\bar{W}}\{f_1W_{1,1}+\tfrac{1}{2}f_2W_{1,2}+\tfrac{1}{4}f_4W_{1,4}\} \qquad (5)$$
$$\times\{f_6W_{3,6}+\tfrac{1}{2}f_5W_{3,5}+\tfrac{1}{4}f_4W_{3,5}\}$$

$$\bar{W}f'_5=(1-t)\{f_4\frac{W_{2,4}W_{3,4}}{4\bar{W}_4}+f_5\frac{W_{2,5}W_{3,5}}{2\bar{W}_5}\}+\frac{2t}{\bar{W}}\{f_6W_{3,6}+\tfrac{1}{2}f_5W_{3,5}+\tfrac{1}{4}f_4W_{3,4}\}$$
$$\times\{\tfrac{1}{2}f_2W_{2,2}+f_3W_{2,3}+\tfrac{1}{2}f_4W_{2,4}+\tfrac{1}{2}f_5W_{2,5}\}$$

$$\bar{W}f'_6=(1-t)\{\frac{f_4W_{3,4}}{16W_4}+\frac{f_5W_{3,5}}{4\bar{W}_5}+f_6W_{3,6}\}+\frac{t}{\bar{W}}\{f_6W_{3,6}+\tfrac{1}{2}f_5W_{3,5}+\tfrac{1}{4}f_4W_{3,4}\},$$

with $f_4=1-f_1-f_2-f_3-f_5-f_6$ and $\bar{W}$ as before (4).

Pure sib mating: In the case of pure sib-mating (t=0), system (5) can be with $\mathbf{f} = [f^1,f^2,f^3,f^4,f^5,f^6]^T$ and

$$\mathbf{f}_{t+1} = \frac{\mathbf{A}}{\bar{W}_t}\mathbf{f}_t \tag{6}$$

with $f = [f_1,f_2,f_3,f_4,f_5,f_6]^T$ and

$$\mathbf{A} = \begin{bmatrix} W_{1,1} & \frac{W_{1,2}^2}{4\bar{W}_2} & 0 & \frac{W_{1,4}^2}{16\bar{W}_4} & 0 & 0 \\ 0 & \frac{W_{1,2}W_{2,2}}{2\bar{W}_2} & 0 & \frac{W_{1,4}W_{2,4}}{4\bar{W}_4} & 0 & 0 \\ 0 & 0 & 0 & \frac{W_{1,4}W_{3,4}}{8\bar{W}_4} & 0 & 0 \\ 0 & \frac{W_{2,2}^2}{4\bar{W}_2} & W_{2,3} & \frac{W_{2,4}^2}{4\bar{W}_4} & \frac{W_{2,5}^2}{4\bar{W}_5} & 0 \\ 0 & 0 & 0 & \frac{W_{2,4}W_{3,4}}{4\bar{W}_4} & \frac{W_{2,5}W_{3,5}}{2\bar{W}_5} & 0 \\ 0 & 0 & 0 & \frac{W_{3,4}^2}{16\bar{W}_4} & \frac{W_{3,5}^2}{4\bar{W}_5} & W_{3,6} \end{bmatrix}$$

where $\bar{W}_t$ acts as a normalizing scaler to guarantee that $\mathbf{f}_t + 1$ is again a frequency vector.

Special cases of (6) have been previously studied. With all $W_{i,m}$'s $= 1$ (neutral genes), the system reduces to the generation matrix first studied by FISHER (1949). HAYMAN and MATHER (1953) first formulated recurrence equations for constant selection in pure sib-mating populations (see also KARLIN 1968).

ANALYSIS

The recurrence equations derived in their most general form in the last section are intractable. To obtain results, I have studied either (a) the local behavior of these equations near fixation of either allele, which provides conditions for increase of the allele when rare or when common, or (b) the "total" altruism in which the A_1 allele has zero individual fitness when expressed.

Local analysis, mixed selfing: The local analysis of equations (2) and (3) results in the following linear equations for the perturbations:

$$\begin{bmatrix} \delta g'_1 \\ \delta g'_3 \end{bmatrix} = \begin{bmatrix} A & B \\ C & D \end{bmatrix} \begin{bmatrix} \delta g_1 \\ \delta g_3 \end{bmatrix}, \tag{7}$$

with

$$A = (1-t)(W_{1,1} - \tfrac{1}{4}W_{1,4})/W_{3,6}$$
$$B = -\tfrac{1}{4}(1-t)W_{1,4}/W_{3,6}$$
$$C = [t(W_{2,5} - 2W_{2,3}) + (1-t)(\tfrac{1}{4}W_{1,4} + \tfrac{1}{2}W_{2,4} - W_{1,1})]/W_{3,6}$$
$$D = [tW_{2,5} + (1-t)(\tfrac{1}{4}W_{1,4} + \tfrac{1}{2}W_{2,4})]/W_{3,6}$$

at the (0,1) equilibrium, and

$$A = [tW_{2,2} + (1-t)(\tfrac{1}{2}W_{2,4} + \tfrac{1}{2}W_{3,4})]/W_{1,1}$$
$$B = [t(W_{2,2} - 2W_{2,3}) + (1-t)(\tfrac{1}{2}W_{2,4} + \tfrac{1}{4}W_{3,4} - W_{3,6})]/W_{1,1}$$
$$C = -\tfrac{1}{4}(1-t)W_{3,4}/W_{1,1}$$
$$D = (1-t)(W_{3,6} - \tfrac{1}{4}W_{3,4})/W_{1,1}$$

at the (1,0) equilibrium, and $\delta g_2 = 1 - \delta g_1 - \delta g_3$.
The dominant eigenvalues are given by analysis of (7) to be

$$\lambda = \frac{\tfrac{1}{2}}{W_{3,6}}\{(1-t)(W_{1,1} + \tfrac{1}{2}W_{2,4}) + tW_{2,5} + [[tW_{2,5} - (1-t)(W_{1,1} - \tfrac{1}{2}W_{2,4})]^2 + 2(1-t)W_{1,4}W_{2,3}]^{1/2}\} \quad (8a)$$

at (0,1) and

$$\lambda = \frac{\tfrac{1}{2}}{W_{1,1}}\{(1-t)(W_{3,6} + \tfrac{1}{2}W_{2,4}) + tW_{2,2} + [[tW_{2,2} - (1-t)(W_{3,6} - \tfrac{1}{2}W_{2,4})]^2 + 2t(1-t)W_{3,4}W_{2,3}]^{1/2}\} \quad (8b)$$

at (1,0). It is apparent by inspection (or differentiation) of equations (8) that no simple relationship between the dominant eigenvalues and t exists. Note that if $t = 1$, equations (8) become

$$\lambda = \begin{cases} W_{2,5}/W_{3,6} \text{ at } (0,1) & (9a) \\ W_{2,2}/W_{1,1} \text{ at } (1,0), & (9b) \end{cases}$$

which are identical to the results obtained elsewhere for random mating populations (TEMPLETON 1979; MICHOD, submitted for publication).

Local analysis, mixed sib mating: The local analysis of (5) at both fixation equilibria, (0,0,0,0,1) and (1,0,0,0,0), results in the following linear equations for the perturbations,

$$\delta\mathbf{f}_{t+1} = \mathbf{B}\delta\mathbf{f}_t \ , \quad (10)$$

with $\delta\mathbf{f} = (\delta f_1, \delta f_2, \delta f_3, \delta f_5, \delta f_6)$ at (0,0,0,0,1) and $\delta\mathbf{f} = (\delta f_6, \delta f_5, \delta f_3, \delta f_2, \delta f_1)$ at (1,0,0,0,0) with $\delta f_4 = 1-\delta f_1-\delta f_2-\delta f_3-\delta f_5-\delta f_6$. The elements of matrix **B** are given in Table 4 for the two equilibria. Applying row and column operations to obtain the characteristic equation, $\mathbf{B}\text{-}\mathbf{I}\lambda$ reduces to a matrix of the following form

$$\begin{array}{lllll} a-\lambda & b & 0 & 0 & c \\ 0 & d-\lambda & 0 & 0 & e \\ f & g & -\lambda & 0 & h \\ 0 & i & j & k-\lambda & l \\ m+\lambda & n+\lambda & p+\lambda & q+\lambda & r-\lambda \end{array} \quad (11)$$

TABLE 4

Elements of **B** *matrix of equation (10) for (0,0,0,0,1) and (1,0,0,0,0)*

$(t-1)\left[\frac{W_{1,4}^2}{16\overline{W}_4}-W_{1,1}\right]$	$(t-1)\left[\frac{W_{1,4}^2}{16\overline{W}_4}-\frac{W_{1,2}^2}{4\overline{W}_2}\right]$	$(t-1)\frac{W_{1,4}^2}{16\overline{W}_4}$	$(t-1)\frac{W_{1,4}^2}{16\overline{W}_4}$	$(t-1)\frac{W_{1,4}^2}{16\overline{W}_4}$
$(t-1)\frac{W_{1,4}W_{2,4}}{4\overline{W}_4}$	$(t-1)\left[\frac{W_{1,4}W_{2,4}}{4\overline{W}_4}-\frac{W_{1,2}W_{2,2}}{2\overline{W}_2}\right]$	$(t-1)\frac{W_{1,4}W_{2,4}}{4\overline{W}_4}$	$(t-1)\frac{W_{1,4}W_{2,4}}{4\overline{W}_4}$	$(t-1)\frac{W_{1,4}W_{2,4}}{4\overline{W}_4}$
$(t-1)\frac{W_{1,4}W_{3,4}}{8\overline{W}_4}+t\left[2W_{1,1}-\frac{W_{1,4}}{2}\right]$	$(t-1)\frac{W_{1,4}W_{3,4}}{8\overline{W}_4}+t\left[W_{1,2}-\frac{W_{1,4}}{2}\right]$	$(t-1)\frac{W_{1,4}W_{3,4}}{8\overline{W}_4}-t\frac{W_{1,4}}{2}$	$(t-1)\frac{W_{1,4}W_{3,4}}{8\overline{W}_4}-t\frac{W_{1,4}}{2}$	$(t-1)\frac{W_{1,4}W_{3,4}}{8\overline{W}_4}-t\frac{W_{1,4}}{2}$
$(t-1)\frac{W_{2,4}W_{3,4}}{4\overline{W}_4}-tW_{2,4}$	$(t-1)\frac{W_{2,4}W_{3,4}}{4\overline{W}_4}+t(W_{2,2}-W_{2,4})$	$(t-1)\frac{W_{2,4}W_{3,4}}{4\overline{W}_4}+t(2W_{2,3}-W_{2,4})$	$(t-1)\left[\frac{W_{2,4}W_{3,4}}{4\overline{W}_4}-\frac{W_{2,5}W_{3,5}}{2\overline{W}_5}\right]+t(W_{2,5}-W_{2,4})$	$(t-1)\frac{W_{2,4}W_{3,4}}{4\overline{W}_4}-tW_{2,4}$
$(t-1)\frac{W_{3,4}^2}{16\overline{W}_4}+(t+1)(\overline{W}_4-W_{1,1})-\frac{1}{2}tW_{3,4}$	$(t-1)\frac{W_{3,4}^2}{16\overline{W}_4}+(t+1)(\overline{W}_4-\overline{W}_2)-\frac{1}{2}tW_{3,4}$	$(t-1)\frac{W_{3,4}^2}{16\overline{W}_4}+(t+1)(\overline{W}_4-W_{2,3})-\frac{1}{2}tW_{3,4}$	$(t-1)\left[\frac{W_{3,4}^2}{16\overline{W}_4}-\frac{W_{3,5}^2}{4\overline{W}_5}\right]+(t+1)(\overline{W}_4-\overline{W}_5)+t(W_{3,5}-\frac{1}{2}W_{3,4})$	$(t-1)\frac{W_{3,4}^2}{16\overline{W}_4}+(t+1)\overline{W}_4-\frac{1}{2}tW_{3,4}$

The elements above are for (0,0,0,0,1) and should each be divided by $W_{3,6}$. To obtain the elements for (1,0,0,0,0), exchange $W_{3,4}$ and $W_{1,4}$, $W_{3,5}$ and $W_{1,2}$, $W_{2,5}$ and $W_{2,2}$, $\overline{W}_5$ and $\overline{W}_2$ and $W_{1,1}$ and $W_{3,6}$ in the above table. After doing so, then divide by $W_{1,1}$ instead of $W_{3,6}$.

TABLE 5

Elements of reduced form of (**B**-Iλ) *for use in (11); all elements should be divided by* $W_{3,6}$ *in the case of (0,0,0,0,1) and* $W_{1,1}$ *in the case of (1,0,0,0,0)*

Matrix element	Equilibrium (0,0,0,0,1)	Equilibrium (1,0,0,0,0)
a	$(1-t)W_{1,1}$	$(1-t)W_{3,6}$
b	$(1-t)W_{1,2}^2/4\overline{W}_2$	$(1-t)W_{3,5}^2/4\overline{W}_5$
c	$(t-1)W_{1,4}^2/16\overline{W}_4$	$(t-1)W_{3,4}^2/16\overline{W}_4$
d	$(1-t)W_{1,2}W_{2,2}/2\overline{W}_2$	$(1-t)W_{3,5}W_{2,5}/2\overline{W}_5$
e	$(t-1)W_{1,4}W_{2,4}/4\overline{W}_4$	$(t-1)W_{3,4}W_{2,4}/4\overline{W}_4$
f	$2tW_{1,1}$	$2tW_{3,6}$
g	$tW_{1,2}$	$tW_{3,5}$
h	$(t-1)W_{1,4}W_{3,4}/8\overline{W}_4-tW_{1,4}/2$	$(t-1)W_{1,4}W_{3,4}/8\overline{W}_4-tW_{3,4}/2$
i	$tW_{2,2}$	$tW_{2,5}$
j	$2tW_{2,3}$	$2tW_{2,3}$
k	$(1-t)W_{3,5}W_{2,5}/2\overline{W}_5+tW_{2,5}$	$(1-t)W_{1,2}W_{2,2}/2\overline{W}_2+tW_{2,2}$
l	$(t-1)W_{2,4}W_{3,4}/4\overline{W}_4-tW_{2,4}$	$(t-1)W_{2,4}W_{1,4}/4\overline{W}_4-tW_{2,4}$
m	$-(1+t)\overline{W}_{1,1}$	$-(1+t)\overline{W}_{3,6}$
n	$-(1+t)\overline{W}_2$	$-(1+t)\overline{W}_5$
p	$-(1+t)W_{2,3}$	$-(1+t)W_{2,3}$
q	$(1-t)W_{3,5}^2/4\overline{W}_5+tW_{3,5}-(1+t)\overline{W}_5$	$(1-t)W_{1,2}^2/4\overline{W}_2+tW_{1,2}-(1+t)\overline{W}_2$
r	$(t-1)W_{3,4}^2/16\overline{W}_4-\frac{1}{2}tW_{2,4}+(1+t)\overline{W}_4$	$(t-1)W_{1,4}^2/16\overline{W}_4-\frac{1}{2}tW_{1,4}+(1+t)\overline{W}_4$

with the elements given in Table 5 for the two equilibria. Upon evaluating the determinant of (11), the following characteristic equation is obtained:

$$\lambda^5 + \alpha\lambda^4 + \beta\lambda^3 + \gamma\lambda^2 + \Delta\lambda + \varepsilon = 0 \tag{12}$$

with

$$\begin{aligned}
\alpha &= -d-k-c-r-a-l-h-e \\
\beta &= d(k+c+r+a+l+h)+e(a-i+k-n-g-b) \\
&\quad +k(c+r+a+h)+h(a-j-p)-l(q-a)-mc+ar-fc \\
\gamma &= -cdk-drk-akd+mcd-ard+qld \\
&\quad -lad+fcd-had+hjd-hkd+hpd \\
&\quad +bek-bem-bef+ckm-ark+qla \\
&\quad -jcf+jha-kcf-kha-cpf-pha \\
&\quad -hjq+hkp+eai-eak+ean+eag \\
&\quad -ejg-epg+ekg-eqi+ekn \\
\Delta &= -cdkm+arkd-glad+jfcd-jhad \\
&\quad -kfcd+khad+pfcd-phad+hjqd \\
&\quad -hkpd+bekm-bejf+bekf-bepf \\
&\quad -cfjq+cfkp+hajq-hakp+eajg \\
&\quad +eapg-eakg+eaqi-eakn-egjq+egpk \\
\varepsilon &= cdjqf-cdkpf+dkpha-djqha \\
&\quad -bejqf+bekpf+eagjq-eagpk.
\end{aligned}$$

Solutions to fifth-degree polynomials are generally not obtainable analytically. Consequently, to attain some insight into the effects of inbreeding, I have resorted to solving (12) numerically for the two models of altruism in Table 3 for different values of c, b, h and t. In all cases, the predictions of the local analysis at the fixation equilibria were checked by computer simulation of (5). Although a systematic survey was not conducted, for all cases encountered in which the two equilibria were unstable, there was a single stable interior equilibrium gene frequency. A similar point was made by CAVALLI-SFORZA and FELDMAN (1978) for random-mating populations.

In all cases studied for the additive model (Table 3a), decreasing t served to increase λ [(8), or the dominant root of (12)] and hence the evolutionary prospects of the altruistic gene (see legend of Figure 1). Hence, it appears that inbreeding *via* selfing or sib-mating facilitates the increase of such additive genes. However, for the multiplicative model, cases in which inbreeding decreased the evolutionary prospects of the gene were found. The case of zero individual fitness ($c = 1$) will be discussed in more detail shortly. Here, by increasing the frequency of inbred matings, the chance that the gene will increase when rare was decreased (*e.g.*, Figure 2). However, this effect of inbreeding changes as the cost of altruism decreases from $c = 1.0$ (total altruism). In Figure 1, the c/b ratio at which the maximum eigenvalue of (12) equals one, c/b^*, is given as a function of inbreeding for various values of c and h in the multiplicative model. For the hypothetical gene to increase, its cost-benefit ratio must be less than c/b^*. As shown in Figure 1 for $c = 0.75$, t has a variety of effects that depend upon h, the degree of dominance. For lower values of h, decreasing t tends to decrease c/b^*. However, for higher penetrance ($h = 0.75$ and $h = 1.0$), decreasing t facilitates increase of the gene when rare. The (1,0,0,0,0) equilibrium was always unstable for $c = 0.75$ over any reasonable range of benefits. However for $c = 0.25$, sib mating always acts to facilitate the initial evolutionary prospects of the altruist gene. Dominance has little effect on the qualitative shape of the curve in this case ($c = 0.25$). Although not presented in Figure 1, similar curves were generated over a range of costs and dominance for the multiplicative model ($c = 0.90, 0.80, 0.75, 0.50, 0.25, 0.20, 0.10$; $h = 0.0$–1.0 in increments of .1). As expected, c/b^* increased as the costs decreased for a given h and t. For lower costs (*i.e.*, $c = 0.50, 0.25, 0.20, 0.10$), c/b^* increased with decreasing t. In addition, as c decreased over this range ($c < 0.50$), the convexity of the curves increased. Consequently, for lower costs, inbreeding facilitates altruism. The curves for $c = 0.90$, 0.80 were similar to those presented in Figure 1 for $c = 0.75$, although displaced slightly downward.

Total altruism: Consider the case of genes that, when expressed, have zero individual fitness. Although individuals expressing the gene do not reproduce, it is assumed that they do affect the fitness of their associates. Many examples of such traits exist in nature, for example, the sterile castes of insect societies (see also the discussion of the "sacrificial lamb" phenotype in TEMPLETON 1979). Although it is generally thought that the caste-determining mechanism is strongly environmental (WILSON 1971), there are some well-documented cases

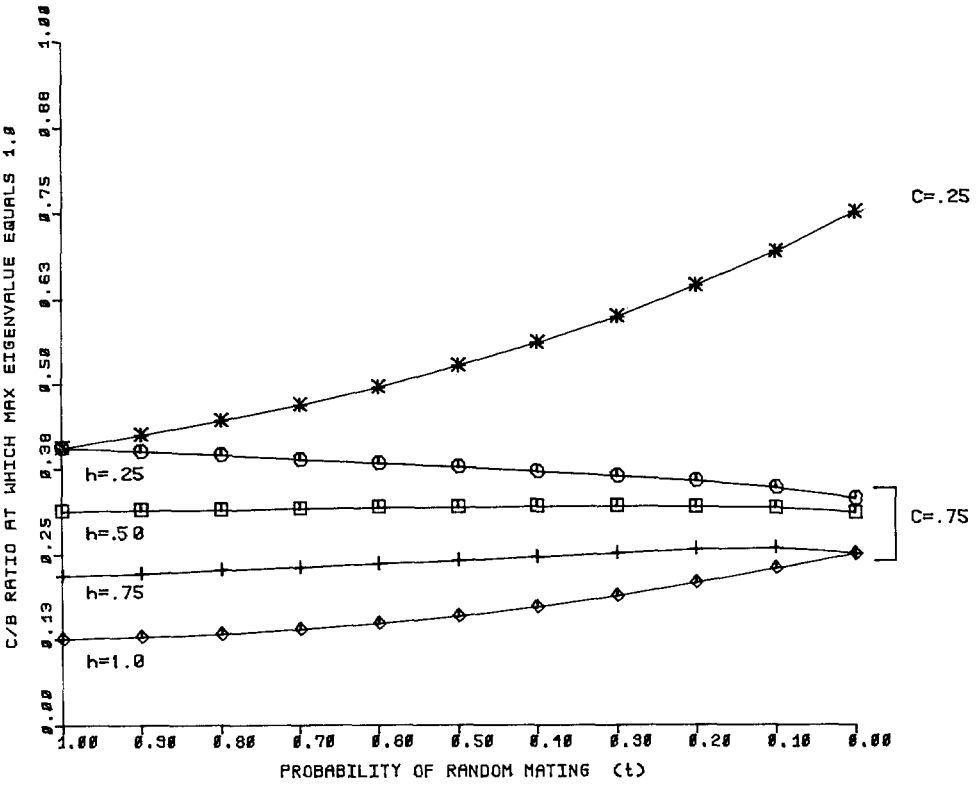

FIGURE 1.—c/b^* for the multiplicative model as a function of the level of outbreeding, t, for the mixed-sib mating system. The additive model (Table 3a) always generated curves similar in shape to the $c = 0.25$ curve given here, except that the "y intercept" ($t = 1.0$) was always $c/b^* = 0.50$. See text for explanation.

in which it is strongly genetic (*e.g.*, Melipona: KERR 1950, 1969). In addition, this case may have some relevance to the early stages of insect sociality. For example, consider a solitary wasp species and a mutant gene that confers either of the following behaviors on a female: associate with your sister and work for her offspring, or stay at home and work for your mother by helping to rear your sisters. In either case, the model is relevant. First, this female wasp would have zero individual fitness, but would increase the fitness of its sibling associates. Second, mutations are often recessive, as will be assumed shortly.

For such genes, we have $W_{1,m} = 0$ for $m = 1,2,4$ and consequently the only possible families are 4,5 and 6 (Table 1). In the case of the mixed-selfing mating system, equations (2) and (3) become

$$\begin{aligned} g'_1 &= 0 \\ g'_2 \overline{W} &= \tfrac{1}{2}[(1-t)g_2 + tg_2^2]W_{2,4} + tg_3g_2W_{2,5} \\ g'_3 &= 1 - g'_2 \end{aligned} \tag{13}$$

with

$$\overline{W} = [(1-t)g_2 + tg_2^2]\overline{W}_4 + 2tg_3g_2\overline{W}_5 + [(1-t)g_3 + tg_3^2]W_{3,6} \ .$$

In this case, it is possible to solve for the interior equibrium, $\hat{g}_2$. If the gene is recessive, we have $W_{2,4} = W_{3,4}$ and $W_{2,5} = W_{3,5} = W_{3,6} = 1.0$, the baseline fitness. In this case, $\hat{g}_2$ is given as the positive root of the following quadratic:

$$\hat{g}_2^2\, tA + \hat{g}_2[(1-t)A - tB] - (1-t)B = 0 \tag{14}$$

with

$$A = \tfrac{3}{4}W_{2,4} - 1 \text{ and } B = \tfrac{1}{2}W_{2,4} - 1 \ .$$

Solving (14) yields

$$\hat{g}_2 = \frac{\tfrac{1}{2}W_{2,4} - 1}{\tfrac{3}{4}W_{2,4} - 1} \ . \tag{15}$$

Consequently, as long as $W_{2,4} > 2$, there exists a unique interior equilibrium $\hat{g}_2$ given by (15). It is also possible to show by linearizing (13) at $\hat{g}_2$ that $\hat{g}_2$ is locally stable whenever it exists. Note that this equilibrium does *not* depend upon the amount of selfing, hence upon the amount of inbreeding, and it is identical to that attained under pure selfing ($t = 0$). It is also possible to show (MICHOD, submitted for publication) that (15) holds in the case of random mating [*i.e.*, $t = 1$ in (2)], and that in this case it is globally stable. In addition, this equilibrium is attained under the mixed-sib mating system.

For pure sib-mating, the dominant root of (6) becomes

$$\lambda = 1/4 + 1/6W_{2,4} + 1/2[1/4 + 1/9W_{2,4}^2]^{1/2} \ .$$

The corresponding eigenvector is

$$\mathbf{V} = \begin{matrix} 0 \\ 0 \\ 0 \\ 1 \\ W_{2,4}/3(\lambda - \tfrac{1}{2}) \\ \dfrac{W_{2,4}}{12(\lambda-1)} + \dfrac{W_{2,4}}{12(\lambda-1)(\lambda-\tfrac{1}{2})} \end{matrix} \ ,$$

which, after some algebra, yields the equilibrium adult genotypic frequencies given in (15). I have not been able to solve explicitly for the internal equilibria of the mixed-sib mating system [(5) with $0<t<1$] even in this special case ($W_{1,m}=0$, A_1 recessive). By using the multiplicative model with $c = 1.0$, A_1A_1 individuals have zero fitness in all families. This is not normally possible with the additive model, although, if the A_1 homozygotes are forced to have zero fitness, the fitnesses of the remaining genotypes are identical under either the additive or multiplicative model (A_1 recessive; see Table 3). Using the multiplicative model with $c = 1.0$, I have verified by simulation of (5) that $\hat{g}_2$ (15) is attained under the mixed-sib mating system for various t. In these simulations, the family frequencies were not the same at equilibrium; however, the adult

genotypic frequencies were identical and independent of t for the many cases studied. The rate of approach to this equilibrium, however, was sensitive to t and increased with increasing inbreeding under both the mixed-sib and mixed-selfing mating system.

Consequently, the mating system appears to exert no important effect on the evolutionary outcome of these recessive genes. It is important to note that this conclusion appears to be independent of the particular model of altruism (for example, those considered in Table 3) and that it holds for any recessive gene with zero individual fitness, as long as heterozygotes and the other homozygotes have higher fitness when in association with individuals homozygous for the gene. While the term "altruistic" captures these properties, there is nothing in the model at this point to make it explicit. Furthermore, there is nothing explicit that restricts these properties to the effects of behavior, although it is natural to interpret them in that light.

We now turn to consider the fixation equilibria for these genes. It is obvious that we need not concern ourselves with fixation of the A_1 allele, since genes with zero individual fitness could never reach fixation. In the case of mixed-selfing and arbitrary dominance, equation (8a) becomes

$$\lambda = W_{3,6}^{-1} \{tW_{2,5} + \tfrac{1}{2}(1-t)W_{2,4}\} . \tag{16}$$

Applying (16) to the case just discussed, a recessive gene will increase in frequency when rare if $W_{2,4} > 2$, which is the same condition required above for there to be a stable, interior equilibrium. To determine how inbreeding *via* selfing affects the condition for increase when rare, differentiate (16) to obtain

$$\frac{d\lambda}{dt} = W_{3,6}^{-1} \{W_{2,5} - \tfrac{1}{2}W_{2,4}\} , \tag{17}$$

which is positve as long as $\frac{1}{2}W_{2,4} < W_{2,5}$. Consequently, if $\frac{1}{2}W_{2,4} < W_{2,5}$, inbreeding (as a result of selfing) actually decreases the evolutionary prospects for the altruistic gene. Simulations of the multiplicative model with $c = 1.0$ and partial penetrance (*i.e.*, $W_{1,m}=0$ and $0<h<1$) indicate that if selfing decreases λ, it also decreases the equilibrium frequency ultimately reached by the gene.

In retrospect, all this makes sense. As t decreases (inbreeding increases), the frequency of the homogenotypic matings increase at the expense of heterogenotypic matings (equation [2]). Consequently, inbreeding will increase families 4 and 6, while decreasing the frequency of family 5 (everything else being equal). The effect of this shift in family frequencies (due to inbreeding) on the evolution of the social gene will depend upon how well the gene does in the context of the two families that carry the gene, $W_{2,4}$ and $W_{2,5}$, as (17) indicates. Consequently, the important effect of inbreeding on sociality lies in its altering the genotypic distribution of the interactions by changing the family frequencies.

The exact effect of inbreeding on the genotypic distribution of interactions in the case of the mixed-sib mating system is more difficult to tease out analytically. However, the net result appears to be the same. Using the multiplicative model (Table 3b) with $c = 1$ and $0 < h < 1$, the benefit (b^*) was found at which

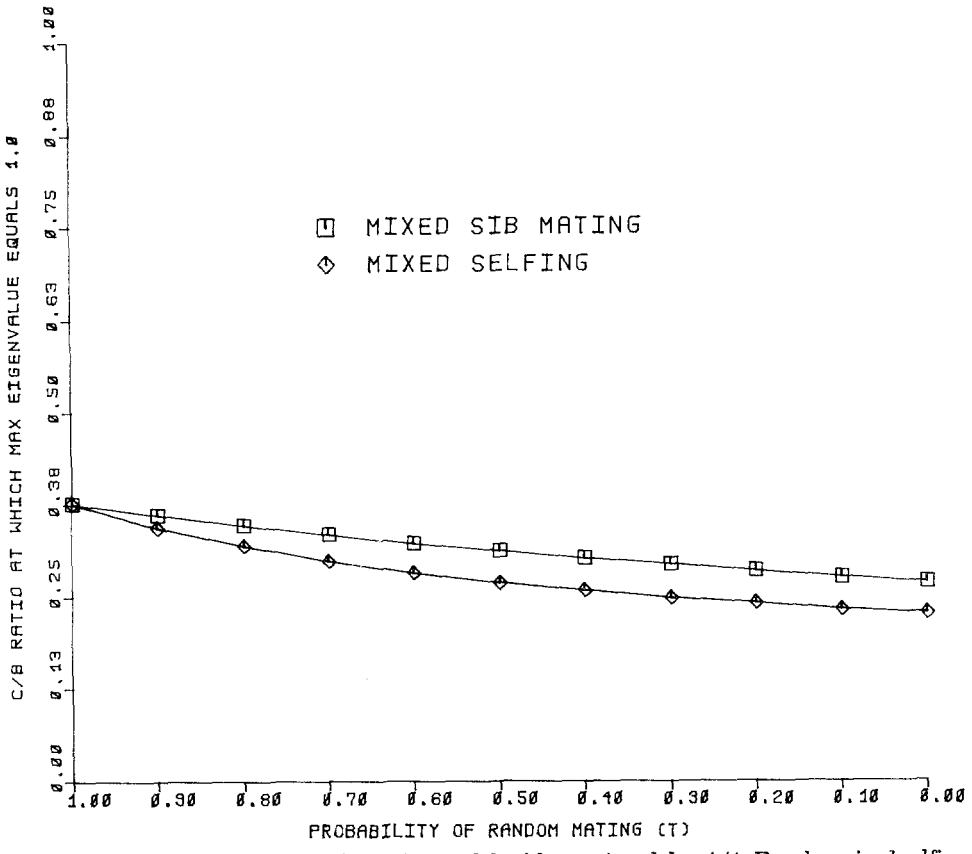

FIGURE 2.—c/b^* for the multiplicative model with $c = 1$ and $h = 1/4$. For the mixed-selfing system, using equation (16), b^* was found to be $b^* = [(1 - h)^{-1} - \frac{1}{2}(1 + t)]\,[\frac{1}{4}h(1 + t) + \frac{1}{8}(1 - t)]^{-1}$. For the mixed-sib system, b^* was obtained from equation (12) as explained in text.

the dominant root of (12) equals unity for the (0,0,0,0,1) equilibrium (Table 5). Again, in the case of recessive genes (h=0), no effect of t on c/b^* was found. For partial penetrance, inbreeding may decrease the ability of the gene to increase when rare. In Figure 2, the case of $h = 0.25$ is presented for both mating systems. For either mating system, inbreeding (decreasing t) decreases c/b^*. In addition, c/b^* is lower for the mixed-selfing system than for the mixed-sib mating system.

DISCUSSION

Some of the results derived in the last section appear to contradict the hypothesized effects of inbreeding discussed in the sociobiological literature. To assess the expected effects of the mating system according to current tenets of sociobiology, we need to know how each system affects the relatedness between interacting individuals.

Following the procedures of WRIGHT (1965), it is possible to analyze the genetic structure of specified mating systems by F statistics. The total population at any point in time is divided into families of six genotypic classes. Since the context of selection is the same in families with the same parental genotypes, there are six subpopulations, S (S=1,2, ... ,6), in the family-structured model (Table 1). F_{IS} is defined as the correlation between alleles in an individual of family S relative to alleles picked at random from the family, F_{ST} is the correlation between alleels picked at random from a family relative to alleles from the total population, and F_{IT} is the correlation between alleles in an individual relative to alleles picked at random from the total population. F_{IS} can be calculated either by the statistical method of WRIGHT (1965) or from the deviation of the observed heterozygote frequencies, H_S, within families,

$$F_{IS} = 1 - \frac{H_S}{2p_S(1-p_S)} \quad . \tag{18}$$

Consider, for example, family 2 in which the frequency of the altruistic allele is $p_2 = 3/4$ with $H_2 = 1/2$. Consequently, by (18) $F_{I2} = -1/3$. Likewise, $F_{I1} = F_{I6} = F_{I4} = 0.0$, $F_{I2} = F_{I5} = -1/3$ and $F_{I3} = -1.0$. Consequently, in a family-structured population, alleles in individuals are equally or less correlated than if they were randomly combined in a population with the same gene frequencies as the families. As usual, F_{ST} is calculated as

$$F_{ST} = \frac{\sigma_p^2}{\bar{p}(1-\bar{p})} \quad , \tag{19}$$

where σ_p^2 is the variance in gene frequency among families and $\bar{p}$ is the average frequency of the altruistic gene at time t. Using the relation

$$F_{IT} = F_{ST} + \bar{F}_{IS}\,(1-F_{ST}) \, , \tag{20}$$

where $\bar{F}_{IS}$ is the average of F_{IS} and F_{ST} is given by (19), the F statistics can be calculated each generation. This allows one to analyze the interrelationships of selection, the mating system and genetic relatedness.

A measure of relatedness between individuals X and Y who interact within subpopulations is (HAMILTON 1971)

$$b_{XY} = \frac{2F_{ST}}{1+F_{IT}} \quad . \tag{21}$$

HAMILTON (1971) cautioned against using a single measure of relatedness for studying evolution in structured populations. However, as he suggested, b_{XY} provides some assessment of relatedness in structured populations for use in discussions of kin selection. In Figure 3, b_{XY} is given for the two mating systems and several values of t. These curves apply only to neutral genes. Based on these considerations of the effects of the mating system on relatedness, it is expected that inbreeding should always facilitate the evolution of altruism.

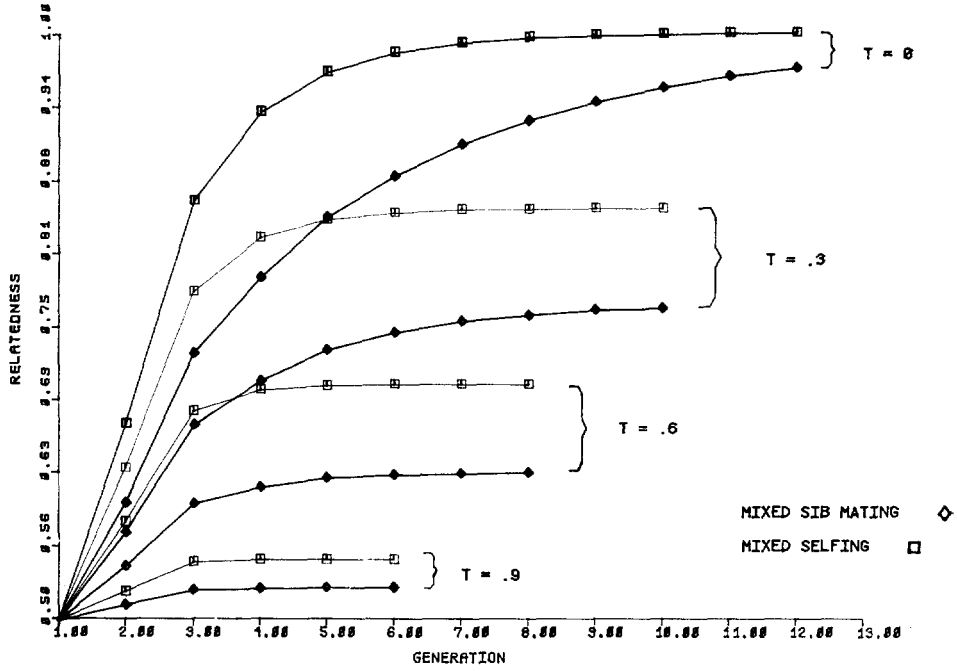

FIGURE 3.—Relatedness (Equation 21) for the two mixed mating systems and various outbreeding rates, t. These curves are for neutral genes only and measure the between group variance on which social evolution depends. See text for explanation.

The reason for this is understandable. Recent work has clarified the population processes underlying kin selection in family structured populations (WADE 1979, 1980; MICHOD, submitted for publication). A central result of this work is that the process of kin selection between sibs relies upon the differential production of family groups. The efficacy of this group-selection process (and hence kin selection) depends upon the variance in gene frequency between families, which is measured by F_{ST} (19). As mentioned in the introduction, inbreeding may be expected to increase this variance, and this increase is directly responsible [through (19) and (21)] for the increases in the overall relatedness within families given in Figure 3. This increase in the between-group variance results in increased selection at the group level and hence should facilitate altruism. This theme is pursued more explicitly by BREDEN and WADE (submitted for publication), where the various within- and between-family components of variance are studied in the mixed-sib mating system for certain cases of the additive model of altruism. BREDEN and WADE (submitted for publication) show that inbreeding facilitates the increase of altruistic genes by increasing the variance between families, but decreasing it within families. Since altruism is always selected against within families, but favored by between-family selection, the evolutionary prospects of altruism are enhanced by inbreeding.

The results of the last section are consistent with these considerations of

between- and within-group variance, but serve to emphasize the complicating effects of selection. For those genes with low or zero individual fitness ($W_{1,m} \simeq 0$), only families 4,5 and 6 exist to pass on the gene. The variance between families is thus severely constrained, since the families with high frequencies of the A_1 allele, 1 and 2 ($p_1 = 1.0$, $p_2 = 0.75$), are nonexistent. This was reflected in simulations of the multiplicative model for $c = 1.0$, in which F_{ST} rarely exceeded 0.3 and b_{XY} often dropped below 0.5, the value for outbreeding populations. In the case of mixed selfing, inbreeding increases the frequencies of families 4 and 6 at the expense of family 5 [see equation (2)]. This will tend to increase the between-group variance, because families 4 and 6 have the extreme gene frequencies in the case of total altruism (families 1, 2 and 3 are nonexistent; $p_4 = 0.5$, $p_5 = 0.25$, $p_6 = 0.0$). However, for total altruism, only heterozygotes can pass on the altruistic allele and inbreeding increases the frequency of altruistic homozygotes by increasing family 4. The net effect of these considerations will depend upon how well the heterozygotes do in the context of the two families that can pass on the gene, $W_{2,4}$ and $W_{2,5}$, as (17) indicates. The point to stress is that inbreeding alters the genotypic distribution of the interactions by changing family frequencies. The case of total altruism may be extreme, but it serves to illustrate the interaction of selection with the mating system in determining the final effect of this alteration on sociality.

The results of the last section (Figures 1 and 2), whereby inbreeding retards sociality, rely on the altruist having a low overall fitness. As the altruist's fitness is increased for the multiplicative model, inbreeding serves to facilitate sociality (Figure 1). These results are consistent with the results of the additive model and those of BREDEN and WADE (submitted for publication), in which inbreeding always facilitates altruism. For the additive model, it is not naturally possible to study extreme forms of altruism in which the altruist has a low fitness in all families. In both models, if a large cost is assumed ($c \simeq 1.0$), large benefits are required for the gene to increase. However, in the additive model these large costs and benefits average out, leaving the altruist with an overall fitness that is intermediate. This effect is reversed in the multiplicative model, where high costs have a more pronounced affect, even if the benefits are high.

In conclusion, inbreeding will often serve to facilitate sociality by increasing the between-group variance and within-group relatedness, so necessary for the evolution of social traits. This effect may be overcome for certain extreme social traits, such as total altruism. In this case, inbreeding may actually retard sociality by decreasing the frequency of the heterozygous class that can pass on the gene. In either circumstance, the important effect of inbreeding is altering the genotypic distribution of the interactions, which can only be understood once the mating system is specified.

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LITERATURE CITED

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