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Kin Selection and the Evolution of Monogamy

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A two-locus genetic model is studied in which one locus controls the tendency of individuals to act altruistically toward siblings and the other locus controls the mating habits of females. It is demonstrated that genetic variation at the altruism locus is often sufficient to induce an increase in the frequency of genes that cause females to produce all of their offspring with a single mate. This occurs because of nonrandom associations that develop between genes that cause altruism and those that affect female mating behavior. The results provide a new explanation for the evolution of monogamy, and they suggest a previously unexplored mechanism for the evolution of a variety of other behavioral traits as well.

RECENT THEORETICAL WORK ON the evolution of altruism between relatives has shown, in general, that altruism between closely related kin is more likely to evolve than altruism between distantly related kin (*1, 2*). This trend motivates the present work, in which we examine the evolution of monogamy, a trait that can control the relatedness of interacting kin. The mating behavior of numerous mammals, birds, fishes, and crustaceans has been described as monogamous, and monogamy has most often been explained by supposed needs for biparental investment in young, although other explanations have been offered as well (*3–7*). We analyze here a mathematical model and show that if there is genetically based variation in the propensity to be altruistic toward siblings, then monogamy may increase in frequency even when there is no paternal care and when maternal care ends at parturition.

Our model distinguishes two kinds of adult females: those that mate monogamously and those that mate polygamously. Monogamous females are assumed to choose a mate at random and then produce all of their offspring with that mate. By contrast, polygamous females are assumed to produce each of their offspring with a different randomly selected mate. All of the offspring produced by a female are deposited into what we call an "offspring group." Monogamous females produce offspring groups consisting entirely of full sibs, whereas polygamous mothers produce offspring groups consisting entirely of half sibs. Without affecting the results reported here, the mates of monogamous mothers may be assumed either to practice monogamy or to remain available to be chosen by other females. Thus, the results apply to a broader class of mating systems than those usually described as monogamous.

The hypothetical population is diploid with discrete generations. Genetic variation is allowed at two autosomal loci, each of which has two alleles. One locus controls altruism, and the other controls monogamy. The alleles at the altruism locus are called A and a, and the alleles at the monogamy locus are called M and m. There are ten possible genotypes, enumerated as follows:

AA	Aa	aa	AA	Aa
mm	mm	mm	Mm	Mm
1	2	3	4	5

Aa	aa	AA	Aa	aa
mM	Mm	MM	MM	MM
6	7	8	9	10

The rate of recombination between the loci is denoted by r, and the frequency of the ith genotype immediately before mating and reproduction is denoted by u_i. The frequency of A is called p_A, and the frequency of M called p_M.

$$p_A = u_1 + u_4 + u_8 + 1/2(u_2 + u_5 + u_6 + u_9)$$

and

$$p_M = 1/2(u_4 + u_5 + u_6 + u_7) + u_8 + u_9 + u_{10}$$

Mothers with the mm genotype are assumed to be polygamous. Mothers with the Mm and MM genotypes are monogamous with probabilities g_1 and g_2, respectively, and are polygamous with probabilities $1 - g_1$ and $1 - g_2$, respectively. The monogamy locus is not expressed in males. For convenience, we assume that $g_1 > 0$ and that the population size and the number of offspring produced per female are sufficiently large to allow us to ignore stochastic effects. We assume further that the two sexes are produced in equal numbers.

Individuals with genotypes AA, Aa, and aa at the altruism locus are altruists with probabilities h_1, h_2, and h_3, respectively (*8*). We assume that h_2 lies between h_1 and h_3. Without loss of generality, this may be written:

$$h_1 > h_3 \quad \text{and} \quad h_1 \geq h_2 \geq h_3 \qquad (1)$$

Let w be a quantity that is proportional to the probability that a given juvenile will survive to reproductive age. We use the additive formulation common in kin selection models (*1, 9*) and assume that w may be expressed as

$$w = 1 - \delta\gamma + z\beta$$

where z is the frequency of altruists in the juvenile's offspring group, and δ equals 1 if the juvenile is an altruist and equals 0 if the juvenile is not an altruist. The parameters β and γ are positive constants that represent, respectively, the benefits and costs of altruism. We assume $\gamma \leq 1$.

Let us first examine the case where the allele m is fixed, and thus the population is entirely polygamous ($u_1 + u_2 + u_3 = 1$, $u_i = 0$ for $i > 3$). With methods similar to those of Uyenoyama and Feldman (*8*), it may be shown that when m is fixed, both fixation equilibria ($p_A = 0$ or $p_A = 1$) and polymorphic equilibria ($0 < p_A < 1$) are possible at the altruism locus. Numerical and analytic work indicates that both types of equilibrium can be stable. Additive genetic variance is preserved at polymorphic equilibria.

Next, we ask whether a population that is fixed for m and that has achieved a stable equilibrium at the altruism locus can be successfully invaded by M. It is simple to show that p_M will neither increase nor decrease from any initial value if the population is fixed on A or a. This is not surprising because, when A or a is fixed, there are no potential sources of differential fitness among the genotypes that have nonzero frequencies.

In contrast to the result for fixation equilibria, when the initial equilibrium at the altruism locus is polymorphic, M will always increase to a nonnegligible frequency after invasion (*10*). On intuitive grounds, it seems likely that these successful invasions by M will lead to a subsequent increase in p_A. Although analytic proof of this point has eluded us, an extensive numerical study provides strong support for the idea. Parameters were generated randomly for each of 10,000 computer trials. During each trial,

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the model's recursion equations were iterated for 100 generations. The initial genotype frequencies for each trial were chosen to be in the neighborhood of a stable polymorphic equilibrium. In every case, the final values of both p_M and p_A exceeded their initial values. Additional details concerning this numerical study are presented in (*11*), along with details of the numerical studies that will be described next.

Stable polymorphic equilibria are, by definition, long lasting. Thus, they are attractive candidates for maintaining the variation at the altruism locus that is required for change in gene frequency at the monogamy locus. For wide ranges of the parameters, however, the polymorphic equilibria may not exist. Furthermore, substantial variation at the altruism locus can occur even when the population has not achieved a polymorphic equilibrium. This is the case, for example, when the value of p_A is changing over time either because of a recent successful invasion by A or a or because a polymorphic equilibrium has been disrupted by an environmentally imposed change in the value of β or γ. Although this transient genetic variation may be short-lived if one of the alleles quickly approaches fixation, successive transitions in gene frequency at the altruism locus may collectively produce large changes in gene frequency at the monogamy locus. With these considerations in mind, we carried out a second numerical study consisting of 10,000 trials in which the initial frequencies of A and M were independently chosen at random from the interval (0, 1). We chose β from the interval (0, 10) and γ from the interval (0, 1). The other parameters of the model were also chosen at random, and each trial went on for 100 generations. In 8,448 of the trials, p_A increased (that is, the final value of p_A exceeded its initial value); p_M also increased during each of these trials. During the remaining 1,552 trials p_A decreased, and p_M increased during 875 of these trials and decreased during 677.

The median value of the ratio of the benefits to the costs of altruism (β/γ) for all 10,000 trials was 9.891. For those trials during which p_A increased, the median of the ratios was 11.783 (range 2.181 to 32,927.752). For those trials during which p_A decreased, the median of the ratios was 1.563 (range 0.001 to 3.993). The value of p_M increased whenever β/γ was greater than 1.880 and decreased whenever β/γ was less than 0.153. When β/γ was between 0.153 and 1.880, p_M increased in some cases and decreased in others. Thus, the results of this numerical study suggest that high values of β/γ favor increases in p_A and p_M, whereas low values of β/γ favor decreases in these gene frequencies. Furthermore, it appears that a decrease in p_A is necessary but not sufficient to produce a concurrent decrease in p_M.

In 194 of the trials the value of r was less than 0.01. Although p_A decreased during 27 of these trials, p_M decreased in only two cases. Furthermore, p_M never decreased when r was less than 0.007. These observations led us to conjecture that p_M will always increase so long as r is sufficiently small. To further test this idea, we chose new parameters for 10,000 additional trials in the same way as for the trials just decribed, except that we set $r = 0$ in every case. Although p_A decreased during 1,573 of these trials, p_M increased in every case. This supports our hypothesis that low values of r will allow p_M to increase even when p_A decreases.

In summary, it appears that when there is genetic variance at the altruism locus, the frequency of the M allele will always increase unless p_A is decreasing, β/γ is small, and linkage is loose. It is possible to produce an intuitive explanation for these increases which, though not accurate in every detail, at least captures the spirit of the underlying processes. Individuals that carry M are often born to monogamous mothers and thus interact with full siblings. As a consequence, they are more likely to be phenotypically similar to other members of their offspring group than are individuals that do not carry M. This means that altruists that carry M benefit from the presence of other altruists in their offspring group to a greater degree than altruists that do not carry M, whereas nonaltruists that carry M suffer from the absence of other altruists to a greater extent than nonaltruists that do not carry M. As a result of these differences, an association develops such that individuals that carry M are more likely to be altruists than individuals that do not carry M. Because this means that M tends to occur in individuals that benefit from altruism to a greater degree than other individuals in the population, p_M tends to increase.

Evolutionary processes similar to the one described here may operate in a number of different circumstances, as, for example, when genetic variance in altruism is maintained by migration or mutation. A similar process may influence the evolution of traits other than monogamy if those traits also affect between-individual similarity. Assortative mating and the tendency to inbreed are examples of such traits, and, in a structured population, phenotypic similarity within demes may be enhanced by any trait that decreases effective deme size. Effective deme size is lowered, for example, when individuals act to repel potential immigrants or when certain males are preferred as mates by most females.

Our study may be useful in understanding those monogamous species in which there is apparently no paternal care (*6, 7*). In addition, for those species in which paternal care and monogamy co-occur, our results suggest the possibility that monogamy arose first and that paternal care evolved only when monogamy had produced a decrease in uncertainty concerning the parentage of offspring. This scenario may be of special interest to students of human origins in light of Lovejoy's assertions that a lengthening of childhood (which might intensify sib-sib interactions) along with the development of monogamy and paternal care were crucial steps in the evolutionary process that led to modern *Homo sapiens* (*12*).

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10. The proof that M will increase when rare if the altruism locus is at a polymorphic equilibrium is achieved by showing that when the altruism locus is at a polymorphic equilibrium and m is fixed, the matrix governing invasions by M has at least one eigenvalue in excess of unity. We begin by specifying the full recursion equations that describe the system. Four types of gametes are produced during mating:

$$\begin{array}{cccc} Am & am & AM & aM \\ 1 & 2 & 3 & 4 \end{array}$$

Let x_j be the frequency of the jth gamete. We have:

$$x_1 = u_1 + \tfrac{1}{2}[u_2 + u_4 + ru_5 + (1 - r)u_6]$$
$$x_2 = u_3 + \tfrac{1}{2}[u_2 + (1 - r)u_5 + ru_6 + u_7]$$
$$x_3 = u_8 + \tfrac{1}{2}[u_4 + (1 - r)u_5 + ru_6 + u_9]$$
$$x_4 = u_{10} + \tfrac{1}{2}[ru_5 + (1 - r)u_6 + u_7 + u_9]$$

Since both monogamous and polygamous matings are random, we can write:

$$\tilde{u}_1 = x_1^2 \quad \tilde{u}_2 = 2x_1x_2 \quad \tilde{u}_3 = x_2^2 \quad \tilde{u}_4 = 2x_1x_3$$
$$\tilde{u}_5 = 2x_2x_3 \quad \tilde{u}_6 = 2x_1x_4 \quad \tilde{u}_7 = 2x_2x_4$$
$$\tilde{u}_8 = x_3^2 \quad \tilde{u}_9 = 2x_3x_4 \quad \tilde{u}_{10} = x_4^2$$

where $\tilde{u}_i$ is the frequency of the ith genotype immediately after birth and before sib-sib interactions. If we denote the value of u_i in the next generation as u_i' and let w_i represent the expected value of w for newborns with the ith genotype, then the basic recursion equations may be written as:

$$u_i' = \frac{\tilde{u}_i w_i}{\bar{w}} \qquad (2)$$

where

$$\bar{w} = \sum_{i=1}^{10} \tilde{u}_i w_i$$

We complete our specification of the recursion equations by describing how the values of w_i are derived. We can discriminate 80 types of offspring groups. Ten of these we produced by a polygamous

mother having one of the ten genotypes. The frequency of these offspring groups is simply the frequency of the mother's genotype times the probability that a female of that genotype will be polygamous (1, $1 - g_1$, and $1 - g_2$ for *mm*, *mM*, and *MM* females, respectively). Each of the remaining 70 offspring groups are produced by a monogamous mating between a female with one of the seven genotypes capable of monogamy and a male having one of the ten genotypes. The frequency of each of these offspring groups equals the product of the frequencies of the two genotypes involved in the mating times the probability that the female is monogamous (g_1 and g_2 for *mM* and *MM* mothers, respectively). Let us number the offspring groups in arbitrary order from 1 to 80, and let f_j be the frequency of offspring groups of the jth type. Let $y_{i,j}$ be the frequency of the ith genotype in the jth offspring group. The $y_{i,j}$ values are derived in the usual way from knowledge of the parents' genotypes and the value of r. Finally, let z_j denote the frequency of altruists in the jth type of offspring group [$z_j = h_1(y_{1,j} + y_{4,j} + y_{8,j}) + h_2(y_{2,j} + y_{5,j} + y_{6,j} + y_{9,j}) + h_3(y_{3,j} + y_{7,j} + y_{10,j})$]. We may now write:

$$w_i = 1 - h_k\gamma + \left(\frac{\sum_{j=1}^{j=80} f_j y_{i,j} z_j}{\sum_{j=1}^{j=80} f_j y_{i,j}}\right)\beta$$

$$= 1 - h_k\gamma + \left(\frac{\sum_{j=1}^{j=80} f_j y_{i,j} z_j}{\bar{u}_i}\right)\beta$$

where $k = 1$, 2, and 3, respectively, for genotypes with *AA*, *Aa*, and *aa* at the altruism locus.

When m is fixed, the recursions specified by Eq. 2 become a two-dimensional system and the frequency of A at polymorphic equilibria ($\hat{p}_A$) must satisfy:

$$0 = (h_1\hat{p}_A^2 + 2h_2\hat{p}_A\hat{q}_A + h_3\hat{q}_A^2)(2\gamma - \beta)^2 - \beta\gamma(h_1\hat{p}_A + h_3\hat{q}_A) + \beta - 4\gamma \quad (3)$$

where $\hat{q}_A = 1 - \hat{p}_A$. Furthermore, polymorphic equilibria cannot exist unless the following holds:

$$\beta > 2\gamma \quad (4)$$

We can use Eq. 2 to derive linearized recursions for the frequencies of the *Mm* genotypes in the vicinity of an equilibrium at which m is fixed and the altruism locus is polymorphic. In matrix form, the linearized recursions are:

$$\begin{bmatrix} \epsilon_4' \\ \epsilon_5' \\ \epsilon_6' \\ \epsilon_7' \end{bmatrix} = \begin{bmatrix} c_1(1-r)c_2 & rc_2 & 0 \\ c_3(1-r)c_4 & rc_4 & 0 \\ 0 & rc_5 & (1-r)c_5\ c_6 \\ 0 & rc_7 & (1-r)c_7\ c_8 \end{bmatrix} \begin{bmatrix} \epsilon_4 \\ \epsilon_5 \\ \epsilon_6 \\ \epsilon_7 \end{bmatrix} \quad (5)$$

where $\epsilon_i = u_i$ and $\epsilon_i' = u_i'$ in the vicinity of the equilibrium. The c_i values are positive constants that depend on h_1, h_2, h_3, β, γ, and the particular root of Eq. 3 chosen as $\hat{p}_A$.

Let B be the 4×4 matrix in Eq. 5. When $r = 0$, B breaks down into two 2×2 submatrices. Under the requirements of inequalities 1 and 4, each of these submatrices may be shown to have its largest eigenvalue greater than unity, so that the full 4×4 stability matrix has two eigenvalues greater than unity when $r = 0$.

Let $d(\lambda)$ represent the characteristic polynomial of B:

$$d(\lambda) = \det[B - \lambda I]$$

where I is a 4×4 identity matrix. It is possible to show that $d(1)$ is linear in r, and using this fact, along with a continuity argument, the Perron-Frobenius theorem, and the above results for $r = 0$, one can demonstrate that B has two distinct and real eigenvalues in excess of unity for r small and positive. Thus, for B to have no eigenvalue in excess of unity for some value of r, $d(1) = 0$ must hold for at least two values of r. However, this is impossible in light of the linearity of $d(1)$, and thus B has at least one eigenvalue in excess of unity for all r.

11. We made all selections of random numbers in the numerical studies by using a nonlinear additive feedback random number generator. In the first study (invasions by M when the altruism locus is polymorphic) possible values for h_1, h_2, h_3, g_1, g_2, and γ were selected according to a uniform distribution on (0, 1); β was selected according to a uniform distribution on (0, 10), and r according to a uniform distribution on (0, ½). Each set of possible parameter values was tested for satisfaction of inequalities 1 and for satisfaction of $0 < g_1 \leq g_2$. The parameters were tested further to see whether they allowed for the existence of polymorphic equilibria (that is, Eq. 3 was tested for solutions satisfying $0 < \hat{p}_A < 1$). Parameter sets that passed all of these tests were used in the computer trials. The polymorphic equilibria always occur in pairs, and numerical evaluation of the associated eigenvalues suggests that one equilibrium is always stable while the other is unstable. We picked the stable polymorphic equilibrium to initialize the computer trials.

It is relatively simple to show that the frequencies of the first three genotypes at polymorphic equilibria when m is fixed are given by:

$$\hat{u}_1 = \frac{\hat{p}_A[\beta - \hat{q}_A(2\beta - 4\gamma)]}{\beta}$$

$$\hat{u}_2 = \frac{\hat{p}_A\hat{q}_A(4\beta - 8\gamma)}{\beta}$$

$$\hat{u}_3 = \frac{\hat{q}_A[\beta - \hat{p}_A(2\beta - 4\gamma)]}{\beta}$$

These expressions were used to calculate the initial genotype frequencies [$u_i(0)$], which are given by:

$$\begin{aligned} u_1(0) &= 0.998001\hat{u}_1(1 + \Upsilon_1)N \\ u_2(0) &= 0.998001\hat{u}_2(1 + \Upsilon_2)N \\ u_3(0) &= 0.998001\hat{u}_3(1 + \Upsilon_3)N \\ u_4(0) &= 0.001998\hat{u}_1(1 + \Upsilon_4)N \\ u_5(0) &= 0.000999\hat{u}_2(1 + \Upsilon_5)N \\ u_6(0) &= 0.000999\hat{u}_2(1 + \Upsilon_6)N \\ u_7(0) &= 0.001998\hat{u}_3(1 + \Upsilon_7)N \\ u_8(0) &= 0.000001\hat{u}_1(1 + \Upsilon_8)N \\ u_9(0) &= 0.000001\hat{u}_2(1 + \Upsilon_9)N \\ u_{10}(0) &= 0.000001\hat{u}_3(1 + \Upsilon_{10})N \end{aligned}$$

where the Υ_i values are independent random variables chosen anew for each computer trial from a uniform distribution on (0, 10^{-8}), and N is a normalizing factor calculated from the requirement that

$$1 = \sum_{i=1}^{10} u_i(0)$$

The use of these equations to choose the initial genotype frequencies assures that the initial values of p_M will approximate 0.001 and that the initial values of u_1, u_2, and u_3 will be only slightly perturbed from their equilibrium values. Furthermore, the initial values of D, the linkage disequilibrium coefficient, will be small but nonzero ($D = x_1x_4 - x_2x_3$).

In the second numerical study the parameters were chosen in the same manner as in the first study, except that no test for the existence of polymorphic equilibria was required. To assign the initial genotype frequencies [$u_i(0)$], we started by independently choosing two random variables, $p_A(0)$ and $p_M(0)$, from a uniform distribution on (0, 1). The following equations were then used to generate the u_i (0) values:

$$\begin{aligned} u_1(0) &= [p_A(0)q_M(0)]^2(1 + \Upsilon_1)N \\ u_2(0) &= 2p_A(0)q_A(0)\ [q_M(0)]^2(1 + \Upsilon_2)N \\ u_3(0) &= [q_A(0)q_M(0)]^2(1 + \Upsilon_3)N \\ u_4(0) &= 2[p_A(0)]^2p_M(0)q_M(0)(1 + \Upsilon_4)N \\ u_5(0) &= 2p_A(0)q_A(0)p_M(0)q_M(0)(1 + \Upsilon_5)N \\ u_6(0) &= 2p_A(0)q_A(0)p_M(0)q_M(0)(1 + \Upsilon_6)N \\ u_7(0) &= 2[q_A(0)]^2p_M(0)q_M(0)(1 + \Upsilon_7)N \\ u_8(0) &= [p_A(0)p_M(0)]^2(1 + \Upsilon_8)N \\ u_9(0) &= 2p_A(0)q_A(0)[p_M(0)]^2(1 + \Upsilon_9)N \\ u_{10}(0) &= [q_A(0)p_m(0)]^2(1 + \Upsilon_{10})N \end{aligned} \quad (6)$$

where $q_A(0) = 1 - p_A(0)$ and $q_M(0) = 1 - p_M(0)$; N and the Υ_i values are defined as in the first numerical study. This scheme assures that the initial values of D will be small and that the initial values of p_A and p_M will approximate $p_A(0)$ and $p_M(0)$, respectively.

The use of initial genotypic distributions that have small values of D allowed for the production of a simple and concisely communicable set of results in the numerical studies. However, a similar set of results can be obtained even when arbitrarily large initial deviations from the $D = 0$ surface are allowed. For example, we repeated the first numerical study using the following initial genotype frequencies:

$$\begin{aligned} u_1(0) &= 0.999\hat{u}_1 & u_2(0) &= 0.999\hat{u}_2 \\ u_3(0) &= 0.999\hat{u}_3 & u_4(0) &= \Upsilon_1N \\ u_5(0) &= \Upsilon_2N & u_6(0) &= \Upsilon_3N \\ u_7(0) &= \Upsilon_4N & u_8(0) &= \Upsilon_5N \\ u_9(0) &= \Upsilon_6N & u_{10}(0) &= \Upsilon_7N \end{aligned}$$

In this case the Υ_i values were chosen from a uniform distribution on (0, 1), and N is again a normalizing factor. This scheme assures that the initial values of p_M will not exceed 0.001 but otherwise leaves the starting values of D unconstrained. Although this allowed a variety of outcomes during the early generations of the computer trials, the effects of the initialization tended to "wash out" later on. Thus, although both p_A and p_M increased during the first generation in only 5,854 of the 10,000 trials, by generation 100 these two gene frequencies were increasing in all but 569 cases.

We also repeated the second numerical study using completely arbitrary initial genotype frequencies:

$$\begin{aligned} u_1(0) &= \Upsilon_1N & u_2(0) &= \Upsilon_2N & u_3(0) &= \Upsilon_3N \\ u_4(0) &= \Upsilon_4N & u_5(0) &= \Upsilon_5N & u_6(0) &= \Upsilon_6N \\ u_7(0) &= \Upsilon_7N & u_8(0) &= \Upsilon_8N & u_9(0) &= \Upsilon_9N \\ & & u_{10}(0) &= \Upsilon_{10}N & & \end{aligned}$$

where the Υ_i values are again chosen from a uniform distribution on (0, 1), and N is a normalizing factor. Here also the effects of the initial conditions were generally short-lived and eventually the gene frequencies behaved in a manner analogous to the results obtained when Eqs. 6 were used to assign the initial genotype frequencies. For example, in 8,504 of the trials p_A was increasing by generation 100, and in all but 186 of these trials the same was true of p_M.

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Technical Comments

PCB Dechlorination in Hudson River Sediment

The report "Polychlorinated biphenyl dechlorination in aquatic sediments" by John F. Brown *et al.* (*1*) purports to show that polychlorinated biphenyls (PCBs) in Hudson River sediments are undergoing dechlorination and detoxification by anaerobic microbes. The postulation of anaerobic activity is based on the departure of the PCB conge-