

features of individual marks alone provide insufficient evidence for tool use versus trampling. If such evidence is combined with criteria based on context, pattern of multiple marks and placement on bones, however, it should be possible to distinguish the two processes in at least some cases bearing on early human behaviour.

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Asymmetry in the evolution of female mating preferences

Jon Seger

Department of Biology, Princeton University, Princeton, New Jersey, 08544, USA

Robert Trivers

Institute of Marine Sciences, University of California, Santa Cruz, California 95064, USA

Trivers¹ has suggested that where genetic or developmental constraints on the expression of a trait prevent male and female fitnesses from being maximized simultaneously, female mating preferences should evolve to favour males who exhibit variants of the trait that confer relatively low fitness on males but relatively high fitness on females. This asymmetry is expected because alleles that affect mating preferences are expressed only in females, but are genetically correlated with alleles that differentially affect the fitnesses of the two sexes. Here we describe a two-locus population-genetic model that embodies this idea. The model's qualitative behaviour is exactly like that of previous models²⁻¹⁰ for the joint evolution of male traits and female mating preferences: evolution is equally likely to proceed in either direction along (or away from) a line of neutral equilibria that relates given frequencies of the preference alleles to corresponding frequencies of the trait alleles.

But there is a quantitative asymmetry, of the expected kind, in the shape of the line of equilibria. When we extend the model to include migration between partially isolated demes (breeding groups), 'selective diffusion'^{11,12} moves the demes along the line of equilibria in the direction that increases average female fitness while lowering average male fitness.

The model species is a sexual haploid with two unlinked loci and discrete, non-overlapping generations. Alleles at the trait locus (*T*) determine male and female viabilities according to the following scheme:

	T_1	T_2
Males	α_1	α_2
Females	β_1	β_2

In males, T_1 and T_2 also determine a phenotypic difference that is visible to females. At the beginning of each generation the frequencies of T_1 and T_2 are t_1 and $t_2 = 1 - t_1$. After viability selection, the frequencies of T_1 in adult males and females are

$$t_1^m = \alpha_1 t_1 / (\alpha_1 t_1 + \alpha_2 t_2) \tag{1}$$

and

$$t_1^f = \beta_1 t_1 / (\beta_1 t_1 + \beta_2 t_2) \tag{2}$$

with $t_2^m = 1 - t_1^m$ and $t_2^f = 1 - t_1^f$.

Female mating preferences are determined by alleles at the preference locus (*P*). Females carrying P_1 prefer to mate with T_1 males, and females carrying P_2 prefer to mate with T_2 males. There are many different behavioural models of female choice^{3-10,13,14}; we used two simple ones that give qualitatively different results in some contexts⁸. Under the fixed-relative-preference model^{6,8}, the probability that a P_1 female mates with a T_1 male is

$$U_{11} = a_1 t_1^m / (a_1 t_1^m + t_2^m) \tag{3}$$

where $a_1 (\geq 1)$ is a parameter that sets the strength of the female preference, which vanishes at $a_1 = 1$. The probability that a P_1 female mates with a T_2 male is $U_{12} = 1 - U_{11}$. The behavioural model is one in which P_1 females encounter T_1 and T_2 males sequentially, at random, accepting them as mates with fixed probabilities whose ratio is $a_1 : 1$. Similarly,

$$U_{22} = a_2 t_2^m / (a_2 t_2^m + t_1^m) \tag{4}$$

is the probability that a P_2 female mates with a T_2 male, and $U_{21} = 1 - U_{22}$ is the probability that she mates with a T_1 male.

Under the better-of-two model of female choice,

$$U_{11} = t_1^m + c_1 t_1^m t_2^m \tag{5}$$

and

$$U_{22} = t_2^m + c_2 t_1^m t_2^m \tag{6}$$

where c_1 and c_2 ($0 \leq c_i \leq 1$) are parameters that set the strength of the mating preference. Here the behavioural model is that of a lek. Each lek is composed of two males, sampled at random from the population after viability selection. If the lek contains one T_1 and one T_2 male, the female chooses her preferred type with probability $\frac{1}{2}(1 + c_i)$. Thus the preference vanishes at $c_i = 0$.

The frequencies of P_1 and P_2 are p_1 and $p_2 = 1 - p_1$, at the beginning of each generation. Although P_1 and P_2 have no effects on male or female viabilities, their frequencies change slightly in each sex during viability selection at the *T* locus, because of the genetic covariance (phase disequilibrium) that exists between the two loci. This covariance is maintained, even at gene-frequency equilibrium, by the female mating preferences²⁻¹⁰.

Given the assumptions and definitions described above, analysis of the model proceeds in a straightforward way, along

Fig. 1 Lines of stable equilibria for models in which only one sex is affected by T_1 and T_2 . **A**, Only males are affected. This is Kirkpatrick's⁶ model in which $\alpha_1 = \beta_1 = \beta_2 = 1$, $\alpha_2 = 0.6$, $a_1 = 1$ and $a_2 = 3$. **B**, Only females are affected. Here $\alpha_1 = \alpha_2 = \beta_1 = 1$, $\beta_2 = 0.6$, $a_1 = 1$ and $a_2 = 3$. T_1 goes to fixation when the frequency of P_1 is greater than 0.8 ($p_2 < 0.2$), but T_2 goes to fixation only when P_2 is also at fixation ($p_2 = 1$). If a_2 (the preference parameter) were larger than 3, or if β_2 (the fitness of T_2 females) were larger than 0.6, then the upper end of the line of equilibria would intersect the upper boundary ($t_2 = 1$) at $p_2 < 1$. Conversely, if a_2 were smaller than 3 or if β_2 were smaller than 0.6, then the line would intersect the right-hand boundary ($p_2 = 1$) at $t_2 < 1$, and T_2 would have an interior equilibrium even at fixation for P_2 . In all of the models discussed here, the interior equilibria satisfy the equation

$$p_2^f = \frac{t_2(2 - 1/F) - U_{12}}{U_{22} + U_{11} - 1} \quad (8)$$

where p_2^f is the frequency of P_2 in adult females and $F = \beta_1 t_1 + \beta_2 t_2$ is the average female fitness. Equation (8) was used to draw the lines of equilibria shown above and in Fig. 2. If female fitnesses are not affected by T_1 and T_2 , then $F = 1$, and $p_2^f = p_2$. But if the T locus is expressed in females ($\beta_1 < 1$ or $\beta_2 < 1$), then $F < 1$, and p_2^f is slightly different from p_2 . The change in the frequency of P_2 in females arises during viability selection at the T locus, owing to the phase disequilibrium (covariance) between the two loci. Thus, the line of equilibria shown in **A** (Kirkpatrick's model, in which $\beta_1 = \beta_2 = 1$) is exact, but the lines shown in **B** and Fig. 2 are approximate. (If the horizontal axes were relabelled as p_2^f , then all of the lines would be exact.) The error involved in this approximation (or misrepresentation) was estimated by numerical iteration of the recurrence equations in the genotype frequencies, which model the full dynamics of the two-locus system. Even with strong selection and strong female mating preferences, as in **B**, p_2^f differs from p_2 by only a few per cent for intermediate gene frequencies, becoming identical to p_2 at the boundaries. (The exact end points of the lines of equilibria can easily be derived analytically.) With weaker selection and weaker mating preferences, as in Fig. 2, the approximation is excellent everywhere.

the lines described more fully elsewhere^{6,8}. Additional details are given in the figure legends.

The asymmetry mentioned above can easily be demonstrated by comparing models in which the fitnesses ($\alpha_1, \alpha_2, \beta_1, \beta_2$) have been symmetrically rearranged. Consider the case (1, $\alpha_2, 1, 1$), with $\alpha_2 < 1$. Here females are unaffected by their T -locus genotypes, but T_2 males suffer reduced viability relative to T_1 males. Kirkpatrick's⁶ first model is this fitness scheme with fixed-relative-preference female choice, where $a_1 = 1$ and $a_2 > 1$ (P_1 females mate randomly, while P_2 females prefer the less viable T_2 males). Figure 1A shows the line of stable equilibria illustrated in Kirkpatrick's Fig. 1. If we reverse the pattern of sex-limitation, so that T_2 females suffer reduced viability, then the fitness array is (1, 1, 1, β_2) and we obtain the line of stable equilibria shown in Fig. 1B. Although T_2 invades at lower frequencies of P_2 when its deleterious effect is limited to females than it does when limited to males, much higher frequencies of P_2 are required to take it to fixation. Of course, T_2 can have any equilibrium frequency between 0 and 1, under either of these fitness schemes, given an appropriate frequency of P_2 . But under almost any probability distribution of \hat{p}_2 centred on $\hat{p}_2 = \frac{1}{2}$, the average equilibrium frequency of T_2 is higher when its

deleterious effect is limited to males than when the same deleterious effect is limited to females.

In a more fully symmetrical model, both T_1 and T_2 would affect the fitnesses of their carriers, and both P_1 and P_2 females would exhibit mating preferences. Consider the fitnesses (1, $\alpha_2, \beta_1, 1$). Here T_1 males are fitter than T_2 males, as in Kirkpatrick's model. But now T_2 females are fitter than T_1 females. If we set $\alpha_2 = \beta_1$, then the fitness effects are completely symmetrical, and in the absence of any female mating preferences, T_1 and T_2 have a strongly attracting equilibrium¹⁵⁻¹⁹ at $\hat{t}_1 = \hat{t}_2 = \frac{1}{2}$. If we also set $a_1 = a_2 > 1$, then P_1 and P_2 females have equally strong preferences for T_1 and T_2 males, respectively, and the model is fully symmetrical. But the line of stable equilibria is not symmetrical. T_2 is taken to high frequencies more easily than is T_1 (Fig. 2).

The interior equilibria are always stable under the fixed-relative-preference model of female choice^{6,8}, but they can be unstable under the better-of-two model⁸. Figure 3 shows a line of equilibria for the symmetrical fitnesses described above, under symmetrical better-of-two female choice ($c_1 = c_2 = 1$). Again the asymmetry is obvious, and is illustrated dramatically by the dynamical behaviour of a population that begins at the centre of the gene-frequency plane ($t_2 = p_2 = \frac{1}{2}$). The population evolves rapidly to a boundary equilibrium at $t_2 = 1, p_2 > \frac{1}{2}$ (Fig. 3).

The causes of the asymmetry are easy to see. P_2 females who choose T_2 males instead of T_1 males thereby increase the survival of their daughters. Of course, they also decrease the survival of their sons, so there would seem to be no net advantage associated with the preference for T_2 . But in the next generation it will be females, not males, who do the choosing, and allele P_2 increases its frequency in females by associating itself nonrandomly with T_2 .

The stable equilibria are points at which a population would sit forever, if affected only by the deterministic forces of natural and sexual selection. But in a world of finite, partially isolated demes, differential migration may give rise to a weak directional force that moves an average deme along the line of equilibria, towards higher frequencies of P_2 and T_2 . Consider a set of n demes, each of which has the same carrying capacity, K . The expected number of adult females in the i th deme is $KF_i/(F_i + M_i)$, where F_i and M_i are the average viabilities of females and males born in that deme. If males do not help females to rear offspring, then the productivity of each deme is proportional to its number of adult females, which is proportional to its frequency of T_2 , under the fitness scheme discussed above [(1, $\alpha_2, \beta_1, 1$) with $\alpha_2 = \beta_1$]. Let each deme contribute to the migrant pool in proportion to F_i , and let it then receive mK migrants from the pool. It follows that

$$\Delta \bar{t}_2 = \frac{m}{1+m} \left[\frac{(1-\beta_1) \text{var}(t_2)}{\beta_1 + (1-\beta_1)\bar{t}_2} \right] \quad (7)$$

For example, if $m = 0.01$, $\beta_1 = 0.8$, $\bar{t}_2 = 0.5$ and $\text{var}(t_2) = 0.01$, then $\Delta \bar{t}_2 = 2.2 \times 10^{-5}$. The overall frequency of T_2 increases because demes with higher than average frequencies of T_2 make larger than average contributions to the migrant pool, which

Fig. 2 Line of stable equilibria for a fully symmetrical version of Kirkpatrick's model. Here $\alpha_1 = \beta_2 = 1$, $\alpha_2 = \beta_1 = 0.8$ and $a_1 = a_2 = 1.05$. Note that the strengths of natural selection and female choice are weaker here than in the examples shown in Figs 1 and 3. T_1 and T_2 have an interior equilibrium at fixation for P_1 ($p_2 = 0$), but T_2 is taken to fixation when P_2 is above a frequency of 0.925. Qualitatively similar lines of equilibria exist even if $\alpha_2 \neq \beta_1$ and $a_1 \neq a_2$.

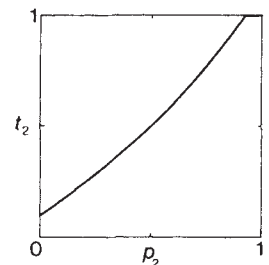
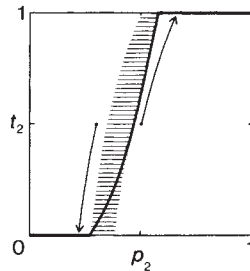


Fig. 3 Line of equilibria for the symmetrical model with better-of-two female choice. Here $\alpha_1 = \beta_2 = 1$, $\alpha_2 = \beta_1 = 0.6$ and $c_1 = c_2 = 1$. From points to the left of the line, populations move down and to the left. From points to the right of the line, they move up and to the right. From points within the hatched region, populations are attracted to stable interior equilibria on the lower region of the line (approximately, the region below $t_2 = 0.55$).



From points outside the hatched region, populations move to the stable boundary equilibria at $t_2 = 0$ or $t_2 = 1$. The illustrated trajectories begin at $p_2 = 0.3$, $t_2 = 0.5$ and at $p_2 = t_2 = 0.5$. Each of these trajectories is less than 100 generations in length. The upper region of the line (between approximately $t_2 = 0.55$ and $t_2 = 1$) is unstable. From points just to the right of the line, populations move to the boundary at $t_2 = 1$, and from points just to the left, they move to stable interior equilibria on the lower region of the line. The line of equilibria, the regions of attraction and the illustrated trajectories were all found numerically, by iteration of the recurrence equations in the genotype frequencies. The approximate line of equilibria (equation (8); see Fig. 1 legend) has the same end points as the line illustrated here, but it passes through the central point ($p_2 = t_2 = \frac{1}{2}$).

therefore has a higher frequency of T_2 than does the population at large.

Suppose that at the beginning of a generation the demes are at various points along an approximately straight segment of a line of stable equilibria. Then after migration, selection and reproduction, the demes will still be on the line, because the migrants that entered each deme will have changed its frequency of P_2 by an amount proportional to the amount by which they changed its frequency of T_2 , the constant of proportionality being the reciprocal of the slope of the line. The changes of gene frequency caused each generation by differential migration are not opposed by selection within demes, because within each deme the new frequency of T_2 is stabilized by a new and appropriate frequency of P_2 . Even where the line of equilibria is moderately curved, migration moves the demes along paths that are almost tangent to the line. Then, depending on the direction of the curvature, there are either slight decreases or slight additional increases in the average frequencies of T_2 and P_2 , as selection pulls the demes in toward the line.

This process of 'selective diffusion'^{11,12} or 'group selection'²⁰⁻²³ depends on the variance of gene frequencies among demes, but the variance is reduced by migration. In group-selection models for the evolution of reproductive altruism or female-biased sex ratios of parental investment, the overall direction of gene-frequency change is reversed when the variance falls below a critical minimum, because within each deme there is selection against the trait that is favoured by differential migration between demes. This problem does not arise in the present model, because within each deme female choice stabilizes the current frequency of T_2 . Thus, \bar{t}_2 continues to increase as long as the balance between migration and sampling drift maintains even a small interdemic variance of gene frequencies. Female mating preferences change the frequencies of T_2 only in demes that are away from the line of equilibria. In demes that are on the line they do not directly change gene frequencies, but they can act as a catalyst for changes induced by other causes.

The main implication of these results is that females in polygynous species are expected to prefer mating with males who, other things being equal, show evidence that they would have been reproductively successful had they been females rather than males. We are not the first to argue that female choice should evolve to favour, in part, traits that promote ecological fitness^{1,2,24-34}, but the present model seems to be the

first to show explicitly how female choice could evolve to place special emphasis on traits that promote female fitness^{1,26}.

Many well-known patterns of sexual dimorphism are consistent with an interpretation of the kind implied by this model. For example, rates of senescence are higher in the males of most species than in the females^{35,36}. Unfortunately, this dimorphism has plausible alternative explanations based on male-male competition rather than female choice^{1,35-37}. The same ambiguity seems likely to appear in all comparative tests of the model, as the following example will illustrate.

Male *Anolis* lizards tend to be much larger than conspecific females on islands, but not on the mainland³⁸. Island populations appear to be more strongly food-limited than mainland populations. Individuals belonging to island populations grow relatively slowly, mature at relatively late ages, feed relatively often on small items, and show sharp increases in growth rates when additional food is supplied experimentally³⁸. If the fitnesses of island females depend relatively strongly on the efficient acquisition and assimilation of food, we would expect island females to prefer mating with males who had demonstrated special competence in these abilities. The greater size dimorphism of island populations is consistent with this expectation. But it is also consistent with the argument that female-defence polygyny is a more successful male strategy on islands than it is on the mainland, because island populations live at higher densities (with smaller territory and home-range sizes) than do mainland populations. Direct male-male competition would then be the selective force that favours relatively large male sizes on islands³⁹. The two models are not mutually exclusive, in this example or in general, so considerable ingenuity will be needed to devise tests that clearly distinguish between them.

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