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Local Mate Competition and the Sex Ratio†

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Hamilton (1967) pointed out that Fisher's (1930) argument predicting an equality of the sex ratio may break down when there is local competition for mates. He considered in particular a model in which the environment consists of a number of isolated patches, each of which is colonized by a number of inseminated females; the offspring breed within the patch before dispersal. The present paper provides a careful derivation of the equilibrium sex ratio under this model in both diploid and haplo-diploid populations, and extends the model to consider the effects of having a finite number of patches.

We suggest that the equilibrium sex ratio is not simply a function of the amount of inbreeding or sib-mating, as suggested by Maynard Smith (1978), but that the detailed breeding structure of the population must be taken into account.

1. Introduction

Fisher (1930) argued that the total efforts invested in producing males and females should be the same; in species without parental care of the young, this implies that the primary sex ratio should be $\frac{1}{2}$. Hamilton (1967) pointed out that Fisher's argument depends on the rather unlikely assumption of random mating within the whole population, and showed that considerable departures from an equal sex ratio may occur when mates are chosen preferentially within local groups (local mate competition).

Hamilton considered in particular a model in which the environment consists of a number of isolated patches, each of which is colonized by a fixed number of inseminated females; the offspring mate at random within the patch in which they were born, and inseminated females then disperse at random among the patches to restart the cycle. In this model the sex ratio

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should show a female bias, and in the extreme case when each patch is colonized by a single inseminated female, the equilibrium sex ratio is all female; in practice this means that a female should produce nearly all female offspring, and only a few males sufficient to fertilize all their sisters. Examples of this mechanism are provided by some insects and mites in which sib-mating is accompanied by a predominantly female primary sex ratio.

Hamilton's derivation of the theoretical sex ratio under the above model was approximate and rather heuristic. The purpose of this paper is to derive the results by a more exact argument.

In the next section we describe a simple, heuristic argument which demonstrates the underlying reason for the female bias under Hamilton's model in a diploid population; in section 3 we make the argument more rigorous, considering separately the cases with a finite and with an infinite number of patches. Finally, in section 4 we consider haplodiploid populations and meiotic drive.

2. A Heuristic Argument

Hamilton's model in its simplest form can be stated as follows. The environment consists of M patches, each of which is colonized by N fertilized females. Each female produces K offspring, the male and female offspring within a patch mate at random and fertilized female offspring disperse at random among the patches to restart the cycle; when each patch has been colonized by N females, any remaining females die without reproducing.

We shall now consider a simple, non-rigorous derivation of the equilibrium sex ratio in a diploid population. Suppose that the normal sex ratio in the population is r, and that a single mutant female produces offspring with sex ratio s; thus normal females produce Kr sons and $K(1-r)$ daughters, while the mutant female produces Ks sons and $K(1-s)$ daughters. To find the fitness of the mutant female, imagine that her genes, and those of her mate, are coloured red, so that there are four red genes in the population at a typical autosomal locus. We shall now compute the average number of red genes in the next generation.

The mutant patch (the patch containing the mutant female) produces $K(1-s)$ females with two red genes and $K(N-1)\ (1-r)$ females wiith no red genes; likewise, it produces Ks males with two red genes and $K(N-1)r$ males with no red genes. After random mating among these offspring, the

number of red genes among fertilized females is

$$A = 2K(1-s)+[K(1-s)+K(N-1)(1-r)]\left[\frac{2Ks}{Ks+K(N-1)r}\right]$$
$$= 2K(1-s)+2Ks\left[\frac{(1-s)+(N-1)(1-r)}{s+(N-1)r}\right], \quad (1)$$

the first term coming from red genes in females and the second from red genes in their mates. The total number of fertilized female offspring from all patches is

$$T = K(1-s)+K(1-r)[(N-1)+N(M-1)]. \quad (2)$$

When these females have dispersed to start the next generation, the number of red genes in the next generation is AMN/T, and the fitness of the mutant female can be defined as $f = AMN/4T$. If r is the equilibrium sex ratio, then it must be able to resist invasion by any mutant, so that $f<1$ whenever $s \neq r$. Thus the equlibrium sex ratio can be found by solving the equation

$$df/ds_{|s=r} = 0.$$

The equilibrium sex ratio is

$$r = \frac{M(N-1)}{2(MN-1)}. \quad (3)$$

We see that if there is only one patch (a finite random mating population), then r has the standard Fisher (1930) value of $\frac{1}{2}$, but if $M>1$, the equilibrium sex ratio is female biased, the bias increasing with the number of patches. With a very large number of patches ($M \to \infty$), $r = \frac{1}{2}(N-1)/N$, the value obtained by Hamilton (1967).

By comparing the fitness of the three different categories of females in the model, we can get some insight into the mechanism favouring a female biased sex ratio. The quantity A calculated above is the number of red genes among fertilized female offspring after mating. The analogous calculation for a normal female in the mutant patch gives

$$B = 2K(1-r)+2Kr\left[\frac{(1-s)+(N-1)(1-r)}{s+(N-1)r}\right]. \quad (4)$$

Finally a normal female in a normal patch expects to produce

$$C = 2K(1-r) + 2Kr\left[\frac{N(1-r)}{Nr}\right] = 4K(1-r) \tag{5}$$

red genes in mated female offspring.

Now suppose that the population sex ratio is $r = \frac{1}{2}$, and the mutant ratio is female biased, $s < \frac{1}{2}$. Then, in A and B, the term in square brackets is greater than one, so that $B < A$. But also $A > 2K = C$. Thus $C < A < B$. The reason that C is less than A and B is that the mutant patch produces more females than a normal patch. The reason that A is less than B is that the excess of female offspring in the mutant patch has the effect of raising the expected reproductive success of all male offspring, and the ordinary females produce more male offspring than the mutant female. This is the standard Fisher effect which penalizes low sex ratios when the population (patch) sex ratio falls below $\frac{1}{2}$. Thus the mutant female raises her own fitness above that of the general population, but raises the fitness of normal females in her own group even more. If there is only one patch, then of course, s will be at a disadvantage, but if there is more than one patch, there will be values of s close to but below $\frac{1}{2}$ which will have a net advantage over $r = \frac{1}{2}$.

The mutant female exhibits a type of local altruism. Some quite general models which provide conditions under which this form of altruism can spread in a population have been considered by Wilson (1975) and Maynard Smith (1976).

Finally, let us observe that an essential feature of this model is that offspring mate locally within the patch before the mated females disperse. If we drop this requirement, and assume that an individual mates in the patch with probability p, and at random in the whole population with probability $1-p$, then the amount of female bias in the sex ratio is proportional to p. Indeed an argument similar to the above gives the equilibrium ratio as

$$r = \frac{1}{2} - \frac{p(M-1)}{2(MN-1)}. \tag{6}$$

3. A More Rigorous Argument

In this section we shall construct a more rigorous argument based on a precise genetic model of the determination of the sex ratio. We consider a locus with two alleles, R and S, with S dominant to R. We shall suppose that the gene acts in females, so that RR females produce offspring with sex ratio r, while SR or SS females produce offspring with sex ratio s, whatever the genotypes of their mates; however, the argument will go through with minor

notational changes if the gene acts in males rather than females. We shall now show by a direct argument that equation (3) of the last section gives the equilibrium sex ratio under this model. By changing details of the proof it can be shown that the result remains true if the assumption of dominance is relaxed. We shall first consider a deterministic model with an infinite number of patches ($M = \infty$); we shall then discuss the stochastic analogue of this model with a finite number of patches.

(A) INFINITE NUMBER OF PATCHES

There are nine possible genotypes of a fertilized female, which may be represented by (i, j), where i denotes the number of S genes in the female and j the number in her mate ($i, j = 0, 1, 2$). A pair will be called normal if the female is RR ($i = 0$) and abnormal otherwise. A normal pair produces rK male and $(1-r)K$ female offspring, an abnormal pair produces sK male and $(1-s)K$ female offspring. A pair will be called non-mutant if both the female and the male are RR ($i + j = 0$), and mutant otherwise. We shall suppose that S is rare, so that the possibility that there are two or more mutant pairs in a patch can be ignored. Write $x(i, j)$ for the frequency of patches with one mutant pair of type (i, j) and $N-1$ non-mutant pairs, which will be called a mutant patch of type (i, j); since S is rare the $x(i, j)$'s will be small. The patch is called normal if $i = 0$ and abnormal if $i > 0$..

The standard method of determining whether S will spread when it is rare is to write down the linearized recurrence relations for the $x(i, j)$'s and to find the dominant eigenvalue of the corresponding 8×8 transformation matrix. Fortunately a short cut is available. We first compute $\alpha(i, j)$, the expected number of S genes in fertilized female offspring produced by a mutant patch of type (i, j). For normal patches $\alpha(i, j) = (1-r)K(i+j)$, and for abnormal patches

$$\alpha(i, j) = 2 \times \text{no. of female offspring in patch} \times (p_f + p_m), \qquad (17)$$

where

$$\text{no. of female offspring in patch} = (1-s)K + (N-1)(1-r)K$$

$$p_f = \text{frequency of } S \text{ in female offspring}$$

$$= \frac{\frac{1}{4}(i+j)(1-s)K}{[(1-s)K + (N-1)(1-r)K]}$$

$$p_m = \text{frequency of } S \text{ in male offspring} = \frac{\frac{1}{4}(i+j)sK}{[sK + (N-1)rK]}$$

Hence, in abnormal patches,

$$\alpha(i,j) = (1-r)K(i+j)f(r,s) \tag{8}$$

where

$$f(r,s) = \frac{1}{2(1-r)}\left[(1-s) + \frac{s[(1-s)+(N-1)(1-r)]}{s+(N-1)r}\right]. \tag{9}$$

The chance that a fertilized female offspring will survive to breed in the next generation is $1/(1-r)K$, since nearly every patch is normal. It follows from (8) that the number of S genes in the population will increase or decrease from one generation to the next according as $f(r,s) \gtrless 1$. It is now easily shown that $r = \frac{1}{2}(N-1)/N$ is the only equilibrium value of the sex ratio, and that when r takes this value $f(r,s) < 1$ for all $s \neq r$. Thus this sex ratio is an evolutionarily stable strategy. It should be noted that this analysis is confined to the behaviour of a mutant allele S when it is rare, though it seems likely that the result is of general validity.

(B) FINITE NUMBER OF PATCHES

When the number of patches is finite, the total population size is finite, and the process must be treated as a stochastic process. Let n_{ijl} be the number of fertilized females of type (i,j) in patch l. A state of the system is a specification of these numbers, and a complete description of the process would require writing down the matrix of transition probabilities for all possible states. Fortunately, it is possible to obtain enough information about the behaviour of the system by considering simply the total number of S genes, defined as $X = \sum (i+j)n_{ijl}$. In particular we shall show that

$$E(X_{t+1}|X_t) = X_t - \beta(r-\hat{r})(s-r) + o(s-r), \tag{10}$$

where β is strictly positive and $\hat{r}$ is the equilibrium sex ratio given in (3)., Thus the S gene is at a selection advantage if $r > \hat{r}$ and $s < r$, or if $r < \hat{r}$ and $s > r$, provided at least that s is close to r. This does not ensure that the equilibrium sex ratio (3) will hold exactly, since suboptimal alleles may be fixed by chance fluctuations, but we should usually expect to observe a sex ratio near this value under the joint pressures of mutation, selection and drift.

To prove this theorem we first compute the expected value of X_{t+1} given the n_{ijl}'s in generation t. We then write $\varepsilon = s - r$, expand this expected value in a Taylor series about $\varepsilon = 0$, and keep only the linear term in ε. In this way we find that

$$E(X_{t+1}|n_{ijl}\text{'s in generation } t) = X_t + \alpha\varepsilon, \tag{11}$$

where

$$(1-r)\alpha = \frac{A}{MN} X_t + \frac{1}{2r}\left[(1-2r)X_t^- - \sum_l \frac{A_l}{N} X_{lt}\right], \tag{12}$$

and

A_l = no. of abnormal pairs in patch l
$A = \sum_l A_l$ = total no. of abnormal pairs
X_t^- = total no. of S genes in abnormal pairs
X_{lt} = no. of S genes in patch l
X_{lt}^- = no. of S genes in patch l in abnormal pairs.

We now write n_{ij} for the total number of pairs of type (i, j) in all patches, and we consider the expectation of X_{t+1} conditional on the n_{ij}'s in generation t. We first observe that

$$E\left[\sum (A_l)X_{lt} | n_{ij}\text{'s}\right] = \frac{1}{(MN-1)}[(N-1)AX_t + N(M-1)X_t^-]. \tag{13}$$

To obtain this result we first compute $E(X_{lt}|A_l)$, and we then use the fact that A_l, conditional on A, has a hypergeometric distribution. From (11), (12) and (13) we find that

$$E(X_{t+1}|n_{ij}\text{'s in generation } t) = X_t + \alpha^* \varepsilon \tag{14}$$

where

$$(1-r)\alpha^* = \left[1 - \frac{M(N-1)}{2r(MN-1)}\right]\left[\frac{A}{MN} X_t - X_t^-\right]. \tag{15}$$

The second term in square brackets on the right-hand side of (15) will almost certainly be negative. When S is rare this follows from the fact that A/MN is very small. In general, we may assume that the population is approximately in Hardy–Weinberg equilibrium, whence $X_t^- \simeq \frac{1}{2}X_t(1 + A/MN)$, and the term in question is approximately $-(MN-A)X_t/2MN$. Hence (10) is obtained.

4. The Haplodipoid Case

We now suppose that males are haploid and females diploid with the standard reproductive pattern: fertilized eggs become females; unfertilized eggs become males. We will assume that there are an infinite number of patches, and provide an exact argument at the level of that given in section 3. As before we postulate two alleles R and S at a single locus, except that, to

obtain some mathematical simplification, we assume R to be dominant over S. To begin with we assume that the gene acts in the female. Thus RR and RS females produce offspring with sex ratio r and SS females produce offspring with sex ratio s.

As in section 3 we assume that S is rare, so that at most one fertilized female in each patch has any S genes. Let $x(i,j)$ $(0 \leq i \leq 2,\ 0 \leq j \leq 1,\ i+j>0)$, denote the relative frequency of patches in which there are $N-1$ non-mutant fertilized females and one female of type (i,j) with i S genes in the female and j S genes in her mate. To determine whether S will spread we will write down the linearized recurrence relations for the $x(i,j)$ and find the dominant eigenvalue of the 5×5 transformation matrix. The short cut which worked in section 3 is not available when the sexes have different ploidy.

The transformation matrix is obtained by recording the output of each of the five types of mutant patch; the ith column records the contribution of the ith patch type to each next generation patch type. If we let $x(i,j)$ denote the number of patches of type (i,j) at one generation and $x'(i,j)$ at the next then

$$\begin{bmatrix} x'(2,1) \\ x'(2,0) \\ x'(1,1) \\ x'(1,0) \\ x'(0,1) \end{bmatrix} = \begin{bmatrix} pu & 0 & 1/4N & 0 & 0 \\ (1-p)u & 0 & (2N-1)/4N & 0 & 0 \\ 0 & pu & 1/4N & 1/4N & 0 \\ 0 & (1-p)u & (2N-1)/4N & (2N-1)/4N & 1 \\ (N-1)p & (N-1)p & (N-1)/2N & (2N-1)/4N & 0 \end{bmatrix} \begin{bmatrix} x(2,1) \\ x(2,0) \\ x(1,1) \\ x(1,0) \\ x(0,1) \end{bmatrix}$$

where we have introduced the abbreviations $p = s/[s+(N-1)r]$, and $u = (1-s)/(1-r)$.

The condition that r be at equilibrium is that the dominant eigenvalue of this matrix be ≤ 1 for all s. This condition is met for $r = (N-1)(2N-1)/N(4N-1)$. We will discuss how this calculation is made at the end of the section. Hamilton (1967, p. 485) did some computer simulations to test this model for the case $N=2$. His result located the equilibrium sex ratio between 0·205 and 0·215. At the time, Hamilton was working with the diploid formula $r=(N-1)/2N$, and he pointed out the discrepancy between the theoretical ratio $r=\frac{1}{4}$ and the result of his simulation. In fact, our formula gives $r=\frac{3}{14}=0{\cdot}214$, and the discrepancy disappears. Hamilton (1979) has subsequently obtained a formula which is equivalent to ours, though his argument is cryptic.

(A) MEIOTIC DRIVE

Suppose we have a diploid population with a gene controlling sex ratio on the X chromosome. This is formally identical to a haplodiploid population with male control of the sex ratio. We proceed exactly as before except that now mated females of type (2, 1), (1, 1) and (0, 1) produce offspring with sex ratio s. The equilibrium sex ratio is calculated to be $r = (N-1)/N(4N-1)$. For infinite N we get $r = 0$, which corresponds to the fact that in an infinite random-mating population, X-drive leads to an all female ratio and extinction. For $N = 1$, we have complete sib-mating and we again get $r = 0$, but for a different reason. For intermediate values of N, the population can survive with a non-zero equilibrium sex ratio.

Hamilton (1967) discusses X-drive. He did not have a mathematical solution available, but determined by computer simulation that the equilibrium ratio for $N = 2$ is between 0·070 and 0·072. Our formula gives $r = \frac{1}{14} = 0{\cdot}0714$.

A model for X-drive in *Drosophila* has been proposed by Thompson & Feldman (1975). They show that a stable equilibrium can occur if the driving X-chromosome confers differential viability and, in males, differential fertility. They quote biological evidence for both these effects which seem to provide a more likely explanation of the polymorphism than the situation of Hamilton's model.

It is of interest to compare our formula with the formula $r = (N-1)/N$ for Y-drive obtained by Hamilton (1967). This case does not need the matrix machinery of the X-linked case, since there is only one type of mutant mated female.

(B) THE CALCULATIONS

Let us describe how the mathematical calculations proceed in the haplodiploid case with female control. Denote the 5×5 transition matrix by $\mathbf{A}(r, s)$. We want the value of r for which the dominant eigenvalue of this matrix is ≤ 1 for all s. Notice first that when $s = r$ the population is in a steady state, so that $\lambda(r, r) = 1$. Indeed it is easy to check that (3, 2, 2, 1, 1) is a left eigenvector for $\lambda = 1$ of $\mathbf{A}(r, r)$. Thus a necessary condition that $\lambda(r, s) \leq 1$ for all s is that $(\partial\lambda/\partial s) = 0$ when $s = r$. If we let $f(r, s, \lambda)$ be the characteristic polynomial of $\mathbf{A}(r, s)$ then the function $\lambda(r, s)$ is obtained by solving the equation $f(r, s, \lambda) = 0$ for λ as a function of r and s (and taking the largest value). Hence the equation we get by differentiating with respect to s,

$$\frac{\partial f}{\partial s} + \frac{\partial f}{\partial \lambda}\frac{\partial \lambda}{\partial s} = 0,$$

will give us $(\partial\lambda/\partial s)$. The condition $(\partial\lambda/\partial s)=0$ is equivalent to $(\partial f/\partial s)=0$. Since f is the determinant of $\mathbf{A}-\lambda\mathbf{I}$, the condition becomes

$$\frac{\partial}{\partial s}\det|\mathbf{A}(r,s)-I|=0 \quad \text{at } s=r. \tag{16}$$

The use of (16) to calculate the equilibrium value of r requires the evaluation of the determinant of a large matrix (in this case 5×5), and the work required can be tedious. Simplifications can sometimes be made with row and column operations (which do not alter the determinant). In the present case, if we replace row 3 of $\mathbf{A}-\mathbf{I}$ with row $3+\frac{1}{2}$ row $4+\frac{1}{2}$ row 5, we get zeros in the last two columns of row 3. (Essentially, this happens because (3, 2, 2, 1, 1) is a left eigenvector of $\mathbf{A}(r,r)-\mathbf{I}$ for the eigenvalue 0, and the last two columns of $\mathbf{A}-\mathbf{I}$ are independent of s.) The matrix $\mathbf{A}-\mathbf{I}$ then has block triangular form, but has the same determinant:

$$\det(\mathbf{A}-\mathbf{I})=\det\begin{vmatrix}B & O\\ C & D\end{vmatrix}=\det B\,\det D$$

where D is a 2×2 matrix which does not depend on s. Thus (1) becomes $(\partial/\partial s)\det B=0$, with B a 3×3 matrix. The calculations are now reasonable.

Let us finally remark that if we assume S is dominant, the last two columns of $\mathbf{A}$ are no longer independent of s, and the above row reduction trick does not work. This is what makes the calculations more difficult in this case. However, we have verified that the equilibrium value of r is the same as in the case S recessive.

5. Discussion

Maynard Smith (1978) discusses the effect of sib-mating on the equilibrium sex ratio. He argues that in a diploid population if a proportion p of all females sib-mate while $1-p$ mate at random, then the equilibrium sex ratio should be $r=(1-p)/2$. His argument is in general terms and does not require a knowledge of the specific breeding structure which causes the partial sib-mating.

One reason that Hamilton's model is of theoretical interest, is that it does provide a specific breeding structure in which Maynard Smith's general formula can be tested. In the first part of section 3 we discuss Hamilton's original model, in which the probability of sib-mating is $p=1/N$. We obtain Hamilton's result $r=(N-1)/2N$, which gives Maynard Smith's formula when $N=1/p$. If, however, we assume a finite population, organized into M patches, the equilibrium sex ratio is rather less female biased than Maynard Smith's formula predicts. In case $M=1$, we obtain the result $r=\frac{1}{2}$ for a

random mating finite population of size N (which is of course, completely inbred).

In two forthcoming papers (Taylor & Bulmer 1980; Bulmer & Taylor, 1980), we argue that what affects sex ratio is not inbreeding as such, but rather the extent of sib competition for a genetic share of the next generation. Between males this might involve competition for mates; between females, competition for nest space. Such competition will often tend to produce some inbreeding, but there is no direct link between the amount of such inbreeding and the equilibrium sex ratio. To actually calculate the equilibrium sex ratio one needs to know several parameters of this sib competition; it is not enough simply to know p, the probability that a female will sib-mate.

Finally we will make a point about dominance. If a genetic argument, using alternative alleles, is employed to calculate equilibrium sex ratios, one is forced to make an assumption about the dominance of the mutant allele. In sections 2 and 3, we have assumed S is completely dominant, whereas in section 4, some mathematical simplifications were obtained in our assumption that S is recessive. In some models it is easy to see that the equilibrium ratio will remain the same under any dominance assumption. In others, for example in section 4, considerable calculation is required to see whether this is so.

There is a general problem here which appears to be difficult. Our biological intuition tells us that under a wide range of conditions there should be a stable equilibrium of the sex ratio which resists invasion by mutant alleles of all degrees of dominance. But it is not easy to see how a precise result along these lines might be formulated or demonstrated.

REFERENCES

BULMER M. G. & TAYLOR P. D. (1980). *Nature* **284**, 448.

FISHER, R. A. (1930). *The Genetical Theory of Natural Selection.* Oxford: Oxford University Press.

HAMILTON, W. D. (1967). *Science 156*, 477.

HAMILTON, W. D. (1979). In *Sexual Selection and Reproductive Competition in Insects*, (M. S. Blum & N. A. Blum, eds), p. 167. New York: Academic Press.

MAYNARD SMITH, J. (1964). *Nature, Lond.* **201**, 1145.

MAYNARD SMITH, J. (1976). *Quart. Rev. Biol.* **51**, 277.

MAYNARD SMITH, J. (1978). *The Evolution of Sex.* Cambridge: Cambridge University Press.

TAYLOR, P. D. & BULMER, M. G. (1980). Submitted to *Am. Nat.*

THOMPSON, G. and FELDMAN, M. (1975). *Theor. Pop. Biol.* **8**, 202.

WILSON, D. S. (1975). *Proc. natn. Acad. Sci. U.S.A.* **72**, 143.