The Selective Advantage of Sex-Ratio Homeostasis
Author(s): Peter D. Taylor and Ann Sauer
Source: *The American Naturalist*, Vol. 116, No. 2 (Aug., 1980), pp. 305-310
Published by: The University of Chicago Press for The American Society of Naturalists
Stable URL: http://www.jstor.org/stable/2460679
Accessed: 24-06-2016 16:14 UTC

THE SELECTIVE ADVANTAGE OF SEX-RATIO HOMEOSTASIS

Fisher (1930) first postulated that under normal circumstances the equilibrium sex ratio should result from equal overall investment in male and female offspring. Assuming males and females are equally expensive (which we henceforth do), this means a 1 : 1 sex ratio. Since Fisher's work, models of varying sophistication have been produced to show that the 1 : 1 ratio obtains under quite a variety of assumptions.

According to these models, if the population as a whole has a 1 : 1 sex ratio then an individual's expected fitness is the same no matter what the sex ratio of his offspring. That is, provided the population has an overall 1 : 1 sex ratio, there is no selective pressure for what might be called "sex-ratio homeostasis," equal expenditure by an individual parent on males and females.

Of course, this assumes an outbred population with mates chosen randomly from the population at large, so that male and female offspring have an equal chance of finding mates. However, this is not generally strictly the situation. Usually a family of offspring will choose mates from a local group, and the male-female ratio within that group will not be 1 : 1 but will show some variation (with, of course, mean ratio 1 : 1). In that case an all male family will do better than average when the local mating group has more females and will do worse than average when there are more males.

Verner (1965) thought that in such a situation the all male family would lose more, on the average, when the local group was mostly male, than it would gain, on the average, when the local group was mostly female. He gave a simple numerical example to illustrate this point.

Our purpose in this paper is to take a diploid population with a 1 : 1 equilibrium sex ratio in which mates are chosen from local groups and to construct a simple model to measure the selective advantage of an allele h for sex-ratio homeostasis. We will find that the selection coefficient s of h is density dependent. This is to be expected. If h is common, the local mating group will tend to have nearly equal numbers of males and females, and it ought to make little difference whether an individual's offspring are mixed or all of the same sex. It is when h is rare that it has a maximum advantage.

Indeed, if p is the proportion of h alleles in the population and G is the total number of offspring produced by the founding females in a local group (not all these offsprings may survive to breed), then we show that $s = (1 - p)^2/2G$ if h is dominant and $s = p(1 - p)/2G$ if h is recessive. To test these values, we use the standard diffusion equation method (Crow and Kimura 1970, chap. 8) to calculate the probability of fixation of the h allele starting at some initial frequency p. We report results of a computer simulation with $G = 20$, $p_0 = 1/2$, which agree closely with our theoretical calculations.

Am. Nat. 1980. Vol. 116, pp. 305–310.

A MODEL TO MEASURE THE SELECTIVE ADVANTAGE

We will build the simplest possible model that exhibits the behavior that interests us. Our parameters all exhibit some variance in real life, but we will assume they are almost all constant. In most cases this variance can be incorporated fairly easily into the model to produce a minor effect on the results.

Assume the females, after mating, form at random into local groups of size N. Each female then gives birth to $2T$ offspring. Assume each of these offspring, independently, has probability K/T of surviving to breeding age. Those who do survive ($2KN$ on average) mate within the group, after which the females disperse to form new groups of size N with females of the population at large. We will assume the equilibrium sex ratio is 1:1, although it is known that the sex ratio is slightly female biased in such local mate competition models. Indeed, Hamilton (1967) considered the above model and calculated the equilibrium sex ratio to be $N - 1:N + 1$ (males to females). P. D. Taylor and M. G. Bulmer (in prep.) provide a method for obtaining this result more rigorously.

Now consider a particular group of N females of the F_0 generation. Suppose M of these females produce offspring in the normal (Mendelian) way, and H produce (as nearly as possible) equal numbers of males and females. We will calculate the expected number of descendants in the F_2 generation of the alleles in the M Mendelian females and in the H homeostatic females.

Let us count sons. The H homeostatic females produce HT sons and the M Mendelian females produce $B(2MT, \frac{1}{2})$ sons (binomially distributed with mean MT). Since each male survives to breed with probability K/T, the homeostatic females produce $B(HT, K/T)$, and the Mendelian females produce $B(2MT, K/2T)$ breeding sons. Writing these last two numbers as $HK + \delta$ and $MK + \epsilon$, δ has mean 0 and variance $HK(T - K)/T$ and ϵ has mean 0 and variance $MK(2T - K)/2T$.

The numbers of daughters of these two types of females are distributed in the same way. These sons and daughters, the F_1 generation, then mate with one another randomly within the group and produce the F_2 generation. Assuming the population remains constant in size, this group F_1 can expect to contribute $2N$ offspring to the F_2 generation. Now look at these $2N$ offspring. Of their $4N$ alleles, $2N$ come from the F_0 females; of these, N come through F_1 males and N through F_1 females. How many of these come from the H homeostatic F_0 females? The number that come through F_1 males is

$$\frac{N \cdot \text{no. sons of homeostatic } F_0 \text{ females}}{\text{total no. } F_1 \text{ males}} = \frac{N(HK + \delta)}{(MK + \epsilon) + (HK + \delta)}$$

$$= \frac{N(HK + \delta)}{NK + \epsilon + \delta} \simeq \frac{N}{NK}(HK + \delta)\left[1 - \frac{\epsilon + \delta}{NK} + \frac{(\epsilon + \delta)^2}{(NK)^2}\right]$$

if we assume δ/NK and ϵ/NK are much less than 1. Now averaging this over δ and ϵ (which are independent with mean 0), we get

$$\frac{1}{K}\left\{HK + \text{var}(\epsilon)\frac{HK}{(NK)^2} + \text{var}(\delta)\left[\frac{HK}{(NK)^2} - \frac{1}{NK}\right]\right\} = H\left(1 + \frac{M}{2N^2T}\right).$$

Since the same average number comes through F_1 females, the expected number of descendants of alleles in the H homeostatic F_0 females in the F_2 generation is

$$E(\text{hom}) = 2H\left(1 + \frac{M}{2N^2T}\right). \tag{1}$$

The expected number of descendants of alleles in the M Mendelian F_0 females in the F_2 generation is the difference between this and $2N$, which is

$$E(\text{Mend}) = 2M\left(1 - \frac{H}{2N^2T}\right). \tag{2}$$

So far we have been working with phenotypic fitness. We now introduce different alleles, assign phenotypes to genotypes, and calculate genic fitness of the different alleles. Suppose we have two alleles m and h at a certain locus which code for Mendelian and homeostatic behavior. Suppose h has frequency p in the population and a typical local group has Hardy-Weinberg proportions p^2N of type hh, $2pqN$ of type hm, q^2N of type mm, where $q = 1 - p$. Referring to our previous numbers M and H, if h is dominant then $M = q^2N$ and $H = (1 - q^2)N$, and if h is recessive then $M = (1 - p^2)N$ and $H = p^2N$.

Now we calculate the expected number of h alleles in the F_2 generation that have descended from the N F_0 females. In case h is dominant, the h alleles comprise a proportion $(p^2 + pq)/(p^2 + 2pq) = 1/(1 + q)$ of the alleles in the H F_0 females of types hh and hm, so the expected number is

$$\frac{1}{1+q}E(\text{hom}) = \frac{1}{1+q}\left[2(1 - q^2)N\left(1 + \frac{q^2N}{2N^2T}\right)\right]$$

$$= 2pN + \frac{pq^2}{T} \qquad (h \text{ dominant}). \tag{3}$$

In case h is recessive, the h alleles consist of all the alleles of the H hh females and a proportion $pq/(2pq + q^2) = p/(1 + p)$ of the alleles in the M females of types hm and mm. Thus the expected number is

$$E(\text{hom}) + \frac{p}{1+p}E(\text{Mend}) = 2p^2N\left[1 + \frac{(1 - p^2)}{2NT}\right]$$

$$+ \frac{p}{1+p}\left[2(1 - p^2)N\left(1 - \frac{p^2}{2NT}\right)\right]$$

$$= 2pN + \frac{p^2q}{T} \qquad (h \text{ recessive}). \tag{4}$$

Now (3) and (4) give the expected number of h alleles in the F_2 generation that descend from N F_0 females. Of course, half the alleles in the F_2 generation are descended from F_0 males, but, under the assumption that the h allele is expressed only in females, these will appear in equal numbers in the two generations and can be ignored when calculating changes in numbers. Thus the average increase in numbers of h alleles that is due to the expression of the allele in a group of N females is the difference between (3) or (4) and $2pN$, and is pq^2/T (h dominant),

p^2q/T (h recessive). The selection coefficient s is the increase per allele in the population and is the above number divided by $4pN$, the number of h alleles in N females and N males. Hence

$$s = \frac{q^2}{4NT} = \frac{q^2}{2G} \qquad (h \text{ dominant}), \tag{5}$$

$$s = \frac{pq}{4NT} = \frac{pq}{2G} \qquad (h \text{ recessive}), \tag{6}$$

where $G = 2NT$ is the total number of offspring born into a local group. These two formulae are graphed in figure 1.

You might think at first that, since the increase in numbers of h alleles takes two generations to show up, the selection coefficient per generation should be one-half of the above value. However, this is not the case. The above value of s reflects the increase resulting from the action of h on a single generation. In the following generation h will act again to produce a similar increase. It is incidental that these increases are not realized until two generations have passed.

THE PROBABILITY OF FIXATION—BY SIMULATION AND FORMULA

To test our values for s, we decided to use a formula developed by Kimura (1962) for the probability of fixation of a mutant allele in a population. First we did a computer simulation to measure this probability for the gene h, and then we calculated it using Kimura's formula.

In the simulation we took a population of 10 females and 10 males and assigned them genotypes mm, hm, or hh in Hardy-Weinberg proportions (approximately) with initial h frequency $p_0 = \frac{1}{2}$. They mated at random and the females had offspring, Poisson distributed in number with mean 2. The homeostatic females alternated the sex of their offspring, whereas the Mendelian females had the sex of each offspring assigned independently, male or female each with probability $\frac{1}{2}$. We recorded the genotypes of the offspring, then mated them at random (with no mortality between birth and breeding), and the females started over again. We did not hold the population size constant, but allowed it to drift. However, the number of offspring per female at each generation was drawn from a Poisson distribution with mean $40/n$, where n was the number of males and females in the current population. Thus the population tended to stay near 20 individuals. We continued until either the h allele was lost from the population or was fixed in the population. Once or twice the population reached an all male or all female state and disappeared, but this happened too rarely to cause concern. This scenario corresponds roughly to our model with $T = K = 1$, $N = 10$, and hence $s = q^2/40$ (h dominant) and $s = pq/40$ (h recessive).

A dominant h was fixed 544 times out of a total of 968 runs, a fixation rate of .562. A recessive h was fixed 538 times out of 960 runs, a fixation rate of .560.

Now let us calculate theoretical values for these fixation rates. Suppose in a population of effective size N_e an allele h has a selective advantage s (which may be a function of its frequency p) over its competitors. If h starts with frequency p_0, what is its probability $u(p_0)$ of eventually becoming fixed in the population? This

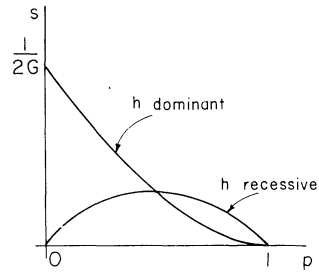

FIG. 1.—Selective advantage s of h allele against frequency p in population.

problem can be neatly solved using diffusion equation methods (Crow and Kimura 1970, p. 424). The answer is

$$u(p_0) = \frac{\int_0^{p_0} \exp(-\int 2M/V)dp}{\int_0^1 \exp(-\int 2M/V)dp},$$

where $M = sp(1 - p)$ and $V = p(1 - p)/2N_e$. In our case, if we take $p_0 = \frac{1}{2}$, $N_e = 2N = 20$, and $s = (1 - p)^2/4NT = (1 - p)^2/40$ if h is dominant, and $s = p(1 - p)/4NT = p(1 - p)/40$ if h is recessive, we get

$$u\left(\frac{1}{2}\right) = \frac{\int_0^{1/2} e^{2(1-p)^3/3}}{\int_0^1 e^{2(1-p)^3/3}} \cong .576 \qquad (h \text{ dominant}), \tag{7}$$

$$u\left(\frac{1}{2}\right) = \frac{\int_0^{1/2} e^{-p^2+2p^3/3}}{\int_0^1 e^{-p^2+2p^3/3}} \cong .552 \qquad (h \text{ recessive}). \tag{8}$$

The integrations were performed using Simpson's rule on intervals of size $\frac{1}{8}$.

Observe that we have taken the effective population size N_e to be the total number of adults in the population. In natural populations N_e is often somewhat less than this, sometimes as little of three-fourths of the total number of adults (Crow and Kimura 1970, p. 362). In fact this correction lowers the values of $u(\frac{1}{2})$ obtained in (7) and (8), but not by much. For example, taking $N_e = (\frac{3}{4})(2N) = 15$ we get $u(\frac{1}{2}) = .556$ (h dominant) and $u(\frac{1}{2}) = .539$ (h recessive).

DISCUSSION

We have built a simple model to measure the selective advantage of an allele h which causes a female to alternate the sex of her offspring. We made a considerable number of simplifying assumptions in the mathematical model, but our purpose was to obtain as clear a model as possible so that the crucial mechanism giving h its advantage could be observed. For example, we have assumed the local mating groups are always of the same size, that every female has the same number of offspring, that the sex ratio is 1:1, and that the homeostatic females produce exactly the same number of sons and daughters (this will be very closely true unless the number of such females is very small).

The selection coefficient s we get in (5) and (6) is density dependent. This is to be expected. The advantage of sex-ratio homeostasis is most pronounced when there is large variance in local sex ratio, and this will be greatest if h is rare. Its

TABLE 1

PROBABILITY u OF FIXATION OF h ALLELE

	h dominant	h recessive
Simulated	.562	.560
Theoretical	.576	.552

advantage is also inversely proportional to the size of the local group, but it is important to notice that, for this effect, size must be measured by the total number of offspring G born into a group. Thus, even if groups are founded by small numbers of females (N small), if they have high fecundity (with a resulting high offspring mortality either before or after breeding) the advantage of homeostasis will be swamped.

We tested s by asking what the probability u should be of fixation of h starting at $p_0 = \frac{1}{2}$. We estimated u with a large number of computer simulations in which we avoided some of the unrealistic assumptions of the mathematical model. We then calculated u using a formula based on diffusion equation methods in which we inserted the value of s obtained from our mathematical model. The results are shown in table 1. The simulated and theoretical values show remarkable agreement. This result, as well as providing a nice test for our model, provides an interesting test for the fixation probability formula of Kimura (1962).

ACKNOWLEDGMENTS

We are indebted to John Maynard Smith who suggested that we ought to consider the effect of mortality between birth and breeding. This work was partially supported by a grant from the Natural Sciences and Engineering Research Council of Canada.

LITERATURE CITED

Crow, J. F., and M. Kimura. 1970. An introduction to population genetics theory. Harper & Row, New York.

Fisher, R. A. 1930. The genetical theory of natural selection. Oxford University Press, Oxford.

Hamilton, W. D. 1967. Extraordinary sex ratios. Science 156:477–488.

Kimura, M. 1962. On the probability of fixation of mutant genes in a population. Genetics 47:713–719.

Verner, J. 1965. Selection for sex ratio. Am. Nat. 99:419–421.

PETER D. TAYLOR

DEPARTMENT OF MATHEMATICS
QUEEN'S UNIVERSITY
KINGSTON, ONTARIO K7L 3N6, CANADA

ANN SAUER

DEPARTMENT OF ECOLOGY AND EVOLUTION
STATE UNIVERSITY OF NEW YORK AT STONY BROOK
STONY BROOK, NEW YORK 11794

Submitted November 28, 1978; Accepted April 26, 1979