

TOWARDS A GENETIC THEORY FOR THE EVOLUTION OF THE SEX RATIO

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ABSTRACT

A genetical model is formulated in which the sex ratio in broods and the relative size of broods are determined by the genotype at an autosomal locus. The results also apply to the case in which the sex-ratio locus is sex linked and expressed in the homogametic sex and to the case in which the locus is expressed in the diploid sex of a haplodiploid organism. FISHER (1930) argued that the sex ratio evolves under natural selection to a value such that parental expenditure is equalized between the sexes. SHAW and MOHLER (1953) and MACARTHUR (1965) proposed that the sex ratio evolves to increase a certain expression for fitness. The sex ratio suggested by FISHER (1930) is in fact identical to the sex ratio specified by these maximization principles. Further, in our model, the Fisherian sex ratio corresponds exactly to the sex ratio at certain equilibria that are approached whenever they exist.

THE adaptive significance of the sex ratio has been a point of controversy among evolutionists since DARWIN. DARWIN (1871) suggested that certain sex ratios may benefit a population in terms of allowing more efficient group defense or mate selection, but failed to discover any selective advantage or disadvantage to the individual associated with distortion of the sex ratio. FISHER (1930) suggested that the sex ratio evolves under natural selection to a value such that parental expenditure is equalized between male and female offspring. FISHER's discussion of the problem, involving economic terms such as "expenditure" and a rather imprecise use of "reproductive value," was criticized by SHAW and MOHLER (1953) as "non-genetical." They proposed that fitness in sex-ratio evolution should be interpreted as the relative contribution of individuals through sons and through daughters and implied that fitness defined in this way should increase under natural selection. That expression for fitness, which we will call representational fitness, has been widely used (*e.g.*, BODMER and EDWARDS 1960; KOLMAN 1960; MACARTHUR 1965; HARTL and BROWN 1970; CHARNOV 1975), but the precise relationship between representational fitness and the underlying genetics has not been explored. Using a graphical fitness set approach, MACARTHUR (1965) suggested that the product of the number of sons and the number of daughters produced by the population will be maximized by natural selection.

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Theoretical investigations analyzing evolution at specific sex-ratio modification loci indicate that the qualitative predictions are critically dependent on the particular mode of transmission of the sex-ratio locus. SHAW (1958), SPIETH (1974), NUR (1974) and ESHEL (1975) have analyzed models in which sex-ratio distortion is influenced by an autosomal locus. Evolution at an autosomal locus that influences susceptibility to material inheritance of a cytoplasmic endosymbiont that distorts the sex ratio in the broods of its carriers has been investigated by UYENOYAMA and FELDMAN (1978). HARTL and BROWN (1970) considered a sex-ratio locus that is expressed in the diploid sex of a haplodiploid organism. In general, these papers indicate that the 1:1 sex ratio is highly attractive. However, models involving sex-linked sex-ratio loci (EDWARDS 1961; HAMILTON 1967; THOMSON and FELDMAN 1975; BENGTTSSON 1977) suggest that skewed sex ratios may be common in such systems; in fact, populations in which the sex chromosomes segregate in the normal 1:1 manner are particularly susceptible to the introduction of X- or Y-linked drivers (THOMSON and FELDMAN 1975). The role of FISHER's (1930) measure of reproductive value or SHAW and MOHLER's (1953) expression for representational fitness in these models is not clear (EDWARDS 1961; BENGTTSSON 1977). HAMILTON (1967) pointed out that FISHER's (1930) argument contains tacit genetic assumptions that restrict its applicability to sex-ratio modification loci that are transmitted in genetically equivalent forms through sons and daughters. In particular, HAMILTON (1967) suggested that FISHER's argument does apply to sex-linked loci expressed in the homogametic sex, to loci expressed in the diploid sex of a haplodiploid organism, and to autosomal loci expressed in the heterogametic sex. The results obtained in the present paper bear out HAMILTON's predictions, provided the restriction of the expression of autosomal sex-ratio loci to only the heterogametic sex is removed.

For the purpose of examining the significance to sex-ratio evolution of the concept of reproductive investment and the sex-ratio strategy arguments, we have formulated a genetical model involving an autosomal sex-ratio locus that influences the brood sex ratio and the relative fertility of the various matings. The results apply equally well to sex-linked loci that are expressed in the homogametic sex and to loci that are expressed in the diploid sex of a haplodiploid organism.

The equilibria of the model, under the restriction that the genotype of one parent alone determines the brood sex ratio and relative brood size, are determined as the valid roots of a cubic function. The analysis in the present paper extends the work of ESHEL (1975), who considered the equal brood-size case under the assumption that offspring genotype rather than parental genotype determines brood sex ratio. The evolutionary criteria proposed by SHAW and MOHLER (1953) and MACARTHUR (1965) will be compared and found to specify identical strategies. Maximization of representational fitness and of MACARTHUR's (1965) product is possible in our model if and only if a certain relationship exists between the brood sex ratio and relative brood size among the genotypes. Under this condition, the roots of the equilibrium cubic can be obtained explicitly. Further, it will be shown that, when that condition holds, the sex ratio specified by the two maximization principles corresponds exactly to the sex ratio attained at certain equi-

libria in our genetical model. Local stability of the equilibria will be examined and interpreted in light of the maximization principles. A number of reasonable models that satisfy the key condition for maximization, including the case of parental expenditure considered by FISHER (1970) will be discussed. It will be shown that, for the case of parental expenditure, the population sex ratio converges towards the value predicted by FISHER.

THE MODEL

Consider an infinitely large, random-mating population, and let there be a locus, A , with two alleles, A and a , that determines the proportions of males and females within broods and the relative size of broods. If the locus is autosomally inherited and the organism is diploid, there are three genotypes in females and three genotypes in males. Let the frequencies of AA , Aa and aa females be f_1 , f_2 and f_3 , and the corresponding values for males be m_1 , m_2 and m_3 ($\sum f_i = \sum m_j = 1$). The frequency of males in broods, censused at reproductive age and arising from matings of females of genotype i and males of genotype j , is denoted by r_{ij} , and the relative size of such broods, censused at the same time, by s_{ij} . The outcomes of the different matings are summarized in Table 1. There are no additional selective effects, such as viability differences, associated with the A locus. The model is similar to one formulated by SPIETH (1974).

If the A locus is X-linked in an organism where the females are homogametic or if it is expressed in the diploid sex of a haplodiploid organism, then there are only two male genotypes, A and a , and six mating types. A mating table similar to Table 1 may also be constructed for this case.

Table 1 presents our model in its full complexity. In the present paper, attention will be restricted to the case in which the effect of the A locus is expressed only in females. This does not necessarily imply that the females are sex determining in a physiological sense, but that the A alleles modify the sex ratios *via* an effect in females. Under this simplifying assumption, the frequency of males produced by a mating is determined by the mother's genotype only. Let females of genotype AA produce a fraction α of males among their offspring, Aa females

TABLE 1
Brood sex ratios and relative fertilities

Female	Male	Frequency of mating	Frequency of males	Relative brood size
AA	AA	$f_1 m_1$	r_{11}	s_{11}
	Aa	$f_1 m_2$	r_{12}	s_{12}
	aa	$f_1 m_3$	r_{13}	s_{13}
Aa	AA	$f_2 m_1$	r_{21}	s_{21}
	Aa	$f_2 m_2$	r_{22}	s_{22}
	aa	$f_2 m_3$	r_{23}	s_{23}
aa	AA	$f_3 m_1$	r_{31}	s_{31}
	Aa	$f_3 m_2$	r_{32}	s_{32}
	aa	$f_3 m_3$	r_{33}	s_{33}

produce a fraction β , and *aa* females a fraction γ , and let the corresponding relative brood sizes be σ_1 , σ_2 and σ_3 . These definitions apply both to the autosomal inheritance scheme and the *X*-linked or haplodiploid model. In Table 1, the definitions imply that

$$\begin{aligned} r_{11} = r_{12} = r_{13} = \alpha \text{ and } s_{11} = s_{12} = s_{13} = \sigma_1 \\ r_{21} = r_{22} = r_{23} = \beta \text{ and } s_{21} = s_{22} = s_{23} = \sigma_2 \\ r_{31} = r_{32} = r_{33} = \gamma \text{ and } s_{31} = s_{32} = s_{33} = \sigma_3. \end{aligned}$$

The situation in which the further assumption that $\sigma_1 = \sigma_2 = \sigma_3$ is made has been analyzed by NUR (1974) and ESHEL (1975).

In the full model given by Table 1, the evolution of the system is described by the frequencies of all genotypes in males and females. However, in the restricted cases investigated here, the evolution of the system is completely described by the gene frequencies in males and the genotype frequencies in females. The recursion equations for the autosomal inheritance model are:

$$p_m' = (1/2)p_m + (1/2)[f_1\sigma_1\alpha + (1/2)f_2\sigma_2\beta]/(f_1\sigma_1\alpha + f_2\sigma_2\beta + f_3\sigma_3\gamma) \quad (1a)$$

$$f_1' = \frac{[f_1\sigma_1(1-\alpha) + (1/2)f_2\sigma_2(1-\beta)]p_m}{f_1\sigma_1(1-\alpha) + f_2\sigma_2(1-\beta) + f_3\sigma_3(1-\gamma)} \quad (1b)$$

$$f_2' = \frac{[f_1\sigma_1(1-\alpha) + (1/2)f_2\sigma_2(1-\beta)]q_m + [(1/2)f_2\sigma_2(1-\beta) + f_3\sigma_3(1-\gamma)]p_m}{f_1\sigma_1(1-\alpha) + f_2\sigma_2(1-\beta) + f_3\sigma_3(1-\gamma)} \quad (1c)$$

$$f_3' = \frac{[(1/2)f_2\sigma_2(1-\beta) + f_3\sigma_3(1-\gamma)]q_m}{f_1\sigma_1(1-\alpha) + f_2\sigma_2(1-\beta) + f_3\sigma_3(1-\gamma)} \quad (1d)$$

$$F' = \frac{f_1\sigma_1(1-\alpha) + f_2\sigma_2(1-\beta) + f_3\sigma_3(1-\gamma)}{f_1\sigma_1 + f_2\sigma_2 + f_3\sigma_3} \quad (1e)$$

$$M' = \frac{f_1\sigma_1\alpha + f_2\sigma_2\beta + f_3\sigma_3\gamma}{f_1\sigma_1 + f_2\sigma_2 + f_3\sigma_3} \quad (1f)$$

where $p_m = m_1 + 0.5 m_2$ and $q_m = (1 - p_m)$. F is the frequency of females at the time of mating, and $M = 1 - F$ is the frequency of males at the same time.

If the sex-ratio locus is *X*-linked or is expressed in the diploid sex of a haplodiploid organism, then the recursion equations are identical to (1) with the exception of (1a), which is replaced by

$$p_m' = \frac{f_1\sigma_1\alpha + (1/2)f_2\sigma_2\beta}{f_1\sigma_1\alpha + f_2\sigma_2\beta + f_3\sigma_3\gamma} \quad (1a')$$

The equilibria of (1a) and (1a') are identical.

ANALYSIS OF THE MODEL

Equilibria of the system

The equilibria of system (1) are found by first expressing the female genotype frequencies at equilibrium ($\hat{f}_1, \hat{f}_2, \hat{f}_3$) in terms of the equilibrium gene frequencies

in males, \hat{p}_m and \hat{q}_m , and then producing a cubic in p_m , the roots of which are the nonfixation equilibrium values \hat{p}_m . The frequencies of the female genotypes are given below:

$$\begin{aligned} T\hat{f}_1 &= (1/2)\sigma_2(1-\beta)p_m[\tilde{F}-q_m\sigma_3(1-\gamma)] \\ T\hat{f}_2 &= [\tilde{F}-p_m\sigma_1(1-\alpha)][\tilde{F}-q_m\sigma_3(1-\gamma)] \\ T\hat{f}_3 &= (1/2)\sigma_2(1-\beta)q_m[\tilde{F}-p_m\sigma_1(1-\alpha)] \end{aligned} \quad (2)$$

where T is the sum of the right-hand expressions ensuring that $\Sigma\hat{f}_i = 1$ and \tilde{F} is the larger root of the following quadratic:

$$\begin{aligned} (\tilde{F})^2 - \tilde{F}[\hat{p}_m\sigma_1(1-\alpha) + (1/2)\sigma_2(1-\beta) + \hat{q}_m\sigma_3(1-\gamma)] \\ + (1/2)\hat{p}_m\sigma_1(1-\alpha)[\hat{p}_m\sigma_2(1-\beta) + \hat{q}_m\sigma_3(1-\gamma)] \\ + (1/2)\hat{q}_m\sigma_3(1-\gamma)[\hat{p}_m\sigma_1(1-\alpha) + \hat{q}_m\sigma_2(1-\beta)] = 0. \end{aligned} \quad (3)$$

An expression for \hat{p}_m in terms of the \hat{f}_i is available from (1a) or (1a'), which are identical at equilibrium. Elimination of the \hat{f}_i from that expression using (2) produces the following cubic after the fixation equilibria $\hat{p}_m = 0$ and $\hat{p}_m = 1$ have been factored out. The roots of this cubic are the equilibrium gene frequencies \hat{p}_m .

$$\begin{aligned} g(p_m) &= A_1A_2[\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)] \\ &\left[p_m - \frac{\sigma_3(1-\gamma) - \sigma_2(1-\beta)}{\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)} \right] \times \\ &+ (1/2)\sigma_2(1-\beta)(p_mA_2 + q_mA_1)(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta) \times \\ &\left[p_m - \frac{\sigma_3\gamma - \sigma_2\beta}{\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta} \right] \end{aligned} \quad (4)$$

where $A_1 = p_m\sigma_1\alpha + q_m\sigma_2\beta - (1/2)\sigma_2\beta$
 $A_2 = p_m\sigma_2\beta + q_m\sigma_3\gamma - (1/2)\sigma_2\beta$.

It can be shown that all roots of $g(p_m)$ in (0,1) are valid equilibria in our system, and conversely all valid equilibria correspond to roots of $g(p_m)$ in (0, 1). Once the equilibrium gene frequency in males \hat{p}_m is determined from (4), the equilibrium genotype frequencies in females \hat{f}_1 , \hat{f}_2 and \hat{f}_3 can be calculated from (2), and the frequency of males and females in the equilibrium population can then be obtained from (1e) and (1f).

ESHEL (1975) found for his sex-ratio model, in which offspring genotype rather than parental genotype determines the brood sex ratio, that two classes of equilibria may exist. The first class, denoted symmetric, is characterized by equal gene frequencies in males and females, *i.e.*, $\hat{p}_m = \hat{f}_1 + (1/2)\hat{f}_2$. The second class, denoted asymmetric, is characterized by the 1:1 sex ratio. Following ESHEL (1975), we say that a root of the cubic $g(p_m)$ in (4) is symmetric and

denote this root by \hat{p}_s if $\hat{p}_s = \hat{p}_m = \hat{f}_1 + (1/2)\hat{f}_2$. When symmetric roots exist, the remaining roots of $g(p_m)$ are denoted the asymmetric roots.

From (2) and (4), the following necessary and sufficient condition for the existence of a symmetric root in the present case may be obtained:

$$\sigma_1\sigma_2(\alpha-\beta) + \sigma_2\sigma_3(\beta-\gamma) + \sigma_1\sigma_3(\gamma-\alpha) = 0 . \quad (5)$$

When (5) is satisfied, the symmetric root $\hat{p}_s = \hat{p}_m = \hat{f}_1 + (1/2)\hat{f}_2$ is given by

$$\hat{p}_s = \frac{\sigma_3\gamma - \sigma_2\beta}{\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta} = \frac{\sigma_3(1-\gamma) - \sigma_2(1-\beta)}{\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)} = \frac{\sigma_3 - \sigma_2}{\sigma_1 + \sigma_3 - 2\sigma_2} \quad (6)$$

from (4). Under (5), \hat{p}_s corresponds to a valid equilibrium if $\beta > \alpha$ and γ , or $\beta < \alpha$ and γ .

The symmetric root may now be factored out of $g(p_m)$ to produce the following quadratic, the valid roots of which correspond to the remaining equilibria, which we denote asymmetric:

$$h(p_m) = A_1A_2[\sigma_1(1-\alpha) + \sigma_3(1-\gamma) - 2\sigma_2(1-\beta)] \\ + (1/2)\sigma_2(1-\beta)(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta)(p_mA_2 + q_mA_1). \quad (7)$$

The remaining analysis in the present paper will be restricted to those cases in which $g(p_m)$ has a symmetric root. It will be argued in the next section that optimization of sex ratio strategies is possible only in those cases in which the condition for the existence of the symmetric root holds.

Maximization principles

In this section, we relate (5), the condition for existence of a symmetric root, with the condition under which the proposed maximization principles are meaningful in the present model.

SHAW and MOHLER (1953) suggested that the following expression for an individual's contribution through sons and through daughters is a measure of fitness and implied that fitness defined as follows increases under natural selection:

$$\left[\frac{m}{M} + \frac{f}{F} \right] , \quad (8)$$

where m and f are the frequencies of males and females in broods produced by the individual and M and F are the population frequencies of males and females. Because the relationship between genotypic fitness and individual contribution is not made explicit, we regard SHAW and MOHLER's (1953) suggestion as a maximization principle. In terms of our model, a natural interpretation of SHAW and MOHLER's principle is that selection should maximize the following expression for mean fitness:

$$W = f_1\sigma_1 \left[\frac{\alpha}{M} + \frac{(1-\alpha)}{F} \right] + f_2\sigma_2 \left[\frac{\beta}{M} + \frac{(1-\beta)}{F} \right] + f_3\sigma_3 \left[\frac{\gamma}{M} + \frac{(1-\gamma)}{F} \right] . \quad (9)$$

MACARTHUR (1965) proposed on the basis of his fitness set analysis that natural selection maximizes the product of the number of sons and daughters reared to reproductive age by the population. MAYNARD SMITH (1978, pp. 166–167) has obtained a similar result from a genetic model describing the initial increase of a sex-ratio modification gene. SPIETH (1974) has interpreted this principle as implying the maximization of the product $\tilde{F}\tilde{M}$, where \tilde{F} is the unnormalized frequency of females defined by (3) and \tilde{M} is the analogous quantity for males. It can be shown that the points of maximization over the f_i specified under the two principles are identical (see also MAYNARD SMITH 1978, p. 167).

Maximization of W or $\tilde{F}\tilde{M}$ with respect to f_1 , f_2 and f_3 and subject to the constraint that $\Sigma f_i = 1$ (see COURANT 1937, p. 190) leads to the following “optimal” frequencies of females:

$$\begin{aligned} F_{12}^* &= \frac{\sigma_1(1-\alpha) - \sigma_2(1-\beta)}{\sigma_1(1-\alpha) - \sigma_2(1-\beta) + \sigma_2\beta - \sigma_1\alpha} \\ F_{23}^* &= \frac{\sigma_3(1-\gamma) - \sigma_2(1-\beta)}{\sigma_3(1-\gamma) - \sigma_2(1-\beta) + \sigma_2\beta - \sigma_3\gamma} \\ F_{13}^* &= \frac{\sigma_1(1-\alpha) - \sigma_3(1-\gamma)}{\sigma_1(1-\alpha) - \sigma_3(1-\gamma) + \sigma_3\gamma - \sigma_1\alpha} \end{aligned} \quad (10)$$

where F_{ij}^* is the frequency of females in the population obtained by maximization with respect to f_i and f_j . Existence of a sex ratio at which the maximization principles are satisfied requires that the expressions in (10) be identical. These three expressions are identical if and only if condition (5) is satisfied. Therefore, maximization in the senses suggested by SHAW and MOHLER (1953) and MACARTHUR (1965) is possible in our model (in which the genetic basis of the sex-ratio distortion is well defined) if and only if the model is such that it has a symmetric root. The frequency of females at the “optimal” sex ratio will be denoted F^* ($= F_{12}^* = F_{23}^* = F_{13}^*$).

Further, it can be shown that the frequency of females, F^* , derived from maximization principles when (5) holds is exactly equal to the frequency of females associated with the roots of the quadratic $h(p_m)$ given in (7), which defines the asymmetric equilibria of our model.

Existence conditions for the asymmetric equilibria

We have found that the frequency of females at the asymmetric equilibria is exactly the value suggested by the maximization arguments. The following geometric method is helpful in the presentation of the existence conditions for 0, 1 or 2 valid asymmetric equilibria.

From (1) it can be seen that over symmetric points the frequencies of the \hat{f}_i assume Hardy-Weinberg proportions. Define the curve $F(p)$, the frequency of females over symmetric populations, as follows:

$$F(p) = \frac{p^2\sigma(1-\alpha) + 2pq\sigma_2(1-\beta) + q^2\sigma_3(1-\gamma)}{p^2\sigma_1 + 2pq\sigma_2 + q^2\sigma_3} \quad (11)$$

The symmetric equilibria of the system, given by $\hat{p}_m = \hat{f}_1 + (1/2)\hat{f}_2 = 0, 1$ and \hat{p}_s , correspond to the extrema of the curve in $(0, 1)$. In particular, $F'(\hat{p}_s) = 0$. Asymmetric equilibria are associated with the line $F = F^*$, but do not fall on $F(p)$ because Hardy-Weinberg proportions do not obtain over asymmetric equilibria. It can nevertheless be shown that the number of valid asymmetric equilibria is exactly equal to the number of intersections between the curve $F(p)$ and the line $F = F^*$. Figures 1 and 2 depict two examples showing the symmetric equilibria at the extrema of $F(p)$ $(0, 1)$ and the intersections between $F(p)$ and $F = F^*$.

In Figure 1, a polymorphic symmetric equilibrium is represented by the maximum of $F(p)$ in $(0, 1)$. $F(p)$ intersects with $F = F^*$ in a single point, indicating that one valid asymmetric equilibrium exists. In Figure 2, $F(p)$ has no internal critical point and hence no internal symmetric equilibrium exists.

Local stability conditions for the symmetric equilibria for both autosomal and sex-linked cases

The fixation equilibria associated with $p_m=0$ is stable in the full model only if the following condition is satisfied:

$$(1-\gamma)(\sigma_3\gamma-\sigma_2\beta) + \gamma[\sigma_3(1-\gamma)-\sigma_2(1-\beta)] > 0. \tag{12}$$

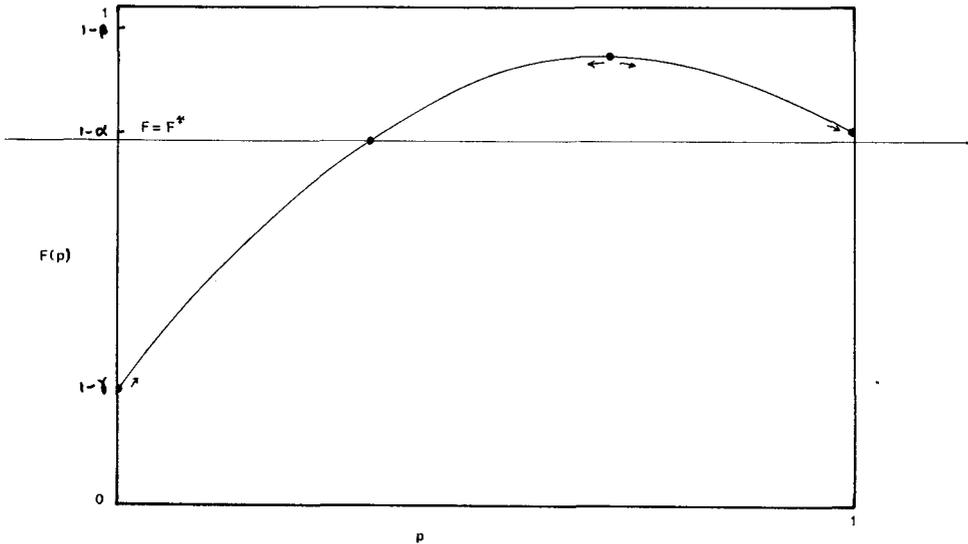


FIGURE 1.—The frequency of females over symmetric populations, $F(p)$ plotted as a function of p for the case in which $1 - \beta > 1 - \alpha > F^* > 1 - \gamma$. Arrows point toward stable symmetric equilibria and away from unstable symmetric equilibria. Symmetric equilibria $p = 0$, $p = 1$ and $p = \hat{p}_s$ occur at the extrema of the curve in $(0, 1)$. One intersection between F^* and $F(p)$, corresponding to the single valid asymmetric equilibrium, exists in $(0, 1)$. Trajectories approach F^* under all starting conditions.

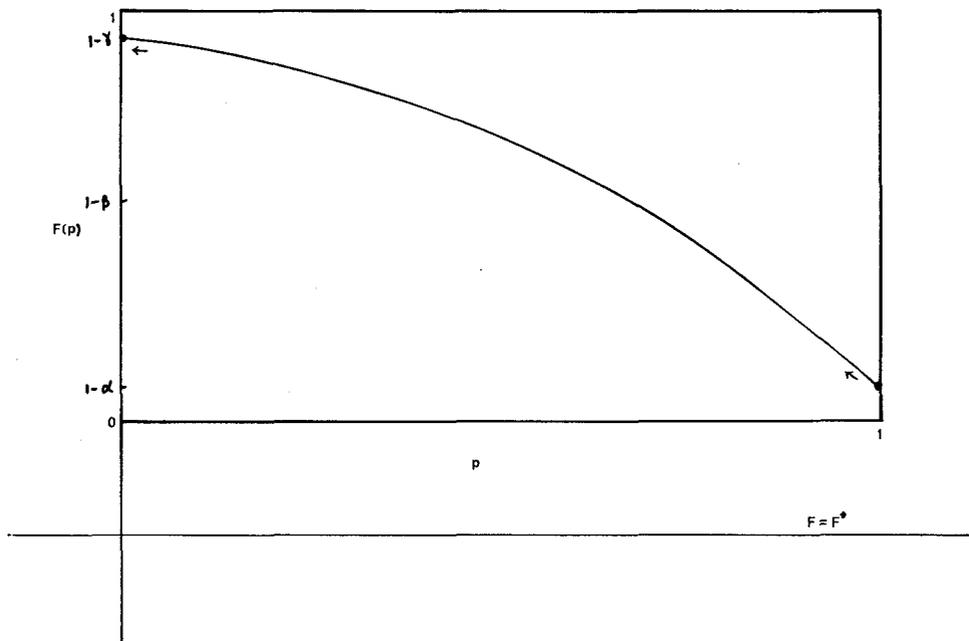


FIGURE 2.— $F(p)$ plotted as a function of p for the case in which $\alpha > \beta > \gamma$, $\sigma_1(1-\alpha) - \sigma_2(1-\beta) + \sigma_2\beta - \sigma_1\alpha < 0$, and $\sigma_2\beta - \sigma_1\alpha > 0$. Two symmetric equilibria, $p = 0$ and $p = 1$, exist. F^* is negative and no valid asymmetric equilibria exist. Note that the symmetric equilibrium $p = 0$ is stable, although it maximizes the deviation between the frequency of females in the population and F^* .

When (5) holds, (12) may be arranged as follows:

$$(\sigma_3\gamma - \sigma_2\beta) \left[\frac{(1-\gamma)}{\gamma} - \frac{F^*}{M^*} \right] > 0. \tag{13}$$

By symmetry, the condition for stability of the $p_m = 1$ equilibrium is given by

$$(\sigma_1\alpha - \sigma_2\beta) \left[\frac{(1-\alpha)}{\alpha} - \frac{F^*}{M^*} \right] > 0. \tag{14}$$

The $p_m = \hat{p}_s$ equilibrium is stable when it exists if

$$(\sigma_1\alpha - \sigma_2\beta) \left[\frac{F_s}{M_s} - \frac{F^*}{M^*} \right] < 0, \tag{15}$$

where F_s is the frequency of females at the $p_m = \hat{p}_s$ equilibrium, given by

$$F_s = \frac{\sigma_1\alpha\sigma_3\gamma - (\sigma_2\beta)^2}{\sigma_1(\sigma_3\gamma - \sigma_2\beta) + \sigma_2(\sigma_1\alpha - \sigma_2\beta)},$$

and $M_s = 1 - F_s$ is the frequency of males.

Using (13), (14) and (15), it can be shown that if two asymmetric equilibria exist, all symmetric equilibria are unstable. If one asymmetric equilibrium

exists, then the two symmetric equilibria closest in gene frequency to the asymmetric equilibrium frequency for p_m are unstable. For example, in Figure 1, the $p_m = 0$ and $p_m = \hat{p}_s$ equilibria are unstable and the $p_m = 1$ equilibrium is stable. If no asymmetric equilibria exist, then the local stability conditions (13), (14) and (15) suggest that the domains of attraction for the various symmetric equilibria has eluded analysis, but the considerations presented above and numerical iterations suggest that the asymmetric equilibria are locally stable whenever they exist.

Under parameter combinations leading to positive F^* , the stable equilibria of the system are those that minimize the local deviation between the frequency of females in the population and F^* , even if F^* is greater than 1. However, when F^* is negative, it is not true that natural selection always minimizes the deviation between the frequency of females in the population and F^* . In the example presented in Figure 2, the $p = 0$ equilibrium is the only stable point in the system even though the deviation between $F(p)$ and F^* is minimized at the $p = 1$ equilibrium. The local stability of the symmetric equilibria under all possible parameter combinations can be correctly predicted using a one-locus, frequency-dependent model—constructed solely for the purpose of illustration—in which fitness depends on the sex ratio in a population in a manner suggested by (8). Specifically, assign the genotypic fitnesses as follows

$$\text{Fitness } \sigma_1 \left[\frac{\alpha}{M} + \frac{(1-\alpha)}{F} \right] \quad \sigma_2 \left[\frac{\beta}{M} + \frac{(1-\beta)}{F} \right] \quad \sigma_3 \left[\frac{\gamma}{M} + \frac{(1-\gamma)}{F} \right]$$

Then the marginal fitnesses will be given by

$$\begin{aligned} W_1 &= p\sigma_1 \left[\frac{\alpha}{M} + \frac{(1-\alpha)}{F} \right] + q\sigma_2 \left[\frac{\beta}{M} + \frac{(1-\beta)}{F} \right] \\ W_2 &= p\sigma_2 \left[\frac{\beta}{M} + \frac{(1-\beta)}{F} \right] + q\sigma_3 \left[\frac{\gamma}{M} + \frac{(1-\gamma)}{F} \right] \end{aligned} \quad (16)$$

and the one generation change in gene frequency, Δp , becomes

$$\Delta p = \frac{pq(W_1 - W_2)}{pW_1 + qW_2} \quad (17)$$

Equation (17) may be arranged in the following form:

$$\Delta p = \frac{pq(\sigma_1\alpha + \sigma_3\gamma - 2\sigma_2\beta)(p - \hat{p}_s)}{F} \left[\frac{F}{M} - \frac{F^*}{M^*} \right] \quad (18)$$

From (18), it can be seen that the symmetric equilibria of our sex-ratio model are also equilibria of this constructed frequency-dependent model. Using (6), the $p = 0$ point in the model described by (18) is stable if (13) is satisfied. Similarly, $p = 1$ in the constructed example is stable if (14) holds, and $p = \hat{p}_s$ is stable if (15) holds. Therefore, the stability conditions (13), (14) and (15) near the symmetric equilibria of the sex-ratio model may be understood by analogy with the selective pressures arising in a purely symmetric system such

as (17), which acts to increase locally the frequency-dependent mean fitness as defined by (9).

RELATIONSHIP BETWEEN THE BROOD SEX RATIO AND FERTILITY

It may appear that the existence condition for a symmetric root under which we have derived our main results is highly restrictive. However, it is our position that condition (5) represents a special case of the full model given by (4) rather than a degeneracy, in the sense that the small parameter theory of KARLIN and MCGREGOR (1972) applies to our system and allows us to infer the qualitative behavior of more general cases when the structure of the full model is "close" to our special case. We will discuss several relationships between the brood sex ratio and fertility that satisfy (5) and then construct the case discussed by FISHER (1930) from which the extension to more general cases will be made.

Equal fertilities among genotypes

Suppose that physiological conditions in the female are such that the broods produced by the various genotypes are of equal size independent of the sex ratio in the brood. Under this assumption $\sigma_1 = \sigma_2 = \sigma_3$ and (5) is satisfied for all α , β , and γ . In this case F^* reduces to 0.5 in agreement with NUR (1974) and ESHEL (1975), who found that the system converges towards a state characterized by the 1:1 sex ratio.

Dominance of A

Condition (5) is satisfied under the assumption that the *A* allele is completely dominant with respect to sex ratio and brood size, *i.e.*, $\alpha = \beta$ and $\sigma_1 = \sigma_2$. There can be at most one asymmetric equilibrium, and the frequency of females at this highly attracting equilibrium is given by

$$F^* = \frac{\sigma_3(1-\gamma) - \sigma_1(1-\alpha)}{\sigma_3(1-\gamma) - \sigma_1(1-\alpha) + \sigma_1\alpha - \sigma_3\gamma}$$

Equal homozygotes

Under the assumption that $\alpha = \gamma$ and $\sigma_1 = \sigma_3$, condition (5) is satisfied for all β and σ_2 , and F^* reduces to

$$F^* = \frac{\sigma_1(1-\alpha) - \sigma_2(1-\beta)}{\sigma_1(1-\alpha) - \sigma_2(1-\beta) + \sigma_2\beta - \sigma_1\alpha}$$

Manipulation of the sex ratio through infanticide

DARWIN (1871, p. 608) speculated that "There is reason to suspect that in some cases man has by selection indirectly influenced his own sex-producing powers." DARWIN believed that the tendency to give birth to more sons than daughters observed in certain cultures in India, New Zealand and the Hawaiian Islands reflected the selective pressures arising from the tradition of female infanticide which was practiced by those cultures in earlier times. In terms of our model, suppose that the frequency of males at birth in broods produced by *AA*, *Aa*, and *aa*

females are given by α_1 , β_1 and γ_1 , respectively, and that the frequency of males is adjusted in all broods to the value r through infanticide of females. If we assume that no reproductive compensation is practiced by the genotypes, then the fertilities of the genotypes are given by

$$\sigma_1 = \frac{\alpha_1}{r}, \quad \sigma_2 = \frac{\beta_1}{r} \quad \text{and} \quad \sigma_3 = \frac{\gamma_1}{r}. \quad (19)$$

In this case, the secondary frequencies of males are $\alpha = \beta = \gamma = r$ and (5) is satisfied for all r . F^* reduces to $(1-r)/(1-2r)$, which is never valid for r in $(0, 1)$. The problem reduces to a pure fertility model and the stability conditions (13), (14), and (19), indicate that the mean fertility will be maximized. Fertility as defined in (19) is an increasing function of the frequency in males at birth. Therefore, genotypes that produce more sons in a culture practicing infanticide of females will indeed be favored.

Control of the sex ratio through parental expenditure

FISHER (1930) introduced the factor of parental expenditure in offspring into the problem of sex-ratio evolution, stating that a population evolves under natural selection until the parental expenditure in female offspring is equal to the parental expenditure in male offspring. In the present genetical model, we use the economic concept of parental expenditure to construct a functional relationship between the sex ratio in a brood and the size of the brood. Specifically assume that the amount of parental expenditure required to rear a female offspring compared to the amount needed to rear a male offspring is Φ to 1, and consider the following relationship between the sex ratio in a brood and the relative size of the brood:

$$\sigma_1 = [\alpha + (1-\alpha)\Phi]^{-1}, \quad \sigma_2 = [\beta + (1-\beta)\Phi]^{-1} \quad \text{and} \quad \sigma_3 = [\gamma + (1-\gamma)\Phi]^{-1}. \quad (20)$$

Condition (5) for the existence of a symmetric root is always fulfilled under (20). This relationship between parental expenditure, sex ratio and brood size has been used by BODMER and EDWARDS (1960) and by SPIETH (1974).

The relationship (20) between brood size and brood sex ratio may be justified in a variety of ways. The concept common to all is that a limited amount of resources is allocated between the production of daughters and the production of daughters and the production of sons. TRIVERS' (1972) concept of "parental investment" is based on a similar assumption. BODMER and EDWARDS (1960) obtained (20) by calculating the reproductive value of offspring, defined as in (8), per unit parental expenditure. SPIETH (1974) assumed that the production of one female requires Φ units of parental capital relative to one male and called (20) the case of "constant total expenditure" among parental genotypes. Further, one may interpret (20) as reflecting the effects of preferential care shown to one sex by mothers in species which produce more offspring per brood than can possibly survive. Suppose females of all genotypes produce the same sex ratio and the same number of offspring at birth, but the genotypes display different propensities to care for offspring of one sex. At the end of the period of parental care, the brood

sizes among the maternal genotypes are given by (20), where Φ is a conversion factor that determines the number of daughters gained for each son lost.

According to FISHER (1930), if the amount of parental expenditure needed to rear a female offspring compared to a male offspring is Φ to 1, the population evolves to a state such that the population frequency of females is $1/(1 + \Phi)$, because at this value there is equal investment in male and female offspring. Substitution of the expressions (20) into (10) gives exactly the suggested value for F^*

$$F^* = \frac{1}{1 + \Phi} . \quad (21)$$

FISHER's (1930) principle concerning equal investments in males and females, represented by (20), is therefore equivalent to the maximization principles discussed earlier. Because F^* under the interpretation of parental expenditure must be positive, the population will either evolve to a state where the frequency of females is exactly F^* or converge towards a state where the deviation between the frequency of females and F^* is locally minimized.

Extensions to more general cases

Any set of fertilities σ_1 , σ_2 and σ_3 may arbitrarily be represented in the following form for some (not necessarily positive) values of Φ_1 , Φ_2 and Φ_3 :

$$\sigma_1 = [\alpha + (1-\alpha)\Phi_1]^{-1}, \quad \sigma_2 = [\beta + (1-\beta)\Phi_2]^{-1} \text{ and } \sigma_3 = [\gamma + (1-\gamma)\Phi_3]^{-1}. \quad (22)$$

The existence condition for the symmetric root, which can be written

$$(\Phi_1 - \Phi_2)(\beta - \gamma)(1 - \alpha) - (\Phi_3 - \Phi_2)(\beta - \alpha)(1 - \gamma) = 0, \quad (23)$$

is not satisfied in general. However, appealing to the small parameter theory of KARLIN and MCGREGOR (1972), we conclude that the stable equilibria (which include the asymmetric equilibria when they exist) of the special case considered in the present paper will correspond to stable equilibria in the more general model described by (12) for perturbations of the parameters in the special cases considered such that (23) is sufficiently small. Numerical iterations of the general case in which Φ_1 , Φ_2 and Φ_3 differ slightly from one another confirm that the qualitative behavior of the system is preserved. We therefore believe that condition (5) describes a special case rather than a degeneracy and that the qualitative insights gained through the analysis of the class of models considered in the present paper are applicable to more general cases.

DISCUSSION

In an effort to interpret FISHER's (1930) argument concerning the role of parental expenditure in the evolution of the sex ratio, SHAW and MOHLER (1953) constructed an expression for fitness that involves the relative contribution of individuals through sons and through daughters. BODMER and EDWARDS (1960) calculated the equilibrium sex ratio under the assumption that the Shaw-Mohler measure increases under natural selection. Proceeding from a similar expression for representational fitness, MACARTHUR (1965) used strategy argu-

ments to arrive at the conclusion that the product of the number of sons and the number of daughters evolves to a maximum. While it is clear that representational fitness as defined by SHAW and MOHLER (1953) is at least qualitatively related to genetic transmission, the exact nature of this relationship has not been fully explained previously. As discussed earlier, results derived from models involving autosomal sex-ratio loci are consistent with the predictions arising from the maximization principles, while results from models of sex-linked loci contradict these predictions. HAMILTON (1967) suggested that FISHER's (1930) argument applies only if the sex-ratio locus is transmitted in genetically equivalent forms through sons and daughters. It has been our purpose in the present paper to clarify the functional significance of the maximization principles to the evolution of the sex ratio in such transmission systems.

The genetic model we used allows the genotype at an autosomal locus in one parent to determine the brood sex ratio and the size of the broods produced by the mating. In agreement with HAMILTON (1967), HARTL and BROWN (1970), TRIVERS and HARE (1976) and CHARLESWORTH (1977), the results also apply to loci that are expressed in the diploid sex of haplodiploid organisms and to sex-linked loci that are expressed in the homogametic sex. A sex-ratio strategy in our model is characterized by a brood sex ratio and an associated relative fertility that may be a direct function of the brood sex ratio. We have shown that the sex ratios specified in our model under the two maximization principles are identical and that this sex ratio exists if condition (5) is satisfied. Under condition (5), the sex ratio suggested by the maximization principles corresponds exactly to the sex ratio at the asymmetric equilibria that are approached from all starting conditions whenever they exist. Further, the local stability conditions are consistent with the interpretation that natural selection leads to the local maximization of representational fitness as defined by SHAW and MOHLER (1953). Appealing to the small parameter theory of KARLIN and MCGREGOR (1972), we believe that our results are qualitatively correct for more general forms of the system which are "close" to the special case defined by (5), in the sense described earlier.

The relationship between brood sex ratio and fertility required by (5) has been shown to hold under a variety of reasonable biological assumptions. In particular, it is satisfied for the case of parental expenditure that was introduced by FISHER (1930). TRIVERS' (1972) concept of parental investment is closely related to FISHER's concept of parental investment. TRIVERS defined parental investment as any action on the part of the parent that increases the probability of survival to reproductive age of a given offspring at the cost of the reduction in the parent's ability to invest in other offspring. Implicit in both concepts are the assumptions that the resources available to the parent for reproduction are limited and that different patterns of allocation of those resources lead to differential reproductive success. While energy may often represent a good indicator of the allocation of investment, it should be recognized that parental expenditure/investment may depend on a variety of factors (TRIVERS 1972).

We interpret the case of parental expenditure in our model as follows:

$$\sigma_1[\alpha + (1-\alpha)\Phi] = \sigma_2[\beta + (1-\beta)\Phi] = \sigma_3[\gamma + (1-\gamma)\Phi] , \quad (24)$$

where Φ is a measure of parental expenditure on females relative to males. SPIETH (1974) termed (24) the constant total expenditure case and showed that MACARTHUR'S maximization criterion is satisfied for

$$F^* = \frac{1}{1 + \Phi} .$$

In fact, parental expenditure between the sexes is equalized at this frequency of females, as follows:

$$M^* = \Phi F^* .$$

F^* is exactly the frequency of females that characterizes the highly attracting asymmetric equilibria. Therefore, in our genetical model the frequency of females predicted by FISHER (1930) at which parental expenditure is equalized between the sexes is approached from all starting conditions.

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