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# PARENT-OFFSPRING CONFLICT OVER SEX RATIO. II. OFFSPRING RESPONSE TO PARENTAL MANIPULATION

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*Abstract.*—A long-term selection, three-locus diploid model is developed in order to analyze the joint evolution of the primary sex ratio among offspring, parental manipulation, and offspring response to parental manipulation when the cost of rearing a male offspring is different from the cost of rearing a female offspring. It is shown that, when the mother has enough information about the potential offspring response to manipulation, natural selection operates to increase offspring intolerance to sex conversion up to a level at which a mother will have either no power of manipulation (if the cost of losing an offspring is high) or only limited power of manipulation through, for example, selective abortion or neglect of male offspring (if the cost of losing an offspring is not too high). Quite paradoxically, when the information available to mothers on offspring tolerance of manipulation is limited, long-term natural selection operates to minimize the conflict up to the stage of full maternal control of the sex ratio.

In a previous article (Eshel and Sansone 1991), we studied a situation of parent-offspring conflict over the sex ratio in the brood, when the cost of rearing a male offspring is different from (say, $\lambda$ times) that of rearing a female offspring. In this case, Fisher (1958) argued that natural selection, in a panmictic, diploid, fully sexual population, should operate to adjust parents' investment so that the total expenditure incurred for offspring of each sex will be equal. This is so because, if the sex ratio is $1:\lambda'$, where $\lambda' \neq \lambda$, then each male in the population will have, on the average, $\lambda'$ times the number of offspring born to a female (since all males together have exactly the same number of offspring as all females together in the population). Hence, the expected number of grandoffspring resulting from a unit of investment in males is $\lambda':\lambda$ times the expected number of grandoffspring resulting from investing the same amount of resources in females. "Selection would thus raise the sex-ratio until the expenditure upon males became equal to that upon females" (Fisher 1958, p. 159).

As has been established by later quantitative studies, this argument turns out to be valid under somewhat more restrictive conditions. It proves false when sex is determined by a sex-linked locus (Hamilton 1967; Thomson and Feldman

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1975; Bengtsson 1977; Charlesworth 1977; Uyenoyama and Bengtsson 1981, 1982; Eshel and Feldman 1982*b*). Yet the bulk of the quantitative studies based on the exact dynamics of genetic models for diploid, panmictic, random-mating populations (Nur 1974; Uyenoyama and Bengtsson 1981; Eshel and Feldman 1982*a*; see also Charnov 1982; Karlin and Lessard 1984) validates Fisher's argument, at least concerning autosomal genes. Moreover, as has been suggested (Hamilton 1967) and later analyzed (Eshel 1984*a*, 1984*b*), it seems likely that the possibility that sex-linked genes substantially affect the sex ratio is minimized by the intervention of autosomal modifier genes. Another possible source of deviation from equal investment by parents in males and females has been suggested by Trivers and Willard (1973) when mothers of high status in a polygynous population are concerned. Indeed, a tacit assumption in Fisher's argument, as well as in the dynamic models following it, was that parents lack any information about the expected status of their offspring; hence natural selection can operate only on averages. Yet high status is crucially important for the reproductive success of males, and much less so for the reproductive success of females in a polygynous population. The argument of Trivers and Willard (see also Wilson and Pianka 1963; Trivers 1972) has been extended by Maynard Smith (1980) and later authors (Clutton Brock et al. 1981, 1982; Lloyd 1983; Frank 1987; Stamps 1990) to predict deviations from equal parental investment over the entire population toward higher investment in the sex whose variance in reproductive success is higher. For example, investment in males may be favored in polygynous populations and females, to a lesser extent, in monogamous populations in which reproductive success depends mainly on female fertility. That polygynous mothers do invest more in sons is well established by a growing body of evidence (Clutton Brock et al. 1982; Fiala and Congdon 1983; Lee and Moss 1986; Anderson and Fedak 1987; Teather and Weatherhead 1988; Wolff 1988; Boyd and McCann 1989; Le Boeuf et al. 1989; Gomendio et al. 1990; Yasukawa et al. 1990). But this phenomenon of extra investment in any single male offspring (relative to the investment in any female) does, indeed, create overall bias in favor of parental investment in males only if one assumes an even sex ratio at birth, as tacitly assumed by Maynard Smith. Yet, if this were the case, Fisher's argument would remain true concerning the sex ratio at earlier stages. Hence, natural selection on the early stages of parental investment is likely to superimpose over the secondary sex ratio a bias toward female offspring that balances the extra investment in males at that stage, which keeps the average overall investment in both sexes equal. This prediction agrees with evidence of higher rates of male abortion (Crew 1954; Bacci 1965) and higher early mortality among male offspring in polygynous populations (Clutton-Brock et al. 1982; Burley 1986; Labov et al. 1986; Slagsvold et al. 1986; Wolff 1988; Krakov and Hoeck 1989; Le Boeuf et al. 1989; Teather and Weatherhead 1989).

As total parental investment in males and females evolves toward equality, the sex ratio in the population converges to $1:\lambda$, at which any male in the population has, on the average, $\lambda$ times the number of offspring born to any female in the population. This outcome occurs because, repeating the argument of Fisher, all males in the population together will have exactly the same number of offspring

as all females in the population. Hence, natural selection, when operating on sex determination in the offspring themselves, is likely to favor a deviation from the $1:\lambda$ sex ratio in favor of the sex that produces more offspring per capita. In fact, it can be shown (Trivers 1974; Eshel and Sansone 1991) that natural selection, when operating on autosomal genes that affect individual sex determination, operates to establish a sex ratio of $1:\sqrt{\lambda}$, which, for $\lambda \neq 1$, is indeed different from that favored by parental genes.

In our previous article (Eshel and Sansone 1991), we attempted to study the possible genetic dynamics of this parent-offspring conflict by resorting to an exact diploid, two-autosome locus, random-mating genetic model. In that model the primary sex ratio is determined by genes carried by the offspring at one locus. At the same time, genes carried at another locus determine parental (say, maternal) manipulation of the sex ratio within the brood. Maternal manipulation of the sex ratio is most commonly affected by differential abortion of the two sexes (see, e.g., Crew 1954; Bacci 1965; Krakov and Hoeck 1989) or, quite equivalently (with respect to the model), by a tendency to neglect or respond less to the begging and demands of offspring of the more costly sex (see Burley 1986; Labov et al. 1986; Slagsvold et al. 1986; Wolff 1988; Krakov and Hoeck 1989; Le Boeuf et al. 1989). Another apparently more economical, yet much less observed, mode of maternal manipulation may occur in a form of the forced sex conversion of individual offspring. In both cases we have assumed (Eshel and Sansone 1991) that maternal manipulation must be of some cost to the family, otherwise the conflict is indeed settled by parental full control of the sex ratio, a situation that has already been extensively studied in previous works (see, e.g., Uyenoyama and Bengtsson 1979; Eshel and Feldman 1982*b*). Thus, in the case of sex conversion, we have assumed that the viability, the sexual success, or the fertility of the sex-converted individual is reduced by a factor $1 - \theta$. Maternal manipulation through a differential rate of abortion (or infant mortality) can, therefore, be treated as a special case of sex conversion with $\theta = 1$. The loss of maternal investment with the death of an offspring (say, its cost) was assumed to be a positive number $c$, less than one.

We have shown that the two-locus fixations that are stable against all nonepistatic mutations are exactly those that determine a mother-offspring evolutionarily stable system (ESS) in an asymmetric population game (see Maynard Smith and Parker 1976; Maynard Smith 1977; Grafen and Sibly 1978; Selten 1980) determined by maternal and offspring payment functions that are analytically derived from the dynamics of the genetic model. Moreover, these formally derived payment functions can be interpreted in terms of inclusive fitness or some generalization of it (see, for comparison, Motro 1991, 1993; Matessi and Eshel 1992). We have shown also that for any values $\lambda$, $\theta$, and $c$, a unique ESS $(\hat{m}, \hat{x})$ exists, in which $0 < \hat{m} < 1$ is the primary proportion of males in the population and $0 \leq \hat{x} < 1$ is the rate of maternal manipulation. A specific value $0 < c^* < 1$, $c^* = c^*(\lambda, \theta)$, has been calculated so that, as the cost $c$, incurred by the death of an offspring, increases from 0 to $c^*$, the primary sex ratio $\hat{m}:(1 - \hat{m})$ decreases from $1:1$ to $1:\sqrt{\lambda}$, while the mother rate of manipulation decreases from $(\lambda - 1)(\lambda + \theta)^{-1}$ to zero. In this case the secondary sex ratio among adults increases from the

mother-desired sex ratio of $1:\lambda$ to the offspring-desired one of $1:\sqrt{\lambda}$ at $c^*$, from which point and above the mother does not interfere and the secondary sex ratio coincides with the primary one, both remaining $1:\sqrt{\lambda}$.

A crucial question, about which we have only speculated, is why the case of differential mortality of male and female offspring $\theta = 1$, though being the most expensive both in terms of parental investment and offspring mortality, is by far the most common in nature. Indeed, it seems quite obvious that, for both mother and offspring, the possibility of sex conversion is preferable to abortion. Moreover, in the case of sex conversion, it seems plausible that any biological factor that reduces mortality because of sex conversion will be favored by natural selection. Yet, in order for natural selection to operate on factors that affect the risk $\theta$ of sex conversion, one has to assume some inherited variance with respect to this parameter among offspring primarily determined as males. Moreover, if the parameter $\theta$ can be estimated with partial success by the mother (say, through the correlation of $\theta$ with some detectable feature of the offspring), there appears to be an obvious selective advantage for maternal strategy of first manipulating those male offspring that are likely to manifest low values of $\theta$. This in turn endows high values of $\theta$ with an apparent selective advantage since male offspring with high $\theta$ are less likely to be manipulated. The question is, then, Under what condition can this advantage overbalance the even more apparent disadvantage of running a higher risk when manipulated? Under what conditions is natural selection likely to increase rather than decrease the risk of sex conversion up to the value $\theta = 1$ of differential offspring mortality?

In this work we attempt to study this question by analyzing genetic dynamics at a third locus, determining intolerance of male offspring to maternal manipulation. More specifically, we assume that the relative loss of fitness $\theta$ due to sex conversion is different from one individual male offspring to another. We refer to it as to the individual "maleness" in the sense that individual male offspring with higher so-called maleness are those that suffer higher risk when converted to females. The maleness of a male offspring (i.e., its specific $\theta$ value) is determined by genes it carries in its third locus. Finally, we assume that the maleness of the offspring is at least partly detectable by the mother. In developing the model and studying mutual effects of natural selection at the three loci, we follow all the assumptions of our previous model regarding the first two loci, except that we now assume that the proportion $x$ of male offspring manipulated by the mother is not just chosen at random. Instead, the mother's choice is made according to some probabilistic information she has about the offspring maleness $\theta$, which is in turn determined by genes this offspring carries at the third locus.

We see that, if maternal detection ability is sufficiently high and if the cost of losing an offspring is not higher than the critical value $c^*$, then natural selection on the third locus will operate even to increase the level of maleness up to $\theta = 1$. If maternal detection ability is high but the cost of losing an offspring is not sufficiently low, natural selection on the third locus will operate to increase the level of maleness to a point (less than one) above which maternal manipulation is disfavored. If maternal detection ability is low, natural selection will operate to decrease the level of maleness of male offspring to a level at which, paradoxi-

cally, the mother has full control of the sex ratio in her progeny. Yet it is argued that, under plausible (though maybe not universal) conditions, natural selection tends to increase maternal detection ability, which thus pushes the population to a situation in which the level of conflict is maximal and maternal control is minimal. The findings of the model are discussed in light of Zahavi's handicap principle (see Zahavi 1977*a*, 1977*b*, 1981).

As in our previous work, long-term fixation stability at the third locus can be interpreted in terms of an ESS in a population game with a male offspring payment function that can formally be derived from the dynamics of the genetic model.

## THE MODEL

We assume a diploid, random-mating population in which the cost of rearing a male offspring is $\lambda$ times the cost of rearing a female ($\lambda > 1$). The sex of an offspring is primarily determined by its own genotype, yet we assume that the mother can change the sex ratio within her brood by either aborting or, if possible, forcing a sex conversion on some proportion of male offspring. We assume that maternal manipulation causes a reduction by a factor $1 - \theta$ in the viability of the offspring ($0 \leq \theta \leq 1$); in particular, the case $\theta = 1$ corresponds to differential abortion or mortality by other means. Unlike our previous view, we assume that $\theta$ is different from one individual male offspring to another, and we refer to it as the individual maleness. Finally, we assume that the loss in maternal investment upon the death of male offspring is $c$ times, $0 < c < 1$, that of a female offspring.

We assume that the primary sex determination depends, in a probabilistic sense, on alleles carried by the offspring at locus $A$. Maternal behavior is determined by alleles she carries at locus $B$. Finally, the maleness $\theta$ of a male offspring (i.e., its intolerance to sex conversion) is determined by alleles it carries at locus $D$. We assume no epistatic effects of these alleles, so that the level of manipulation by a mother is not affected by alleles she carries at the $A$ and $D$ loci, and the primary sex and maleness of an offspring is not affected by alleles it carries in the $B$ locus. As long as the mother can detect offspring maleness, natural selection will indeed operate in favor of maternal strategies that minimize a loss of offspring by manipulating only those who are estimated of lower maleness (for a situation in which this may not be the case, however, see the Discussion). We assume that the mother can detect the value $\theta$ up to a certain variance $\sigma^2 > 0$. Specifically, the mother's estimate $\tilde{\theta}$ is a random variable:

$$\tilde{\theta} = \theta + \sigma Y, \tag{1}$$

where $Y$ is a continuous random variable with a distribution function $F(y) = p(Y \leq y)$ and density $f(y) = F'(y)$, a mean $EY = 0$, and a variance $EY^2 = 1$. The function $f$ may be assumed to be the normal density function or any other density function that is positive for any $y$ with a connected support. Also, $\sigma$ is referred to as maternal estimation error.

A typical maternal strategy determined by the maternal alleles at locus $B$ is, therefore, to manipulate all male offspring of an estimated maleness $\tilde{\theta}$ lower than a critical value $0 \leq u \leq 1$. We refer to this as the maternal threshold strategy.

If an offspring's maleness is $\theta$, then the chance of his being manipulated by a mother with a threshold strategy $u$ is

$$x = p(\theta + \sigma y \leq u) = p\left(y \leq \frac{u - \theta}{\sigma}\right) = F\left(\frac{u - \theta}{\sigma}\right). \tag{2}$$

Finally, we assume that each mother has a fixed amount of resources that is the only factor limiting the size of her brood (see, e.g., Trivers 1972). Thus, if the primary sex ratio in the brood is $m:(1 - m)$, if all male offspring have the same maleness $\theta$, and if the maternal strategy is $u$ with $x = F((u - \theta)/\sigma)$, then the average maternal investment in an offspring (alive or dead) in the brood is

$$W = (1 - x)m\lambda + x\theta mc + (1 - \theta)mx + 1 - m. \tag{3}$$

Using the notation of the previous article,

$$\begin{aligned} a &= \lambda - 1 \\ b &= a + (1 - c)\theta = \lambda - 1 + (1 - c)\theta, \end{aligned} \tag{4}$$

and equation (3) becomes

$$W = 1 + [a - bx(\theta)]m = W_{u,m}(\theta). \tag{4a}$$

Assume now fixation at the $A$ and $B$ loci (hence fixed values for $m$ and $u$) but polymorphism at the $D$ locus. Let different offspring in the brood carry different combination of alleles $D_iD_j$ $(i, j = 1, \ldots, n)$ at the $D$ locus, with the frequency of the $D_iD_j$ combination at the brood $2p_{ij} \geq 0$, if $i \neq j$, and $p_{ii} > 0$, if $i = j$ (note that, under fixation at the $A$ locus, these frequencies are the same for male and female offspring), $\Sigma_1^n ijp_{ij} = 1$; and let the level of maleness, determined by this combination, be $\theta_{ij}$. Then the average maternal expenditure is

$$W = W_{u,m}(\underline{\theta}, \underline{p}) = \sum_1^n ijp_{ij}W_{u,m}(\theta_{ij}). \tag{5}$$

The total number of adult $D_iD_j$ male offspring emerging from the brood will then be

$$M_{ij} = \frac{R}{W(\underline{\theta}, \underline{p})}p_{ij}(1 - x_{ij})m, \tag{6}$$

where $x_{ij} = F((u - \theta_{ij})/\sigma)$ and $R$ is the total amount of resources available for the mother.

The total number of adult $D_iD_j$ females (either originally so or successfully converted males) will be

$$F_{eij} = \frac{R}{W(\underline{\theta}, \underline{p})}p_{ij}[1 - m + mx_{ij}(1 - \theta_{ij})]. \tag{7}$$

Indeed, the total numbers of male and female offspring emerging from the brood are proportional to

$$M = \sum_{1}^{n} ijp_{ij}M_{ij} \quad \text{and} \quad F_e = \sum_{1}^{n} ijp_{ij}F_{eij}.$$

As in the previous article, we are interested in a multilocus (in this case three-loci) fixation of the alleles $A$, $B$, and $D$, determining primary sex ratio $m$, maternal threshold strategy $u$, and maleness $\theta$, respectively, that will be simultaneously stable against any mutation or a combination of them at the three loci.

It has been shown (Matessi and Eshel 1992) under similar conditions that, if the effect of the mutation at each locus is weaker, in order of magnitude, than the rate of recombination among any two loci (i.e., if we restrict our interest to mutations of relatively small effect in loci that are not fully linked as a supergene), then the conditions for stability against any combination of mutations at the three loci are equivalent to the three conditions for stability at each locus separately, given the fixation at the other two.

More specifically, it can be shown that those eigenvalues of the matrix of local analysis that belong to the frequencies of double or triple mutations are of the form $(1 - r_i)L(\omega)$, where $r_i$ is the appropriate rate of recombination, and $L(\omega)$ tends to one as the effect of a single mutation, in terms of selection forces $\omega$, tends to zero. Hence, for nonzero rates of recombination and small-effect mutations, these eigenvalues are always less than one in absolute value, and the eigenvalues that determine stability of the three loci are those of the three matrices of local analysis for each locus separately, taking into account the parameters determined by fixation at the other loci.

Being interested in fixation stability in the three loci, we start by assuming fixation of alleles $A$, $B$, and $D$, respectively, at the appropriate three loci, such that $DD$ determines a level of maleness $0 \leq \theta \leq 1$, while $AA$ and $BB$, respectively, determine a primary sex ratio $m^*:(1 - m^*)$ and a maternal threshold strategy $u^*$ ($m^* = m^*(\theta)$, $u^* = u^*(\theta)$), which, given $\theta$, are stable against any nonepistatic mutation $A \rightarrow a$ and $B \rightarrow b$. We then look for a specific value $\hat{\theta}$, for which the triplet $\hat{u} = u^*(\hat{\theta})$, $\hat{m} = m^*(\hat{\theta})$, $\hat{\theta}$ is stable. Note that, in the case of fixation at the third locus (on whatever value of $\theta$), the maternal threshold strategy $u$, determining a rate of maternal manipulation

$$x = F\left(\frac{u - \theta}{\sigma}\right), \tag{8}$$

is equivalent to a maternal strategy of just manipulating a proportion $x$ of the male offspring, chosen at random. We can, therefore, restrict our attention to such triplets ($m^*$, $u^*$, $\theta$) in which, for the given value of $\theta$, the values $m^* = m^*(\theta)$ for the primary frequency of males and $x^* = x^*(\theta) = F([u^*(\theta) - \theta]\sigma^{-1})$ for the maternal strategy satisfy the mutual stability conditions analyzed in the previous article.

While the primary frequency of males ($m^*$) and maternal manipulation level ($x^*$) are determined entirely by $\lambda$ and $c$ per a given value of $\theta$, the maternal

threshold strategy $u^* = u^*_\sigma(\theta)$ must adjust itself to the error $\sigma$ and to the distribution $F$, in order to satisfy

$$x^*(\theta) = F\left(\frac{u^*(\theta) - \theta}{\sigma}\right). \tag{8'}$$

### OFFSPRING'S RESPONSE TO PARENTS' MANIPULATION: THE EVOLUTION OF A STABLE LEVEL OF MALENESS

We start from any fixation $A$, $B$, $D$ at the three loci, determining strategies $m$, $u$, and $\theta$, respectively. We then introduce a mutation $D \to d$ at the third locus, assuming that the maleness of $Dd$ is $\theta'$ close to $\theta$.

Let $\epsilon > 0$ and $\delta > 0$ be the proportions of the mutant $Dd$ among adult males and females, respectively, with $\epsilon'$ and $\delta'$ the proportions of this genotype among males and females of the next generation. By straightforward calculations, one can verify

$$\frac{\epsilon' + \delta'}{\epsilon + \delta} = \frac{W}{W + W'}\left[\frac{1 - x'}{1 - x} + \frac{1 - m + (1 - \theta')mx'}{1 - m + (1 - \theta)mx}\right] = K(\theta', \theta), \tag{9}$$

where

$$W = 1 + (a - bx)m, \qquad W' = 1 + (a - b'x')m \tag{10}$$

and

$$x' = F\left(\frac{u - \theta'}{\sigma}\right), \tag{11}$$

which is the probability of maternal manipulation of mutant offspring. For the proof of equations (9)–(11), refer to Appendix A.

Note that the representation at the right side of equation (9) is possible since $x$ and $x'$, $W$ and $W'$ are given by equations (2) and (11) as functions of $m$, $u$, $\theta$, and $\theta'$.

The fixation of the allele $D$ is stable against the specific mutation $D \to d$ if $(\epsilon' + \delta') < (\epsilon + \delta)$ or equivalently if, given $u$ and $m$,

$$K(\theta', \theta) < 1 \tag{12}$$

and only if

$$K(\theta', \theta) \leq 1, \tag{12'}$$

where $K(\theta', \theta)$ is given by equation (9).

But, from equations (9)–(11), it follows that

$$K(\theta, \theta) = 1. \tag{13}$$

Hence, a necessary condition for a third locus fixation stability on an allele $D$, determining a value $\hat{\theta}$ of carrier's maleness, is that, for all $\theta' \neq \hat{\theta}$,

$$K(\theta', \hat{\theta}) \leq K(\hat{\theta}, \hat{\theta}) \tag{14}$$

(a sufficient condition is that eq. [14] will hold as a strict inequality). In other words, $\hat{\theta}$ is the best reply against itself in a two-player symmetric game in which $K(\theta', \theta) = K_{m,u}(\theta', \theta)$, as given by equation (9), is the individual payment function to be maximized by each player.

One can thus interpret $K_{m,u}(\theta', \theta)$ as the male offspring's payment function, determined by natural selection (see, for comparison, Eshel and Feldman 1991; Eshel and Sansone 1991; Motro 1991, 1993; Matessi and Eshel 1992). Note that the term $(1 - x')(1 - x)^{-1}$, in the definition (9) of $K_{m,u}(\theta', \theta)$, is proportional to the expected number of grandoffspring born to sons of a mutant of maleness $\theta'$, different from $\theta$, which thus determines a value $x'$ different from $x$ ($m$ and $u$ being fixed). The term $(1 - m + [1 - \theta']mx')(1 - m + [1 - \theta]mx)^{-1}$ is proportional to the number of grandoffspring born to daughters of such a mutant. The factor $2W/(W + W')$, multiplying the sum of this term in equation (9) and which we refer to as the kin selection factor, measures the amount by which a mutant individual, with a single, normal sib, increases (or decreases) the expected parents' investment. In this sense, $K_{m,u}(\theta', \theta)$ can be freely interpreted in terms of inclusive fitness (Hamilton 1964, 1972). Note that, not surprisingly, the payment function $K$ of a male offspring changing his level of maleness as given by equation (9) is equivalent, in this biological interpretation, to the payment function $G(m'; m, x)$ of a newborn offspring changing its probability of being a male as given in our previous article (see eq. [B11] of App. B). The difference between the formulas is, indeed, due to the parameters being changed.

From equation (9) it follows, moreover, that (given $m$ and $u$), if $(\partial K/\partial\theta')_{\theta'=\theta} > 0$, natural selection will operate in favor of small-effect mutations that increase the maleness and against those that decrease it. The opposite is true if $(\partial K/\partial\theta')_{\theta'=\theta} < 0$.

In order to check this, we now distinguish between two cases:

$$\text{Case 1: } c < \frac{1}{2}\sqrt{\lambda}(\sqrt{\lambda} - 1) = c^*(1)$$

(note that this case is characterized by the sharp inequalities $x^*(\theta) > 0$ and $B^*(\theta) < 0$, for all $0 \leq \theta \leq 1$) and

$$\text{Case 2: } c \geq \frac{1}{2}\sqrt{\lambda}(\sqrt{\lambda} - 1) = c^*(1).$$

Obviously case 1, which may be typical of early, low-cost maternal manipulation, is more relevant to the biological problem with which this article is concerned, and we start from it.

*Proposition 1*

With the assumption of the model one can prove proposition 1: If $c < 1/2\sqrt{\lambda}(\sqrt{\lambda} - 1) = c^*(1)$ then two positive values $\sigma_0$ and $\sigma_1$ exist, $\sigma_1 > \sigma_0 > 0$, such that (i) if $\sigma < \sigma_0$ (i.e., if the maternal estimation error is sufficiently low), then, for all $0 \leq \theta < 1$, the triplet $m^*(\theta)$, $x^*(\theta)$, $\theta$ is unstable to the mutation $\theta \rightarrow$

$\theta'$ when $\theta'$ is slightly larger than $\theta$. Then (ii), if $\sigma > \sigma_1$, the triplet $m^*(\theta)$, $x^*(\theta)$, $\theta$, where $\theta > 0$, is unstable to the mutation $\theta \to \theta'$ when $\theta'$ is slightly lower than $\theta$.

Proposition 1 states that, under the assumption $c < c^*(1)$ of case 1, long-term natural selection pushes the male offspring maleness all the way to one if the maternal estimation error is low and all the way to zero if the maternal estimation error is high. Here long-term selection means the slow process in which new mutations, favored by natural selection, repeatedly replace each other at the third locus, at least as long as $\theta$ has not yet reached its extreme value favored by natural selection (see Discussion). This in turn allows for adjustment, because of mutation and selection at the first two loci as well as at the third locus (see Eshel 1991). In these two cases, the triplets $m^*(1)$, $x^*(1)$, 1 and $m^*(0)$, $x^*(0)$, 0, respectively, have the property of global evolutionary genetic stability (EGS; Eshel and Feldman 1982*a*, 1984).

We thus see that, in case 1 (early maternal manipulation and low cost $c$ of losing an offspring), if the maternal error is small, long-term natural selection stabilizes the value $\theta = 1$ of pure abortion. If the maternal error is large, long-term natural selection will stabilize the value $\theta = 0$ of full maternal control over the sex ratio in her brood. In neither of these two cases will actual sex conversion evolve.

For the sake of completeness we now extend our analysis to the range

$$c \geq \frac{1}{2}\sqrt{\lambda}(\sqrt{\lambda} - 1) = c^*(1).$$

This case is characterized by a restriction of the range of conflict to the interval $0 < \theta \leq \theta_c$, where $\theta_c < 1$. As the offspring maleness reaches the critical value $\theta_c$ and above, maternal manipulation becomes disadvantageous and the sex ratio comes to be determined by the offspring alone. Since $F([u^*(\theta_c) - \theta_c]\sigma^{-1}) = x^*(\theta_c) = 0$, it is possible that $f_\theta^* \to 0$ as $\theta \to \theta_c$, and a result somewhat weaker than the previous one can be proved for this case.

*Proposition 2*

If $c \geq c^*(1)$ and hence $x^*(\theta) = 0$ for all $\theta_c \leq \theta \leq 1$, $\theta_c \leq 1$, then for any level of maleness $0 < \theta \leq \theta_c$ there are two positive values $\sigma^\theta$ and $\sigma_\theta$, $\sigma^\theta > \sigma_\theta > 0$, such that, if $\sigma < \sigma_\theta$, the triplet $m^*(\theta)$, $x^*(\theta)$, $\theta$ is unstable to the third-locus mutation $\theta \to \theta'$, where $\theta'$ is slightly larger than $\theta$; and if $\sigma > \sigma^\theta$, the triplet $m^*(\theta)$, $x^*(\theta)$, $\theta$ is unstable to the third-locus mutation $\theta \to \theta'$, where $\theta'$ is slightly lower than $\theta$.

Appendix B contains the proofs of propositions 1 and 2.

Proposition 2 asserts only that, for any $\epsilon > 0$, there exists a value $\sigma_\epsilon$ such that, if $\sigma < \sigma_\epsilon$, long-term natural selection will bring $\theta$ to a value within an $\epsilon$ range from $\theta_c$ with maternal manipulation restricted to any low level; yet rare cases of sex conversions cannot be precluded by the model. On the other hand, large enough values of $\sigma$ only guarantee that long-term selection will decrease $\theta$ as

long as the level of maternal manipulation is not too low (say, $\theta$ close to $\theta_c$ and $x^*[\theta]$ close to zero).

This, indeed, may apply only to the case of prenatal manipulation, with a very small difference between the costs of rearing a male and female offspring.

It is worth mentioning that our analysis of either the case $c < c^*(1)$ or $c \geq c^*(1)$ is not complete since we do not know the behavior of the system for intermediate values of $\sigma$. It seems likely that natural selection on the other loci will operate to decrease $\sigma$ (i.e., to increase maternal estimation ability), although in some cases one cannot preclude the possibility that it works the other way round (see Discussion).

## DISCUSSION

This article, like our previous one (Eshel and Sansone 1991), studies long-term evolution of parent-offspring conflict over the sex ratio when the cost of rearing a male offspring is different from the cost of rearing a female. More specifically, by *long-term evolution* (see Eshel 1991) we mean the process of so-called trial and error in which the population is repeatedly shifting from the vicinity of one internally stable equilibrium (or fixation) to another, because of the selective establishment of new mutations periodically introduced into the population. Assuming availability of all sorts of relevant mutations during the long course of evolution (to be distinguished from the presence of mutations in the population at any given time), the long-term process has the potential to continue as long as the population has not yet reached the state of EGS (see Eshel and Feldman 1982*a*, 1984), say, a state that is stable against *all* possible mutations. As has been established in several previous works (Eshel and Feldman 1982*a*, 1984, 1991; Eshel 1991; Eshel and Sansone 1991; Matessi and Eshel 1992), the dynamics of this *long-term* process may be qualitatively different from that of the extensively studied process of changes in genotype frequencies within a given set of genotypes.

One crucial difference between the two is that an EGS equilibrium of the long-term process is never genetically unique (i.e., different sets of mutations may bring the population to different genetic set-ups of the EGS). Instead, the EGS, in contrast to the usual internal (say, short-term) genetic equilibrium, is characterized by a typical phenotype or a distribution of phenotypes that, once determined by any distribution of genotypes, is stable against any nonepistatic mutation that may affect the phenotype in question. Moreover, in many cases (as in Eshel and Sansone 1991 as well as in this work), the EGS distribution of phenotypes can be formally interpreted in terms of an ESS of a population game in which the payment function is analytically drawn from the (long-term) dynamics rather than assumed, as is the case in traditional ESS models.

Another important difference, with respect to the biological aspects of the two processes, has to do with their different predictions concerning the immediate population response to a direct selection pressure. Indeed, any attempt to explain an evolutionary phenomenon on the basis of changes of frequencies within a

given set of genotypes represented in the population must be based on the assumption that this set of genotypes is rich and varied enough as to allow for a direct response to selection pressure imposed on the population at any given time. In some cases, though, empirical results supporting the opposite prove rather problematic to evolutionary arguments based on this sort of model. This is the case with the observation (brought to our attention by an anonymous referee) that attempts to modify the sex ratio by animal breeders have been almost entirely unsuccessful (Clutton-Brock and Iason 1986). This, on the other hand, is quite expected if one tends to assume that the long-term evolution of a phenomenon like the sex ratio is more likely to take place because of the successive introduction of mutations, each quite likely of small effect, that shift the population from one nearly monomorphic state to another.

Moreover, a factor that may contribute to the stability of the sex ratio in real populations is the apparently harmful pleiotropic effects concerning sex conversions. Indeed, in considering the evolution of sex ratio, one should not overlook technical and structural restrictions of this sort, even though they may be less crucial in long-term evolution, during which mutations with weaker pleiotropic effects may appear. Perhaps more important is the possibility, analyzed in the present work, that these very structural restrictions that govern selection at one locus are likely to result in turn from mutual adjustment and long-term selection at another locus. Indeed, the evolution of what we have referred to as maleness may account for the manifestation of some structural restrictions of this sort.

The results of this work indicate two apparent paradoxes. First, under the assumption that the mother has sufficiently high estimation ability of (or that she reacts accordingly to) some detectable feature of her male offspring that affects their ability to survive her manipulation, natural selection operates to decrease rather than increase the survival ability of manipulated offspring. Second, as the accuracy of the mother's estimation increases, the level of her conflict with her male offspring increases and, more surprisingly, her ability to manipulate the sex ratio decreases either up to a level of full offspring control of the sex ratio (if the cost of the offspring's death is sufficiently high) or at least to a level at which maternal manipulation is minimal and can be executed only in the way that is the most costly for both her and the manipulated offspring: abortion of a certain quota of male offspring. On the other hand, only when maternal estimation error is sufficiently high will natural selection on offspring intolerance to manipulation operate to decrease the intensity of the conflict to a level at which it is the mother who gains full control over the sex ratio in her brood.

Both of these seeming paradoxes can be resolved by appeal to Zahavi's handicap principle (1977*a*, 1977*b*; 1981). In both cases some advantages are shown to follow from an apparent handicap, on the part of either male offspring (higher mortality rate under parental manipulation) or the parents (low detection ability). Yet the analysis of the model indicates a crucial difference between the two. While the advantage of male offspring with higher detectable intolerance to maternal manipulation (higher maleness) is shown to be directly favored by natural selection, no such selection mechanism has been demonstrated with respect to

low maternal detection ability. Indeed, the fact that such a shortcoming (handicap) on the part of mothers would endow them, in the long course of evolution, with some advantages over their offspring does not guarantee a selection mechanism in its favor (in fact, we believe that this sort of distinction should be made also in respect to other arguments concerning the handicap principle). The difference between the two cases stems from the assumption that a mother may (and, in fact, is likely to) have some ability to detect certain features of her male offspring that affect their intolerance to manipulation. As long as this is the case, a discriminating maternal manipulation of those male offspring that appear more able to withstand it, indeed, creates a selection force in favor of high offspring intolerance to manipulation (maleness). Under the condition analyzed in this work, this selection force is shown to be sufficient to overcome the apparent advantage of low maleness in manipulated males.

On the other hand, no similar ability has been assumed on the part of the offspring. We believe that, under plausible conditions, one can safely assume that offspring, especially in the prenatal stage, are most unlikely to detect variations in maternal estimation ability, and they are even less likely to have at their disposal the phenotypic flexibility required to adjust their own maleness in response to the specific detection abilities of their mothers. We therefore did not assume such flexibility on the part of the offspring.

The situation may be different at later, say, postnatal, stages of the conflict, when offspring detection ability appears more feasible. Yet it seems that sex conversion at this stage is not feasible because of obvious biological restrictions. Note, however, that with the appropriate choice of $\lambda$, $c$, and $\theta = 1$, the dynamics of possible postnatal parental manipulation of the sex ratio becomes quantitatively equivalent to selective abortion of males (Eshel and Sansone 1991). Moreover, in this connection, the expected failure of a male offspring to find a future mate, as a result of inadequate parental investment, is indeed equivalent to a genetic death (Maynard Smith 1980). Thus, with the relatively high value of $c$ involved in such a sort of genetic death, our model, in agreement with that of Maynard Smith, predicts full offspring control of the sex ratio (provided, of course, that parental manipulation did not occur before). This, however, may not be the case when parental manipulation is limited to discrimination against the more costly male offspring at times of starvation (see many references above). In such a situation, the relevant cost $c$ of the death of a male offspring must not be measured in terms of parental resources already invested, since those are most likely to be lost in any case. Instead, it must be measured in terms of the difference between the loss of resources expected in this way and the loss expected under a perhaps more optimal allocation of parental resources (Dawkins 1976). Thus, $c$ may be quite small and parental allocation of resources may be much closer to 1:1.

The possibility that, under certain conditions, natural selection would operate in favor of low detection ability of one participant, to his own advantage, has already been suggested by Trivers (1985). While Zahavi (1977*a*, 1977*b*) emphasizes mainly the role of selection in improving the ability of participants to esti-

mate the situation (at least in a case of commonly repeating conflict), it seems to us that natural selection in favor of low detection ability, under certain conditions, may also be a typical manifestation of Zahavi's handicap principle. For example, a parental tendency to be absent at certain times, thus to avoid some crucial information about the offspring, can be encouraged by natural selection if the offspring are able to react accordingly and thus to reduce their own handicaps.

It is worth mentioning, though, that the process we are dealing with is likely to be multidimensional in the sense that, at least initially, the maternal tendency to selectively manipulate specific features of male offspring may be manifested in various stages and means of manipulation, corresponding to various detectable features of the offspring. We thus expect evolution to increase maternal estimation ability with respect to specific features of their male offspring that reduce its reproductive success if sex converted, are not phenotypically flexible, and are not determined by loci tightly linked to those responsible for maternal detection ability. A perfect candidate for such a feature may be the suppression of much of the Y chromosome, which makes a male-to-female conversion rather inefficient (in XY though not in OX systems, in which female-to-male conversion is more difficult to carry out; for a different explanation of the suppression of the Y chromosome, see Hamilton 1967). It is possible, though, that in some cases, when the above prerequisites are not satisfied, natural selection will operate just to reduce maternal estimation ability or, equivalently, maternal discrimination.

Note that in neither of the two cases (selection for high and, possibly, low maternal detection ability) is the establishment of a substantial rate of sex conversion to be expected, a finding that provides an answer to the question raised in our previous article. Instead, we see complete control by the mother (in the case of selection for low maternal detection ability); or, more likely, complete offspring control of the sex ratio (in the case of selection for high maternal detection ability and high enough cost $c$ of losing an offspring); or, possibly, an "in-between" sex ratio with maternal manipulation through selective abortion or neglect of male offspring (in the case of selection for high detection ability of the mother and relatively low cost $c$ of losing an offspring).

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## APPENDIX A

### Proof of Equation (9)

Assuming random mating, the frequency of the mating $Dd \times DD$ or its reciprocal is $(\epsilon + \delta) + o(\epsilon, \delta)$, whereas that of other matings involving the allele $d$ is of the negligible order of $o(\epsilon, \delta)$. Hence, the conditional probability that a $Dd$ offspring, either male or female, will come from a $Dd \times DD$ mating or its reciprocal is $1 + o(\epsilon, \delta)$, in which case

half of the sibs in the brood will be of genotype *Dd* and half will be of the common genotype *DD*. As a special case of equation (5), we therefore know that the average maternal expenditure for an offspring at this brood is

$$W_{u,m}\left(\theta, \theta'; \frac{1}{2}, \frac{1}{2}\right) = \frac{1}{2}(W + W'), \tag{A1}$$

where

$$W = 1 + (a - bx)m, \qquad W' = 1 + (a - b'x')m \tag{A2}$$

and

$$x' = F\left(\frac{u - \theta'}{\sigma}\right), \tag{A3}$$

which is the probability of maternal manipulation of mutant offspring.

From equations (6) and (7), the average numbers of adult male and female *Dd* offspring emerging from such a brood are

$$M' = R\frac{(1 - x')m}{W + W'}$$

and

$$F'_e = R\frac{1 - m + (1 - \theta')mx'}{W + W'}, \tag{A4}$$

respectively.

Using the same formula, we see that the average numbers of adult male and female offspring (all of genotype *DD*) emerging from a common, *DD* × *DD* brood are

$$M = R\frac{(1 - x)m}{W}$$

and

$$F_e = R\frac{1 - m + (1 - \theta)mx}{W}. \tag{A5}$$

Hence,

$$\epsilon' = (\epsilon + \delta)\frac{M'}{M} = \frac{W}{W + W'}\frac{1 - x'}{1 - x},$$

$$\delta' = (\epsilon + \delta)\frac{F'_e}{F_e} = \frac{W}{W + W'}\frac{1 - m + (1 - \theta')mx'}{1 - m + (1 - \theta)mx}, \tag{A6}$$

and

$$\frac{\epsilon' + \delta'}{\epsilon + \delta} = \frac{W}{W + W'}\left[\frac{1 - x'}{1 - x} + \frac{1 - m + (1 - \theta')mx'}{1 - m + (1 - \theta)mx}\right] = K_{m,u}(\theta', \theta). \tag{A7}$$

## APPENDIX B

### Proof of Propositions 1 and 2

In order to calculate $(\partial K/\partial\theta')_{\theta'=\theta}$, we define

$$\Psi = \frac{1 - x'}{1 - x}, \qquad \Phi = \frac{1 - m + (1 - \theta')mx'}{1 - m + (1 - \theta)mx}, \tag{B1}$$

and get

$$K = W[\Psi(\theta') + \Phi(\theta')](W + W')^{-1}. \tag{B2}$$

We now use equation (11) to establish

$$\left(\frac{\partial x'}{\partial \theta'}\right)_{\theta'=\theta} = \left[\frac{\partial}{\partial \theta'} F\left(\frac{u - \theta'}{\sigma}\right)\right]_{\theta'=\theta} = \sigma^{-1} f\left(\frac{u - \theta}{\sigma}\right). \tag{B3}$$

Employing equations (10) and (B3), we get

$$\begin{aligned}\frac{\partial}{\partial \theta'}\left(\frac{W}{W + W'}\right)_{\theta'=\theta} &= -(4W)^{-1}\left(\frac{\partial W'}{\partial \theta'}\right)_{\theta'=\theta} \\ &= \frac{1}{4W}\left\{\left[am\frac{\partial x'}{\partial \theta'} + (1 - c)m\left(\theta\frac{\partial x'}{\partial \theta} + x\right)\right]\right\}_{\theta'=\theta} \\ &= \frac{1}{4W}\left[-am\frac{f}{\sigma} + (1 - c)m\left(x - \theta\frac{f}{\sigma}\right)\right],\end{aligned} \tag{B4}$$

where $f = f((u - \theta)/\sigma)$.

In the same way we calculate at $\theta' = \theta$

$$\frac{\partial \Psi}{\partial \theta'} = \frac{1}{1 - x}\frac{f}{\sigma} \tag{B5}$$

and

$$\frac{\partial \Phi}{\partial \theta'} = -m\frac{x + (1 - \theta)f\sigma^{-1}}{1 - m + (1 - \theta)mx}. \tag{B6}$$

Finally, from equations (B3)–(B6) we get, by straightforward calculations,

$$\left(\frac{\partial K}{\partial \theta'}\right)_{\theta'=\theta} = Af\sigma^{-1} + B, \tag{B7}$$

where

$$A = A(m, x, \theta) = \frac{1}{2}\left[\frac{1}{1 - x} - \frac{m(1 - \theta)}{1 - m + (1 - \theta)mx}\right] - \frac{m}{2W}[a + \theta(1 - c)] \tag{B8}$$

and

$$B = B(m, x, \theta) = \frac{mx}{2}\left[\frac{1 - c}{W} - \frac{1}{1 - m + (1 - \theta)mx}\right]. \tag{B9}$$

Equalities (B7)–(B9) hold for any values of $x$ and $m$, that is, for any fixation of the triplet $m$, $x$, $\theta$. In considering the conditions for the mutual stability of the triplet $m^*$, $x^*$, $\theta^*$, one must first concentrate on the values $m^* = m^*(\theta)$ and $x^* = x^*(\theta)$, which, given $\theta$, are mutually stable against each other. We shall then ask about the selection forces operating on $\theta$, namely about the behavior of $K_{m^*, u(x^*)}(\theta', \theta)$ at the vicinity of $\theta' = \theta$. We shall be interested in such a value $\theta = \theta^*$ that, given $x^* = x^*(\theta)$, $m^* = m^*(\theta)$, will be the best reply against itself, that, say, maximizes $K_{m^*, u(x^*)}(\theta', \theta)$. Concerning the mutually stable pair $x^* = x^*(\theta)$, $m^* = m^*(\theta)$, we already know (Eshel and Sansone 1991) that, for any $0 < \theta \leq 1$, there is such a unique pair. Moreover, $m^*$, $x^*$ is the (unique) ESS of the asymmetric mother-offspring population game, determined by the payment functions

$$H(x'; m, x) = \frac{1}{2}\frac{1 + am - bmx}{1 + am - bmx'}\left[\frac{1 - x'}{1 - x} + \frac{1 - m + (1 - \theta)mx'}{1 - m + (1 - \theta)mx}\right] \tag{B10}$$

and

$$G(m'; m, x) = \frac{1}{2}\frac{W}{W + W'}\left[\frac{m'}{m} + \frac{1 - m' + (1 - \theta)m'x}{1 - m + (1 - \theta)mx}\right], \tag{B11}$$

of a mother that chooses a strategy $x'$ and an offspring that chooses a strategy $m'$, respectively, in a population in which the others choose the pair of strategies $(x, m)$, where $W = 1 + (a - bx)m$, $W' = 1 + (a - bx)m'$.

We know (Eshel and Sansone 1991) that a necessary and sufficient condition for no maternal interference ($x^* = 0$) to be an ESS is

$$c \geq \frac{1}{2\theta}(\sqrt{\lambda} - 1)(\sqrt{\lambda} + 1 - \theta) = c^*, \tag{B12}$$

which in the case $\theta = 1$ becomes

$$c \geq \frac{1}{2}\sqrt{\lambda}(\sqrt{\lambda} - 1) = c^*(1). \tag{B13}$$

When equation (B12) is not satisfied, $x^*$ is positive, and we know (Eshel and Sansone 1991) that

$$2\frac{m^*}{W^*}[a + \theta(1 - c)] + \frac{(1 - \theta)m^*}{1 - m^* + (1 - \theta)x^*m^*} - \frac{1}{1 - x^*} = 0, \tag{B14}$$

where $W^* = W^*_{u^*, m^*}(\theta) = 1 + (a - bx^*)m^*$, $u^* = u^*(\theta)$, as given by equation (4a) above.

Finally, from our previous article (Eshel and Sansone 1991), we know that

$$[1 - (1 - \theta)x^*]m^* = [2 + (a - bx^*)m^*]^{-1} = [1 + W^*]^{-1}. \tag{B15}$$

Employing equations (B14) and (B15), we get

$$A^* = A^*(\theta) = A[m^*(\theta), x^*(\theta), \theta] = \frac{m^*}{2W^*}[a + \theta(1 - c)] > 0 \tag{B16}$$

and

$$\begin{aligned} B^* &= B^*(\theta) = B[m^*(\theta), x^*(\theta), \theta] \\ &= \frac{m^*x^*}{2W^*}[(1 - c) - (W^* + 1)] = -\frac{m^*x^*}{2W^*}(W^* + c) \leq 0. \end{aligned} \tag{B17}$$

Strict inequality holds if $x^*(\theta) > 0$.

Equality (B7) thus becomes

$$\left[\frac{\partial K(m^*(\theta), x^*(\theta), x'(\theta, \theta'), \theta, \theta'}{\partial \theta'}\right]_{\theta' = \theta} = A^*f^*\sigma^{-1} + B^*, \tag{B18}$$

where $f^* = f\{[u^*(\theta) - \theta]\sigma^{-1}]\}$, $A^* = A^*(\theta) > 0$ and $B^* = B^*(\theta) \leq 0$, with $B^*(\theta) = 0$ if and only if $x^*(\theta) = 0$. We know that this is true if and only if equation (B12) holds. It is true for some nonempty interval $\theta_c \leq \theta \leq 1$, $\theta_c \leq 1$, if equation (B13) holds. But, under the condition $c < c^*(1)$, one can easily verify that $A^*(\theta) > 0 > B^*(\theta)$ for all $\theta$ on the closed interval [0, 1]. Since $u^*(\theta)$ is the solution of $x^*(\theta) = F([u^*(\theta) - \theta]\sigma^{-1})$, we already know that the value $[u^*(\theta) - \theta]\sigma^{-1}$, and, therefore, $f = f^*(u^*[\theta] - \theta)\sigma^{-1}$, depends on $\theta$ alone and is independent on $\sigma$. Moreover, since $x^*(\theta) > 0$ for all $0 \leq \theta \leq 1$ and $f(y)$ is a positive density, on the support $\{y: 0 < F(y) < 1\}$ of the distribution $F$ (indeed $x^*(\theta) < 1$ for all $\theta$), we know $f^* = f^*_\theta > 0$ for all $0 \leq \theta \leq 1$. Hence $A^*f^*$ has a positive minimum on $0 \leq \theta \leq 1$. From equation (B17) one can easily see that $-B^* < 1$ for all $\theta$ (indeed, $c < W^*$; $x^*$, $m^* < 1$). Hence, we define $\sigma_0 = \min\{f^*A^*, 0 \leq \theta \leq 1\}$ and (i) follows immediately from equation (B18).

In the same way, (ii) immediately follows from the choice $\sigma_1 = \max\{-A^*f^*B^{*-1}, 0 \leq \theta \leq 1\} > \sigma_0$. This completes the proof of proposition 1.

The proof of proposition 2 is immediate from equation (B18) and the fact that $A^*f^* > 0$, $B^* < 0$ for all $0 < \theta < \theta_c$ (indeed, for $\theta \geq \theta_c$, $A^*f^* = B^* = 0$ and natural selection does not affect $\theta$, which is, then, without any effect in the absence of maternal manipulation).

## LITERATURE CITED


Anderson, S. S., and M. A. Fedak. 1987. Grey seal, *Halychoerus grypus*, energetics: females invest more in male offspring. Journal of Zoology (London) 211:667–679.

Bacci, G. 1965. Sex determination. Pergamon, Oxford.

Bengtsson, B. O. 1977. Evolution of sex-ratio in the wood lemming. Pages 333–343 *in* F. B. Christiansen and T. M. Fenchel, eds. Measuring selection in natural populations. Springer, New York.

Boyd, I. L., and T. S. McCann. 1989. Pre-natal investment in reproduction by female antarctic fur seals. Behavioral Ecology and Sociobiology 24:377–385.

Burley, N. 1986. Sex-ratio manipulation in colour-banded populations of zebra finches. Evolution 40:1191–1206.

Charlesworth, B. 1977. Population genetics, demography and the sex-ratio. Pages 345–363 *in* F. B. Christiansen and T. M. Fenchel, eds. Measuring selection in natural populations. Springer, New York.

Charnov, E. L. 1982. The theory of sex allocation. Princeton University Press, Princeton, N.J.

Clutton-Brock, T. H., and S. D. Albon. 1982. Parental investment in male and female offspring in mammals. Pages 223–247 *in* King's College Sociobiology Group, eds. Current problems in sociobiology. Cambridge University Press, Cambridge.

Clutton-Brock, T. H., and G. R. Iason. 1986. Sex-ratio variation in mammals. Quarterly Review of Biology 61:339–373.

Clutton-Brock, T. H., S. D. Albon, and F. E. Guiness. 1981. Parental investment in male and female offspring in polygynous mammals. Nature (London) 289:487–489.

Clutton-Brock, T. H., F. E. Guiness, and S. D. Albon. 1982. Red deer: behavior and ecology of two sexes. University of Chicago Press, Chicago.

Crew, F. A. 1954. Sex determination. 3d ed. Methuen, London.

Dawkins, R. 1976. The selfish gene. Oxford University Press, Oxford.

Dawkins, R., and J. R. Krebs. 1979. Arms races between and within species. Proceedings of the Royal Society of London B, Biological Sciences 205:489–511.

Eshel, I. 1984*a*. On the evolution of intragametic conflicts. Journal of Theoretical Biology 108: 65–74.

———. 1984*b*. Are intragametic conflicts common in nature? are they important in the evolution of natural populations? Journal of Theoretical Biology 108:159–162.

———. 1991. Game theory and population dynamics in complex genetical systems: the role of sex in short term and in long term evolution. Pages 6–28 *in* R. Selten, ed. Game equilibrium models. Vol. 1. Evolution and game dynamics. Springer, Berlin.

Eshel, I., and M. W. Feldman. 1982*a*. On the evolutionary genetic stability of the sex-ratio. Theoretical Population Biology 21:430–439.

———. 1982*b*. On the evolution of sex determination and sex-ratio in haplodiploid populations. Theoretical Population Biology 21:440–450.

———. 1984. Initial increase of new mutants and some continuity properties of ESS in two locus systems. American Naturalist 124:631–640.

———. 1991. The handicap principle in parent-offspring conflict: comparison of optimality and population-genetic analyses. American Naturalist 137:167–185.

Eshel, I., and E. Sansone. 1991. Parent-offspring conflict over the sex-ratio in a diploid population with different investment in male and in female offspring. American Naturalist 138:954–972.

Fiala, K. L., and J. D. Congdon. 1983. Energetic consequences of sexual size dimorphism in nestling red-winged blackbirds. Ecology 64:642–647.

Fisher, R. A. 1958. The genetical theory of natural selection. 2d ed. Dover, New York.
Frank, S. A. 1987. Individual and population sex allocation patterns. Theoretical Population Biology 31:1–28.
Gomendio, M., T. H. Clutton-Brock, S. D. Albon, F. E. Guiness, and M. J. A. Simpson. 1990. Mammalian sex ratios and variation in the costs of rearing sons and daughters. Nature (London) 343:261–263.
Grafen, A., and R. Sibly. 1978. A model of mate selection. Animal Behaviour 26:645–652.
Hamilton, W. D. 1964. The genetical evolution of social behavior. Journal of Theoretical Biology 7:1–16.
———. 1967. Extraordinary sex ratios. Science (Washington, D.C.) 156:477–488.
———. 1972. Altruism and related phenomena, mainly in the social insects. Annual Review of Ecology and Systematics 3:193–232.
Karlin, S., and S. Lessard. 1984. On the optimal sex-ratio: a stability analysis based on characterization for one locus multiallele viability models. Journal of Mathematical Biology 20:15–38.
———. 1986. Theoretical studies on sex-ratio evolution. Princeton University Press, Princeton, N.J.
Krackov, S., and H. N. Hoeck. 1989. Sex-ratio manipulation, maternal investment and behaviour during concurrent pregnancy and lactation in house mice. Animal Behaviour 37:177–186.
Labov, J. B., U. W. Huck, P. Vaswani, and R. D. Lisk. 1986. Sex-ratio manipulation and decreased growth of male offspring of undernourished golden hamsters. Behavioral Ecology and Sociobiology 18:241–249.
Le Boeuf, B. J., R. Condit, and J. Reiter. 1989. Parent investment and the secondary sex-ratio in northern elephant seals. Behavioral Ecology and Sociobiology 25:109–117.
Lee, P. C., and C. J. Moss. 1986. Early maternal investment in male and female African calves. Behavioral Ecology and Sociobiology 18:353–361.
Lloyd, D. G. 1983. Evolutionary stable sex ratios and sex allocations. Journal of Theoretical Biology 105:525–539.
Matessi, C., and I. Eshel. 1992. Sex-ratio in social Hymenoptera: a population-genetics study of long-term evolution. American Naturalist 139:276–312.
Maynard Smith, J. 1977. Parental investment: a prospective analysis. Animal Behaviour 25:1–9.
———. 1980. A new theory of sexual investment. Behavioral Ecology and Sociobiology 7:247–251.
Maynard Smith, J., and G. A. Parker. 1976. The logic of asymmetric contest. Animal Behaviour 24:159–175.
Motro, U. 1991. Avoiding inbreeding and sibling competition: the evolution of sexual dimorphism for dispersal. American Naturalist 137:108–115.
———. 1993. Helpers at parents' nest: a game theoretic approach. Journal of Theoretical Biology 163:127–134.
Nur, U. 1974. The expected changes in the frequency of alleles affecting the sex-ratio. Theoretical Population Biology 5:143–147.
Selten, R. 1980. A note on evolutionary stable strategies in asymmetric animal conflict. Journal of Theoretical Biology 83:93–101.
Slagsvold, T., E. Roskaft, and S. Engen. 1986. Sex-ratio, differential cost of rearing young and differential mortality between the sexes during the period of parental care: Fisher's theory applied to birds. Ornis Scandinavica 17:117–125.
Stamps, J. A. 1990. When should avian parents differentially provision sons and daughters? American Naturalist 135:671–685.
Teather, K. L., and P. J. Weatherhead. 1988. Sex-specific energy requirements of great-tailed grackle nestlings. Journal of Animal Ecology 57:659–668.
Thomson, G. J., and M. W. Feldman. 1975. Population modifiers of meiotic drive. IV. On the evolution of sex-ratio distortion. Theoretical Population Biology 8:202–211.
Trivers, R. L. 1972. Parental investment and sexual selection. Pages 136–179 *in* B. Campbell, ed. Sexual selection and the descent of man. Aldine, Chicago.
———. 1974. Parent offspring conflict. American Zoologist 14:249–264.

———. 1985. Social evolution. Cummings, Menlo Park, Calif.

Trivers, R. L., and D. E. Willard. 1973. Natural selection of parental ability to vary the sex-ratio of offspring. Science (Washington, D.C.) 179:90–91.

Uyenoyama, M. K., and B. O. Bengtsson. 1979. Toward a genetic theory for evolution of the sex-ratio. Genetics 93:721–736.

———. 1981. Toward a genetic theory for the evolution of sex-ratio. II. Haplodiploid and diploid models with siblings and parental control of the brood sex-ratio and brood size. Theoretical Population Biology 20:57–79.

———. 1982. Toward a genetic theory of the sex-ratio. III. Parental and sibling control of brood investment ratio under partial sib-mating. Theoretical Population Biology 22:43–68.

Wilson, M. F., and E. F. Pianka. 1963. Sexual selection, sex-ratio and mating system. American Naturalist 97:405–407.

Wolff, J. O. 1988. Maternal investment and sex-ratio adjustment in American bison calves. Behavioral Ecology and Sociobiology 23:127–133.

Yasukawa, K., J. L. McClure, R. A. Boley, and J. Zanocco. 1990. Provisioning of nestlings by male and female red-winged blackbirds. Animal Behaviour 40:153–166.

Zahavi, A. 1977*a*. Reliability in communication systems and the evolution of altruism. Pages 253–259 *in* D. Stonehouse and C. Perrins, eds. Evolutionary ecology. University Park Press, Baltimore, Md.

———. 1977*b*. The cost of honesty (further remarks on the handicap principle). Journal of Theoretical Biology 67:603–605.

———. 1981. Natural selection, sexual selection, and the selection of signals. Pages 133–138 *in* G. Scudder and G. L. Reveal, eds. Evolution today: proceedings of the Second International Congress on Systems and Evolution. Hunt Institute for Botanical Documentation, Carnegie-Mellon University, Pittsburgh.

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