Optimal sex allocation: steps towards a mechanistic theory

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2.1 SUMMARY

Sex allocation theory is often hailed as the most successful branch of evolutionary ecology, yet its success has been limited to a relatively small number of taxa, mostly haplodiploid insects. Sex ratio variation in vertebrates is still poorly understood. We argue that this is due to the failure of current sex allocation models to sufficiently take into account the complexities of vertebrate sex determination and life histories. Our main purpose here is to discuss how more "mechanistic" models might be constructed to help answer some of the many open questions regarding vertebrate sex allocation. In particular, we discuss the importance of costs of control, the multidimensional nature of allocation decisions, and conflicts over allocation decisions. We give an overview of optimality or ESS techniques that are useful in analyzing sex allocation problems, and we present a series of models to illustrate several of the concepts and techniques.

2.2 INTRODUCTION

Sex allocation theory (Charnov 1982) has been very successful in gaining insight into the ultimate causes of sex ratio variation, but in applications the extent of its success has proven rather taxon-specific. Especially in haplodiploid insects relatively simple models seem to be able to correctly predict qualitative features of sex ratio variation, and sometimes even quantitative predictions have been met with remarkable precision (e.g. Werren 1980, but see Hardy et al. 1998). In contrast, the theory has shed a rather pale light on sex ratio variation in vertebrates. This discrepancy is usually attributed to taxonomic differences in the mode of sex determination (Williams 1979, Maynard Smith 1980, Bull & Charnov 1988). Relative to the accuracy of sex ratio control in haplodiploids, a chromosomal mechanism of sex determination, which is common in vertebrates, supposedly hinders parental sex ratio manipulation. This cannot, however, be the whole story because there are several well-documented examples of adaptive sex allocation in vertebrates (Clark 1978, Conover & Voorhees 1990, Daan et al. 1996, Komdeur et al. 1997, Kruuk et al. 1999).

Perhaps equally important is that most models of sex allocation lack the sophistication required to tackle the complexities of vertebrate sex allocation and life histories. In addition to the mechanism of sex determination there are several other aspects of vertebrate biology that are usually ignored in sex allocation models. (1) Vertebrates are often long-lived and face a trade-off between current and future reproduction. Strategic allocation decisions such as reproductive effort and sex allocation should reflect this trade-off. In contrast, standard sex allocation models typically assume very simple life histories with non-overlapping generations and reproduction at a fixed rate (for rare exceptions, see Olivieri et al. 1994, Zhang & Wang 1994, Lessells 1998). For a better understanding of sex allocation in long-lived species it will be crucial to study sex allocation, reproductive effort and other reproductive allocation decisions within a single framework. (2) In many vertebrates it seems likely that both parents can have some degree of influence on sex allocation. Asymmetries between the parents may cause their optimal allocation patterns to differ (Trivers 1974). Theoretical analyses tend to ignore the fact that males and females differ fundamentally in their means of exerting control of resource allocation to their offspring. For example, in birds with biparental care, females are the most likely sex to be in control of clutch size and the sex ratio, whereas both sexes might be able to practice sex-biased food allocation. (3) If the mode of sex determination prevents manipulation of the primary sex ratio, there are almost invariably costs involved in manipulating the sex ratio at some later stage. There is certainly awareness of this, but it is seldomly explicitly included in models of sex allocation (but see Maynard Smith 1980, Eshel & Sansone 1991, 1994, Leimar 1996, Pen et al. 1999).

In this chapter we discuss these and other biological factors that might be important for improving our understanding of sex allocation in vertebrates. Most of these aspects have been addressed before, but only some have been explicitly included in models, others have not been addressed at all, and many questions remain unanswered. Our main purpose is to discuss how models might be constructed to answer some of these questions and we provide several examples. We refer to our modelling approach as “mechanistic” because we emphasize the evolutionary implications of specific mechanisms of control over allocation decisions. We do not advocate models which include as much realism as possible, because such models tend to obscure, rather than enhance, our understanding. Instead, we advocate models of “intermediate complexity” that include several crucial aspects of vertebrate biology, without making the models too difficult to analyze.

This chapter is structured as follows: In section 2.3 we give a mainly verbal account of

some mechanistic aspects of sex allocation. We discuss the importance of costs of control, the multidimensional nature of allocation decisions, and conflicts over allocation decisions. In section 2.4 we given an overview of optimality or evolutionarily stable strategy (ESS) techniques that are useful in analyzing sex allocation problems. We show how to derive an expression for the fitness of an individual based on its life history, and we show how to analyze fitness such that meaningful conclusions can be inferred from it. Finally, in section 2.5 we present and analyze a series of models, ranging from very simple classical models to more advanced ones, in order to illustrate the concepts and techniques introduced in the previous sections.

2.3 MECHANISTIC ASPECTS

In this section we discuss in a non-technical way some mechanistic aspects of sex allocation. In section 2.4 we show how ESS techniques can be used to formally analyze the problems addressed here.

The cornerstone of sex allocation theory is Fisher's (1930) principle of equal allocation, which states that selection on the sex ratio comes to a halt if and only if allocation to sons equals allocation to daughters. Although it is now recognized (see Edwards 1998) that the basic idea behind the principle goes back to Darwin (1871) and the first mathematical treatment to Düsing (1884), we still call it "Fisher's principle" here. It is well-known that Fisher's principle relies on many tacit assumptions (Bull & Charnov 1988). We will not review all these assumptions here, but discuss a few that are relevant for our mechanistic approach. One of the most important assumptions of Fisher's principle is that parental control over the sex ratio is cost-free. That is, parents do not waste resources and/or suffer a higher mortality due to sex ratio control. We discuss some biological mechanisms that may render sex ratio control costly, and we use some simple models to investigate how much parents can afford to waste in adjusting the sex ratio.

Fisher's argument assumes that parents have a fixed amount of resource to allocate to their offspring. Iteroparous organisms must decide several times how much to invest in reproduction versus other activities that may affect their future reproduction. Does this affect sex allocation? Does it matter how parents can adjust sex allocation, by adjusting the sex ratio or by investing more or less in individual sons and daughters? We discuss the consequences for optimal sex allocation when different allocation components contribute to the overall allocation pattern, not necessarily all under parental control.

Fisher's equal allocation principle assumes parental control, but if gametes or offspring could somehow affect or obstruct sex allocation, selection may have a different outcome. Gametes and offspring are more closely related to themselves than to their siblings, unlike the parents who are usually equally related to all offspring. This asymmetry typically causes gametes and offspring to favor a sex ratio less biased than under parental control. If parents employ a mechanism of selective killing of gametes or offspring, this is likely to be opposed by the potential "victims".

Parents may also disagree over sex allocation if the costs of parental care are unequally shared between them. We discuss how differences between parents in control mechanisms and the information parents have about their partner's decision may affect the outcome of selection.

2.3.1 Costs

If parents are unable to adjust the primary sex ratio (at fertilization), then sex ratio manipulation is likely to be costly to the parents. The nature and the magnitude of the cost depends on the mechanism of secondary sex ratio control. At least two types of cost may be important. First, a loss of invested resources. If parents cannot directly change the sex of their offspring, they must selectively kill offspring of a particular sex, thereby losing some of the resources already invested in those offspring (Myers 1978, Williams 1979, Maynard Smith 1980). This may be costly if parents are resource-limited, or if the number of foetuses that can be simultaneously implanted is limited. Even if no energy or resources are lost, selective abortion may involve a second type of cost. There are a number of reasons why selective abortion may reduce the fitness prospects of the non-aborted offspring. For example, it may take time to replace a jettisoned offspring by a new one. Such delayed reproduction may be costly: it has been suggested that in birds, a female might be able to recognize the sex of a developing egg just before laying, and resorb it if it is of the undesirable sex (Emlen 1997, Oddie 1998, Pen et al. 1999). Since it takes time for the next egg to reach the same developmental stage, this means of manipulating the sex ratio may result in a later laying date, which in turn may be detrimental to the survival of offspring (Klomp 1970). This type of control may also lead to a more pronounced hatching asynchrony, which might also be detrimental to offspring survival.

Despite its potential importance, costs of sex ratio control are hardly ever explicitly included in evolutionary models of sex allocation (exceptions are Maynard Smith 1980, Eshel & Sansone 1991, 1994, Leimar 1996, Pen et al. 1999). One would like to know the cost parents can afford for sex ratio adjustment to be still adaptive, but this problem has never been analyzed systematically. To give at least an indication of the magnitude of such costs, in sections 2.5.2 and 2.5.5 we analyze two models with costly sex ratio control. In the first model, parents are selected to bias the sex ratio because sons and daughters have different costs. In the second model, parents are selected to bias the sex ratio according to some environmental cue which differentially affects the survival of sons and daughters. In both models parents have a fixed amount of resource to allocate to reproduction, the primary sex ratio is fixed at parity, and the sex ratio is secondarily adjusted by selective abortion after a certain initial investment (the cost of control) in the aborted offspring.

In the first model (section 2.5.2), a son requires E_m units of resource and a daughter E_f units. All parents produce the same sex ratio. Without costs of sex ratio control equal allocation is expected: the investment per son (E_m) times the number of sons (n_m) equals the investment per daughter (E_f) times the number of daughters (n_f), $E_m \times n_m = E_f \times n_f$. Figure 2.1 shows for several values of the investment ratio $\rho = E_m/E_f$ how the sex ratio bias decreases with the cost of control e_m (relative to $E_f = 1$), up to the point where it no longer pays to bias the sex ratio at all. This point is precisely where $e_m = (E_m - E_f)/2$, half the cost difference between sons and daughters. Figure 2.1 also depicts the maximal proportion of total resources wasted on sex ratio control. The maximal waste is surprisingly small: even when a son costs twice the resource of a daughter, and a sex ratio bias of 1:2 is to be expected in the absence of costs, it never pays to waste more than about 3.5% of resources on sex ratio adjustment.

In the second model (section 2.5.5), we analyze a variant of Trivers and Willard's (1973) model of conditional sex ratio adjustment. Parents can be in two states, one favorable for sons, the other for daughters. Specifically, in each state the survival of one sex is a proportion α of the opposite sex's survival. If the costs of control (i.e. the initial investment in aborted offspring) are small, the ESS is a "bang-bang" strategy: produce only one sex in

each state. If, however, the costs of control are higher than a certain threshold, then it does not pay to bias the sex ratio at all (figure 2.2). Hence, it is not true that costs of control select for sex ratio trends intermediate between bang-bang strategies and no adjustment at all (Oddie 1998). We have again calculated the maximal waste of resources on sex ratio adjustment, which is plotted in figure 2.2. In contrast to the "Fisherian" model, we now find that waste can be much larger. For example, when the benefit ratio equals 2 ($\alpha = \frac{1}{2}$), the maximal waste is about 17% of total resources. A tentative conclusion of this analysis is therefore that under conditional sex ratio adjustment a much larger cost of sex ratio control can be sustained compared to unconditional sex ratio adjustment.

2.3.2 Components

Sex allocation is usually defined as the division of resources between male and female function (Charnov 1982). For dioecious organisms, the problem is how to split resources between sons and daughters. Suppose a parent "wishes" to allocate A_m resources to sons and A_f to daughters. The parent can do this in several different ways. It can keep the allocation to individual sons and individual daughters fixed, and manipulate the numerical sex ratio. Or, conversely, the parent can keep the sex ratio fixed, but vary allocation to individual sons and daughters. Thus, the overall allocation pattern can be partitioned into at least four allocation components: the number of offspring or clutch size c, the primary sex ratio s (proportion sons), the amount of resources E_m allocated per individual son (assuming all sons receive the same amount) and the allocation E_f per individual daughter. The total amount of resources allocated to sons is then given by

$$A_m = scE_m \ , \tag{2.1}$$

and the amount allocated to daughters by

$$A_f = (1-s)cE_f \ . \tag{2.2}$$

Total allocation to reproduction, the reproductive effort, can now be written in terms of the different allocation components as

$$E_T = A_m + A_f = scE_m + (1-s)cE_f \ . \tag{2.3}$$

In this way, both sex allocation (A_m relative to A_f) and reproductive effort (E_T) can be viewed as the outcome of selection on four allocation components (s, c, E_m, E_f). The outcome is likely to depend on the way the allocation components affect various fitness components. In the simplest scenario, a parent's survival subsequent to reproduction depends only on its reproductive effort, and the survival of an offspring depends only on the amount of resources invested in it (see figure 2.3). However, it is possible that the survival of offspring in addition depends on clutch size and/or the sex composition of the clutch.

As the analysis in section 2.5.6 shows, the partitioning of allocation into components is helpful for several reasons. First, it gives us insight into the minimum number of allocation components that must be able to evolve in order for certain outcomes to be expected. For example, in order for Fisher's (1930) equal allocation principle ($A_m = A_f$) to be valid, selection on the sex ratio alone is not sufficient. It also necessary for selection to act on clutch size (see section 2.5.6). On the other hand, selection on the investment per individual son or daughter is not necessary for Fisher's principle to hold true (see Pen 2000, chapter 3). Numerical results of a specific example are shown in table 2.1.

Another advantage of partitioning allocation into components is that it forces us to think about what fitness components are affected by what allocation components. This may have important consequences. For example, we need to consider whether survival of offspring depends only on the amount of resources invested in them, or also on the clutch size or the sex ratio. For Fisher's equal allocation to hold, it is necessary that clutch size and sex ratio have no effect on offspring survival (Pen 2000, chapter 3).

For small litter sizes, the problem of simultaneous selection of multiple allocation components can be very complicated, especially if parents vary in the amount of resource available for reproduction and can adjust clutch size and the sex ratio accordingly. Without quite precise knowledge of the distribution of resource availability in the population, even qualitative predictions about the relationship between resource-availability and sex allocation are difficult to make (Williams 1979, Frank 1987).

It is possible to construct models with more than just the four allocation components we considered so far, although such a model will obviously be more difficult to analyze. For example, one can explicitly take the time structure of a reproductive episode into account by splitting the episode into a number of time units and defining for each time unit one or more allocation components. This would raise some interesting questions. Since parents usually have to decide on clutch size and the sex ratio before making any further investment in their offspring, not all of the above four allocation components above are simultaneously under control. A systematic investigation of the consequences for sex allocation of such a temporal decoupling of allocation decisions has not yet been made (Stubblefield & Seger 1990, Seger & Eckhart 1996).

The study of sex allocation and reproductive effort in a single framework raises more interesting questions. For example, the benefits of some forms of risky investment are difficult to assign to just one sex, such as nest defense behavior. There is currently no theory on how such investments, of which the benefits are shared equally by all offspring, affect sex allocation.

2.3.3 Conflicts

The concept of a conflict of interest in the context of sex allocation was introduced by Trivers (1974). Trivers noted that if the sexes are not equally costly to raise, then Fisher's principle of equal allocation only holds if the sex ratio of the offspring is under maternal control. If the offspring themselves were able to determine their sex, then selection would favor a different outcome. Since the optimal strategies of mother and offspring do not coincide, they have a conflict of interest. The reason for the discrepancy between mother and offspring is that the very tendency of the mother to produce fewer of the expensive sex renders this sex the one with the highest reproductive value. Offspring will therefore have a greater tendency to become the most expensive sex, even at the expense of the number of siblings their mother can raise. Because of the latter aspect of the problem, kin selection arguments play a role in the analysis. In section 2.5.3 we show how a simple kin selection model can be used to analyze the offspring's strategy.

Eshel & Sansone (1991, 1994) concluded that when both mother and offspring have some influence on the sex ratio, a compromise between the mother's and the offspring's optima is the most likely evolutionary outcome. They also investigated what happens when offspring signal their vulnerability to maternal manipulation, a stronger signal being associated with the handicap of greater vulnerability. The interesting conclusion arose that the mother may be more likely to "win" the conflict if she has no information about the state of her offspring. However, it is not clear under what circumstances selection could favor

a reduction in a mother's discriminatory power. It is also difficult to envisage mechanisms that might allow offspring to determine their own sex or influence the sex of others. Of greater practical relevance seems the possibility of offspring to hide or reveal their sex. This may affect the outcome of the conflict between parent and offspring, but it may also lead to a new conflict, between sons and daughters. This problem has yet to be analyzed.

A conflict may arise at an even earlier stage, between a parent and its gametes. Reiss (1987) has shown that only when fitness differences between sons and daughters are sufficiently large, gametes of the "wrong" sex may be selected to sacrifice themselves in favor of gametes of the "right" sex. However, it is not clear what the biological interpretation of this conclusion is. Does it mean that gametes actively oppose parental manipulation, or does it mean that they signal their identity under the appropriate circumstances? More work is needed to analyze specific biological scenarios of the conflict between parents and gametes to see how the outcome depends on specific mechanistic details.

Most sex allocation models assume that just one parent, by default the mother, has control over sex allocation decisions. In many vertebrates both parents have some share in the allocation of resources to their offspring. Asymmetries in the nature of their contribution and asymmetries in their life history may cause the parents to disagree over the preferred allocation pattern. In order to properly analyze such a scenario, several mechanistic aspects of the asymmetry between the parents have to be considered:

1. What components of allocation are influenced by each parent?
2. In what temporal order do the parents make their decisions?
3. What information about their decisions is transferred between the parents, and when?

In many cases some allocation components will be under control of one parent, and others under control of the other parent. For example, one parent might be able to determine the sex ratio while the other parent determines the clutch size. If the sex ratio is determined before the clutch size, then the outcome of selection on the sex ratio depends on whether or not the parent in control of the clutch size can detect the sex ratio before determining the clutch size. This kind of situation can be analyzed relatively straightforwardly with standard methods (see section 2.5.7).

Some components of allocation might be influenced by both parents. For example, it is conceivable that both parents can adjust the sex ratio to some extent. If the parents favor different sex ratios they have a conflict. This could lead to an arms race or a stable resolution of the conflict. In case of a stable outcome, either one parent "wins" or there will be a compromise between the parental optima. Of major importance for the outcome is the order in which the parents can make their decisions. For example, in mammals it seems likely that males, being the heterogametic sex, have the first opportunity to bias the primary sex ratio. After insemination or fertilization it's the female's turn to adjust the sex ratio. Once the offspring are born, males might again have an opportunity to alter the sex ratio or to differentially provision sons and daughters. In birds, where females are heterogametic, females are most likely to have the first option. We are not aware of any models that investigate the consequences of biparental control over the same allocation problem.

2.4 FITNESS

In this section we give a brief overview of basic evolutionarily stable strategy (ESS) techniques that are useful for analyzing sex allocation problems. More elaborate treatments

can be found in Taylor (1996), Taylor & Frank (1996), McNamara & Houston (1996), and Frank (1998).

2.4.1 Evolutionary stability

The recipe for an ESS analysis has three ingredients (e.g. Parker & Maynard Smith 1990): (1) define a strategy set consisting of all phenotypically feasible traits. (2) Define a fitness function relating the "adaptedness" of a trait to properties of the population and/or the environment. (3) Use an ESS criterion to find those strategies that are evolutionarily stable. So far we have considered the first ingredient, describing how the strategy set may reflect mechanistic aspects of sex allocation. Here we focus on the rest of the recipe.

In an ESS analysis one seeks an expression for the fitness $W(x, x^*)$ of a rare mutant phenotype x in a resident population which is monomorphic for phenotype x^*. A necessary condition for x^* to be an ESS is that in a resident population of individuals with phenotype x^*, rare mutants with phenotype $x \neq x^*$ do not have a higher fitness than the residents. In other words,

$$W(x^*, x^*) \geq W(x, x^*) \, . \tag{2.4}$$

This implies that $W(x, x^*)$ achieves its maximal value with respect to x when $x = x^*$, giving us the conditions

$$\left.\frac{\partial W}{\partial x}\right|_{x=x^*} = 0 \qquad \text{and} \qquad \left.\frac{\partial^2 W}{\partial x^2}\right|_{x=x^*} \leq 0 \, . \tag{2.5}$$

To avoid unneccesary technical details, in the rest of the chapter we focus only on the first condition, the equilibrium condition. For completeness we note that the conditions (2.5) do not imply that in a resident population with phenotype $\hat{x}$ close to an ESS x^*, selection favor mutants which are even closer to the ESS x^*. To check whether an ESS has this extra stability property (convergence stability), additional second order conditions have to be verified (Taylor 1996, Geritz et al. 1998).

We shall see below (sections 2.4.2 and 2.4.3) that in many sex allocation models, the fitness function $W(x, x^*)$ can be written in the "Shaw-Mohler" form

$$W(x, x^*) = \frac{m(x)}{m(x^*)} + \frac{f(x)}{f(x^*)} \, , \tag{2.6}$$

named after Shaw & Mohler (1953) who first derived such an expression in the context of sex ratio evolution. The term $m(x)$ represents the returns on investment in sons, as determined by the mutant's behavior x, and $f(x)$ the returns on investment in daughters. "Returns on investment" seems rather vague, but this is because its interpretation may vary from one model to another. Often it simply means "number of individuals that survive until adulthood". The returns on investment are scaled by the "reproductive values" (see section 2.4.3) $1/m(x^*)$ and $1/f(x^*)$ to take into account that an individual of one sex may have a greater expected contribution to future generations than an individual of the opposite sex. If we take m and f to mean the number of sons and daughters surviving until adulthood, the value of a son relative to that of a daughter equals the ratio $f(x^*)/m(x^*)$ of adult females to adult males in the population, owing to the fact that everyone has one father and one mother.

The first of the ESS conditions (2.5) applied to (2.6) yields

$$\frac{m'(x^*)}{m(x^*)} + \frac{f'(x^*)}{f(x^*)} = 0 \, , \tag{2.7}$$

and this equation is known as the Shaw-Mohler equation (Charnov 1982). Together with the second-order condition, it shows that the ESS x^* maximizes the product $m(x^*) \times f(x^*)$. Similar product-maximization theorems hold for many sex allocation models (Lessard 1989). In such cases an ESS analysis may be skipped altogether and one may rely on the easier product-maximization technique. However, for relatively complex life histories, it is not always possible to derive a fitness function as simple as (2.6), and one has to derive a "custom-made" version. It turns out that for more complex life histories, simple product-maximization criteria are in general not valid (e.g. Charnov 1982, p17).

2.4.2 Life history and population dynamics

It is obvious that the derivation of evolutionarily stable sex allocation patterns crucially depends on a proper measure of the fitness $W(x, x^*)$ of a rare mutant phenotype x in a resident population with phenotype x^*. Unfortunately, many sex allocation models are based on *ad hoc* measures of fitness. A proper measure of fitness is given by the growth rate λ of a subpopulation of mutants relative to the growth rate λ^* of the resident population (Metz et al. 1992). If the growth rate of the mutant subpopulation is smaller than that of the resident population, the mutant cannot increase in frequency, and vice versa. The growth rates are derived from population dynamical models, a model for the resident population, and one for the mutant subpopulation. The structure of the models is determined by the life history of the (class of) organisms we wish to study. It is often helpful to characterize life histories by a number of distinct states or "stages" the organism can be in, and the transitions between the stages (Caswell 1989). Dioecious organisms can obviously be in at least two states: female or male. One may further distinguish between different kinds of females and males, for example according to how old they are, whether they have a territory or not, etc. States can also be continuous; typical continuous state variables are time of birth, mass, size. Here we restrict our attention to life histories with a finite number of discrete states.

To illustrate the methods discussed in this section, we analyze the simplest possible life history with two states (males and females) and overlapping generations. Consider a monomorphic resident population with allocation strategy or behavior x^*. Each adult female produces $f^* = f(x^*)$ daughters and $m^* = m(x^*)$ sons that survive until adulthood, one season later. Adult females survive with probability $P_f^* = P_f(x^*)$, adult males with probability P_m. This gives us the population dynamical model

$$n'_f = (P_f^* + \frac{1}{2}f^*)n_f + \frac{1}{2}f^*Q^*n_m \tag{2.8a}$$

$$n'_m = \frac{1}{2}m^*n_f + (P_m + \frac{1}{2}m^*Q^*)n_m \,, \tag{2.8b}$$

where n'_f and n'_m are the number of adult females and males in the next season as a function of the numbers n_f and n_m in the current season. Note that f^* and m^* are multiplied by $\frac{1}{2}$, to account for the genetic share of parents in their offspring, or equivalently, to prevent counting the same offspring twice. The reproductive output of males is Q^* times that of females because the per capita reproductive output of males differs from that of females whenever the adult sex ratio differs from 1:1. In fact, from the viewpoint of an autosomal gene, every season the output of all females must equal that of all males since every offspring has one mother and one father: $n_f = Q^*n_m$, or equivalently $Q^* = n_f/n_m$.

Substituting this expression for Q^* in eqs (2.8a) and (2.8b) gives the simplified system

$$n'_f = (P_f^* + f^*)n_f \tag{2.9a}$$

$$n'_m = m^* n_f + P_m n_m \ . \tag{2.9b}$$

In the example, the state of the population can be described by a vector $\mathbf{n} = (n_f, n_m)$. More generally, if we discriminate between k different states, the population can be described by a vector $\mathbf{n} = (n_1, \ldots, n_k)$. If the per capita contribution of individuals in state j to individuals of state i in the next season is given by $a_{ij} = a_{ij}(x^*)$, then the number of individuals n_i in state i changes from one season to the next according to the recurrence relations

$$n'_i = \sum_{j=1}^{k} a_{ij} n_j \ , \qquad i = 1, \ldots, k \ . \tag{2.10}$$

In matrix notation, $\mathbf{n}' = \mathbf{A}\mathbf{n}$, where $\mathbf{A} = \mathbf{A}(x^*)$ is the $k \times k$ matrix with elements a_{ij} $(i, j = 1, \ldots, k)$. The matrix corresponding to (2.8a) and (2.8b) is given by

$$\mathbf{A} = \begin{pmatrix} P_f^* + \frac{1}{2} f^* & \frac{1}{2} f^* Q^* \\ \frac{1}{2} m^* & P_m + \frac{1}{2} m^* Q^* \end{pmatrix} \tag{2.11}$$

or equivalently, according to (2.9a) and (2.9b), by

$$\begin{pmatrix} P_f^* + f^* & 0 \\ m^* & P_m \end{pmatrix} \ . \tag{2.12}$$

Under mild conditions (e.g. Caswell 1989), a population with dynamics governed by $\mathbf{A}$ will eventually reach demographic equilibrium and grow with constant rate $\lambda^* = \lambda(x^*)$. In demographic equilibrium the distribution of individuals over the different states is stable, given by a vector $\mathbf{u}(x^*) = (u_1, \ldots, u_k)$, hence every stage grows with rate λ^*. Technically, λ^* equals the dominant eigenvalue of $\mathbf{A}$ and $\mathbf{u}(x^*)$ is a dominant right eigenvector of $\mathbf{A}$. There are standard methods to calculate the eigenvalues of a given matrix (e.g. Lancaster 1969). In case of a 2×2 nonnegative matrix (every element ≥ 0), the dominant eigenvalue is given by

$$\lambda = \frac{1}{2}(a_{11} + a_{22}) + \sqrt{(a_{11} + a_{22})^2 - 4(a_{11}a_{22} - a_{12}a_{21})} \ . \tag{2.13}$$

In our example, a straightforward calculation shows that $\lambda(x^*) = P_f(x^*) + f(x^*)$, i.e. a population in demographic equilibrium grows by a factor $P_f^* + f^*$ per season. This is quite obvious from (2.9a) in our example, but in general dominant eigenvalues are often difficult to calculate.

How does selection enter the picture? After specifying a population dynamical model for the resident population with behavior x^*, we need a model for a mutant subpopulation with behavior x. Writing $\mathbf{m} = (m_1, \ldots, m_k)$ for the numbers of mutants in the different states we can write $\mathbf{m}' = \mathbf{B}\mathbf{m}$, where $\mathbf{B}$ is a state-transition matrix which depends on the mutant behavior x. How does $\mathbf{B}$ look for our example? Similar to (2.11), with a few modifications. We assume that females are in control of behavior x. Then survival of mutant females and their reproductive output (corresponding to the first column of $\mathbf{A}$) depend on x. However, if mutants are rare, then it is very unlikely that mutant males mate with mutant females, hence the reproductive output of mutant males is determined by the resident

behavior x^*. $\mathbf{B}$ can therefore be written as

$$\mathbf{B} = \begin{pmatrix} P_f + \frac{1}{2}f & \frac{1}{2}f^*Q^* \\ \frac{1}{2}m & P_m + \frac{1}{2}m^*Q^* \end{pmatrix} . \tag{2.14}$$

The rareness of mutants causes the adult sex ratio $Q^* = u_f/u_m$ to be determined by the resident behavior x^*, and can be calculated from (2.9b): replacing the n's by u's, setting the left-hand side equal to $\lambda^* u_m$ and dividing both sides by u_m gives

$$Q^* = \frac{u_f}{u_m} = \frac{\lambda^* - P_m^*}{m^*} = \frac{P_f^* + f^* - P_m^*}{m^*} . \tag{2.15}$$

If adult females and males have identical survival ($P_f = P_m$), then the ratio of adult females to males is just f^*/m^*, the ratio of surviving daughters to surviving sons. Otherwise ($P_f \neq P_m$), the adult sex ratio is more biased towards the sex with the highest adult survival. Note that $\mathbf{B}$ does not only depend on the mutant behavior x, but also on the resident behavior x^*, so in general we have $\mathbf{B} = \mathbf{B}(x, x^*)$. Clearly, if the mutant behavior does not differ from the resident behavior, that is, $x = x^*$, then we should have $\mathbf{B}(x^*, x^*) = \mathbf{A}(x^*)$. This is obviously the case in our example.

Given a transition matrix $\mathbf{B}(x, x^*)$ we can calculate the growth rate $\lambda = \lambda(x, x^*)$ of the mutant subpopulation, which determines whether the mutants will increase in frequency ($\lambda > \lambda^*$) or not ($\lambda < \lambda^*$). Hence, $\lambda(x, x^*)$ is a suitable fitness measure, and by (2.5) an ESS must obey

$$\left.\frac{\partial \lambda}{\partial x}\right|_{x=x^*} = 0 . \tag{2.16}$$

In our example, the dominant eigenvalue of (2.14) can be found by inserting the elements of (2.14) into (2.13). The reader may check for herself that a rather nasty formula results. Differentiating this formula with respect to x yields, after some tedious calculations, the ESS condition

$$\left.\frac{\partial \lambda}{\partial x}\right|_{x=x^*} = \frac{f(x^*)m(x^*)}{\sqrt{z^*}} \left[\frac{P_f'(x^*)}{f(x^*)} + \frac{1}{2}\left(\frac{f'(x^*)}{f(x^*)} + \frac{m'(x^*)}{m(x^*)} \right) \right] = 0 , \tag{2.17}$$

where primes denote differentiation and $\sqrt{z^*}$ corresponds to the square root in (2.13). Since $f^*m^*/\sqrt{z^*}$ is positive, (2.17) is equivalent to

$$\frac{P_f'(x^*)}{f(x^*)} + \frac{1}{2}\left(\frac{f'(x^*)}{f(x^*)} + \frac{m'(x^*)}{m(x^*)} \right) = 0 . \tag{2.18}$$

The first term is the marginal cost of increasing x in terms of the female's own survival, the second term is the marginal benefit in terms of offspring production. This is a generalization of the Shaw-Mohler equation (2.7), as we shall discuss below in more detail.

Even for such a simple model, it is quite hard to calculate the dominant eigenvalue $\lambda(x, x^*)$ and to derive the ESS condition (2.17). For models with more than 2 states, it becomes very impractical. In the next section, we shall see that the calculations can be simplified considerably with the aid of reproductive values.

2.4.3 Reproductive values

The expected contribution to future generations may differ between individuals in different states. For example, if males are the rare sex, then they are more "valuable" because they

can, on average, expect more offspring than females. To quantify such differences in value in general, we need the concept of reproductive value. The reproductive value v_i of an individual in state i relative to the reproductive values of individuals in other states j, can be defined such that $u_i v_i / \sum_j u_j v_j$ is the expected proportion of genes in the far future that reside in individuals of state i now. In view of this interpretation, reproductive values can be defined recursively according to

$$v_j = \frac{1}{\lambda^*} \sum_i a_{ij} v_i \,, \tag{2.19}$$

where a_{ij} is the per capita contribution (through survival and/or reproduction) of individual in state j to individuals in state i in the next time period. In other words, the current reproductive value of an individual is the total amount of reproductive value it contributes to the next time period. The contribution is scaled by the inverse of the growth rate λ^* of the population to account for the "diluting" effect of a growing population. Technically, the vector of reproductive values $\mathbf{v} = (v_1, \ldots v_k)$ is a dominant left eigenvector of the transition matrix $\mathbf{A}$. Since only the relative reproductive values are meaningful in this context (the direction of the vector, not its length), we may arbitrarily set one of them to a fixed value, leaving $k-1$ equations in $k-1$ unknowns. For our example, we have

$$\lambda^* v_f = (P_f^* + \frac{1}{2} f^*) v_f + \frac{1}{2} m^* v_m \,. \tag{2.20}$$

Substituting $\lambda^* = P_f^* + f^*$ yields $v_f = (m^*/f^*) v_m$. We may set $v_m = 1/m^*$, giving

$$v_m = \frac{1}{m^*} \quad \text{and} \quad v_f = \frac{1}{f^*} \,. \tag{2.21}$$

The usefulness of the reproductive values arises from the fact that instead of $\lambda(x, x^*)$, which is hard to calculate, we can use as a fitness function

$$W(x, x^*) = \sum_{i,j} v_i(x^*) b_{ij}(x, x^*) u_j(x^*) \,. \tag{2.22}$$

This fitness function is the total amount of reproductive value contributed by mutants to the next time period. Note that the v_i and the u_j do not depend on the mutant behavior x, but only on the resident behavior x^*. In other words, W can be interpreted as the fitness of mutants that display mutant behavior during a single time period, after which they revert to the resident behavior.

The use of W as a fitness function instead of λ is justified by the fact that $\partial\lambda/\partial x$ and $\partial W/\partial x$ have the same sign. In fact, it follows from simple algebraic considerations (e.g. Taylor 1996) that

$$\left.\frac{\partial W}{\partial x}\right|_{x=x^*} = \sum_{i,j} v_i u_j \left.\frac{\partial b_{ij}}{\partial x}\right|_{x=x^*} = \sum_i v_i u_i \left.\frac{\partial \lambda}{\partial x}\right|_{x=x^*} \,. \tag{2.23}$$

Hence $\partial W/\partial x$ is identical to $\partial\lambda/\partial x$, up to the positive factor $\sum_i v_i u_i$.

Notice that all terms b_{ij} that do not depend explicitly on the mutant behavior x can be omitted from the sum (2.22) since they would vanish after differentiation anyhow. This is particularly convenient when the mutant behavior is restricted to individuals of only one state, say state j. We can then skip all terms in (2.22) that do not explicitly depend on x and obtain (up to the positive constant $u_j(x^*)$) the simplified fitness function

$$W(x, x^*) = \sum_i v_i(x^*) b_{ij}(x, x^*) \,. \tag{2.24}$$

This applies to our example, because we assumed that only females are in control. Applying (2.24) yields

$$W(x,x^*) = v_f(x^*)\left(P_f(x) + \frac{1}{2}f(x)\right) + v_m(x^*)\frac{1}{2}m(x) \tag{2.25a}$$

$$= \frac{P_f(x)}{f(x^*)} + \frac{1}{2}\left(\frac{f(x)}{f(x^*)} + \frac{m(x)}{m(x^*)}\right) , \tag{2.25b}$$

and we get the same ESS condition as before (see (2.18)),

$$\frac{P_f'(x^*)}{f(x^*)} + \frac{1}{2}\left(\frac{f'(x^*)}{f(x^*)} + \frac{m'(x^*)}{m(x^*)}\right) = 0 . \tag{2.26}$$

This condition is a generalization to overlapping generations of the Shaw-Mohler equation (2.7). It is a special case of the more general equation

$$P_f'(x^*)v_P(x^*) + \frac{1}{2}\left(\frac{f'(x^*)}{f(x^*)} + \frac{m'(x^*)}{m(x^*)}\right) = 0 , \tag{2.27}$$

where $v_P(x^*)$ is the reproductive value of a surviving female. This relatively simple and transparent condition does not just hold for our simple example, but holds for a fairly general class of life histories, as we show in Appendix 2.6. The only part that depends on life history details is the reproductive value v_P^* of a surviving female.

2.4.4 Density dependence

In section 2.4.3 we showed that calculation of the growth rate $\lambda(x,x^*)$ of the mutant subpopulation can be avoided with the aid of reproductive values, and we only need to compute the dominant eigenvalue and eigenvectors of the resident population. However, one might question whether a value $\lambda^* \neq 1$ can be considered reasonable, since no biological population can grow or decline forever. In reality, density dependent factors will ensure that a resident population will, in the long run, be more or less stationary, corresponding to $\lambda^* = 1$. To ensure that $\lambda^* = 1$ in the model, it is necessary to specify a particular mechanism of density dependence. For example, at high population densities juvenile survival might be compromised or the age of first breeding might be delayed. It is then assumed that the density dependent life history parameters take on such values in equilibrium that a stationary population results. For such a population with $\lambda^* = 1$, it is then relatively easy to calculate the left and right eigenvectors and to derive the fitness function (2.22).

The choice of a paticular mechanism of density dependence can hace important evolutionary implications (see Mylius & Diekmann 1995, Pen & Weissing 2000). Those life history parameters that are supposed to be affected by density dependence, can no longer be considered as independent entities. Instead, the constraint $\lambda^* = 1$ can be used to express the density dependent life history parameters in terms of the other parameters. As a consequence, different mechanisms of density dependence may lead to rather different fitness functions and, hence, to rather different evolutionary precitions.

2.4.5 Direct fitness and relatedness

Sometimes the fitness of an individual with behavior x does not just depend on its own behavior and the behavior x^* of the entire population, but also on the behavior $\overline{x}$ in a local

environment of the individual. That is, fitness can be written as $W(x, \overline{x}, x^*)$. The effect on fitness of a small change in x can then be written as

$$\frac{\mathrm{d}W}{\mathrm{d}x} = \frac{\partial W}{\partial x} R_0 + \frac{\partial W}{\partial \overline{x}} R_1 + \frac{\partial W}{\partial x^*} R_\infty \,, \tag{2.28}$$

where the R_i can be interpreted as coefficients of relatedness (Michod & Hamilton 1980) between the individual doing x and itself ($R_0 = 1$), the local individuals (R_1) and the entire population ($R_\infty = 0$). Hence, if we write $R_1 = R$, the ESS condition (2.5) can be generalized to

$$\left(\frac{\partial W}{\partial x} + R \frac{\partial W}{\partial \overline{x}} \right)_{x = \overline{x} = x^*} = 0 \,. \tag{2.29}$$

This approach is known as the "direct fitness" approach (Taylor & Frank 1996, Frank 1998), as opposed to "inclusive fitness". Clearly, (2.28) can be generalized by letting fitness depend on the behavior x_i of every individual i in the entire population:

$$\frac{\mathrm{d}W}{\mathrm{d}x} = \frac{\partial W}{\partial x} + \sum_i R_i \frac{\partial W}{\partial x_i} \,, \tag{2.30}$$

where R_i is the coefficient of relatedness between the focal individual and individual i. In fact, the R_i measure association between behavior of different individuals, which may come about by common genealogy but also by other factors such as a shared environment. Hence, the direct fitness approach is more general than the inclusive fitness approach (Frank 1998).

In the next section we give an example of the direct fitness approach, applied to parent-offspring conflict over the sex ratio.

2.5 MODEL GALLERY

In this section we present a series of models with a twofold purpose: firstly, to illustrate and verify results discussed in section 2.3 and, secondly, as an overview of the most commonly used ESS techniques. As a prelude to more advanced techniques used later on, we start with some simple models, generating well-known results that apply to organisms with non-overlapping generations. These models are then extended to include several of the mechanistic aspects discussed before and we use the results to analyze the conflict over sex allocation between parents.

2.5.1 Fisher's principle of equal allocation

Consider a mother with a sex ratio (proportion sons) s among her offspring in a population with sex ratio s^*. We assume non-overlapping generations, hence the fitness $W(s, s^*)$ of such a mother is a special case of (2.25b), with maternal survival P_f set equal to zero, that is, $W(s, s^*) = m(s)/m(s^*) + f(s)/f(s^*)$. The mother's clutch size is given by $c(s)$, hence the number of sons is given by $m(s) = sc(s)$ and the number of daughters by $f(s) = (1 - s)c(s)$. Thus,

$$W(s, s^*) = \frac{c(s)}{c(s^*)} \left[\frac{s}{s^*} + \frac{1 - s}{1 - s^*} \right] \,. \tag{2.31}$$

If a mother's clutch size c is independent of the sex ratio s among her offspring, this reduces to

$$W(s, s^*) = \frac{s}{s^*} + \frac{1 - s}{1 - s^*} \,, \tag{2.32}$$

which yields an ESS sex ratio $s^* = 1/2$. However, if sons and daughters require different amount of resources, say E_m units per son and E_f units per daughter, then for a fixed amount of resources E_T we have $E_T = scE_m + (1-s)cE_f$, or in other words,

$$c(s) = \frac{E_T}{sE_m + (1-s)E_f} \,. \tag{2.33}$$

Substitution in (2.31) gives the fitness function

$$W(s, s^*) = \frac{s^*E_m + (1-s^*)E_f}{sE_m + (1-s)E_f} \left[\frac{s}{s^*} + \frac{1-s}{1-s^*} \right] \,. \tag{2.34}$$

The ESS sex ratio s^* is found by solving

$$\left. \frac{\partial W}{\partial s} \right|_{s=s^*} = \frac{(1-s^*)E_f - s^*E_m}{[s^*E_m + (1-s^*)E_f]s^*(1-s^*)} = 0 \,, \tag{2.35}$$

which yields

$$\frac{s^*}{1-s^*} = \frac{E_f}{E_m} \,. \tag{2.36}$$

This is Fisher's (1930) classic result that the evolutionarily stable ratio of sons to daughters is the inverse of their cost ratio, or, in other words, equal allocation to sons and daughters:

$$s^*E_m = (1-s^*)E_f \,. \tag{2.37}$$

2.5.2 Fisher's model with costly sex ratio control

Now we extend the previous model by allowing for costs of sex ratio control. Similar models have been studied by Maynard Smith (1980), Eshel & Sansone (1991) and Charnov (1982). Suppose the primary sex ratio is even and parents can adjust the secondary sex ratio by selective abortion. As before, we assume that a son costs E_m units of resource and a daughter E_f units. Without loss of generality we may assume that $E_m > E_f$, so selection favors a sex ratio biased towards daughters when the costs of abortion are small enough. An aborted son costs $0 \le e_m < E_m$ units of resource. Suppose a mother aborts a fraction a of her sons, then for a fixed amount of resources E_T and clutch size c we have $E_T = \frac{1}{2}(1-a)cE_m + \frac{1}{2}ace_m + \frac{1}{2}cE_f$, or

$$c(a) = \frac{E_T}{\frac{1}{2}[(1-a)E_m + ae_m + E_f]} \,. \tag{2.38}$$

Note that the secondary (after abortion) sex ratio s is given by $s = (1-a)/(2-a)$, or $s/(1-s) = 1-a$. Substitution of the expression (2.38) for clutch size in (2.31) gives the fitness function

$$W(a, a^*) = \frac{(1-a^*)E_m + a^*e_m + E_f}{(1-a)E_m + ae_m + E_f} \left[\frac{1-a}{1-a^*} + 1 \right] \,. \tag{2.39}$$

The ESS sex ratio is found by solving $\partial W/\partial a = 0$, evaluated at $a = a^*$, which gives the evolutionarily stable level of abortion as

$$a^* = \frac{E_m - E_f - 2e_m}{E_m - e_m} \,, \tag{2.40}$$

or equivalently,

$$\frac{s^*}{1-s^*} = 1 - a^* = \frac{E_f + e_m}{E_m - e_m} , \tag{2.41}$$

which is identical to Fisher's result if and only if $e_m = 0$. Figure 2.1 shows for several values of the relative cost $\rho = E_m/E_f$ of sons how the ESS sex ratio s^* varies with e_m.

The condition that it still pays to abort any sons at all ($a^* > 0$) is given by

$$e_m < \frac{1}{2}(E_m - E_f) . \tag{2.42}$$

In other words, it doesn't pay to abort if an aborted son costs half the cost difference between the sexes or more. The same result was found by Eshel & Sansone (1991) by means of a population genetical model.

Given E_m, E_f and e_m, we can calculate the proportion p of resources wasted on adjusting the sex ratio as

$$p = \frac{1}{2}a^* e_m = \frac{1}{2}\frac{E_m - E_f - 2e_m}{E_m - e_m} e_m . \tag{2.43}$$

The value of e_m for which the waste is maximal, $\hat{e}_m$, is found by solving $\mathrm{d}p/\mathrm{d}e_m = 0$, which yields

$$\hat{e}_m = E_m - \sqrt{\frac{E_m(E_m + E_f)}{2}} . \tag{2.44}$$

If we insert the right-hand side in (2.43), we get an expression for the maximal waste, given the costs E_m and E_f of sons and daughters, respectively. This maximal waste is plotted against E_m for $E_f = 1$ in figure 2.1, which shows that even for extreme values of $\rho = E_m/E_f$ it never pays to waste a great deal of resources to adjusting the sex ratio. For the specific case of $\rho = 2$, $p(\hat{e}_m) = 0.036$.

2.5.3 Parent-offspring conflict

Now we use the model to investigate what the favored sex ratio is from the offspring's point of view. We use Taylor and Frank's (1996) direct fitness approach to derive Triver's (1974) classic result. For an individual offspring we can use the same fitness function as (2.34), except that the clutch size is not only a function of its own sex ratio s, but also depends on the sex ratio favored by the other offspring in the clutch. If we write $\overline{s}$ for the average sex ratio favored by the offspring in a clutch, and s^* again for the sex ratio in the rest of the population, then we can rewrite (2.34) as

$$W(s, \overline{s}, s^*) = \frac{s^* E_m + (1-s^*)E_f}{\overline{s}E_m + (1-\overline{s})E_f}\left[\frac{s}{s^*} + \frac{1-s}{1-s^*}\right] . \tag{2.45}$$

The direct fitness equilibrium condition is given by

$$\left(\frac{\partial W}{\partial s} + R\frac{\partial W}{\partial \overline{s}}\right)_{s=\overline{s}=s^*} = 0 , \tag{2.46}$$

where R is the coefficient of relatedness between the offspring. We get

$$E_f - [2E_f - (E_m - E_f)(1-2R)]s^* - 2(E_m - E_f)(1-R)s^{*2} = 0 , \tag{2.47}$$

which is Triver's (1974) result. For the special case of $R = \frac{1}{2}$ we get

$$\frac{s^*}{1-s^*} = \sqrt{\frac{E_f}{E_m}}\,, \tag{2.48}$$

which is less biased than the ESS from the mother's point of view. In general, the larger R, the smaller the disagreement between mother and offspring, and for $R = 1$ the offspring's optimum and maternal optimum coincide (see figure 2.4). Factors that influence R are the level of inbreeding, the number of fathers per clutch and the clutch size (for a single offspring $R = 1$).

2.5.4 Trivers and Willard's model with two states

Fisher's result is based on the assumption that all mothers are in identical conditions, and they all produce the same sex ratio. Trivers & Willard (1973) argued that if mothers in good condition produce fitter offspring than mothers in bad condition, and if sons profit more from good maternal condition than daughters, then mothers in good condition should produce sons and mothers in bad condition should produce daughters. More generally, given the prevailing conditions, a mother should overproduce that sex which yields the highest inclusive fitness from the mother's point of view.

Suppose all mothers have the same amount of resources E_T and sons and daughters are equally costly ($E_m = E_f$), but mothers can be in two possible states (0 or 1), each state occurring with frequency $\frac{1}{2}$. The mother's state affects offspring survival such that the relative survival of sons in state 0 compared to sons in state 1, and that of daughters in state 1 compared to daughters in state 0 is given by $\alpha < 1$. Hence, in state 0 it is better to produce daughters, in state 1 sons. The symmetry of the situation simplifies the analysis because the sex ratio bias in state 0 must equal the bias in state 1. Hence, the average sex ratio s^* will be $\frac{1}{2}$, independently of the sex ratio s_0 produced in state 0 and the sex ratio $s_1 = 1 - s_0$ produced in state 1. For each state we therefore have a fitness function similar to (2.25b), with

$$m(s^*) = f(s^*) = \frac{1}{2}c\frac{1+\alpha}{2}\,, \tag{2.49}$$

where $s^* = 1/2$ is the average sex ratio and c the clutch size. In state 0, the number of sons is given by $m_0(s_0) = \alpha s_0 c$ and the number of daughters by $f_0(s_0) = (1-s_0)c$; in state 1 $m_1(s_1) = s_1 c$ and $f_1(s_1) = \alpha(1-s_1)c$. This gives the fitness functions

$$W_0(s_0) = 2\frac{\alpha s_0 + 1 - s_0}{\alpha + 1} \tag{2.50a}$$

$$W_1(s_1) = 2\frac{s_1 + \alpha(1-s_1)}{\alpha+1}\,. \tag{2.50b}$$

Hence, the selection differentials are given by

$$\frac{\mathrm{d}W_0}{\mathrm{d}s_0} = -\frac{\mathrm{d}W_1}{\mathrm{d}s_1} = \frac{2(\alpha-1)}{\alpha+1} < 0\,, \tag{2.51}$$

which implies that there is a unique ESS (s_0^*, s_1^*), given by the "bang-bang" strategy $(0,1)$.

2.5.5 Trivers and Willard's model with costly sex ratio control

Now we assume that the primary sex ratio is fixed at parity, and the sex ratio can be adjusted secondarily by selective abortion. Let a_0 represent the fraction of sons that are aborted in state 0, and a_1 the fraction of daughters aborted in state 1. An aborted offspring costs e units of resource relative to the cost E of an "accepted" offspring. Given a total amount of resources E_T, the clutch size in state i is given by

$$c_i(a_i) = \frac{E_T}{\frac{1}{2}[2E - a_i(E-e)]} \,. \tag{2.52}$$

The fitness functions are then given by

$$W_i(a_i, a_i^*) = \frac{2E - a^*(E-e)}{2E - a_i(E-e)} \frac{(1-a_i)\alpha + 1}{(1-a^*)\alpha + 1} \,. \tag{2.53}$$

The selection differentials are

$$\left.\frac{\partial W_i}{\partial a_i}\right|_{a_i = a_i^* = a^*} = \frac{-1}{2E - a^*(E-e)} \frac{\alpha(E+e) - E + e}{(1-a^*)\alpha + 1} \,. \tag{2.54}$$

The sign of the right-hand side is independent of the abortion rate a_i. Hence, either all offspring of the undesired sex are aborted, or none. In other words, selection favors the bang-bang sex ratio strategy (0,1) or no sex ratio adjustment at all. For selection to favor abortion, the right-hand side of (2.54) must be positive, or equivalently,

$$e < E\frac{1-\alpha}{1+\alpha} \,. \tag{2.55}$$

This condition is plotted in figure 2.2.

Two interesting conclusions can be drawn from this analysis. First, costly sex ratio control does not necessarily favor sex ratio trends less extreme than bang-bang strategies. Second, if selection favors conditional abortion, then a fraction $\frac{1}{2}e/E$ of resources is "wasted" on sex ratio adjustment, which can be considerably more than under unconditional sex ratio adjustment in Fisher's scenario (see figure 2.2).

2.5.6 Multiple allocation components

In the appendix we have derived a fitness function for the allocation strategy x^* of a reproducing female, valid for a rather general class of life histories:

$$W_f(x, x^*) = P_f(x) v_P(x^*) + \frac{1}{2}\left(\frac{f(x)}{f(x^*)} + \frac{m(x)}{m(x^*)}\right) \,, \tag{2.56}$$

where v_P is the residual reproductive value of a reproducing female, which depends on particular life history details such as the mating system.

We use the fitness function (2.56) to illustrate our approach of section 2.3.2, where we decomposed allocation decisions into multiple components. Accordingly, let s be the primary sex ratio, c clutch size, E_m the investment per son and E_f the investment per daughter. A female's total reproductive effort is then given by $E_T = c(sE_m + (1-s)E_f)$ which determines her survival $P_f = P_f(E_T)$. Survival $\mu = \mu(E_m)$ of a son is assumed to depend only on E_m, not on any other allocation components, and the survival $\phi = \phi(E_f)$

of a daughter only on E_f. The number of surviving daughters and sons produced per mother is then given by

$$m = sc\mu(E_m) \qquad \text{and} \qquad f = (1-s)c\phi(E_f)\ . \tag{2.57}$$

Inserting s, c, E_f and E_m for x in (2.56) and applying (2.5) yields the four ESS conditions

$$s^*\ :\qquad \frac{1}{2}\left(\frac{1}{s^*}-\frac{1}{1-s^*}\right) = -c(E_m-E_f)P_f'(E_T^*)v_P \tag{2.58a}$$

$$c^*\ :\qquad \frac{1}{c^*} = -\frac{E_T^*}{c^*}P_f'(E_T^*)v_P \tag{2.58b}$$

$$E_m^*\ :\qquad \frac{1}{2}\frac{\mu'(E_m^*)}{\mu(E_m^*)} = -csP_f'(E_T^*)v_P \tag{2.58c}$$

$$E_f^*\ :\qquad \frac{1}{2}\frac{\phi'(E_f^*)}{\phi(E_f^*)} = -c(1-s)P_f'(E_T^*)v_P\ . \tag{2.58d}$$

Note that $-P_f'(E_T^*)v_P$ is positive, assuming thatP_f decreases with E_T. Condition (2.58a) therefore shows that selection on the sex ratio leads to an overproduction of the cheaper sex. In general, however, evolutionary stability of s, taken in isolation, will not yield equal allocation to sons and daughters,

$$s^*E_m = (1-s^*)E_f\ . \tag{2.59}$$

In fact, a straightforward calculation yields that (2.58a) implies (2.59) only if it is true that $-P_f'(E_T^*)v_P = 1/E_T^*$ or, equivalently, if (2.58b) is also satisfied. In other words, Fisher's (1930) equal allocation principle (2.59) will only hold if sex ratio and clutch size are optimized simultaneously.

Let us now consider several examples. First we have to find an expression for a mother's residual reproductive value v_P, which depends on the mating system. For example, if a breeding female that survives until the next breeding season will breed again, then her residual reproductive value is given by

$$v_P = \frac{1}{1-P_f}\ , \tag{2.60}$$

her life expectancy. If a breeding female that survives has the same chance as a surviving daughter to breed in the next season, then their reproductive values are equal, hence $v_P = 1/f$ (see Pen 2000, chapter 3). In the numerical examples below we assume that v_P is given by (2.60). In that case, selection on the clutch size gives us condition (2.58b), which for our choice of v_P can be written as

$$\frac{1}{E_T^*} = -\frac{P_f'(E_T^*)}{1-P_f(E_T^*)} \tag{2.61}$$

Hence, for this choice of v_P, the ESS reproductive effort is completely determined by $P_f(E_T)$, the relationship between reproductive effort E_T and the mother's subsequent survival P_F. Notice that the same total reproductive effort $E_T^* = -(1-P_f)/P_f'$ can be achieved by various combinations of the allocation components. For clutch size to be evolutionarily stable, only the level of E_T^* is important, not the way it is achieved. In particular, the ESS value of E_T is independent of the sex ratio s. Graphically, condition (2.61) implies that E_T^* can be found by drawing a straight line from $P = 1$ that touches the graph of $P(E_T)$ (see

figure 2.3A). In case that offspring survival is independent of clutch size, the ESS criteria (2.58c) and (2.58d) can also be given by a simple graphical interpretation (see figure 2.3B).

Table 2.1 shows numerical examples of what happens when either all four allocation components are optimized ("no constraints"), or only a subset of allocation components is optimized (due to "constraints"). If, for instance, the primary sex ratio is fixed at $1/2$ (e.g. due to chromosomal sex determination), (2.58a) will not hold true and the solution of the remaining equations (2.58b) to (2.58d) yields a biased sex allocation ratio, in this specific example male-biased. If clutch size is constrained to fixed value, condition (2.58b) will not hold true, and selection on the remaining allocation components results again in biased sex allocation, this time female-biased.

2.5.7 Parent-parent conflict

In the previous section we assumed that all allocation decisions are under control of one and the same parent. We now extend the model to investigate what happens when sex allocation is under partial control of both parents. We assume there is one father per clutch. The father is in control of the sex ratio s, and the mother controls the clutch size c. Father invests no further in his offspring, and his sex ratio decision does not affect his own subsequent survival. The investments per individual son (E_m) and daughter (E_f) are assumed to be constant now. This does not affect our qualitative conclusions.

As we have seen before, the mother would prefer equal allocation, i.e. (2.36), if she were in control of clutch size and sex ratio. If, on the other hand, the father had full control, he would prefer a 1:1 sex ratio, since his survival is, by assumption, independent of E_T. Hence, in case of $E_f \neq E_m$ there is a conflict over the sex ratio between the parents. To illustrate possible outcomes of this conflict, we consider two scenarios, according to whether or not the mother has information about the sex ratio decision of her mate and uses this information to adjust the clutch size.

Analogous to the mother's fitness function (2.56), the following fitness function holds for a father with allocation strategy y:

$$W_m(y, y^*) = P_m(y) v_M(y^*) + \frac{1}{2}\left(\frac{f(y)}{f(y^*)} + \frac{m(y)}{m(y^*)}\right) , \tag{2.62}$$

where v_M is a breeding male's residual reproductive value. Since the male's survival P_m is independent of his sex ratio decision, the first term of (2.62) can be skipped, and the fitness function simplifies to

$$W_m(s, s^*) \propto \frac{sc\mu(E_m)}{s^* c^* \mu(E_m)} + \frac{(1-s)c\phi(E_f)}{(1-s^*)c^*\phi(E_f)} \tag{2.63a}$$

$$= \frac{c(s)}{c(s^*)}\left(\frac{s}{s^*} + \frac{1-s}{1-s^*}\right) . \tag{2.63b}$$

This yields the ESS condition

$$\left.\frac{\partial W_m}{\partial s}\right|_{s=s^*} = 2\frac{c'(s^*)}{c(s^*)} + \frac{1}{s^*} - \frac{1}{1-s^*} = 0 . \tag{2.64}$$

Now suppose the mother cannot adjust her clutch size to the sex ratio determined by her mate. Then $c'(s^*) = 0$ and it follows from (2.64) that the male's ESS sex ratio is $s^* = \frac{1}{2}$. From section 2.5.6 we know that if the sex ratio is fixed at $1/2$, the mother's ESS sex allocation is not equal allocation. Hence, the father "wins" the conflict.

Now suppose the mother *can* adjust her clutch size according to her mate's strategy. We assume that a breeding female that survives breeds again in the next season. A female's residual reproductive value is then given by (2.60), and selection on clutch size yields condition (2.61) that we have derived in the previous section. Since (2.61) says that a female's ESS reproductive effort E_T^* is independent of the sex ratio, a change in the sex ratio by her mate induces a selection pressure on females to change their clutch size in such a way that their reproductive effort stays constant. Since the mother's reproductive effort is given by $E_T = c(sE_m + (1-s)E_f)$, we can denote the female's clutch size "response" to the male's sex ratio s as

$$c(s) = \frac{E_T}{sE_m + (1-s)E_f} \,. \tag{2.65}$$

Hence,

$$\frac{c'(s^*)}{c(s^*)} = \frac{2(E_f - E_m)}{s^*E_m + (1-s^*)E_f} \,. \tag{2.66}$$

As a consequence, (2.64) yields $s^*E_m = (1-s^*)E_f$, equal allocation. Thus, if the mother can adjust her clutch size to the sex ratio decision of her mate, then she "wins" the conflict.

2.6 APPENDIX: GENERALIZED SHAW-MOHLER EQUATION

In this appendix we show that the generalized Shaw-Mohler equation (2.27) holds for a fairly general class of life histories. Suppose individuals of each sex can be in one of two states, a non-breeding or floater state and a breeding or territorial state. The two non-breeding states, one for females and one for males, can each be regarded as a "pooling" of many non-breeding states, which is what makes this description general (Weissing & Pen, in prep.).

The life history parameters that characterize each state are doubly indexed by f or m to denote sex, and by 0 or 1 to denote the breeding state. P_{ij} ($i \in \{f,m\}$, $j \in \{0,1\}$) represents the probability of survival from one season to the next. For example, P_{f0} denotes the survival probability of a non-breeding female, and P_{m1} of a breeding male. The number of surviving daughters produced per breeding female is denoted by f and the number of surviving sons by m. An individual's probability to be in the breeding state in the next season may depend on its current state and is denoted by α_{ij} ($i \in \{f,m\}$, $j \in \{0,1\}$). Likewise, the probability that an individual is a non-breeder is denoted by β_{ij}. For newborns we use the symbols α_i and β_i ($i \in \{f,m\}$) to denote the probabilities that they become breeders respectively non-breeders in the season after birth.

The numbers of individuals in the four states are stored in the column vector $\mathbf{n} = (n_{f0}, n_{f1}, n_{m0}, n_{m1})$. The population composition $\mathbf{n}'$ in the next season, as a function of the population $\mathbf{n}$ in the current season and the life history parameters defined above, is given by the recurrence relation $\mathbf{n}' = \mathbf{An}$, where $\mathbf{A}$ is the 4×4 matrix

$$\begin{pmatrix} \beta_{f0}P_{f0} & \beta_{f1}P_{f1} + \frac{1}{2}\beta_f f & 0 & \frac{1}{2}\beta_f fQ \\ \alpha_{f0}P_{f0} & \alpha_{f1}P_{f1} + \frac{1}{2}\alpha_f f & 0 & \frac{1}{2}\alpha_f fQ \\ 0 & \frac{1}{2}\beta_m m & \beta_{m0}P_{m0} & \beta_{m1}P_{m1} + \frac{1}{2}\beta_m mQ \\ 0 & \frac{1}{2}\alpha_m m & \alpha_{m0}P_{m0} & \alpha_{m1}P_{m1} + \frac{1}{2}\alpha_m mQ \end{pmatrix} \tag{2.67}$$

Reproductive output is multiplied by $\frac{1}{2}$ to account for the genetic share of parents in their offspring (see section 2.4.2). The reproductive output of males is Q times that of females

to allow for the possibility that the number of reproducing males, n_{m1}, need not equal the number of reproducing females, n_{f1}. In fact, as we explained before $Q = n_{f1}/n_{m1}$, the ratio of breeding females to breeding males.

The fitness function can be written as

$$W(x, x^*) = P_{f1} v_P + \frac{1}{2}\left(f v_f + m v_m\right) , \tag{2.68}$$

where v_P is the (residual) reproductive value of a surviving mother,

$$v_P = \alpha_{f1} v_{f1} + \beta_{f1} v_{f0} , \tag{2.69}$$

v_f the reproductive value of a surviving daughter and v_m the reproductive value of a surviving son,

$$v_f = \alpha_f v_{f1} + \beta_f v_{f0} \tag{2.70a}$$
$$v_m = \alpha_m v_{m1} + \beta_m v_{m0} \tag{2.70b}$$

We will now show that

$$\frac{v_f}{v_m} = \frac{m^*}{f^*} , \tag{2.71}$$

the ratio of sons to daughters at independence. As a consequence, we have

$$W(x, x^*) = P_{f1} v_P + \frac{1}{2}\left(\frac{f}{f^*} + \frac{m}{m^*}\right) . \tag{2.72}$$

The reproductive value of a breeding female in the resident population is given by

$$\begin{aligned} \lambda^* v_{f1} &= P^*_{f1}(\alpha_{f1} v_{f1} + \beta_{f1} v_{f0}) + \frac{1}{2} f^*(\alpha_f v_{f1} + \beta_f v_{f0}) \\ &\quad + \frac{1}{2} m^*(\alpha_m v_{m1} + \beta_m v_{m0}) \qquad (2.73a) \\ &= P^*_{f1} v_P + \frac{1}{2}\left(f^* v_f + m^* v_m\right) , \qquad (2.73b) \end{aligned}$$

which implies

$$\lambda^* v_{f1} - P^*_{f1} v_P - f^* v_f = \frac{1}{2}\left(m^* v_m - f^* v_f\right) . \tag{2.74}$$

To complete the proof we have to show that the left-hand side is zero or, equivalently, that

$$v_{f1}\left(\lambda^* - \alpha_{f1} P^*_{f1} - \alpha_f f^*\right) = v_{f0}(\beta_{f1} P^*_{f1} + \beta_f f^*) . \tag{2.75}$$

The reproductive value of a non-breeding female in the resident population is given by

$$\lambda^* v_{f0} = P_{f0}(\beta_{f0} v_{f0} + \alpha_{f0} v_{f1}) , \tag{2.76}$$

or equivalently,

$$v_{f1} \alpha_{f0} P_{f0} = v_{f0}(\lambda^* - \beta_{f0} P_{f0}) . \tag{2.77}$$

Comparing (2.75) to (2.77), the proof of (2.71) therefore boils down to showing that

$$\frac{\lambda^* - \alpha_{f1} P^*_{f1} - \alpha_f f^*}{\beta_{f1} P^*_{f1} + \beta_f f^*} = \frac{\alpha_{f0} P_{f0}}{\lambda^* - \beta_{f0} P_{f0}} . \tag{2.78}$$

This equality can be derived as follows. Insertion of n_{f1}/n_{m1} for Q shows that the population dynamics can equivalently be described by the matrix

$$\begin{pmatrix} \beta_{f0}P_{f0} & \beta_{f1}P_{f1}+\beta_f f & 0 & 0 \\ \alpha_{f0}P_{f0} & \alpha_{f1}P_{f1}+\alpha_f f & 0 & 0 \\ 0 & \beta_m m & \beta_{m0}P_{m0} & \beta_{m1}P_{m1} \\ 0 & \alpha_m m & \alpha_{m0}P_{m0} & \alpha_{m1}P_{m1} \end{pmatrix}, \tag{2.79}$$

which can be written in block matrix notation as

$$\begin{pmatrix} \mathbf{A}_{ff} & \mathbf{0} \\ \mathbf{A}_{mf} & \mathbf{A}_{mm} \end{pmatrix}. \tag{2.80}$$

The dominant eigenvalue λ^* is determined by the block matrix $\mathbf{A}_{ff}$ which we assume has the largest eigenvalue. This eigenvalue is given by the characteristic equation of $\mathbf{A}_{ff}$ (e.g. Lancaster 1969):

$$(\lambda^* - \beta_{f0}P_{f0})(\lambda^* - \alpha_{f1}P_{f1} - \alpha_f f) = \alpha_{f0}P_{f0}(\beta_{f1}P_{f1} + \beta_f f), \tag{2.90}$$

equivalent to (2.78), which is what we wanted to show.

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Table 1: Evolutionarily stable allocation components, reproductive effort and sex allocation under different scenarios of maternal control. In the first scenario, whcih corresponds to figure 2.3, all components are under maternal control, leading to equal allocation ($A_f/A_m = 1$). In the second scenario, the primary sex ratio is not under maternal control, but fixed at $s = 1/2$. As a consequence, at the ESS less is invested in daughters than in sons ($A_f/A_m < 1$). In the third scenario, the cluch size is fixed at $c = 5$, implying a female-biased investment ($A_f/A_m > 1$) at ESS. The functions P, ϕ and μ, and all parameters are as in figure 2.3

Allocation component	No constraints	Sex ratio constrained	Clutch size constrained
Clutch size (c)	3.00	2.79	5.00
Sex ratio (s)	0.33	0.50	0.25
Investment per daughter (E_f)	1.00	1.19	0.72
Investment per son (E_m)	2.00	1.68	1.80
Total allocation to daughters (A_f)	2.00	1.66	2.69
Total allocation to sons (A_m)	2.00	2.34	2.27
Reproductive effort (E_T)	4.00	4.00	4.96
Allocation ratio (A_f/A_m)	1.00	0.71	1.19

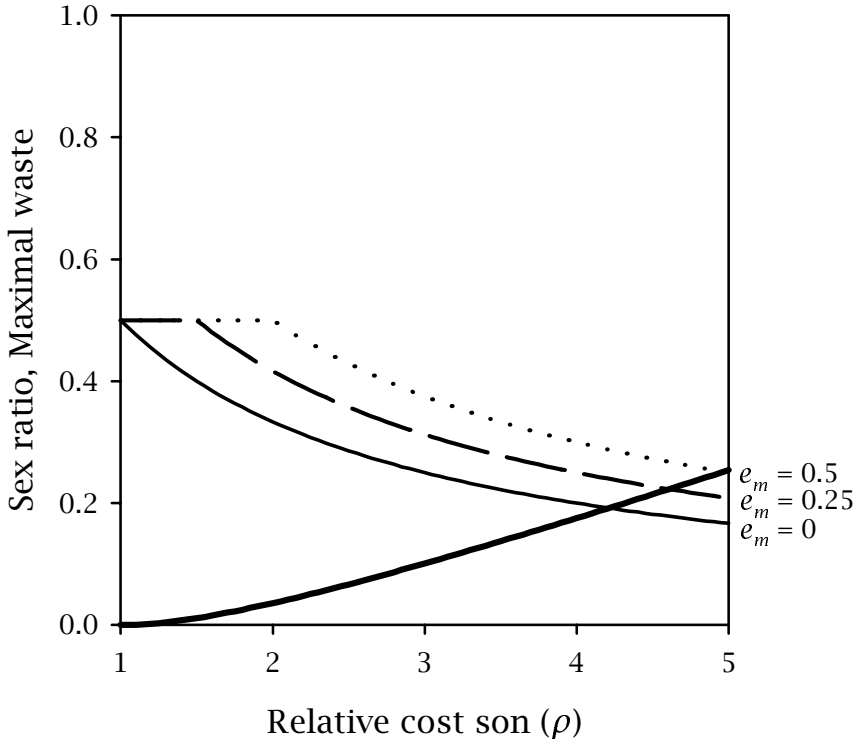

Figure 1: ESS sex ratio with costly selective abortion (see section 2.5.2). The decreasing solid line illustrates Fisher's classic result that – in the absence of any abortion costs ($e_m = 0$) – the ESS sex ratio should decline with the relative cost ρ =cost son/cost daughter of a son according to $s^* = 1/(1 + \rho)$. A substantially smaller sex ratio bias is to be expected if sex ratio control can only be achieved by abortion involving some cost e_m per aborted offspring (relative to a cost of unity per daughter). Notice that for small values of ρ it does not pay at all to adjust the sex ratio. The increasing solid line indicates the maximal proportion of total resources available for reproduction wasted during abortion.

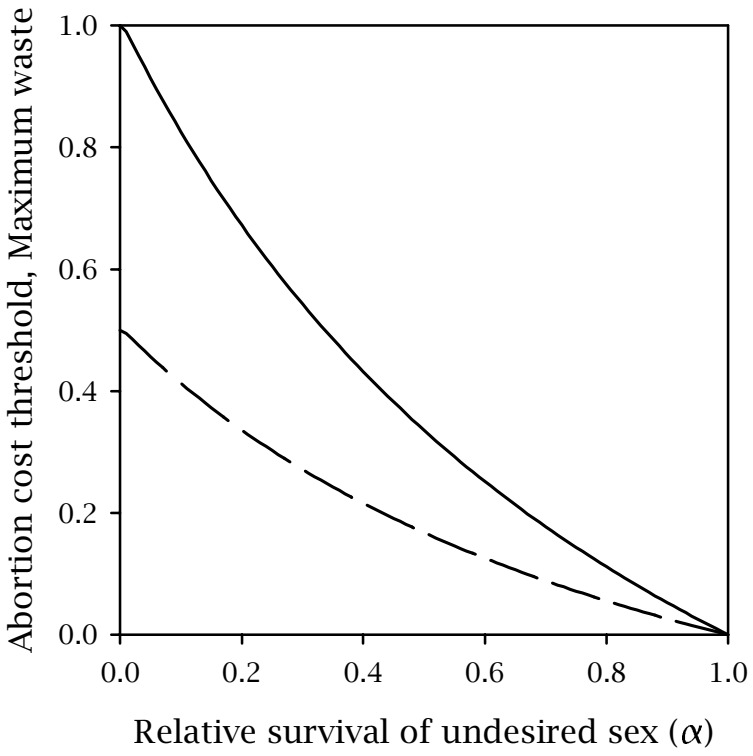

Figure 2: Threshold cost of abortion (solid curve) in the Trivers and Willard model, below which parents abort all offspring of the undesired sex and above which parents do not adjust the sex ratio at all, versus the relative survival α of the undesired sex. The dashed line indicates the maximal proportion of total resources available for reproduction, wasted during abortion of the undesired sex. See section 2.5.5 for details.

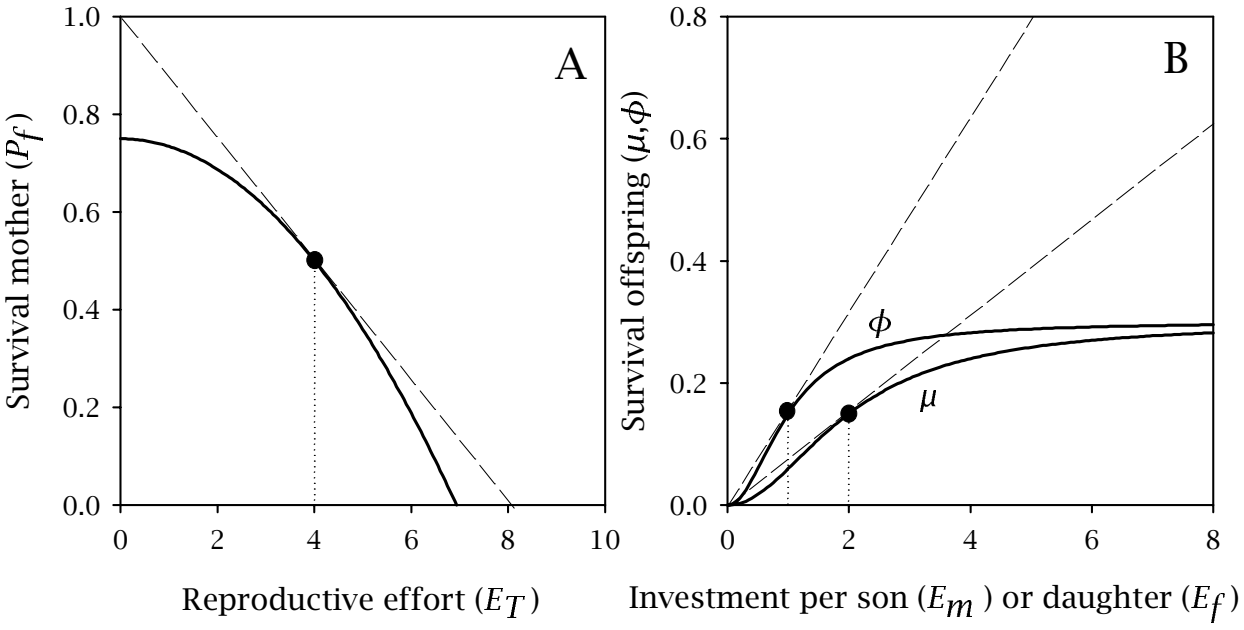

Figure 3: Survival of mother (A) as a function of her reproductive effort and survival of sons and daughters (B) as a function of the investment per son and daughter. Dashed lines indicate graphically the evolutionarily stable reproductive effort E_T^* of the mother and the ESS investment (E_m^* and E_f^*) per offspring for the case that the mother controls all allocation components. In the example the following functions are used: $P_f = P_{\max} - \gamma E_T^2$, $\mu(E_m) = \mu_{\max} E_m^2/(\alpha^2 + E_m^2)$, $\phi(E_f) = \phi_{\max} E_f^2/(\beta^2 + E_f^2)$. Parameters: $P_{\max} = 0.75$, $\gamma = 0.016$, $\alpha = 2$, $\beta = 1$, $\phi_{\max} = \mu_{\max} = 0.3$.

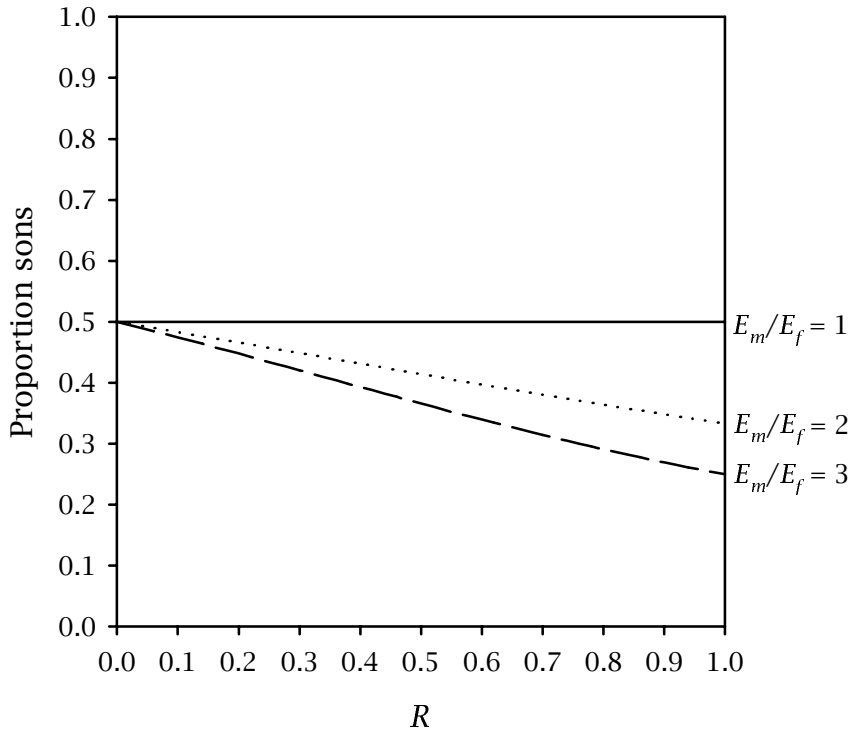

Figure 4: ESS sex ratio (proportion sons) under offspring control as a function of the average relatedness R between offspring in a clutch, for several values of the relative cost of a son, E_m/E_f. Note that for $R = 1$ the offspring's ESS coincides with the maternal ESS.